Handedness in Twins: Joint Analysis of Data From 35 Samples

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Simultaneous analysis of handedness data from 35 samples of twins (with a combined sample size of 21,127 twin pairs) found a small but significant additive genetic effect accounting for 25.47% of the variance (95% confidence interval [CI] 15.69–29.51%). No common environmental influences were detected (C = 0.00; 95% CI 0.00–7.67%), with the majority of the variance, 74.53%, explained by factors unique to the individual (95% CI 70.49–78.67%). No significant heterogeneity was observed within studies that used similar methods to assess handedness, or across studies that used different methods. At an individual level the majority of studies had insufficient power to reject a purely unique environmental model due to insufficient power to detect familial aggregation. This lack of power is seldom mentioned within studies, and has contributed to the misconception that twin studies of handedness are not informative.

The etiology and neurological implications of left-handedness have been the subject of debate for over a century. The first twin study comparing the handedness of monozygotic (MZ) and dizygotic (DZ) twins was published 81 years ago (Siemens, 1924); since this time there have been 34 published studies on 35 samples. However, there has been little consistency of results. The area has become one of the most controversial in laterality with the proposal of special twin effects, (such as differential right-shifts effects or mirror imaging effects; Annett, 2002; Newman, 1928) and questions raised regarding the suitability of the twin method for studying handedness and laterality in general (Nagylaki & Levy, 1973).

Comparison of results across studies is complicated by differences in methodology. Handedness can be assessed as either *hand preference* or *hand skill*; two separate but related traits. Hand preference is typically a directional measure. In its simplest form it is assessed by asking '*are you left or right handed?*', or '*which hand do you write with?*' In its more complex form, hand preference is assessed by asking (or asking the participant to demonstrate) which hand is preferred for a range of items. Handedness questionnaires range in length from four (Coren, 1993) to 55 items (Healey et al., 1986), the number of response choices ranging from two to five, with the responses resulting either in a handedness score/quotient or a grouping classification. The diverse methods of assessing hand preference and the number of questionnaires available within the literature in part reflects the lack of a gold standard and conceptual differences regarding the evolution and nature of handedness.

In contrast, hand skill is typically a quantitative measure of the degree of motor dominance of one hand over the over. Common tests of hand skill involve measuring the time taken to move a series of pegs on a specially designed board (Annett, 1985), or counting the number of circles that can be dotted with a pen in 20 seconds (Tapley & Bryden, 1985). Typically, the task(s) is performed with each hand and a dominance or lambda score is calculated expressing the direction and degree of dominance. The 35 published samples assessing handedness in twins are summarized in Table 1. Of the 35, one has assessed hand skill, 18 have assessed self classification or hand preference for less than three activities, 10 used questionnaires which considered hand preference for three or more activities, and two assessed handedness through observation or parental report. In the remaining four, the method used to assess handedness was not described.

Sociocultural differences across studies further complicate the comparison of results, with social attitudes towards left-handedness differing markedly both across cultures and within cultures over time. In western cultures at the beginning of the last century, left-handedness was considered highly undesirable and left-handed students were often made to write with their right hands. Attitudes towards left-handedness softened midway through the century and the pressure to be right-handed decreased dramatically, with the prevalence of left-handedness rising from

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approximately 3% in those born before 1910 to 12% in those born after the Second World War (data from the [American] participants of the National Geographic survey as cited by McManus, 2002). However, marked cultural differences remain, for example the prevalence of left-handedness ranged from 2.5% in Mexico to 12.8% in Canada in one large international study (Perelle & Ehrman, 1994). From a methodological point of view the changing social attitudes towards left-handedness can be seen in the classification of individuals who identified themselves as ambidextrous. In early studies, ambidextrous individuals were treated as right-handers and in some studies any one identifying as left-handed was required to prove their left-handedness (Lauterbach, 1925), while in later studies ambidextrous participants were usually classified as left-handers. The sociocultural suppression of left-handedness has important implications for behavioral genetic explorations of handedness as in effect it created large numbers of phenocopies (individuals whose phenotype does not match their genotype), and may have acted to decrease the genetic variance of the trait, or increase the environmental variance.

The lack of reliable methods of determining zygosity in the early 1900s has hampered the study of handedness in twins and left a lasting legacy of debate regarding the presence of special-twin, or mirror imaging effects. Based on studies of the shell-markings of nine-banded armadillo and the observation that MZ twins were often discordant for handedness, Newman (1928) proposed a mirror imaging effect. According to this theory in later-splitting embryos, where MZ twinning was hypothesized to occur after lateralization had been established in the blastocyst, the co-twins would show discordant handedness and a range of other heterotaxic or mirrored physical characteristics. This effect would act to increase the rate of handedness discordance within MZ twin pairs and increase the rate of left-handedness across MZ twins. This theory was supported by Newman's own work and a number of early studies (Dahlberg, 1926; Hirsh, 1930), and for a time the presence of discordant handedness was considered a marker of monozygosity, thereby confounding the results of some early handedness studies. However, although there has been little support for this theory since the inception of modern zygosity classification, it still persists in the literature and in the lay mythology surrounding twinning. Similarly, many studies have reported increased rates of left-handedness in twins as compared to singletons. However, twins and singletons are seldom assessed using the same handedness criteria, recruited in the same manner, or matched for age and sex (McManus, 1980). Several of the more recent studies have found no differences in the prevalence of left-handedness between twins and singletons (Ellis et al., 1988; Medland et al., 2003; Morley & Caffrey, 1994).

While these methodological issues make it difficult to draw clear conclusions from the literature, the overarching problem has been the lack of statistical power associated with small sample sizes. Given that the majority of twin studies have used a binary handedness classification (either left vs. right or right vs. nonright) the issue of sample size is nontrivial when trying to determine the confidence that can be placed in results. For example, given a trait with a 10% prevalence (which is typical of left-handedness) where 30% of the variance is accounted for by an additive genetic effect, about 1000 pairs of twins would be required to reject a purely unique environmental model with 80% power (Neale et al., 1994). With few exceptions, sample sizes have not been adequate to detect genetic or environmental effects that account for less than 50% of the total phenotypic variance with 80% power. While the sample size required to detect small to medium genetic effects may be beyond the resources of any one research group, such sample sizes can be reached through collaboration (Medland et al., 2003) or meta-analysis (McManus, 1980; Sicotte et al., 1999).

In 1999 Sicotte and colleagues presented an excellent meta-analysis of the 28 twin studies of handedness published at that time. Since then, data on seven additional samples have been published. Sicotte et al. (1999) addressed the issues of mirror imaging, twin sibling differences and the evidence for genetic effects. They found no evidence of mirror imaging, an increase in left-handedness in twins compared to singletons and higher concordance among MZ than DZ twin pairs, concluding that handedness is subject to genetic influences. The aim of the present article is not to readdress these questions but rather to estimate the magnitude and nature of this genetic effect through joint analysis. Biometrical genetic models were applied to the published contingency table data from each study and the proportions of variance accounted for by additive genetic, unique environmental and common environmental or dominant genetic influences were estimated for each study individually, and then for all 35 samples simultaneously (such modeling techniques had been applied in only four of the studies reviewed: Bishop, 2001; Bishop, in press; Medland et al., 2003; Neale, 1988).

Methods

Studies were included if the sample was nonclinical in nature and included both MZ and DZ twins. The data from Bishop (2001, in press) were included although the twins in these samples were selected for language impairment, as no relationship was found between the language and handedness measures in these studies. The 28 studies of handedness in twins reviewed by Sicotte et al. (1999) were included; and a review of the literature found no additional studies published before 1999. Where studies reported duplicate data, for example, Orlebeke et al. (1996) and James and

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Orlebeke (2002), the larger of the two samples was analyzed. Details of the studies included in the analysis are given in Table 1.

While the majority of the studies assessed hand preference as a binary construct, some utilized multicategory ordinal variables or continuous variables. In the review by Sicotte et al. (1999) the data from these studies were reduced to binary variables. For the purposes of consistency and to avoid problems associated with the nonnormal nature of hand preference data (which is J-shaped) the reclassifications made by Sicotte et al. (1999) have been used in these analyses. In addition, the data of Bishop (2001) were reduced to a binary classification.

Table 1

Previous Twin Studies of Handedness: Method of Handedness Assessment, and Pairwise Handedness (L = Left-Handed, R = Right-Handed) for MZ and DZ Twins

(Author, year)	Handedness criteria	MZ			DZ		
		RR	LR/RL	LL	RR	LR/RL	LL
(Siemens, 1924)	Not stated	41	9	1	16	13	2
(Dahlberg, 1926)	Hand used to cut and throw	53	12	4	111	16	1
(von Verschuer, 1927)	Not stated	58	15	6	28	10	0
(Hirsh, 1930)	Self-classification	25	18	0	51	7	0
(Wilson & Jones, 1932)	Throwing hand	56	13	1	97	24	2
(Stocks, 1933)	Writing hand	35	6	1	76	16	2
(Komai & Fukuoka, 1934)	Hand used for brush writing	112	6	0	60	1	1
(Newman et al., 1937)	Hand skill — tapping task	30	17	3	39	11	0
(Rife, 1940)	Hand used for 9 tasks Any L responses = LH	176	41	6	104	39	3
(Thyss, 1946)	Not stated	72	24	7	60	24	2
(Rife, 1950)	As in Rife, 1940	261	76	6	164	45	2
(Dechaume, 1957)	Not stated	19	12	2	21	11	1
(Zazzo, 1960)	Self-classification	199	51	9	264	69	2
(Koch, 1966)	Observation and parent report	28	3	4	45	6	4
(Carter-Saltzman et al., 1976)	Writing hand	132	46	9	115	54	7
(Loehlin & Nichols, 1976)	Self-classification	380	123	11	261	70	2
(Springer & Searleman, 1978)	Writing hand	53	19	3	35	9	3
(Hay & Howie, 1980)	11-item questionnaire	9	7	0	10	3	0
(Osborne, 1980)	Self-classification	76	27	4	90	40	0
(Boklage, 1981)	Self-classification	145	45	24	132	69	13
(Shimizu & Endo, 1983)	Questionnaire	57	4	1	41	7	0
(Forrai & Bankovi, 1983)	Self-classification	78	16	2	44	21	3
(Tambs et al., 1987)	Writing hand	175	21	1	171	32	0
(NCD as cited by McManus, 1985)	Writing hand	32	9	2	66	18	4
(Neale, 1988)	Self-classification	655	158	23	626	183	23
(Derom et al., 1996)	Self-classification	249	86	17	276	109	23
(Carlier et al., 1996)	Writing hand	48	6	1	15	9	0
(Orlebeke et al., 1996)	Self-classification	475	122	25	764	255	22
(Ross et al., 1999)	Hand preference for 5 tasks: write, draw, throw, scissors, toothbrush.	923	214	21	805	155	13
(Reiss et al., 1999)	Hand preference for 12 tasks	28	5	0	58	9	0
(Basso et al., 2000)	Self-classification †	1049	136	19	1762	247	16
(Bishop, 2001) Manchester sample	Number of items on a questionnaire completed with the right hand †	35	14	1	65	14	7
(Bishop, 2001) Cambridge sample	Writing hand †	36	9	0	33	10	1
(Medland et al., 2003)	Throwing hand	1894	534	89	2050	632	80
(Bishop, in press)	Writing hand	67	27	6	62	24	8

Notes: † Pairwise data were obtained by contacting the authors. The Basso et al. (2000) data presented here include data on all available twin pairs born before 1910. The pairwise data from Bishop (2001, Cambridge sample; 2005) reported here are only for the writing hand item, the original published data reports a laterality quotient. For the Bishop (2001) Manchester sample, participants who completed less than 6 items with the right hand are coded as left-handers. The original published data reports the full distribution.

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In order to estimate the proportion of phenotypic variance arising from additive (A) and nonadditive (dominant D) genetic sources and shared (C) and nonshared (E) environmental influences, the data from previous studies were entered into a joint analysis. Because our twin data are assumed to come from MZ and DZ twins raised together, the effects of C and D are confounded and cannot be estimated together. Both ACE and ADE models in which the prevalence of left-handedness of MZ and DZ twins were allowed to differ, between and within studies, were fit to the data.

The amount of variance explained by A, C or D, and E in each sample were calculated from 2 × 2 contingency tables for MZ and DZ twins using Mx (1.54). These analyses were conducted within the framework of the multifactorial threshold model which posits a continuous normally distributed liability for laterality on which thresholds are imposed that define the prevalence of different definitions of handedness. The procedure is readily extended to multiple groups so that hypotheses about equality of variance components between studies can be tested. Variance components were estimated from each study individually. The variance components were then equated across studies to test for heterogeneity, and pooled variance components were estimated for all studies jointly.

Results

Individual Studies

The proportion of variance accounted for by A, C and E from the ACE model within each study, the 95% confidence intervals surrounding these estimates, and the chi-square fit of the model are summarized in Figure 1. An ACE model provided a good fit to the data from all studies, except that of Osborne (1980; = 7.95, p = .02). There were no obvious differences between this and other studies, and the lack of fit reflects the large negative correlation observed in the DZ twins which is incompatible with both genetic and environmental models (r = –.98; 95% CI –1.00, –0.20). This pattern of data may reflect the effects of co-twin competition within this sample, or selection bias given the small sample size. The proportion of variance accounted for by A, D and E, 95% confidence intervals, and the chi-square fit of the model are summarized in Figure 2. The ADE model provided a poor fit for three studies; Komai and Fukuoka (1934; = 6.76, p = .03), Osborne (1980; = 7.9, p = .02), and Bishop (2001, Manchester sample; = 8.31, p = .02).

As shown in the forest plots the variance components estimates varied widely, with the lower confidence intervals on A and C seldom higher than zero in the ACE model. While for the ADE model the

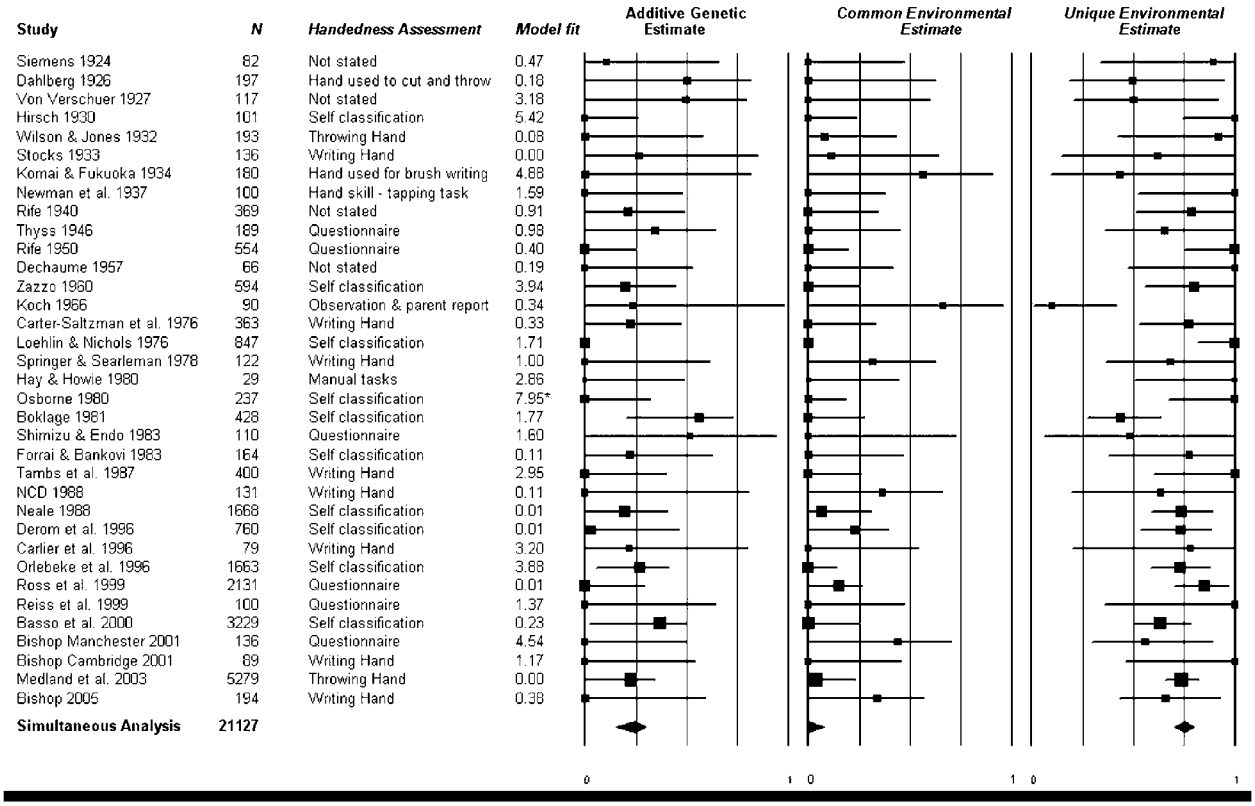

Figure 1

Sample size, handedness criteria, model fit (* p < .05, 2df), and standardized estimates of additive genetic, common environmental and unique environmental variance components (with 95% confidence intervals given by the horizontal bars) by study.

The relative size of the sample is indicated by the size of the data point. For the estimates derived from the joint analysis, the 95% confidence intervals are given by the width of the polygons.

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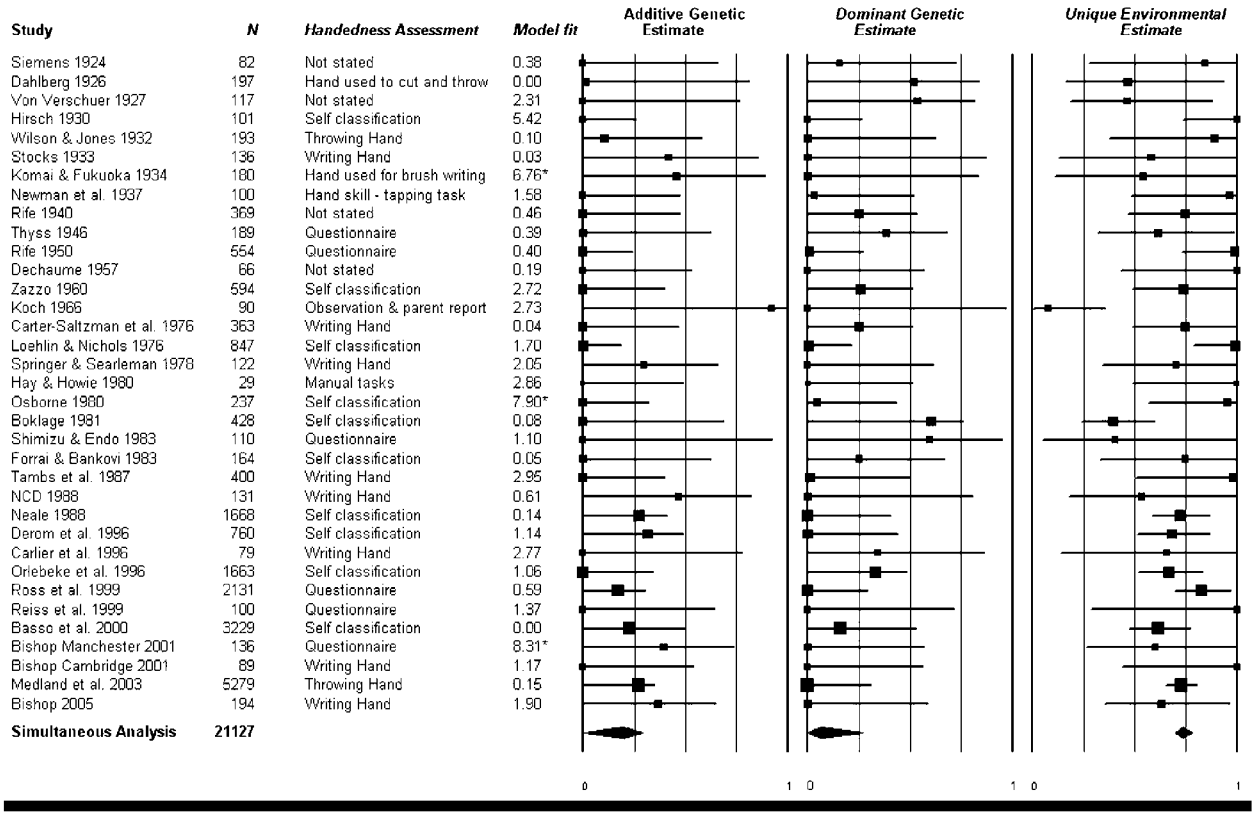

Figure 2

Sample size, handedness criteria, model fit (* $p < .05$, 2*df*), and standardized estimates of additive genetic, dominant genetic and unique environmental variance components (with 95% confidence intervals given by the horizontal bars) by study.

The relative size of the sample is indicated by the size of the data point. For the estimates derived from the joint analysis, the 95% confidence intervals are given by the width of the polygons.

lower confidence intervals were zero for A and D across studies. Individually less than half of the studies could reject a purely environmental model. There was no clear effect of year of publication which may reflect the fact that year of publication is not always a good proxy measure of participant age/birth cohort (i.e., year of birth ranged from 1896 to 2000 in Medland et al., 2003, and 1870 to 1910 in extended sample of Basso et al., 2000). Unsurprisingly, sample size appears an important factor in detecting familial aggregation (an upper confidence interval for E < 1). Sixty-six per cent of studies with 500 or more participants and all of those with a sample greater than 1000 pairs found familial aggregation (using an ACE model), as compared to 19% of those with less than 500 pairs. However, few studies had sufficient power to determine whether the familial aggregation was genetic or environmental in nature. In the ACE model for all 35 studies the 95% CI surrounding C encompassed zero.

Joint Analysis

ACE Model: The data from each study were entered into a joint analysis (in which each group was modeled separately resulting in 70 data groups). The variance components were first equated across studies that used similar methods of assessing handedness: Self-classification, handedness based on less than three activities, handedness for longer questionnaires, and other methods. As shown in Table 2 equating estimates in this manner did not result in any significant differences. Similarly variance components could be equated across all studies regardless of the method used to assess handedness. This resulted in a change in –2 log-likelihood of 70.64 for 68 degrees of freedom (102 equated parameters — 34 constraints; $p = .39$). Across studies additive genetic factors accounted for 25.47% of the variance (95% CI 15.69–29.51%), no significant common environmental effect was found (C = 0.00; 95% CI 0.00–7.67%), with the largest proportion of variance, 74.53%, explained by unique environmental effect (95% CI 70.49–78.67%). To examine the influence of the largest sample on these results the analysis was rerun excluding the data of Medland et al. (2003), although the confidence intervals increased there was no substantial difference in the results. The analyses were also rerun excluding the data from Osborne (1980) to determine the influence of this sample (which was inconsistent with an ACE model) on the simultaneous analysis, once again though the confidence intervals shifted slightly there was little change in the variance component estimates.

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Table 2

Change in Fit Due to Equating Parameters Across Studies and Standardized Variance Components at Each Step of the Analysis

Model	Compared to model	$\Delta\chi^2$	Δdf	p	Standardized variance components (CI 95%) Additive genetic	Common environment	Unique environment
1 ACE Saturated Model (–2LL 31,119.43, *df* 420)							
2 Equating studies that used self-classification	1	26.74	18	.08	.268 (.154, .331)	.000 (.000, .083)	.732 (.669, .796)
3 Equating studies that used less than 3 items	2	24.67	22	.31	.206 (.000, .344)	.066 (.000, .227)	.728 (.656, .802)
4 Equating studies that used self-classification or less than 3 items	3	0.73	2	.69	.278 (.159, .323)	.000 (.000, .092)	.722 (.677, .770)
5 Equating studies that used questionnaires with more than 3 items	4	8.62	10	.56	.000 (.000, .243)	.133 (.000, .223)	.867 (.755, .958)
6 Equating studies that used other methods	5	3.36	8	.91	.204 (.000, .391)	.000 (.000, .222)	.795 (.609, .989)
7 Equating across all studies	1	70.64	68	.39	.255 (.157, .295)	.000 (.000, .076)	.745 (.704, .787)
8 Excluding Medland et al., 2003	1	68.08	66	.41	.259 (.148, .299)	.000 (.000, .087)	.742 (.701, .784)
9 Excluding Osbourne, 1980	1	70.21	66	.34	.248 (.149, .296)	.000 (.000, .076)	.752 (.704, .801)
10 Dropping common environmental effects from the model	7	0.02	1	1.00	.255 (.214, .295)		.745 (.705, .787)

Model	Compared to model	$\Delta\chi^2$	Δdf	p	Standardized variance components (CI 95%) Additive genetic	Dominant genetic	Unique environment
1 ADE Saturated Model (–2LL 31,123.11, *df* 420)							
2 Equating studies that used self-classification	1	28.52	18	.05	.000 (.000, .656)	.158 (.000, .725)	.842 (.275, 1.00)
3 Equating studies that used less than 3 items	2	18.63	22	.67	.000 (.000, .656)	.151 (.000, .725)	.849 (.275, 1.00)
4 Equating studies that used self-classification or less than 3 items	3	1.73	2	.42	.000 (.000, .656)	.158 (.000, .725)	.842 (.275, 1.00)
5 Equating studies that used questionnaires with more than 3 items	4	5.87	10	.83	.000 (.000, .656)	.157 (.000, .725)	.843 (.275, 1.00)
6 Equating studies that used other methods	5	3.38	8	.91	.000 (.000, .355)	.248 (.000, .438)	.752 (.562, .956)
7 Equating across all studies	1	66.60	68	.53	.197 (.005, .293)	.064 (.000, .271)	.739 (.695, .784)
8 Excluding Medland et al., 2003	1	63.08	66	.44	.197 (.005, .293)	.064 (.000, .271)	.739 (.695, .784)
9 Excluding Osborne, 1980; Komai & Fukuoka, 1934; (Bishop, 2001) Manchester sample	1	65.73	66	.49	.181 (.000, .294)	.083 (.000, .290)	.735 (.690, .782)
10 Dropping dominance effects from the model	7	0.37	1	.55	.255 (.214, .295)		.745 (.705, .787)

ADE Model: The data from each study were entered into a joint analysis using the same procedure as described for the ACE Model. As shown in Table 2 equating estimates did not result in any significant differences (Δ–2LL 66.60 for 68 degrees of freedom; 102 equated parameters — 34 constraints; p = .53). Across studies additive genetic factors accounted for 19.7% of the variance (95% CI 0.50–29.3%), no significant dominant genetic effect was found (D = 6.4; 95% CI 0.00–27.1%), with the largest proportion of variance, 73.9%, explained by unique environmental effect (95% CI 69.50–78.40%). Rerunning the analyses excluding the data of Medland et al. (2003), led to no substantial differences in the results. Similarly excluding the data of Osborne (1980), Komai and Fukuoka (1934), and Bishop (2001, Manchester sample) did not alter the results. Dominant genetic effects could be dropped from the model without significant loss of fit (Δ–2LL 0.37 for 1 degree of freedom). Thus, the most parsimonious model was one in which the familial aggregation for hand preference was explained by additive genetic influences.

Discussion

Joint analysis of handedness in 35 samples of MZ and DZ twins has shown that the data are consistent with an additive genetic model of familial aggregation, in which around 25% of the variation in liability to left-handedness is explained by additive genetic influences. Somewhat surprisingly, no significant heterogeneity was observed between studies, suggesting that any cohort and or cultural differences in genetic contribution to liability between studies were minimal.

The nature of the data (contingency tables from published studies) did not allow the prevalence of left-handedness to be corrected for known covariates such as sex, cohort (year of birth), and birthweight. However, allowing the prevalence to vary freely across studies and across zygosity groups within studies would have allowed for covariate differences at a

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group level. Similarly, the structure of the data did not allow an investigation of the effects of covariates on the proportions of variance accounted for by genetic and environmental effects, thereby limiting the scope of the current analyses. In the absence of Gene × Environment interaction, the multifactorial threshold model predicts homogeneity of variance components across studies with different observed prevalence of left-handedness (i.e., differing thresholds).

One of the main conclusions that may be drawn from this analysis is that, at an individual level, it is clear that many studies have not had sufficient power to detect familial aggregation. This lack of power is seldom mentioned within studies, and should not be considered common knowledge. This unacknowledged limitation has contributed to the belief that twin studies of handedness are not informative.

A number of competing genetic models have been proposed in the literature (Annett, 1985; Crow, 2002; Klar, 1999; McManus, 1985) and these have been applied to some of the data described here (e.g., Annett, 2002), with the results generally showing an acceptable fit of one or more of the models. While the polygenic ACE model utilized here provided a good fit to all but one of the studies, the lack of power associated with many studies limits the conclusions that can be made regarding the polygenic nature of the genetic influences on handedness. Linkage analysis of hand skill by Francks et al., (2003, 2002) has found significant parent of origin (maternally imprinted) linkage to the chromosomal region 2p12–q11, with the second most significant linkage on chromosome 17. Although the genetic correlation between hand skill and hand preference has not been thoroughly investigated in unselected samples, the results of linkage analysis for hand skill are inconsistent with the models of Annett, McManus, Klar and Crow, and suggest that hand skill, and by association hand preference, are complex traits that may be influenced by more than one gene.

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References

Annett, M. (1985). *Left, right, hand and brain: The right shift theory*. London: Lawrence Erlbaum.

Annett, M. (2002). *Handedness and brain asymmetry: The right shift theory*. Hove, UK: Psychology Press.

Basso, O., Olsen, J., Holm, N. V., Skytthe, A., Vaupel, J. W., & Christensen, K. (2000). Handedness and mortality: A follow-up study of Danish twins born between 1900 and 1910. *Epidemiology, 11*, 576–580.

Bishop, D. V. M. (2001). Individual differences in handedness and specific speech and language impairment: Evidence against a genetic link. *Behavior Genetics, 31*, 339–351.

Bishop, D. V. M. (in press). Handedness and specific language impairment: A study of 6-year-old twins. *Developmental Psychobiology*.

Boklage, C. E. (1981). On the distribution of nonright handedness among twins and their families. *Acta Geneticae Medicae et Gemellologiae, 30*, 167–187.

Carlier, M., Spitz, E., Vacher-Lavenu, M. C., Villeger, P., Martin, B., & Michel, F. (1996). Manual performance and laterality in twins of known chorion type. *Behavior Genetics, 26*, 409–418.

Carter-Saltzman, L., Scarr-Salapatek, S., Barker, W. B., & Katz, S. (1976). Left-handedness in twins: Incidence and patterns of performance in an adolescent sample. *Behavior Genetics, 6*, 189–203.

Coren, S. (1993). The lateral preference inventory for measurement of handedness, footedness, eyedness and earedness: Norms for young adults. *Bulletin of the Psychonomic Society, 31*, 1–3.

Crow, T. J. (2002). Handedness, language lateralisation and anatomical asymmetry: Relevance of protocadherin XY to hominid speciation and the aetiology of psychosis. Point of view. *British Journal of Psychiatry, 181*, 295–297.

Dahlberg, G. (1926). *Twin births and twins from a hereditary point of view* [as cited by Sicotte, Woods & Mazziotta, 1999]. Stockholm, Sweden: Tidens Forlag.

Dechaume, M. P. (1957). *Contribution a l'etude de la dominance laterale chez les jumeaux* [as cited by Sicotte, Woods, & Mazziotta, 1999]. Unpublished manuscript.

Derom, C., Thiery, E., Vlietinck, R., & Loos, R. (1996). Handedness in twins according to zygosity and chorion type: A preliminary report. *Behavior Genetics, 26*, 407–408.

Ellis, S. J., Ellis, P. J., & Marshall, E. (1988). Hand preference in a normal population. *Cortex, 24*, 157–163.

Forrai, G., & Bankovi, G. (1983). A Hungarian twin study on hand clasping, arm folding and tongue curling [as cited by Sicotte, Woods & Mazziotta, 1999]. *Acta Biologica Hungarica, 34*, 99–106.

Francks, C., DeLisi, L. E., Shaw, S. H., Fisher, S. E., Richardson, A. J., Stein, J. F., & Monaco, A. P. (2003). Parent-of-origin effects on handedness and schizophrenia susceptibility on chromosome 2p12–q11. *Human Molecular Genetics, 12*, 3225–3230.

Francks, C., Fisher, S., MacPhie, L., Richardson, A., Marlow, A., Stein, J., & Monaco, A. (2002). A genomewide linkage screen for relative hand skill in sibling pairs. *American Journal of Human Genetics, 70*, 800–805.

Hay, D. A., & Howie, P. M. (1980). Handedness and differences in birthweight of twins. *Perceptual and Motor Skill, 51*, 666.

https://doi.org/10.1375/twin.9.1.46 Published online by Cambridge University Press

Healey, J. M., Liederman, J., & Geschwind, N. (1986). Handedness is not a unidimensional trait. *Cortex, 22*, 33–53.

Hirsh, N. D. M. (1930). *Twins, hereditary and environment* [as cited by Sicotte, Woods, & Mazziotta, 1999]. Cambridge, MA: Harvard University Press.

James, W. H., & Orlebeke, J. F. (2002). Determinants of handedness in twins. *Laterality, 7*, 301–307.

Klar, A. J. S. (1999). Genetic models for handedness, brain lateralization, schizophrenia, and manic-depression. *Schizophrenia Research, 39*, 207–218.

Koch, H. L. (1966). *Twins and twin relations* [as cited by Sicotte, Woods, & Mazziotta, 1999]. Chicago: University of Chicago Press.

Komai, T., & Fukuoka, G. (1934). A note on the problem of mirror-imaging in human twins [as cited by Sicotte, Woods, & Mazziotta, 1999]. *Human Biology, 6*, 24–32.

Lauterbach, C. E. (1925). Studies in twin resemblances [as cited by Sicotte, Woods, & Mazziotta, 1999]. *Genetics, 10*, 525–568.

Loehlin, J., & Nichols, R. C. (1976). *Heredity, environment and personality: A study of 850 sets of twins* [as cited by Sicotte, Woods, & Mazziotta, 1999]. Austin, TX: University of Texas Press.

McManus, C. (2002). *Right hand, left hand: The origins of asymmetry in brains, bodies, atoms and cultures*. London: Weidenfeld & Nicolson.

McManus, I. C. (1980). Handedness in twins: A critical review. *Neuropsychologia, 18*, 347–355.

McManus, I. C. (1985). Handedness, language dominance and aphasia: A genetic model. *Psychological Medicine Monographs Supplement, 8*, 1–40.

Medland, S. E., Wright, M. J., Geffen, G. M., Hay, D. A., Levy, F., Martin, N. G., & Duffy, D. (2003). Special twin environments, genetic influences and their effects on the handedness of twins and their siblings. *Twin Research, 6*, 119–130.

Morley, R., & Caffrey, E. A. (1994). Handedness in blood donors: No association with blood group or twinning. *Cortex, 30*, 707–710.

Nagylaki, T., & Levy, J. (1973). 'The sound of one paw clapping' isn't sound. *Behavior Genetics, 3*, 279–292.

Neale, M. C. (1988). Handedness in a sample of volunteer twins. *Behavior Genetics, 18*, 69–79.

Neale, M. C., Eaves, L. J., & Kendler, K. S. (1994). The power of the classical twin study to resolve variation in threshold traits. *Behavior Genetics, 24*, 239–258.

Newman, H. H. (1928). Studies of human twins II: Asymmetry reversal of mirror imaging in identical twins. *Biological Bulletin, 55*, 298–315.

Newman, H. H., Freeman, F. N., & Holzinger, K. J. (1937). *Twins: A study of heredity and environment*. Chicago: University of Chicago Press.

Orlebeke, J. F., Knol, D. L., Koopmans, J. R., Boomsma, D. I., & Bleker, O. P. (1996). Left-handedness in twins: Genes or environment? *Cortex, 32*, 479–490.

Osborne, R. T. (1980). *Twins: Black and white* [as cited by Sicotte, Woods, & Mazziotta, 1999]. Athens, Greece: Foundation for Human Understanding.

Perelle, I. B., & Ehrman, L. (1994). An international study of human handedness: The data. *Behavior Genetics, 24*, 217–227.

Reiss, M., Tymnik, G., Kögler, P., & Kögler, W. (1999). Laterality of hand, foot, eye and ear in twins. *Laterality, 4*, 287–298.

Rife, D. C. (1940). Handedness, with special reference to twins. *Genetics, 25*, 178–186.

Rife, D. C. (1950). An application of gene frequency analysis to the interpretation of data from twins. *Human Biology, 22*, 136–145.

Ross, D. C., Jaffe, J., Collins, R. L., Page, W., & Robinette, D. (1999). Handedness in the NAS/NRC Twin Study. *Laterality, 4*, 257–264.

Shimizu, A., & Endo, M. (1983). Comparison of patterns of handedness between twins and singletons in Japan. *Cortex, 19*, 345–352.

Sicotte, N., Woods, R., & Mazziotta, J. (1999). Handedness in twins: A meta-analysis. *Laterality, 4*, 265–286.

Siemens, H. W. (1924). Uber Linkshandigkeit [as cited by Sicotte, Woods, & Mazziotta, 1999]. *Virchow's Archives, 252*, 1–24.

Springer, S., & Searleman, A. (1978). Laterality in twins: The relationship between handedness and hemispheric asymmetry for speech. *Behavior Genetics, 8*, 349–357.

Stocks, P. (1933). A biometrics investigation of twins and their brothers and sisters. *Annals of Eugenics, 5*, 1–55.

Tambs, K., Magnus, P., & Berg, K. (1987). Left-handedness in twin families: Support of an environmental hypothesis. *Perceptual and Motor Skill, 64*, 155–170.

Tapley, S. M., & Bryden, M. P. (1985). A group test for the assessment of performance between the hands. *Neuropsychologia, 23*, 215–221.

Thyss, J. (1946). *Etude bibliographique et critique du problème des gauches* [as cited by Sicotte, Woods & Mazziotta, 1999]: Unpublished manuscript.

von Verschuer, O. (1927). Die vererbungsbiologische zwillingsforschung [as cited by Sicotte, Woods & Mazziotta, 1999]. *Ergebnisse der Inneren Medizin und Kinderheilkunde, 31*, 35–102.

Wilson, P. T., & Jones, H. E. (1932). Left-handedness in twins. *Genetics, 17*, 560–571.

Zazzo, R. (1960). *Les jumeaux: Le couple et la personne* [as cited by Sicotte, Woods, & Mazziotta, 1999]. Paris: Presses Universitaires de France.

https://doi.org/10.1375/twin.9.1.46 Published online by Cambridge University Press