CHAPTER 9: HAND-CLASPING AND ARM-FOLDING

" ... a little folding of the hands ..."

Proverbs, 6:10

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.

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 or to show simple genetic linkage.

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

9:1 Introduction

In previous chapters I have discussed the inheritance of handedness in terms of a simple genetic model. In this chapter I wish first of all to demonstrate that there are consistent and meaningful familial patterns in hand-clasping and arm-folding, and also to show that this data can be fit by a variant of the simple genetic model described for handedness.

9:2 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 earlier in Chapter 7, 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, whilst the other allele, C, for Chance, in its homozygous form 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 Chapter 7).

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):-

	Phenotype	
Genotype	Proportion of type L	Proportion of Type R
DD	0.5 - a	0.5 + a
DC	0.5 - a.x	0.5 + a.x
CC	0.5	0.5

... Model I

'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 = 0.5, and x = 0.5 (i.e. an intermediate model, using the terminology of Wilson (1971a and b)).

Models such as this one (type I) have very strong predictions and limitations. Let p(L) be the proportion of type L in the population as a whole. Clearly p(L) cannot be greater than 0.5 or less than 0.5 - a. In particular, p(L|LxL), 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:-

Genotype	Proportion of type L	Proportion of type R	
SS	0.5 + b	0.5 - b	
SC	0.5 + b.y	0.5 - b.y	
CC	0.5	0.5	... Model II

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(L) must be greater than 0.5, and less than 0.5 + b, for all possible sub-populations.

Considering models I and II together, it may be seen that if for any sub-population p(L) is greater than 0.5, then for all sub-populations $p(L) > 0.5$ and vice-versa for $p(L) < 0.5$. 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(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.

Genotype	Proportion of type L	Proportion of type R	
DD	0.5 - c	0.5 + c	
DS	0.5 + c.z	0.5 - c.z	
SS	0.5 + c	0.5 - c	... Model III

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 0.5 + c to 0.5 - 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, whilst "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:-

Genotype	Proportion of type L	Proportion of type R	
DD	0.5 - a	0.5 + a	
SS	0.5 + b	0.5 - b	
CC	0.5	0.5	
DC	0.5 - a.x	0.5 + a.x	
SC	0.5 + b.y	0.5 - b.y	
DS	0.5 - a + z(a+b)	0.5 + a - z(a+b)	... Model I

'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 (= c of model III) this becomes equivalent to model III. Whilst 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 homozygites. 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 be known as the "three-allele model".

9:3 Hand-Clasping

9:3.1 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 survey II (described in Chapter 2) amongst undergraduates of the University of Cambridge, suggests 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. (I am grateful to Dr. C.G.N. Mascie-Taylor for allowing me to examine this and other of his unpublished data on handed-

ness, hand-clasping and arm-folding); other aspects of the survey by Dr. Mascie-Taylor have been considered elsewhere (Mascie-Taylor and Gibson 1978, 1979; Mascie-Taylor 1979a, and b).

9:3.2 Population Incidence

Estimates of p(L) have been made for a total of fifty-three populations. These give a mean value of 43.85% (SE 1.09%), the distribution being shown in Figure 9.1. 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 two unpublished studies in the United Kingdom, one of which (Dr. Mascie-Taylor) was conducted amongst members of a Cambridge suburb (p(L) = 57.6%, n = 587) and the other of which (Survey II) was conducted amongst graduates of the University of Cambridge, and their parents (p(L) = 56.32%, n = 1453). These data together completely exclude the possibility of a two-allele asymmetric genetic model (Models I and II).

One of the studies seems to give a value of p(L) which is strangely at variance with the other studies; This is the study of Thessalonikan school children by Pelecanos (1969). He found a p(L) of 18.7%, very different

from the value of 47.6% found in Patras by himself only a few years later (Pelecanos, Zacharopoulou and Yannopoulos, 1974). The Thessalonikan data are difficult to explain and need to be independently replicated before being treated seriously.

Figure 9.2 shows a map of the world on which are placed values of p(L) for different populations. Migrant populations are placed at their site of origin; thus Dutch emigrants to Brazil are placed in Holland, and so on. Americans are placed in America only if insufficient information is given to place them elsewhere. Incidences are placed in brackets if the original site cannot be adequately determined. It is apparent that there is a cline extending across the whole of Eurasia and Australiasia, from the United Kingdom in the West (p(L) = 57.4%) to the Solomon Islands in the East (p(L) = 33.7%). Figure 9.3 shows the individual data points for Eurasia/Australasia plotted against degrees of longitude east of Greenwich. The correlation is highly significant. ($r = -0.791$; $n = 40$; $t = 7.97$, $p < 10^{-6}$; analysis weighted for different sample sizes). P(L) in migrants is closer to their sedentes than to indigenous populations, suggesting that the cline might be due to genetic differences. Thus White Australians have p(L) values similar to those of Europe, and very different to those of the Aboriginal Australians.

9:3.3 Generational Differences

Thirteen studies, including our own, present data which allows comparison of the incidence of left hand-clasping (LHC) in different generations, either parent-child data or a transverse study. In 4 studies the difference is significant with $p < 0.05$, and in one study with $p < 0.10$. In all these cases the younger individuals have the higher incidence of p(L). The data points are shown in Figure 9.4. Whilst not conclusive these data might suggest a secular trend towards a greater incidence of p(L).

9:3.4 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 9.5 summarises 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 other studies, 12 have found females to have a higher incidence of LHC, whilst 24 have found males to have a higher incidence of LHC: this difference is not significant ($X^2 = 2.85$, 1 df, NS). Neither is there a tendency for either sex to show more extreme lateralisation of hand-clasping (i.e. to be further from the chance expectation of 50%) (see Table 9.1,

$X^2 = 0.45$, 1 df, NS).

9:3.5 Mating Patterns

Figure 9.6 summarises data on mating patterns for hand-clasping. There is no evidence for assortative mating, proportions being accounted for almost entirely by binomial (chance) proportions. The data of Kawabe (1949) is slightly discrepant, but otherwise the fit of the binomial is adequate.

9:3.6 Familial Data

Figure 9.7 shows that there is no evidence for a difference in the proportion of left hand claspers amongst the progeny of L x R matings or of R x 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 x L and L x R groups will be concatenated.

In the literature there are fourteen studies of incidence of LHC in the progeny of various mating types; to these may be added my own data and that of Dr. Mascie-Taylor, shown in Table 9.2. If all these data are plotted as a function of p(L) then it becomes readily apparent that

the 16 samples are not homogeneous, for the four Japanese (J) studies show marked differences relative to the non-Japanese studies,Figure 9.8. In Figure 9.9 are shown the 12 non-Japanese studies: these show a regular relation between the incidence of progeny LHC in R x R, R x L and L x L matings and the overall incidence of LHC. Figure 9.10 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(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 program has been used fo systematically vary possible values of c and z and to then fit the resulting values of p(L|RxR), p(L|RxL) and p(L|LxL) against empirically derived data, using a X^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 $0.5 \geqslant c \geqslant -0.5$, $0 \leqslant z \leqslant 1$, the minimum X^2_{16} value is 131.61, which is different from chance with a probability $\ll 0.001$; indeed for a completely general 2 allele model (i.e. $0 \leqslant P(L|DD) \leqslant 1.0$, $0 \leqslant p(L|DS) \leqslant 1.0$ and $0 \leqslant p(L|SS) \leqslant 1.0$, where p(L DD), p(L DS) and p(L SS) were all varied independently)

there was no adequate triplet of parameters which would fit all 16 sets of data. However if the 4 J and 12 NJ studies were 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 9.11. 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. Whilst 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 generalised 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(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(L) then the values of D and S are uniquely defined. From these may be calculated the values of p(L|RxR), 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 data. The process of allele frequency estimation may be seen in Figure 9.12 which shows the Japanese data only. The value of C is systematically altered and the X^2 value calculated, the minimal X^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 9.13. Note that in Figure 9.13 many of the lines terminate at relatively low values of X^2; this is due to an absolute limitation on the frequency of the C allele, for clearly if C becomes too high then it is impossible to find a population of greater than a certain value of p(L). In the extreme case, if C = 1.0, then all populations must have a p(L) of 0.50.

Table 9.3 shows, for all 16 groups of family data, the optimal values of D, C and S, and their 5% ranges, as well as the X^2 value for the optimal fit, all but one of which are acceptable with $p > 0.05$. Note that in many cases an absolute limit of allele frequency is more

important than the 5% limits. The rows of Table 9.3 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 9.3.

A further problem of the two-allele symmetric model discussed earlier, and shown in Figure 9.11, 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 9.3 provides an explanation of this anomaly. As the D values increase as one passes down through Table 9.3, 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	$r = -0.865$ $p = 1.5 \times 10^{-5}$	$r = +0.266$ $p = 0.317$
C		$r = -0.611$ $p = 0.0118$

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 (-0.661) and significant, since D + C + S = 1). From Table 9.3 it is apparent that the S allele is more common in the Japanese populations than in the other European groups. This can be seen more clearly in Table 9.4. We can thus summarise. 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 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 Figure 9.9 and 9.10.

9:3.7 Twin Data

Only two studies have provided data on hand-clasping in monozygotic and dizgotic twin pairs. In one study (Martin, 1975) there is no indication of the population incidence of LHC. In neither study is 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 is shown in Table 9.5. 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 (Chapter 7) 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.

9:3.8 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 (but see Chapter 10).

9:4 Arm-Folding

9:4.1 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 Dr. Mascie-Taylor. Unlike the case of hand-clasping, it was not felt practicable in a questionnaire study (such as my Survey II) to try and assess arm-folding, it being difficult to devise an unambiguous question.

9:4.2 Population Incidence

The incidence of LAF has been determined in 23 population groups. The distribution, shown in Figure 9.14, is very interesting. Unlike the case of hand-clasping, 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 4 populations in which p(L) is less than 50%, all being significantly so ($p < 0.001$ for all groups except the Fali, or Huizinga (1968) for whom $p < 0.05$). Three of these groups (the Fali and Kurumba (Huizinga, 1968)), and the Angolan Negroes (Freire-Maia and de Almeida, J. 1966)) are African whilst none of the groups with $p(L) > 0.50$ 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) < 0.50 can be shown to be methodologically inadequately, it is highly likely that a two-allele asymmetric model will be unable to cope with the arm-folding data.

9:4.3 Generational Differences

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

9:4.4 Sex Differences

Figure 9.15 shows data from 17 population groups for whom data are given on sex differences. There is no evidence for overall differences being significant.

9:4.5 Assortative Mating

Table 9.6 shows data from 5 studies, two of which are our own. The incidences of LAF in the five studies do not differ significantly, and neither does the 5 x 3 table show any heterogeneity (X^2 = 12.87, 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 (X^2 = 3.89, 2 df, NS).

9:4.6 Familial Data

There are five studies in the literature which give family data on arm-folding. In addition Dr. Mascie-Taylor has carried out a survey of familial trends in arm-folding (see Table 9.7 for data). The incidences of LAF in these six studies show no significant differences (Dr. Mascie-Taylor, 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%; X^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 x R matings, 51.31% show LAF; of 1553 progeny of R x L matings, 56.53% show LAF; and of 1013 progeny of L x L matings, 60.31% show LAF. These differences are significant overall (X^2 = 13.42, 2 df, $p < 0.005$). The difference between R x R and R x L is significant (X^2 = 5.03, $p < 0.05$) as is the difference between R x R and L x L (X^2 = 13.09, 1 df, $p < 0.001$). The difference between R x L and L x L is almost significant (X^2 = 3.45, 1 df, $p < 0.10$). This is strong evidence for a familial, and probably a genetic, component in the control of arm-folding.

Although the proportions of LAF overall are the same in all six studies, the family data themselves are not homogeneous; thus if one considers the progeny of R x R matings, then there are significant differences between the studies (X^2 = 19.78, 5 df, $p < 0.01$). This situation is analogous to the differences between Japanese and non-Japanese populations in the case of hand-clasping, and for identical reasons it means that it will only be possible to fit a two-allele model (of any type) if one assumes that penetrance and /or dominance may differ between populations. This might be an unreasonable assumption, and thus it was felt more reasonable, as with hand-clasping, to fit a three-allele model, and once again the simple three-allele model described earlier was used. Table 9.8 shows, in an exactly analogous manner to that described earlier for hand-clasping, the optimal estimates and the 5% ranges of allele frequency estimates for the six studies. Five of the six studies are fitted satisfactorily by what is, in effect, a 2-allele asymmetric model, the frequency of D being 0. The study of Falk and Ayala however requires a significant proportion of D allales, which is difficult to explain. If the population had contained a proportion of persons of African derivation these D alleles would have been comprehensible. According to Falk and Ayala their population consists of "all Caucasians, more than 90% are descendants of immigrants from Ireland, Italy and Portugal"; possibly the Portugese contingent contained a proportion of 'African' D alleles

derived perhaps from the Moorish invasions of the Iberian peninsular.

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

9:4.7 Twin Data

To my knowledge there is, in the literature, no arm-folding data from twins.

9:5 Inter-Relations between Asymmetries

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

9:5.1 Handedness 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-clasping.

My own survey II found no correlation (see Table 9:9.1). The study by Dr. Mascie-Taylor found a significant correlation when all individuals were included in the analysis (Table 9.2.2a; $p < 0.005$). However this seemed to be primarily due to a small number of left-handed families; when the data was re-analysed for the parental generation only (i.e. who were genetically unrelated) the relationship became non-significant (Table 9.9.2b). The study of Pelecanos (1969) claimed to find a "highly significant positive correlation"; however no data is 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 ($p < 0.01$) 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.

9:5.2 Handedness and Arm-Folding

Five studies have investigated this relationship. Data from Dr. Mascie-Taylor's study is shown in Table 9.10; 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.

9:5.3 Hand-clasping and Arm-folding

Seven studies have investigated this relationship. Data from a study by Dr. Mascie-Taylor is shown in Table 9.11; 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.

9:5.4 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 (see Chapter 7). 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 in the future.

9:5.5 Linkage

Whilst 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 9.12 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-same; there is no evidence for this in the present analysis. Tables 9.13 and 9.14 show similar analyses for handedness and arm-folding, and arm-folding and hand-clasping; again, there is no significant linkage.

9:6 Discussion and Conclusions

It is beyond reasonable doubt that hand-clasping and arm-folding are familial, and in the absence of any other theory which can cope with the data, a genetic model seems reasonable. If penetrance values are to remain constant a two-allele model within populations is unacceptable

and it is necessary to postulate a three-allele model. This raises several questions which can perhaps be best discussed by considering the nature and evolutionary development of asymmetries.

In the absence of genetic control, and due to the inevitable chance fluctuations that one finds during development, one must expect that organisms will show a degree of asymmetry, albeit often minimal. 50% of organisms will show one side as 'dominant' and 50% the opposite side as 'dominant' i.e. a racemic mixture, due to fluctuating asymmetry. Directional asymmetry is asymmetry in which a non-racemic mixture of the two phenotypes is found, one enantiomorph predominating over the other. In its most extreme form all of a species are directional in the same way (e.g. in the fiddler crab, and other crustacea, almost all organisms are born with the left claw larger (Vernberg and Gostlow, 1966; Hamilton, Nishimoto and Halusky, 1976)). Directional asymmetry poses distinct 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 colleaque have a known and conventional asymmetry. The problem would be unsolvable

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 of the helix of the genetic DNA itself; 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, is discussed by Morgan (1976) and found to be of particular interest. The snail, Limnaea peregra, occurs in two forms, a common dextral, and a rare sinistral, form. The asymmetry is determined by a recessive gene which has the strange property of working one generation out of phase: the asymmetry of an individual depends upon the genotype of its 'mother' (they are hermaphroditic), and not at all upon its own genotype (Boycott, Diver, Garstang and Turner, 1920). The effect is due to cytoplasmic inheritance. Thus a gene produces cytoplasm of a particular asymmetric type. However the gene manifests after the asymmetry of the organism has actually been determined, and the cytoplasm produced will

therefore control the asymmetry of the individual's progeny, etc. Morgan proposed that cytoplasmic inheritance might be of 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 of handedness. Whilst all of asymmetry cannot be explained by cytoplasmic inheritance, it 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.

Earlier, in Chapter 7, 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 sign-post 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 (Elze, 1924; see Fritsch, 1968, p. 55). 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 amongst 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. Whilst 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 most 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 recognisable 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, symbolised s.

If the signpost is acting typically, i.e. S, then all progeny of the hypothesised 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 <u>independently</u>, neither the P nor the Q system having any chirality information of its own. We thus have the situation:-

	Phenotype proportions			
<u>Signpost</u>	<u>PQ</u>	<u>$\bar{P}$Q</u>	<u>P$\bar{Q}$</u>	<u>$\bar{P}\bar{Q}$</u>
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 essentually trivial behaviours; and yet are controlled genetically. This does not appear to be due to them 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-Feuntes, 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 procuding the east-west cline demonstrated earlier. The importance of genetic drift in determining the allele frequency is shown by entering the measured 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 9.15 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 X^2 value. It may also be seen that the heterozygote SC must show intermediate penetrance, whilst 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 claimed to be 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. Appendix 9.1 presents evidence to suggest that all of these flies may well be of the CC genotype. 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, whilst this same phenotype is found in about 5-10% of Acheta assimilis, 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 chiasm shows a dimorphic racemic mixture (Parker, 1903). The flatfish (heterosomata) represent an interesting group for they have a gross asymmetry, one eye migrating 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 chiasm 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, whilst 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, whilst the flounders proper, the Pleuronectidae, are dextral, and have the chiasma with the left optic nerve over the right (Norman, 1934)). Whilst there are almost no known exceptions to the monomorphisms of the chiasma amongst the pleuronectidae, the migrating eye is not quote 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 peninsular, 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 a typical 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 hydridise 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. Whilst 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 kitahaeae 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'.

APPENDIX 9.1 WING-FOLDING IN DROSOPHILA MELANOGASTER

A9:1.1 Introduction

Asymmetries in any organism are of interest for the light that they may throw upon human asymmetries, especially that of the preferential specialisation of one cerebral hemisphere for speech (Morgan and Corballis, 1978). It is of importance that no example has ever been described of an asymmetry being inherited according to a simple Mendelian mechanism (Morgan, 1976). Purnell and Thompson (1973) examined wing-folding in a wild strain of Drosophila melanogaster and concluded that it was possible to select, at least for a number of generations, for either of the two possible phenotypes (Right wing over left wing, R/L, or Left wing over right wing, L/R). Wing-folding is of further interest in that it might be regarded as bearing a similarity to human hand clasping, the inheritance of which can be explained by a simple genetic model (see Chapter 9).

It is clearly of little use to try selecting for one of the two phenotypes if those phenotypes are themselves unstable in time. Purnell and Thompson tested for consistency of wing-folding by examining 20 individuals on ten separate occasions and finding that "each individual fly didshow a strong tendency to close the same wing first each time"; detailed statistics were not however given.

As a preliminary to an attempt to replicate the selection experiment of Purnell and Thompson we looked at the consistency of wing-folding, and, having found no evidence of consistency, did not continue with the experiment proper. Purnell and Thompson gave no indication of the method of assessment of wing-folding, or of whether the flies were mobile or anaethetised; we looked specifically for a possible difference between unanaethetised and etherised flies.

A9:1.2 Method

Specimens of Drosophila melanogaster were obtained from a commercial animal suppliers (T. Gerrard, Sussex), and kept in bottles containing 'blue Formula 4.24' culture medium. Two strains were used, white-eyed and wild-type, but no differences were found between them, and results have thus been combined. Virgin flies were collected within 16 hours of ecdysis and the flies etherised. Flies were then placed individually into 50 mm x 12 mm culture tubes, and their wing-folding assessed before the effects of the ether had disappeared. The next day wing-folding was re-assessed in the unanaethetised flies, the individual culture tubes being placed on the stage of a low-power binocular microscope, lighting being laterally from above, and at an almost glancing angle. Each fly was assessed in this way on at least five separate occasions at intervals of at least 24 hours. Some of the flies were then assessed

whilst etherised. For this the flies were kept in their individual culture tubes and ether vapour injected into the tube through the cotton wool plug by means of a hypodermic syringe. After assessment, as above, air was injected into the tube in order to dispel the ether vapour. Each fly was assessed on five separate occasions of at least 24 hours.

A9:1.3 **Results**

140 individual flies were examined. 97 survived sufficiently long to be assessed on five distinct occasions whilst unanaethetised. Table A9.1.1 shows that the vast majority (92/97) showed some inconsistency in wing-folding, and that the distribution was indistinguishable from the expected values for a binomial distribution with p(L/R) = 0.5, (X^2 = 1.96, 4 df, NS). 40 flies were tested whilst etherised, and of these 33 survived for five successive assessments. Table A9.1.2 shows that once again the majority of the flies were inconsistent (27/33), and that the distribution was indistinguishable from a binomial with p(L/R) = 0.5 (X^2 = 0.51, 2 df, NS; groups 0 and 1 and 4 and 5 combined to give expected values of greater than 5.0).

The 33 flies tested whilst etherised had also been tested earlier whilst unanaethetised; the correlation between the number of times a fly folded L/R whilst

unanatthetised and the number of times the same fly folded L/R whilst etherised was only 0.187, which is not significantly different from chance (n=33).

Thirty-seven flies were also assessed for wing-folding whilst unanaethetised and then immediately etherised and re-assessed. Table A9.1.3 shows that even with an interval of only 2 or 3 minutes between the assessments there was no consistency of wing-folding (X^2 = 1.65, 1 df, NS).

A9:1.4 Discussion

There is no evidence from the present analysis for consistency of wing-folding in Drosophila melanogaster. It is thus difficult to know how selection for wing-folding type could be effective. It is possible that the Eversden-14 wild type stock used by Purnell and Thompson shows genetic differences from the present strain, but this seems unlikely. An alternative explanation of the apparent selection found by Purnell and Thompson is that their statistical analysis may be in error. Consider their Table 2. For L/R bias there is a correlation of 0.962, significant at the 0.01 level, between generation number and proportion of L/R flies. However the decision to analyse only generations o-4 out of the 15 possible generations, is clearly ad hoc. It is therefore artificially selecting only one out of 12 possible consecutive groups of five from fifteen (e.g. 1-5, 2-6, etc.). Moreover it is apparent that the decision to analyse five successive

generations was also ad hoc, for in the R/L analysis, eight successive generations were analysed. Under such conditions the significance level quoted is seriously over-estimated, and is almost certainly insignificant in reality This problem is compounded by the impossibility of re-constructing the data in order to re-analyse it, since there is no indication of the number of flies assessed in order to plot each of the points on Figure 1. The hypothesised selection for R/L bias is of the order of 8% over 8 generations, i.e. 1% per generation. To detect such a difference between one generation and the next would require at least 1000 progeny per generation in order to produce a statistically significant difference between successive generations.

A9:1.5 Conclusions

Wing-folding in Drosophila melanogaster is not consistent. The statistical analysis of Purnell and Thompson (1973), who claimed to find evidence of selection for wing-folding type, is probably in error, and hence there is neither evidence for the inheritance of wing-folding, nor for its modification by selection.

APPENDIX 9:2

Code numbers for data points on figures 9.3, 9.4, 9.5, 9.6, 9.7, 9.8, 9.9, 9.10, 9.11 and 9.12.

Code	Study population
1.	Beckman and Elston (1962).
2.	Beiguelmann (1964).
3.	Bonné (1966).
4.	Chattopadhyay (1968).
5.	Dahlberg (1926).
6.	Downey (1926).
7.	Falk and Ayala (1971).
8.	Ferronato *et al* (1974).
9.	Forrai and Bankovi (1969).
10.	Freire-Maia and de Almeida (1966).
11.	Freire-Maia *et al* (1960).
12.	Freire-Maia *et al* (1958) 'Caucasians'.
13.	Freire-Maia *et al* (1958) 'Mongoloids'.
14.	Freire-Maia *et al* (1958) 'Mulattoes'.
15.	Freire-Maia *et al* (1958) 'Negroes'.
16.	Freure-Maia *et al* (1958) 'Indians'.
17.	Frisancho *et al* (1977) Lowland Mestizos.
18.	Frisancho *et al* (1977) Lowland Quechuas.
19.	Frisancho *et al* (1977) Highland Quechuas.
20.	Huizinga (1968) Fali.
21.	Huizinga (1968) Kurumba.
22.	Kawaube (1949).

APPENDIX 9:2 CONTINUED

23. Lai and Walsh (1965) Aborigines.
24. Lai and Walsh (1965) White Australians.
25. Lai and Walsh (1965) Chinese.
26. Lai and Walsh (1965) Indians.
27. Lai and Walsh (1965) Japanese.
28. Lai and Walsh (1965) New Guinea.
29. Lai and Walsh (1965) Phillipines.
30. Leguebe (1967).
31. Lourie (1972) Kurds.
32. Lourie (1972) Yemenites.
33. Lutz (1908).
34. McManus (Survey II).
35. Malhotra (1968).
36. Mascie-Taylor (1978) Personal communication.
37. Pelecanos et al (1974).
38. Pons (1961).
39. Rhoads and Damon (1973) Baegu.
40. Rhoads and Damon (1973) Kwaio.
41. Rhoads and Damon (1973) Lau.
42. Rhoads and Damon (1973) Nasioi.
43. Saldanha et al (1960).
44. Singh and Goel (1975) Khatri.
45. Singh and Goel (1975) Bhaniya.
46. Srnivasan and Mukherjee (1975).
47. Tiwari and Bhasin (1968) Brahmin.
48. Tiwari and Bhasin (1969) Rajput.

APPENDIX 9:2 CONTINUED

49. Wiener (1932).

50. Yamaura (1940) Japanese.

51. Yamaura (1940) Koreans.

52. Yoshiwara (1957)

53. Kawabe (1953).

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

APPENDIX 9:3

As for Appendix 9:2 but for Arm-folding of Figures 9.14 and 9.15.

Code	Study population
1.	Beckman and Elston (1962).
2.	Beiguelmann (1964).
3.	Bonné (1966).
4.	Chattopadhyay (1968).
5.	Falk and Ayala (1971).
6.	Ferronato et al (1974).
7.	Forrai and Bankovi (1969).
8.	Freire-Maia and de Almeida (1966).
9.	Freire-Maia et al (1960).
10.	Huizinga (1968) Fali.
11.	Huizinga (1968) Kurumba.
12.	Leguebe and Martinez-Fuentes (1971).
13.	Lourie (1972) Kurds.
14.	Lourie (1972)YYemenites.
15.	Mascie-Taylor (1978) Personal communication.
16.	Pelecanos (1969).
17.	Pelecanos et al (1974).
18.	Rhoads and Damon (1973).
19.	Srnivasan and Mukherjee (1975).
20.	Wiener (1932).

Table 9:1 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%).

		Pop'n incidence of PHC		
		<0.50	>0.50	
Sex differences in LHC	Male > Female	11	12	23
	Female<Male	3	9	12
		14	21	35

$X^2 = 0.89$, 1 df, NS.

Table 9:2 Family tables for hand-clasping, by sex.

i. McManus: Survey II

		Progeny			
		Right HC		Left HC	
Father	Mother	Male	Female	Male	Female
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. Dr. Mascie-Taylor's Data

Right	Right	14	16	16	15
Right	Left	10	13	21	17
Left	Right	19	19	28	28
Left	Left	8	18	31	28

n.b. For my own study only one child was analysed per parental pair, whilst for Dr.Mascie-Taylor's study all children were included who were old enough for assessment.

TABLE 9.3 Shows optimal allele frequency estimates for 16 populations. Optima were estimated by observing goodness of fi altering C allele frequency at intervals of 0.01. 5% ranges are also shown, except where an asterisk is given, in which the limit is absolute (see text). Data are arranged in order of ascending D allele estimates. All allele estimates are x

Study	D			S			C			goodness of fit of optimal values (2 df)	P	N (chi
	Optimal	5% range		Optimal	5% range		Optimal	5% range				
		Max	Min		Max	Min		Max	Min			
Survey II	0	13.6	0*	21.8	35.3	21.8	78	78*	51	1.150	0.562	44
Terronato et al (1974)	0	14.9	0*	14.5	29.0	14.5*	85	85*	56	0.965	0.617	20
Falk and Ayala (1971)	6.0	15.57	0*	15.9	25.4	9.9*	78	90*	59	0.128	0.938	118
Dr. Mascie-Taylor	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	6.59*	3.9	16.4	0*	86	93*	61	2.037	0.361	469
Mai & Walsh (1965): White Australians	11.0	35.0	1.5*	9.9	33.9	0*	79	98*	31	1.229	0.540	198
Neguebe (1967)	17.7	**	**	17.2	**	**	65	**	**	6.311	0.042	912
Cons (1961)	24.1	48.1*	0*	27.8	51.8*	3.8*	48	96*	0*	0.991	0.609	106
Mai & Walsh (1965): New Guineans	23.9	23.9*	30.4	0	6.5	0*	76	76*	63	2.631	0.268	321
Rhoads & Damon (1973)	35.7	52.5	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	24.05	0*	53	75*	27	0.363	0.834	466
Lutz (1908)	41.5	53.0	29.03	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 X^2

**5% limits not calculable due to optimal fit with $p > 0.05$

Note: C values are in integer units (see text). Values of D and S less than 1 have been rounded down to 0.

TABLE 9.4 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; $F(2,13) = 7.79$, $p = 0.006$; E vs NJNE $F(1,10) = 4.28$, $p = 0.065$; E vs J $(F1,11) = 12.05$, $p = 0.0052$ J vs NJNE $(F(1,5) = 3.87$, $p = 0.106$ S Allele $F(2,13) = 6.93$, $p = 0.0089$ E vs NJNE $F(1,10) = 7.16$, $p = 0.0232$ E vs J $(F1,11) = 2.35$, $p = 0.152$ J vs NJNE $F(1,5) = 14.44$, $p = 0.0126$ C Allele $F(2,13) = 13.63$, $p = 0.0006$ E vs NJNE $F(1,10) = 0.10$, $p = 0.75$ E vs J $F(1,11) = 24.40$, $p = 0.0004$ J vs NJNE $(F(1,5) = 24.76$, $p = 0.0042$

Allele	European (E) n = 9	Non-Japanese Non-European (NJNE) n = 3	Japanese (J) n = 4
D	0.1319	0.318	0.506
S	0.1811	0.036	0.258
C	0.687	0.645	0.236

Table 9:5 Data on hand-clasping in twins.

Twin type	Study	Pair Type R-R		R-L		L-L		n
Monozygotic	Martin (1975)	9		12		7		28
	Dahlberg (1926)	18		34		17		69
	Total	27	(27.8%)	46	(47.4%)	24	(24.8%)	97
Dizygotic	Martin (1975)	1		11		7		19
	Dahlberg (1926)	34		56		33		123
	Total	35	(24.6%)	67	(47.2%)	40	(28.2%)	142

Table 9:6 Mating patterns for Arm-folding

Mating Type	Ferronato et al (1974)	Leguebe & Martinez-Fuentes (1971)	Dr. Mascie-Taylor	Rhoads & Damon (1973)	Wiener (1932)	Tota
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

Table 9:7 Arm-folding: Family tables, by sex, of a study by Dr. Mascie-Taylor

		Right AF		Left AF	
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.

Table 9:8 As for Table 9:3 but for Arm-folding

	D			S			C				
	Optimal	5% limits Max	Min	Optimal	5% limits Max	Min	Optimal	5% limits Max	Min	goodness of fit of optimal values	
Dr. Mascie-Taylor	0	2.5	0*	16.9	19.4	16.9*	83	83	78	4.84	0.0
Wiener (1932)	0	4.2	0*	12.7	16.7	12.7*	87	87*	79	3.43	0.1
Ferronato et al (1974)	0	4.1	0*	13.8	17.8	13.8*	86	86*	78	4.11	0.1
Rhoads & Damon (1973)	1.1	17.6	0*	18.8	35.3	17.8*	80	82*	47	0.89	0.6
Leguebe & Martinez-Fuentes (1971)	1.1	9.5	0*	13.9	22.4	12.9*	85	87*	68	2.02	0.3
Falk & Ayala (1971)	18.1	27.1	8.1	29.9	38.9	19.9	52	72	34	0.16	0.9

Table 9.9 Relationship between handedness and hand-clasping. Including both parents and progeny

i. Survey II

		Hand-clasping			
		Right	Left	n	%LHC
Handedness	Right	545	688	1233	55.80%
	Left	70	99	169	58.58%

$X^2 = 0.36$, 1 df, NS.

ii. Dr. Mascie-Taylor's data

a. All Individuals

		Hand-clasping			
		Right	Left	n	%LHC
Handedness	Right	290	342	632	54.1%
	Left	13	42	55	76.3%

$X^2 = 9.28$, 1 df, $p < 0.005$.

b. Parents only

		Hand-clasping			
		Right	Left	n	%LHC
Handedness	Right	172	181	353	51.27%
	Left	11	22	33	66.66%

$X^2 = 2.28$, 1 df, NS.

Table 9:10 Relationship between handedness and arm-folding (Dr. Mascie-Taylor's data).

		Arm-Folding			
		Right	Left	n	%LAF
Handedness	Right	276	357	633	56.39
	Left	29	25	54	46.29

$X^2 = 1.66$, 1 df, NS.

Table 9:11 Relationship between arm-folding and hand-clasping (Dr. Mascie-Taylor).

		Arm-Folding			
		Right	Left	n	%LHC
Hand-clasping	Right	130	171	301	56.81%
	Left	174	212	386	54.92%

$X^2 = 0.173$, 1 df, NS.

Table 9:12 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. The Table shows number of progeny in which hand-clasping and/or handedness are the same as the parents.

Survey II

		Hand-clasping		
		Same	Opposite	
Handedness	Same	79	63	142
	Opposite	16	7	23
		95	70	165

$X^2 = 1.047$, 1 df, NS

Dr. Mascie-Taylor's data

		Hand-clasping		
		Same	Opposite	
Handedness	Same	50	65	115
	Opposite	2	4	6
		52	69	121

$X^2 = 0.004$, 1 df, NS.

N.B. For my own survey only one progeny for each parental pair; for Dr. Mascie-Taylor's Survey all progeny included.

Table 9:13 Linkage of Handedness and arm-folding

Data from Dr. Mascie-Taylor's study; otherwise as for Table 11.

		Arm-Folding		
		Same	Opposite	
Handedness	Same	37	72	109
	Opposite	2	4	6
		39	76	115

$X^2 = 0.17$, 1 df, NS.

Table 9:14 Linkage of Arm-Folding and Hand-Clasping

Data from Dr. Mascie-Taylor's study; otherwise as for Table 11.

		Arm-Folding		
		Same	Opposite	
Hand-clasping	Same	9	23	32
	Opposite	21	29	50
		30	52	82

$X^2 = 1.08$, 1 df, NS.

Table 9.15 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 = 0.5 and b = 0.5 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 (or alternatively as if it were CC) or as an intermediate degree of penetrance (symbolized by I in the Table). For intermediate penetrance $p(RHC|DC) = \frac{1}{2}(p(RHC|DD)+P(RHC|CC))$, etc. The next 16 columns show the X^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 X^2 values. Each individual dataset has 2 d.f. and the total has 32 d.f. For the individuals columns any X^2 less than 5.99 is an adequate fit with $p > 0.05$, and for the total column a X^2 of less than 56.66 is an adequate fit with $p > 0.05$. Optimal goodness of fits were determined by finding the minimal X^2 values for all models with the C allele frequency varied in 0.01 steps between 0.01 and 0.99.

Allele Pair Genotype DC	SC	DS	Falk & Ayala (1971)	Ferronato et al (1974)	Freire-Maia et al (1958)	Kawabe (1949)	Kawabe (1953)	Lai & Walsh (1965): New Guineans	Lai & Walsh (1965): White Australians	Leguebe (1967)
Penetrance										
		DD	278.9	53.8	58.4	65.3	87.2	87.6	35.0	160.5
	SS	I	123.2	37.9	26.3	10.0	35.8	58.6	19.1	96.4
		SS	120.7	25.8	47.8	78.4	158.4	60.5	24.8	95.6
		DD	0.1	1.0	0.6	3.0	0.1	6.5	1.1	6.9
DD	I	I	0.1	1.0	0.5	5.0	0.7	6.5	1.2	6.3
		SS	0.2	1.0	0.4	12.0	1.8	6.5	1.4	5.6
		DD	28.2	8.3	20.0	34.3	42.4	33.4	2.0	9.1
	CC	I	41.7	9.9	16.6	10.0	30.0	30.9	6.3	21.7
		SS	147.1	12.1	33.7	59.9	116.8	40.1	7.7	89.2
		DD	278.9	63.7	58.4	65.3	87.2	87.6	35.0	160.5
	SS	I	123.2	37.9	26.3	9.6	58.6	58.6	19.1	95.7
		SS	199.6	6.1	63.6	78.4	158.4	56.7	23.3	208.4
		DD	0.1	1.0	0.6	3.3	0.2	2.6	0.9	7.3
I	I	I	0.1	1.0	0.4	5.5	2.7	2.6	1.2	6.3
		SS	0.2	1.0	0.3	14.2	6.5	2.6	1.5	5.1
		DD	0.3	0.4	18.0	24.1	24.4	20.5	2.0	9.1
	CC	I	26.3	6.5	9.9	10.0	20.3	18.2	4.2	14.9
		SS	93.0	14.5	44.0	78.4	158.4	42.8	9.0	49.0
		DD	278.9	63.7	58.4	65.3	87.2	87.6	35.0	160.5
	SS	I	123.2	37.1	26.3	9.7	35.8	58.6	19.1	90.8
		SS	0.4	0.9	22.4	74.3	158.4	36.6	2.0	8.9
		DD	1.8	2.6	0.5	2.5	3.9	11.8	1.1	9.0
CC	I	I	0.1	0.9	0.4	6.6	6.5	4.6	1.4	5.6
		SS	0.2	0.9	0.4	17.4	9.8	4.6	1.7	4.4
		DD	0.3	0.4	13.5	19.3	19.3	17.0	2.0	9.1
	CC	I	20.0	5.1	8.3	9.9	19.9	16.5	3.6	11.7
		SS	76.2	16.4	36.6	78.4	132.0	36.8	10.6	37.9

Lal & Walsh (1965): White Australians	Leguebe (1967)	Lutz (1908)	Dr. Mascie-Taylor's data	Survey II	Pons (1961)	Rhoads & Damon (1973)	Wiener (1932)	Yamaura (1940)	Yoshiwara (1957)	TOTAL
35.0	160.5	50.0	64.7	116.1	16.2	35.1	112.4	34.1	23.9	1280.0
19.1	96.4	16.1	20.2	50.0	5.3	23.6	60.3	5.6	6.5	584.7
24.8	95.6	57.2	21.2	39.6	7.3	35.1	67.4	27.9	28.8	896.3
1.1	6.9	0.8	0.2	1.2	1.3	1.7	2.0	1.0	0.4	27.8
1.2	6.3	0.1	0.2	1.1	1.1	1.7	2.0	0.7	1.4	29.9
1.4	5.6	0.1	0.2	1.1	1.0	1.7	2.0	1.5	3.0	39.5
2.0	9.1	19.0	16.0	28.0	4.1	17.2	4.0	7.2	7.2	280.4
6.3	21.7	6.8	7.4	13.5	4.5	14.2	27.6	4.7	5.0	250.8
7.7	89.2	32.9	13.4	36.3	5.3	13.3	50.0	14.9	13.1	685.7
35.0	160.5	50.0	64.7	116.1	17.9	35.8	112.4	34.1	23.9	1291.7
19.1	95.7	16.1	20.2	50.0	5.3	23.6	60.3	5.6	6.5	593.7
23.3	208.4	72.7	0.2	1.4	6.8	34.0	96.8	29.6	25.6	1061.6
0.9	7.3	1.0	0.3	1.2	1.4	0.1	1.9	1.3	0.2	23.5
1.2	6.3	0.0	0.2	1.1	1.1	0.1	2.0	0.7	1.6	26.7
1.5	5.1	0.3	0.2	1.1	1.1	0.1	2.1	1.8	3.5	41.7
2.0	9.1	12.6	12.7	17.2	0.3	10.3	4.0	7.2	4.3	167.4
4.2	14.9	3.7	5.1	9.5	3.5	8.0	17.5	3.5	3.8	164.7
9.0	49.0	44.5	15.9	25.1	4.5	21.4	54.8	20.4	15.9	691.6
35.0	160.5	50.0	64.7	116.1	18.6	35.8	112.4	34.1	23.9	1292.4
19.1	90.8	16.1	20.2	50.0	5.3	23.6	60.3	5.6	6.5	588.0
2.0	8.9	32.3	0.2	1.4	0.2	33.7	7.3	6.8	9.7	395.5
1.1	9.0	0.1	0.4	2.3	1.4	3.6	5.7	0.8	0.7	48.2
1.4	5.6	0.0	0.2	1.2	1.1	1.2	2.3	0.9	2.1	35.0
1.7	4.4	0.7	0.2	1.1	1.2	1.3	2.4	2.2	4.3	52.8
2.0	9.1	9.2	0.3	1.9	0.3	8.0	4.0	7.2	3.2	115.1
3.6	11.7	2.9	3.9	7.3	2.9	7.4	14.7	3.1	3.6	141.0
10.6	37.9	36.1	10.2	19.9	4.5	15.9	45.5	19.4	17.2	599.7

Table A9:1.1 shows, for 97 flies, the number of times out of five that each fly was observed to fold its wings L/R. Flies were not etherised. Expected values under a chance (binomial) hypothesis are shown beneath the observed values.

	n(L/R)					
	0	1	2	3	4	5
Observed	2	17	27	35	13	3
Expected	3.0	15.1	30.3	30.3	15.1	3.0

Table A9:1.2 As for Table A9:1.1, but for 33 etherised flies

	n(L/R)					
	0	1	2	3	4	5
Observed	3	4	9	9	5	3
Expected	1.0	5.1	10.3	10.3	5.1	1.0

Table A9:1.3 For 37 flies the wing-folding type was assessed immediately before and just after etherisation

		After	
		R/L	L/R
Before	R/L	11	5
	L/R	10	11

Figure 9:1 Shows the incidence of Left Hand-Clasping in fifty-three separate studies of different populations.

Hand-Clasping: Population incidences

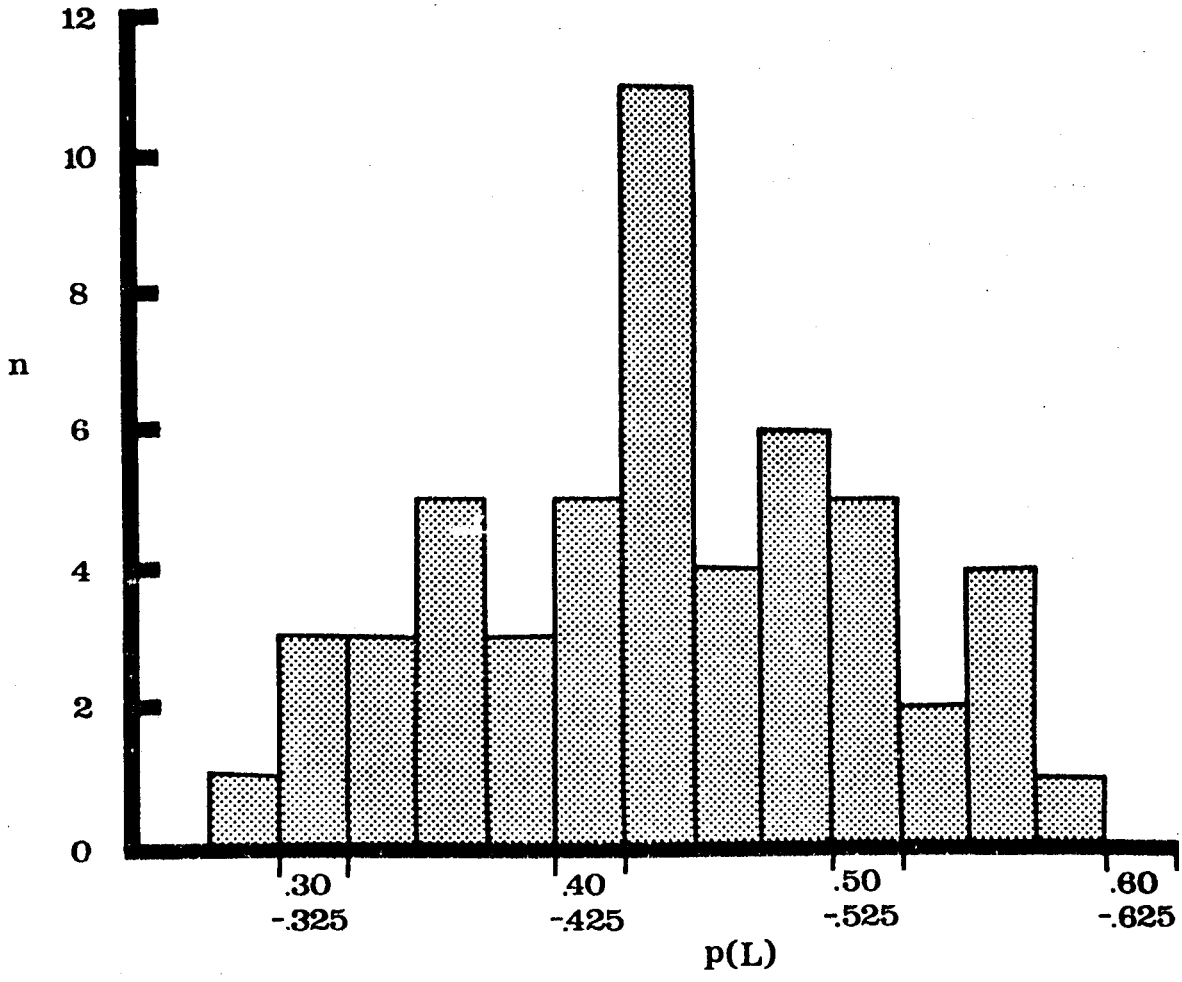


Figure 9:2 Shows a map of the world on which are placed the individual incidences of left hand-clasping in different populations. Values are expressed as percentages. All points are plotted as of the subjects' original home, migrants being plotted as of their sedentes. In a few cases this was not possible for American studies and they have just 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 apapers. 'WA' refers to White Australians who could not more adequately be plotted elsewhere. The profusion of Indian studies have been plotted in the ocean beneath their country, due to lack of space.

Hand Clasping: per cent Left

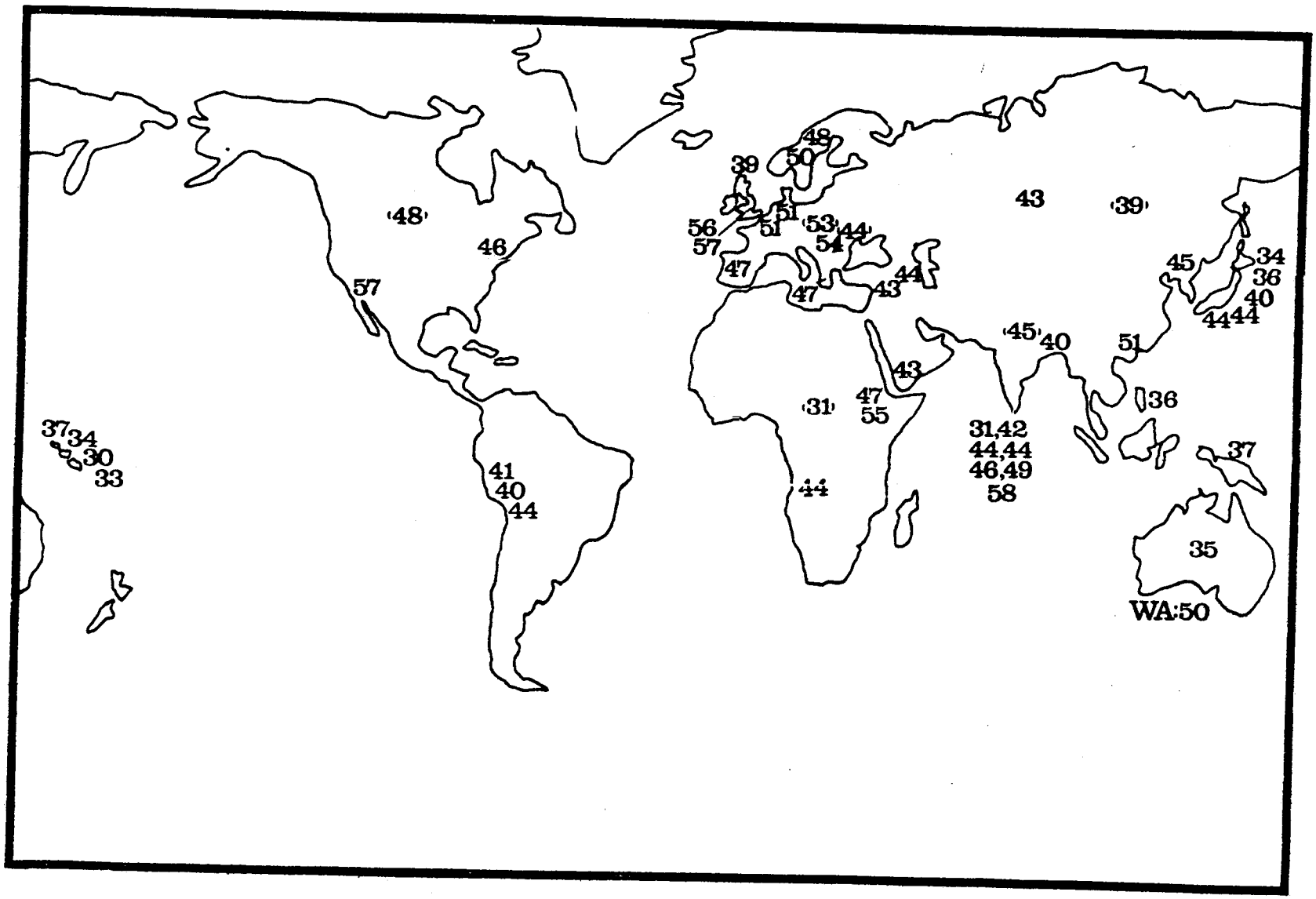


Figure 9:3 Shows for all Eurasian and Australasian studies the incidence of left hand-clasping as a function of the degrees east of Greenwich. Data points are plotted ± 1 standard error. Numbers alongside each point refer to the study from which it was taken (see Appendix 9.2 for key).

Hand-Clasping: Eurasia/Australasia

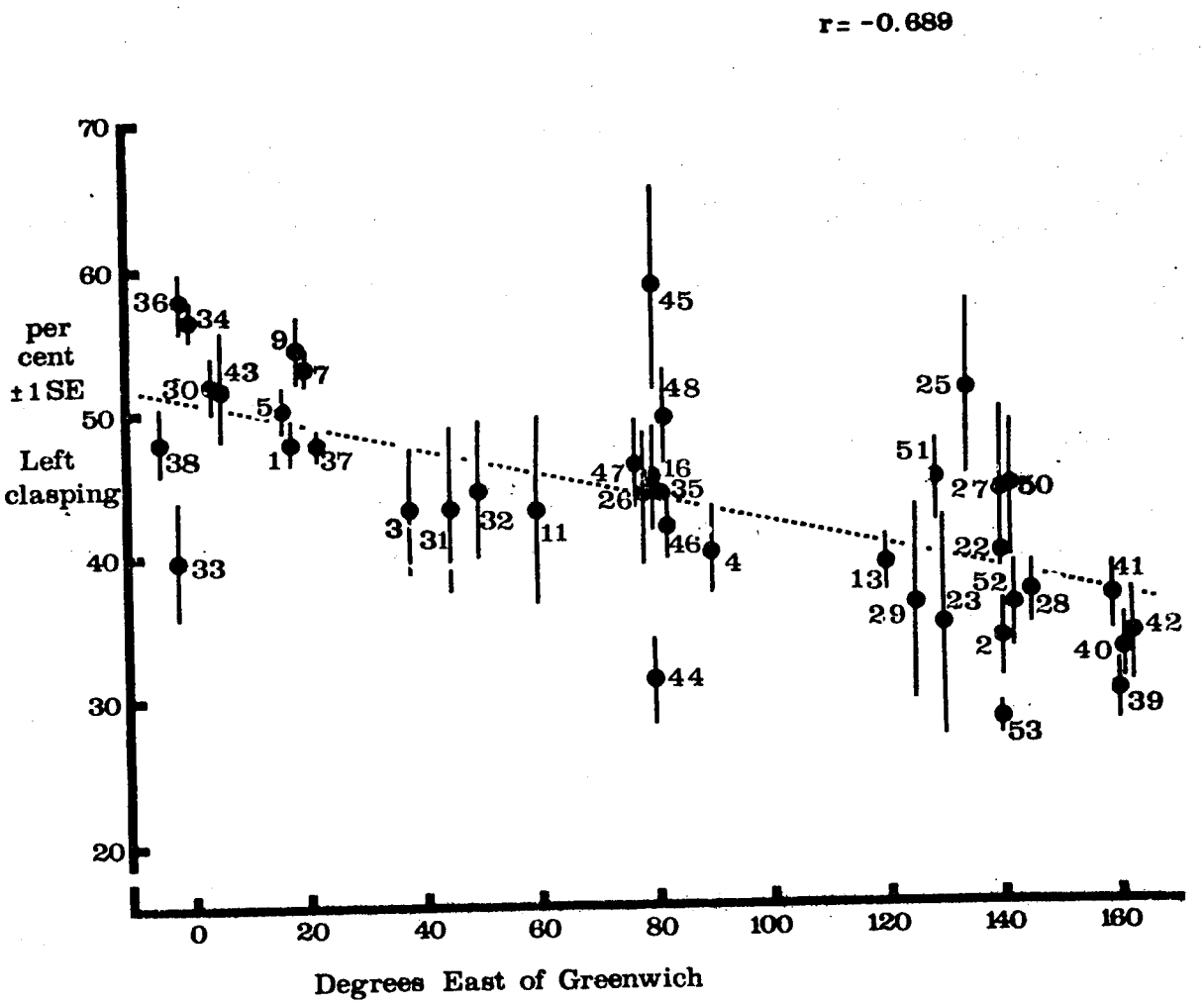


Figure 9:4 Shows the incidence of left-hand clasping in children as a function of the incidence in parents, for 14 studies. Numbers alongside each point indicate the source (see Appendix 9.2 for key). Significance values are placed alongside points in which the incidence in parents and children differs 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.

Hand-Clasping: Generational differences

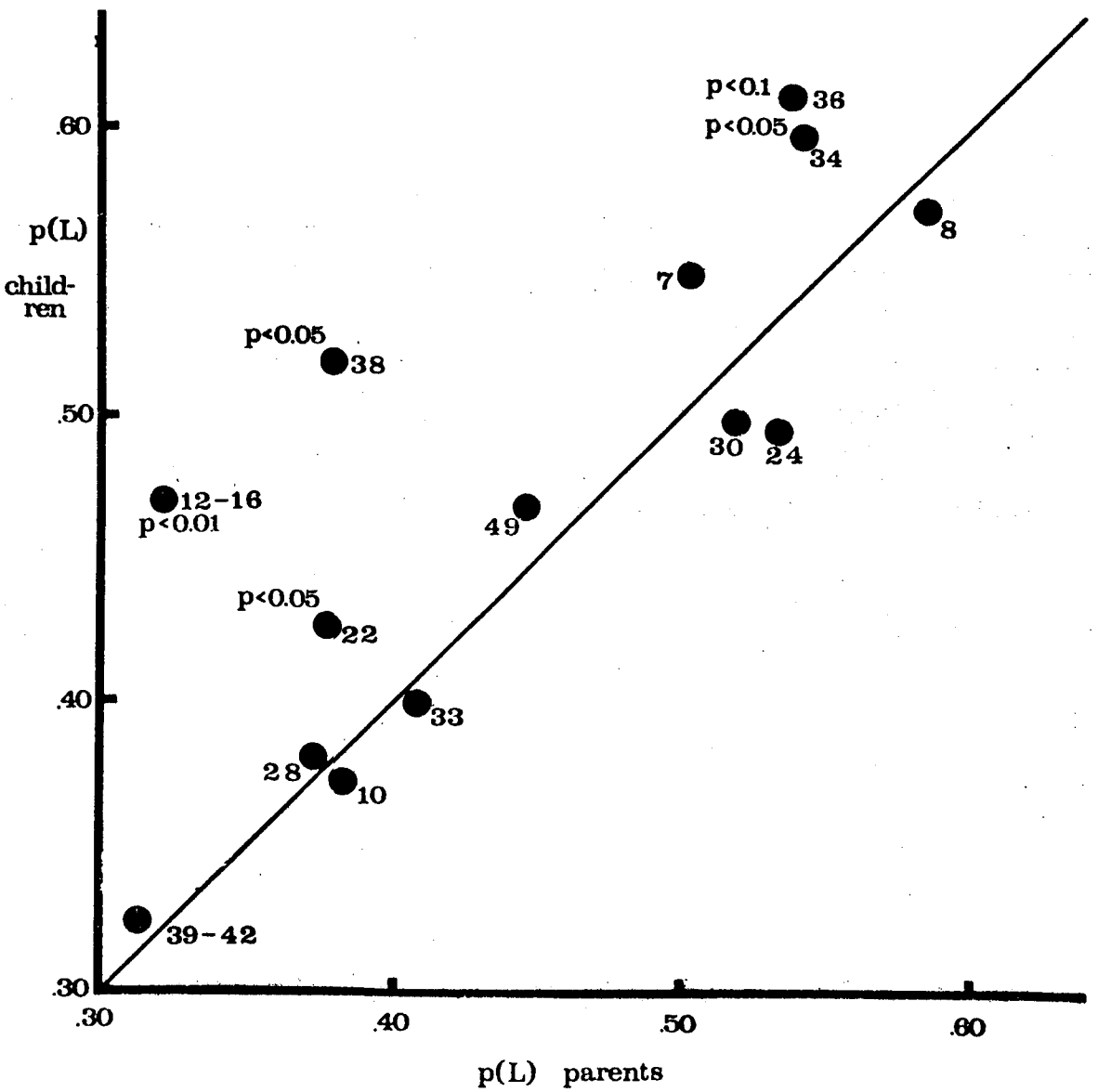


Figure 9:5 Shows the incidence of left hand-clasping in females as a function of the incidence in males, for 36 separate populations. Numbers alongside data points indicate their source (see Appendix 9.2 for key); a subscript 'p' or 'c' indicates parental or child generation respectively. The diagonal line represents the line of equality for male and female incidences

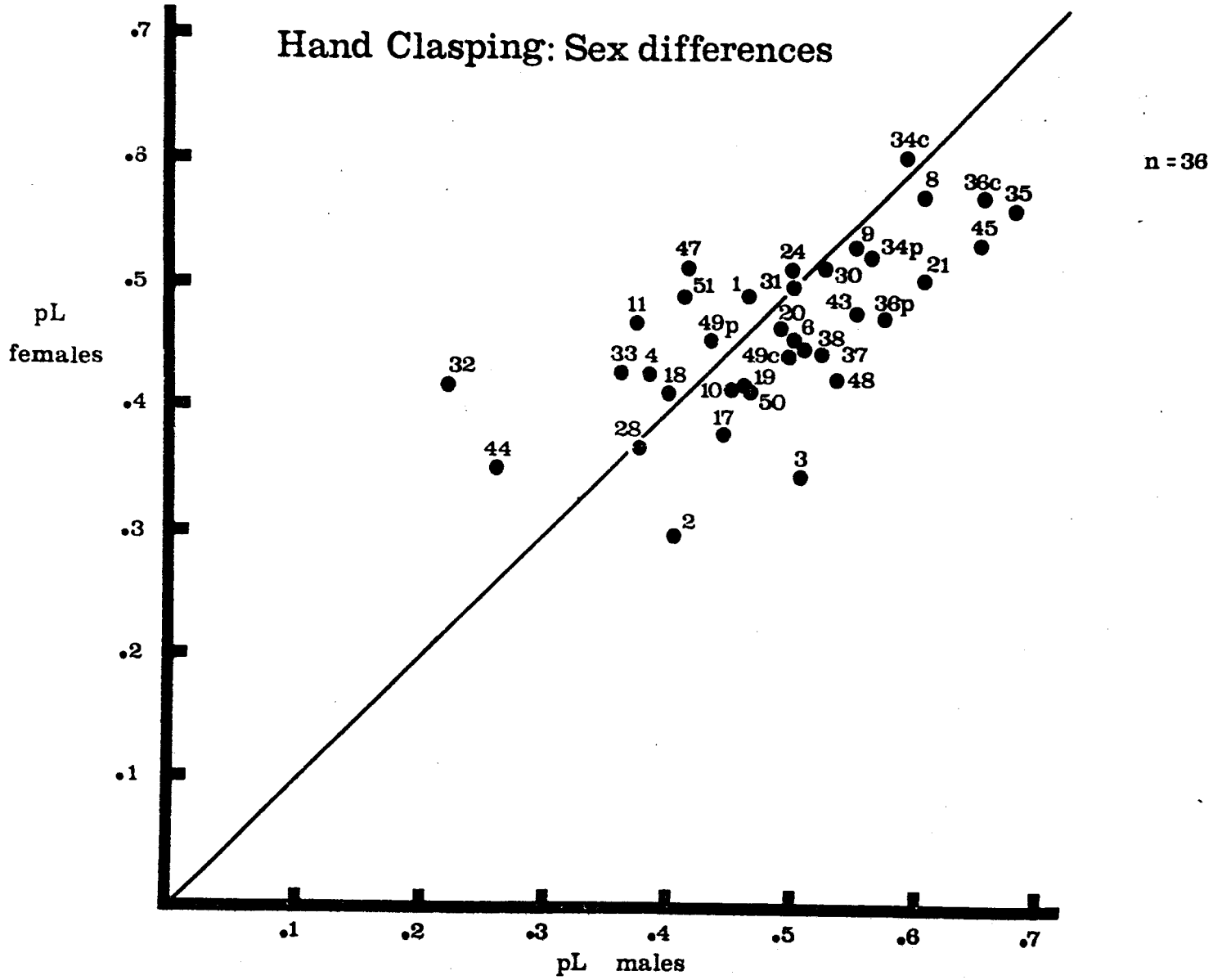

Hand Clasping: Sex differences
n = 36
pL
females
pL males
.1
.2
.3
.4
.5
.6
.7
34c
8
36c
35
45
9
34p
21
47
24
30
51
1
31
43
36p
11
20
49p
6
38
33
4
49c
37
32
18
19
48
10
50
17
28
44
3
2

Figure 9:6 Shows data from fourteen studies on the pattern 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 alongisde each point indicate the source (see Appendix 9.2 for key). Data points are plotted ± 1 standard error.
(■) L-L pairs; (▲) R-L pairs.

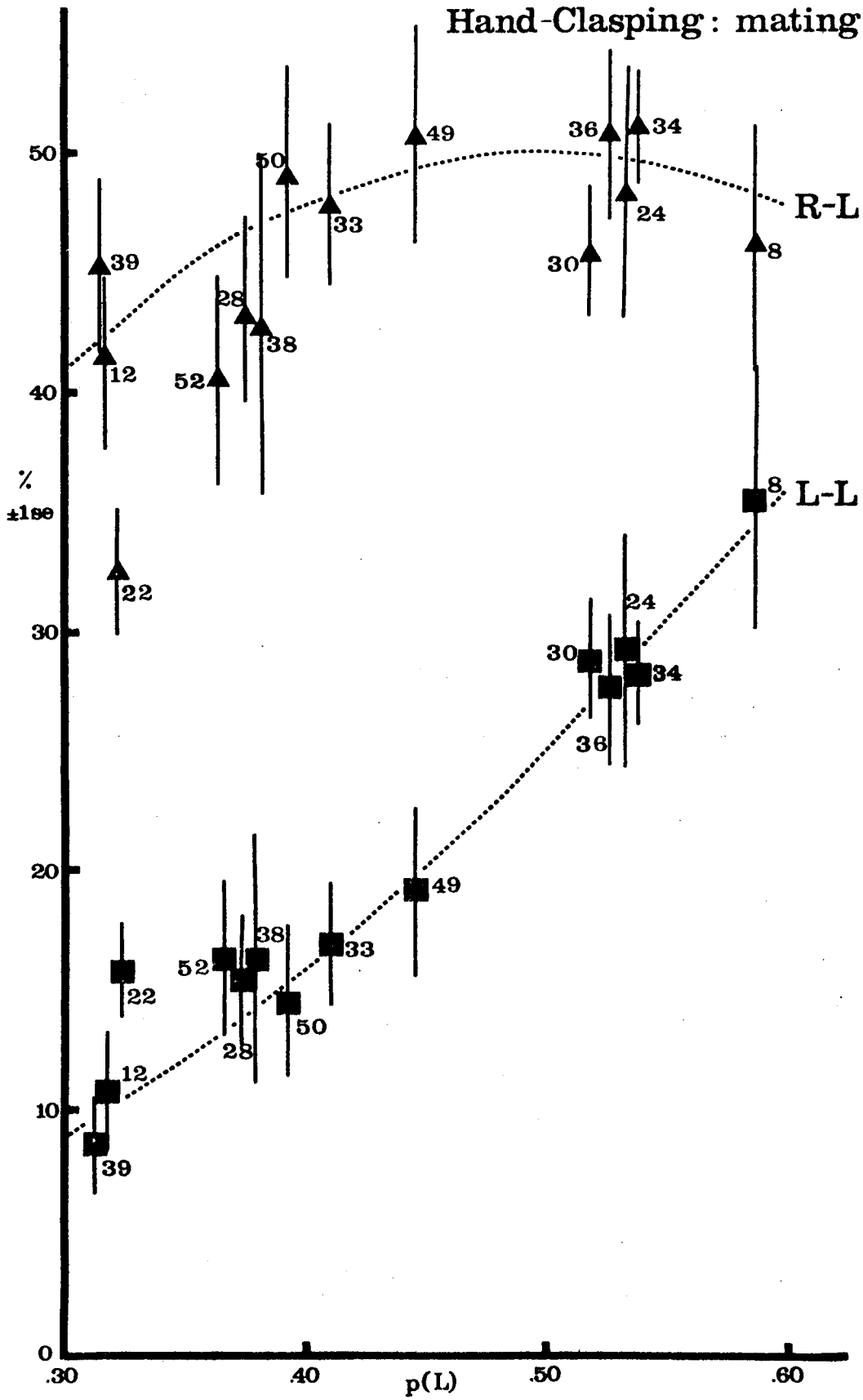

Hand-Clasping: mating
R-L
L-L
%
±1se
p(L)
0
10
20
30
40
50
.30
.40
.50
.60
39
12
22
52
28
38
50
33
49
36
34
24
30
8

Figure 9:7 Shows, for eleven studies, the incidence of left hand-clasping in the progeny of L x R matings as a function of the incidence of left hand-clasping in the progeny of R x L matings. Numbers alongside data points indicate source (see Appendix 9.2 for key). None of the points are significantly different from equality, the line of equality being drawn as solid.

Hand-Clasping: RxL vs. LxR

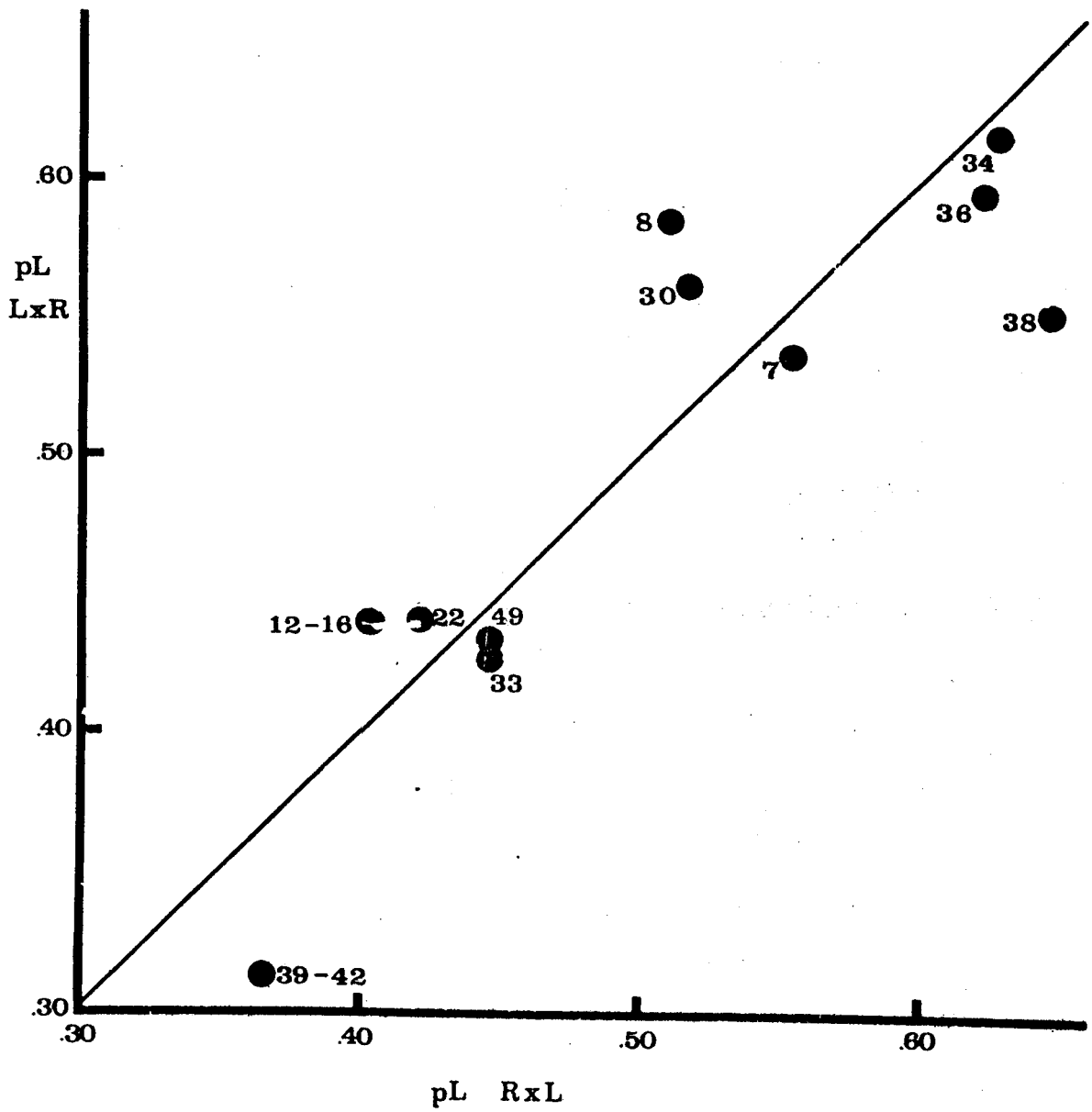


Figure 9:8 Shows the proportion of LHC progeny from RxR (○,●) and LxL (△ ,▲) matings, ± 1 standard error; some standard error bars have been omitted where there is confusing overlap. Japanese studies are shown as solid data points (● ,▲) and non-Japanese studies as open datapoints (○ ,△). p (L) represents the proportion of LHC in the progeny (which does not differ significantly from parental p(L)). For clarity RxL matings have been omitted; in most cases however they are between LxL and RxR, and they have been included when determining the fit of the models. Lines represent the expected values of the best fit two-allele symmetric models described in the text; ------ and ..... for RxR and LxL respectively of Non-Japanese populations, and ——— and _ _ _ _ _ for RxR and LxL respectively of Japanese populations. For key to data-points see Appendix 9.2.

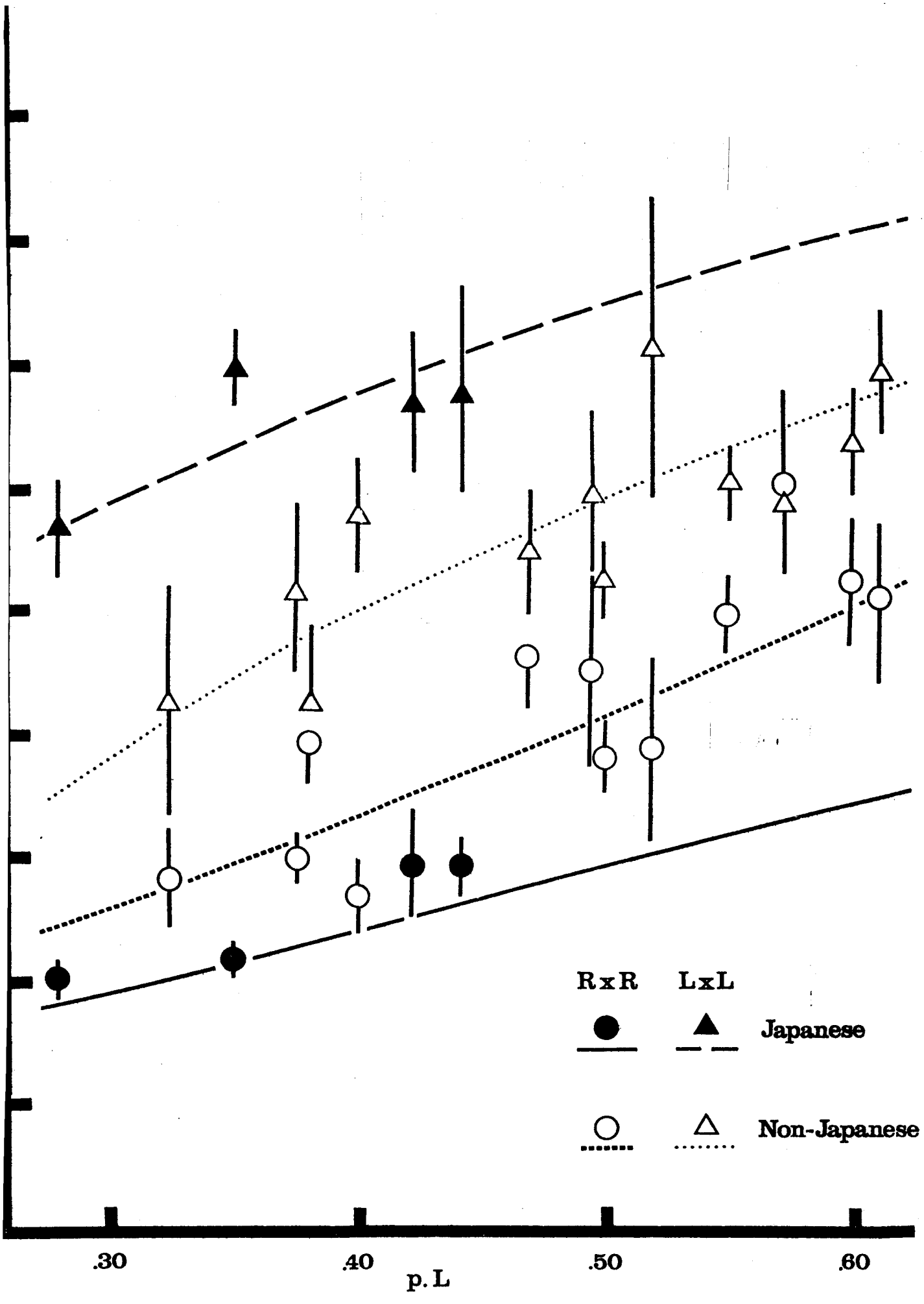

RxR
LxL
Japanese
Non-Japanese
.30
.40
.50
.60
p. L

Figure 9:9 Shows, for twelve non-Japanese studies, the incidence of left hand-clasping in the progeny of RxR matings, R x L matings, and L x L matings. Numbers alongside data points indicate source (see Appendix 9.2 for key). Points are plotted ± standard error. In order to avoid confusing overlap of standard error bars the data for R x L and L x 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 x R, R x L and L x L see Figures 9.8 and 9.10.

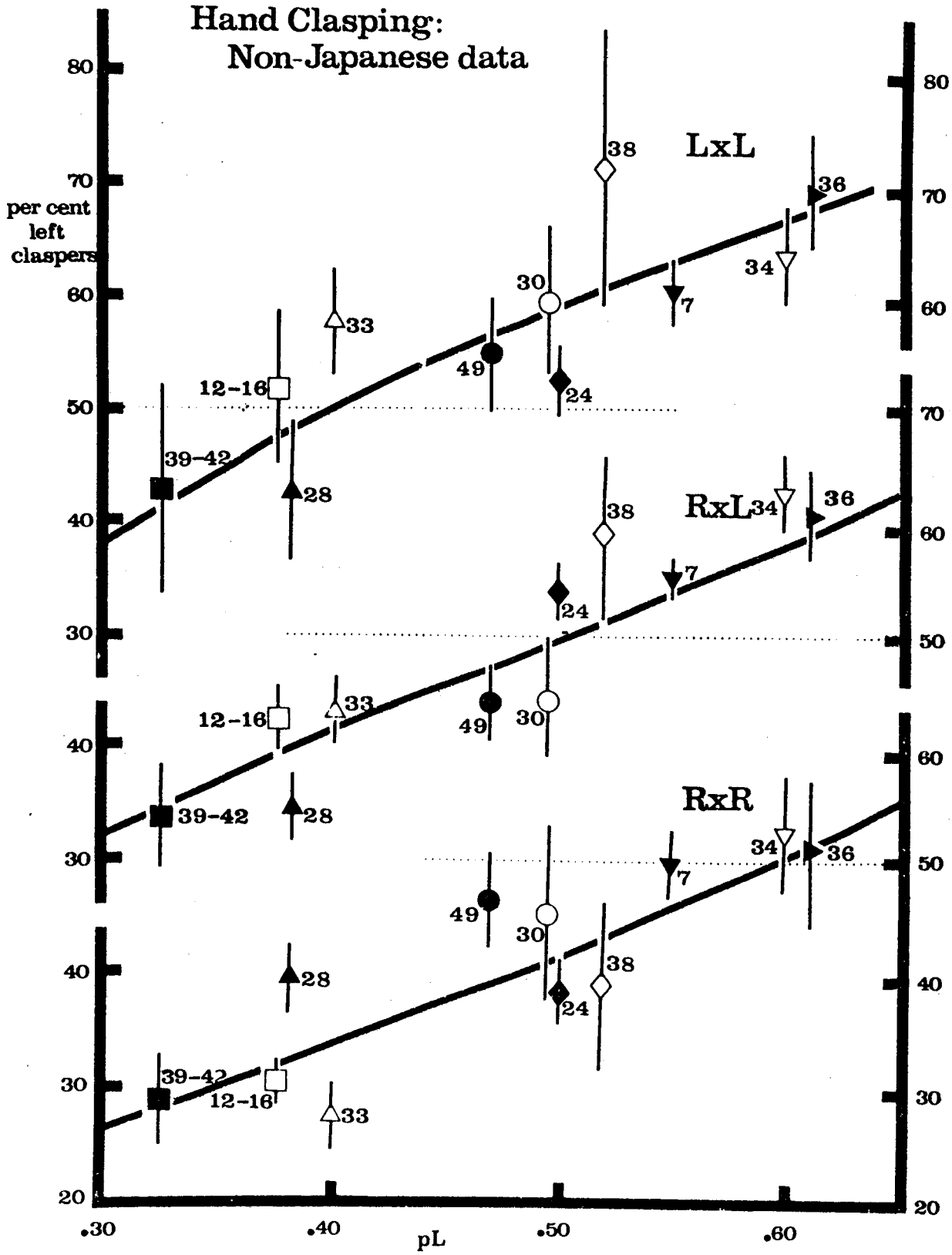

Hand Clasping:
Non-Japanese data
per cent left claspers
LxL
RxL
RxR
pL
80
70
60
50
40
30
20
.30
.40
.50
.60
38
36
34
30
7
33
49
24
12-16
39-42
28

Figure 9:10 shows, for the four Japanese studies, the proportion of left hand-clasping progeny from RxR, RxL and LxL mating pairs. Data points are plotted ± 1 standard error. Numbers alongside datapoints indicate source (see Appendix 9.2 for key). (■) L x L; (●) R x L; (◆) R x R. Solid and dotted lines marked L x L, R x L and R x R represent the fitted lines for non-Japanese studies shown in Figure 9.9 and are present for comparison. Note: unlike figure 9.9 all datapoints in this figure are plotted on the same scale and axes.

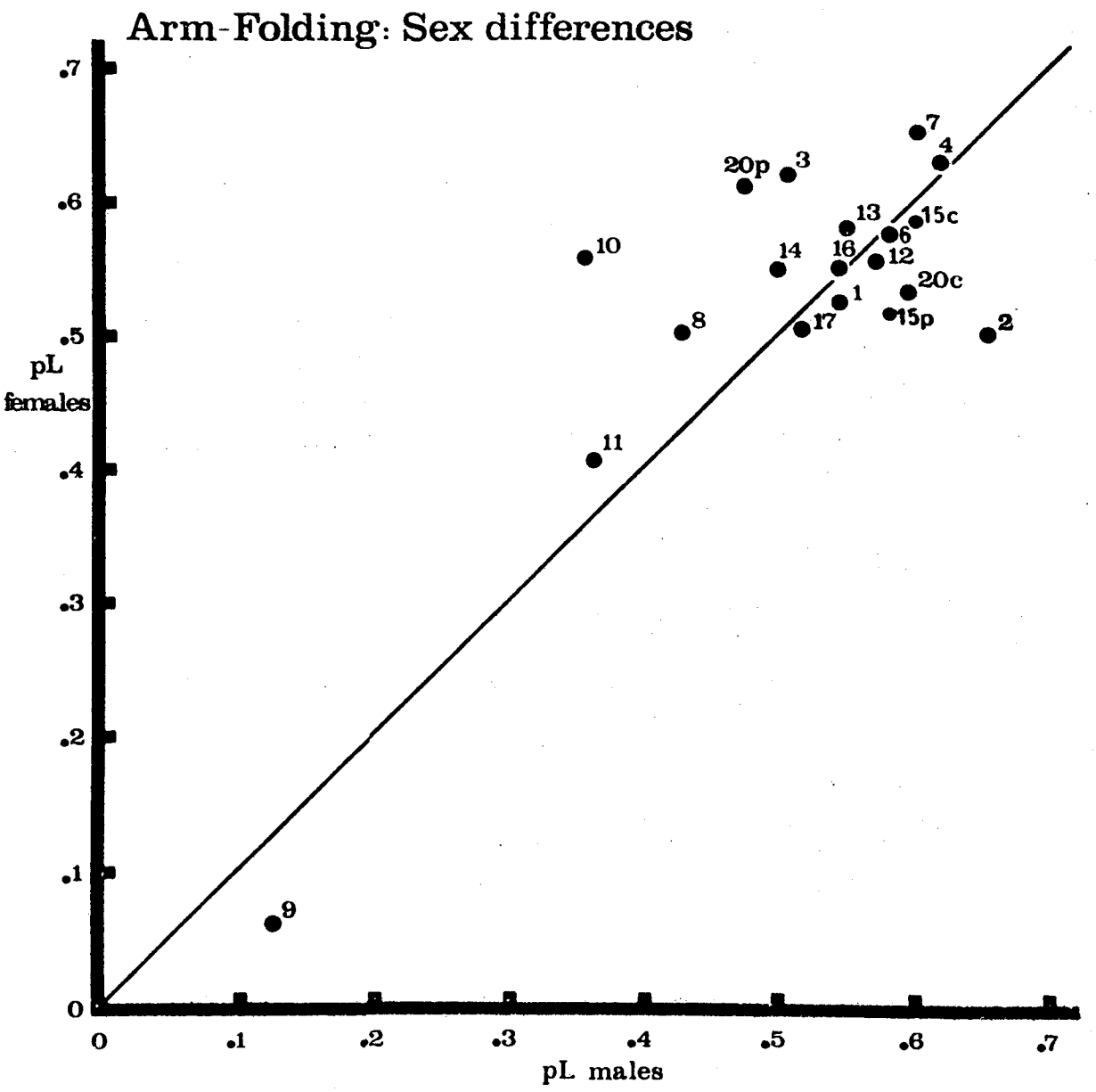
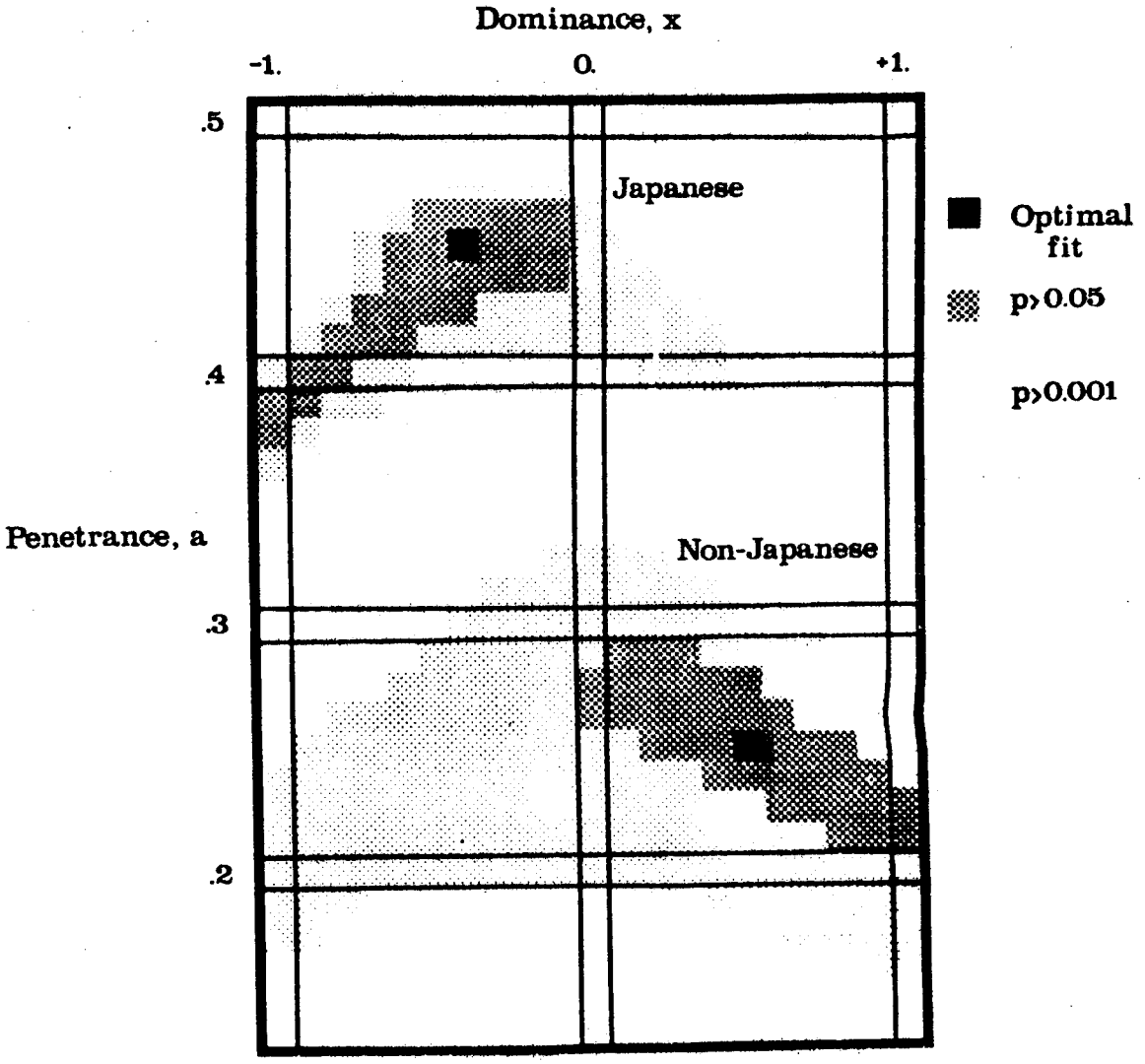

Arm-Folding: Sex differences
.7
.6
.5
.4
.3
.2
.1
0
pL
females
7
4
20p
3
13
15c
10
6
14
16
12
20c
1
8
17
15p
2
11
9
0
.1
.2
.3
.4
.5
.6
.7
pL males

Figure 9:11 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

Hand-Clasping



	L	R
DD	.5-a	.5+a
DS	.5+ax	.5-ax
SS	.5+a	.5-a

Figure 9:12 shows the 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 9.2 for key). A solid circle at the end of a curve means that an absolute limit has been reached (see text for details).

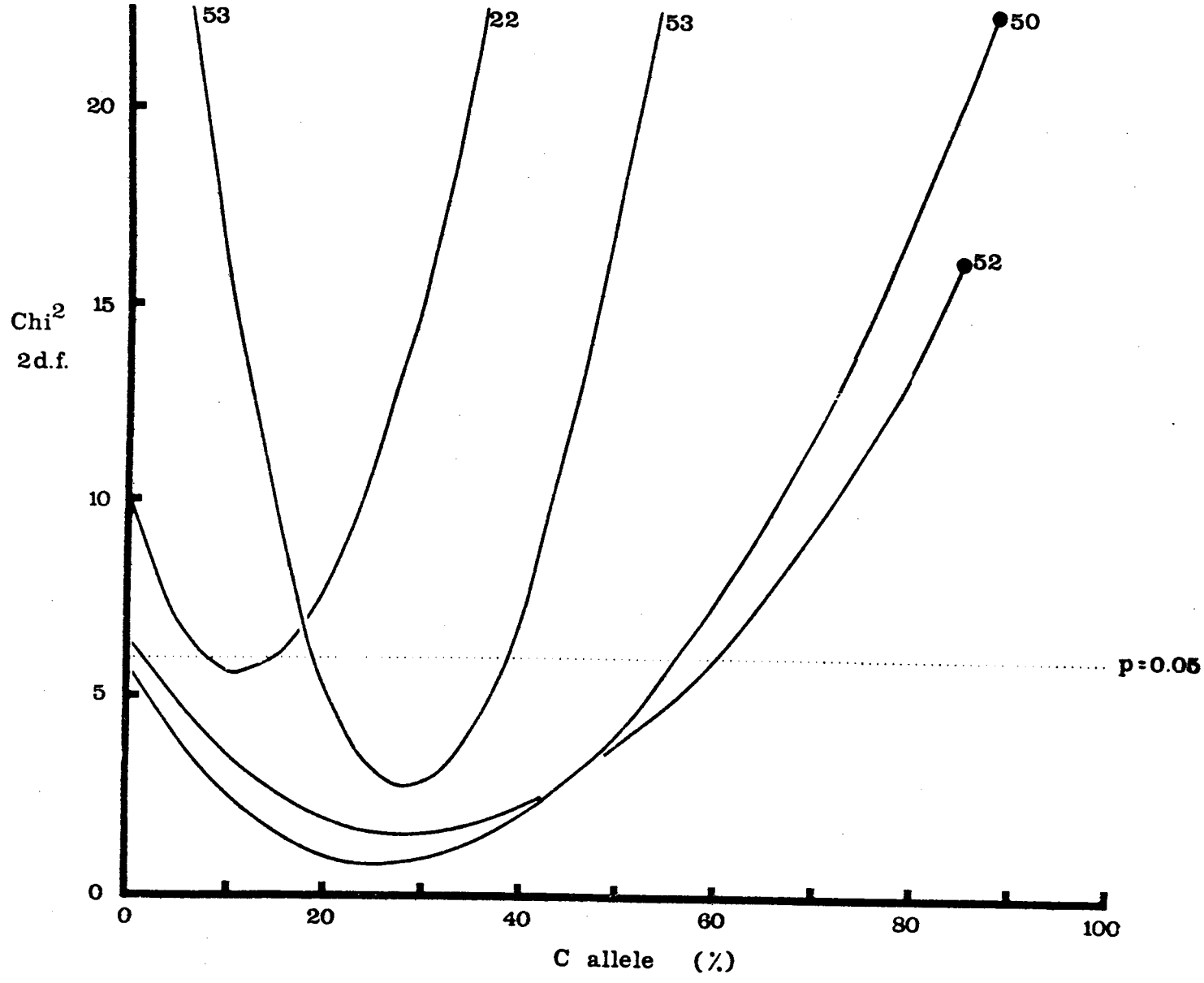

53
22
53
50
52
20
15
10
5
0
Chi2
2d.f.
p=0.05
0
20
40
60
80
100
C allele (%)

Figure 9:13 As for Figure 9:12 but for non-Japanese studies.

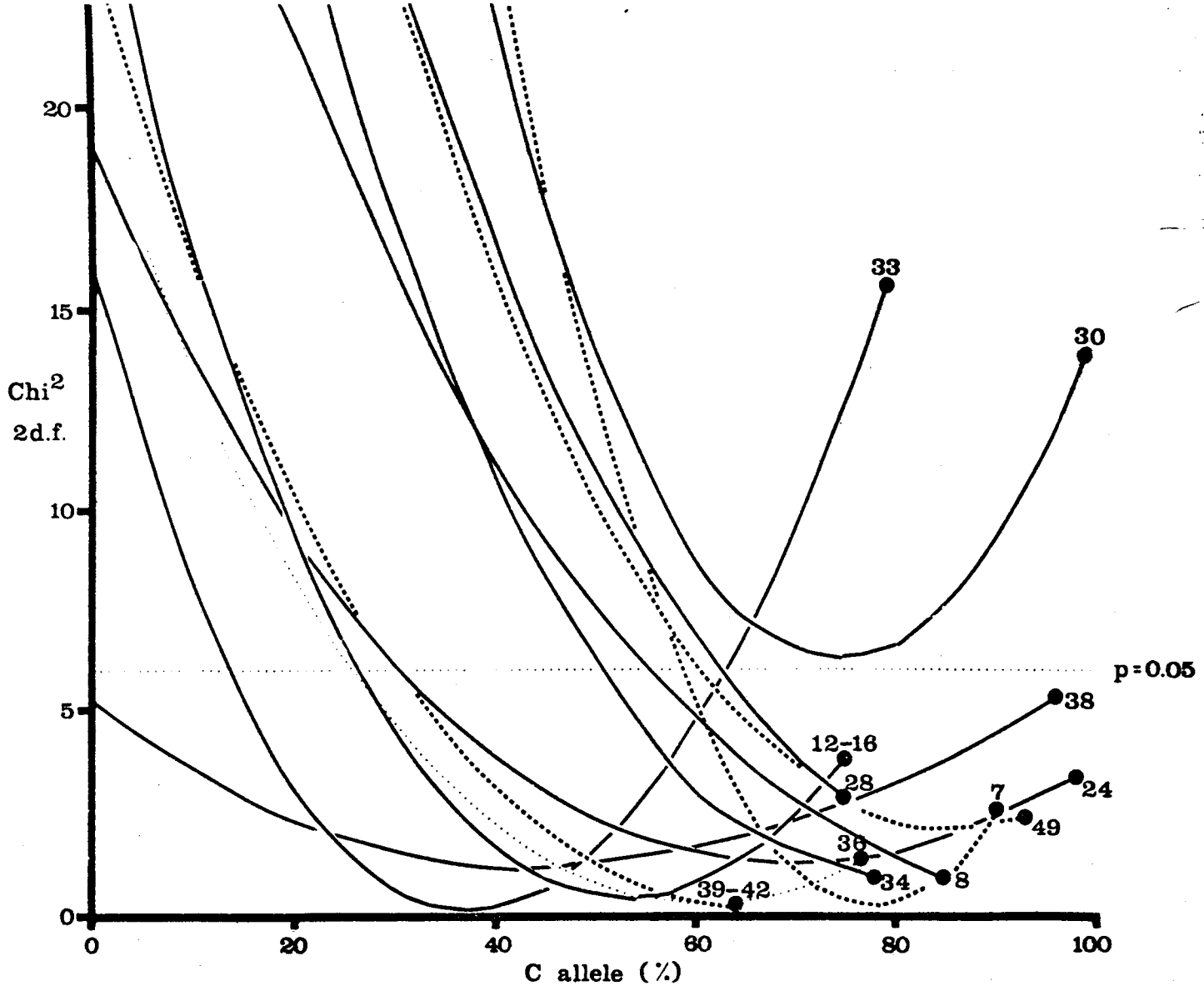

20
15
10
5
0
Chi2
2d.f.
0
20
40
60
80
100
C allele (%)
p=0.05
33
30
38
12-16
28
7
24
49
36
34
8
39-42

Figure 9:14 shows, for twenty-three populations, the incidence of left arm-folding.

Arm-Folding: Population incidences

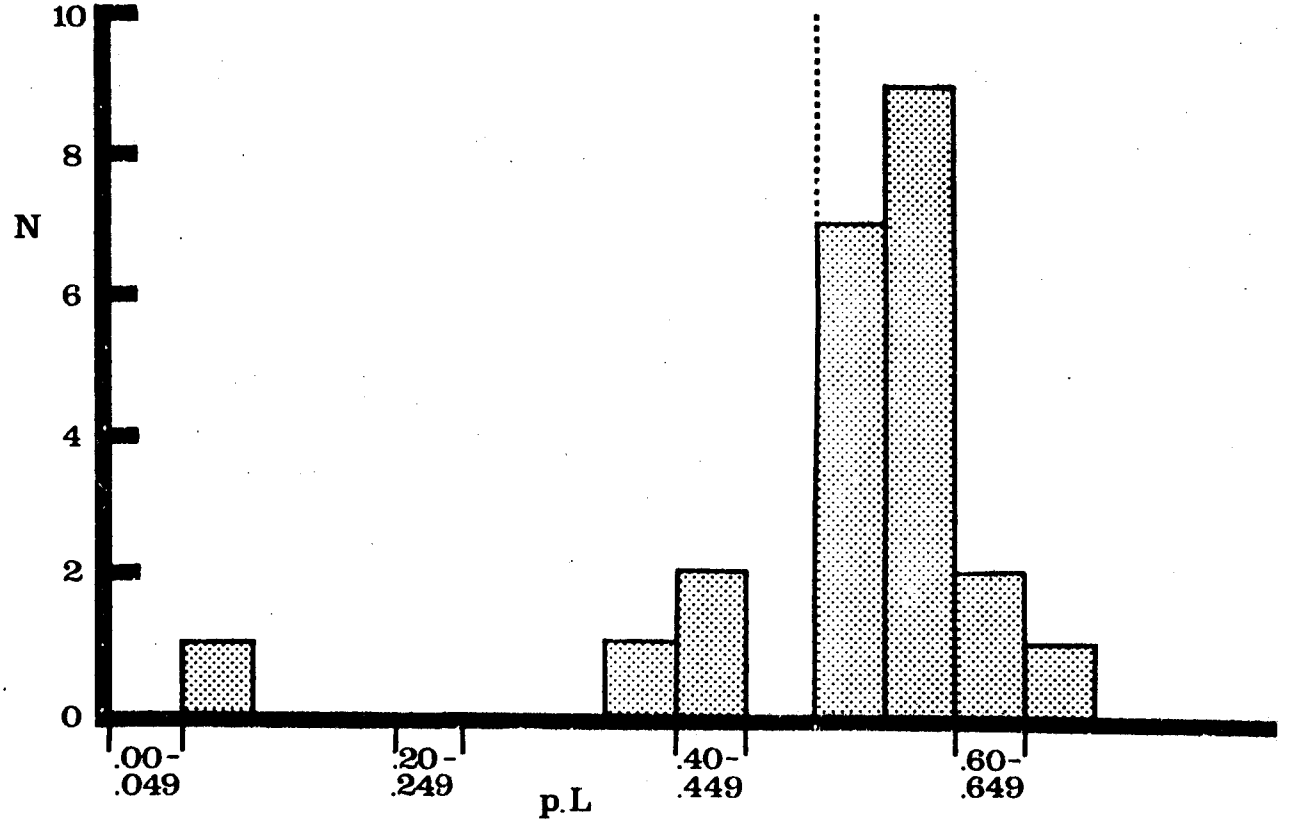
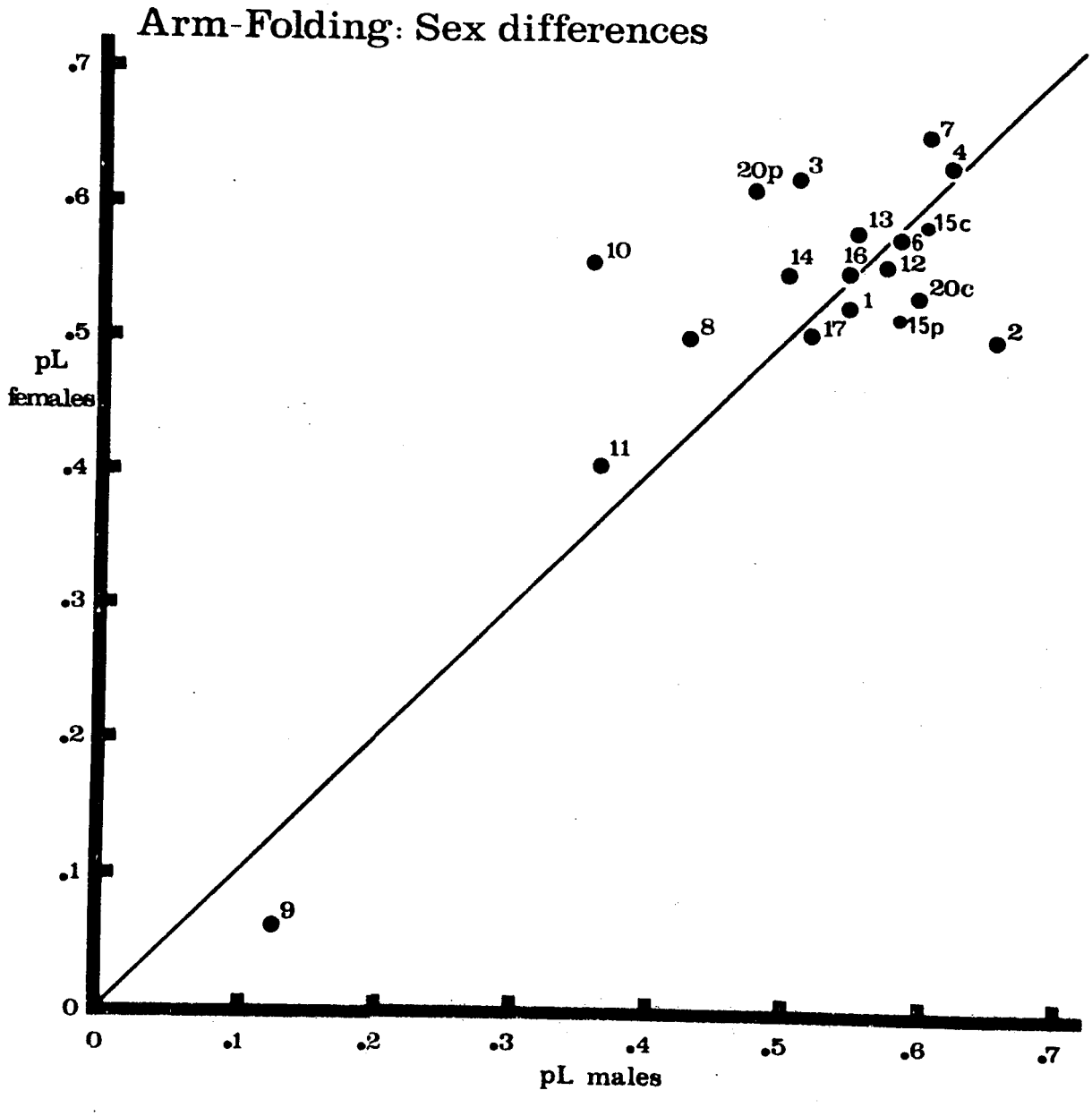


Figure 9:15 shows, the incidence of left arm-folding in females as a function of the incidence of left arm-folding in males, for data from 19 populations. Numbers alongside data points indicate the source (for key see Appendix 9.3).


Arm-Folding: Sex differences
.7
.6
.5
.4
.3
.2
.1
0
pL
females
0
.1
.2
.3
.4
.5
.6
.7
pL males
7
4
20p
3
13
15c
6
10
14
16
12
20c
1
8
17
15p
2
11
9