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# The Epigenesis of Planum Temporale Asymmetry in Twins

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## Abstract

Variation in hemispheric asymmetry of the planum temporale (PT) has been related to verbal ability. The degree to which genetic and environmental factors mediate PT asymmetry is not known. This study examined the heritability for planar asymmetry in 12 dizygotic (DZ) and 27 monozygotic (MZ) male twin pairs who were between 6 and 16 years of age. There was weak but positive evidence for heritability of planar asymmetry. Co-twin similarity for planar asymmetry and Sylvian fissure morphology increased when excluding twins discordant for writing hand and when excluding twins exhibiting birth weight differences >20% from the analyses. Birth weight differences were also related to twin differences in total cerebral volume, but not central sulcus asymmetry. These results suggest that exogenous perinatal factors affect the epigenesis of planar asymmetry development.

## Introduction

Perisylvian cortex engages in language-related information processing (Ojemann, 1983). For this reason, neuroanatomical structures within the perisylvian region have been targets for studies attempting to explain the neurobiological underpinnings of reading and language disability. Symmetry or reversed asymmetry of the planum temporale (PT) is consistently related to language impairment (Plante *et al*., 1991; Gauger *et al*., 1997) and poor verbal ability (Rumsey *et al*., 1997; Eckert and Leonard, 2000; Eckert *et al*., 2001). Individual variability in the PT is thought to reflect the phenotypic expression of genes involved in garden variety reading impairment (low verbal ability) and language impairment (Leonard *et al*., 2001).

The causal factors for the development of reversed PT asymmetry have not been identified. Reversed planar asymmetry has been reported in children with a family history for reading disability (Eckert *et al*., 2001) and in children with congenital adrenal hyperplasia and their siblings (Plante *et al*., 1996). The relative impact of genetic and environmental factors on PT asymmetry is not known, however. Steinmetz and colleagues did not find significant concordance in planar asymmetry among 20 pairs of monozygotic (MZ) twins (Steinmetz *et al*., 1995). This finding could have been influenced by the inclusion of twins discordant for handedness. Right-handed twin pairs exhibited a trend for planar asymmetry concordance.

Studies of brain structure concordance in twins show that MZ twin brains are the same size and look similar in shape, but vary with respect to some major and most minor sulci. Brain volume is the most heritable neuroanatomical feature in twins (Bartley *et al*., 1997; Biondi *et*

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*al*., 1998; Tramo *et al*., 1998; Pennington *et al*., 2000). Genetic models account for the majority of variance in concordance for brain volume between MZ and dizygotic (DZ) twins, but fail to explain much variation in gyral/sulcal topography. Deep sulci appear to be more strongly genetically determined than superficial sulci (Biondi *et al*., 1998; Lohmann *et al*., 1999). Gyral and sulcal features are probably shaped more by experience given a 7–17% heritability estimate for gyral patterns (Bartley *et al*., 1997).

Fetal brains exhibit planar asymmetry by 29–31 weeks in gestation (Wada *et al*., 1975; Chi *et al*., 1977). The weak genetic findings for gyri and sulci suggest that pre- and postnatal events can affect PT asymmetry development and disrupt twin concordance in brain structure. For example, hormones have been proposed to affect planar asymmetry (Geschwind and Galaburda, 1987). The atypical planar asymmetry findings in congenital adrenal hyperplasia, in which absence of 21-hydroxylase leads to high levels of intrauterine testosterone, support this theory.

A large number of antenatal variables, including chorionic status (Machin *et al*., 1995; Charlemaine *et al*., 2000; Victoria *et al*., 2001) and post-zygotic genetic effects can produce phenotypic differences between MZ twin pairs (Martin *et al*., 1997). Chorionic status, due in large part to twin-to-twin transfusion syndrome (TTTS), is frequently associated with cognitive and birth weight differences in twins (James, 1982; O'Brien and Hay, 1987; Charlemaine *et al*., 2000).

This study examined PT asymmetry concordance in 27 male MZ twins and 12 DZ twins. Planar asymmetry was predicted to exhibit significant heritability based on evidence that planar symmetry is related to a family history of reading disability. Birth weight differences, as an index of TTTS, were also examined to determine if this perinatal risk factor was related to twin discordance in planum measures.

## Materials and Methods

### Participants

Twenty-seven MZ and 12 DZ twin pairs were recruited from the American Academy of Child and Adolescent Psychiatry, the Virginia Commonwealth University Twin Registry, Children and Adults with Attention Deficit Disorder and the National Organization of Mothers of Twins Clubs. Female twin pairs were excluded from this study to control for random X-inactivation that might lead to more dissimilarity among female MZ twins (Jorgensen *et al*., 1992). Zygosity was verified using 9–14 unlinked short tandem repeat loci, by BRT Laboratories Inc. (Baltimore, MD). MZ cases which did not yield a probability of twinship >99% were tested further for a total of 21 loci. Written assent from the child and consent from the parents were obtained for each participant. This project was approved by the National Institute of Mental Health (NIMH) and the University of Florida Institutional Review Boards.

Demographic characteristics of the twins are presented in Table 1. The age range was 6.9–16.4 years for the MZ group and 6.1–15.0 years in the DZ group. MZ children were older than DZ children by 1.7 years, but this difference was not statistically significant [$t(1,37) = 1.85$, $P < 0.10$]. Socioeconomic status (SES) was determined using the Hollingshead inventory (Hollingshead, 1975) and did not differ between the MZ (range, 20–77) and DZ (range, 20–73) groups. Quantitative handedness measures were not available for this study. Handedness was defined by writing hand. Operationally defining handedness by writing hand overestimates right-handedness and underestimates the proportion of ambidextrous participants. There were no zygosity differences for SES [$t(1,37) = 0.43$, n.s.] or writing hand [$\chi^2(1,74) = 0.47$, n.s.].

Records indicating whether twins were monochorionic, a risk factor for TTTS, were not available for many twins. It has been suggested (Tan *et al*., 1979) that a birth weight difference >20% can be used as a proxy for TTTS. MZ twins in this study were classified as having birth weight differences >20% as an index of twin transfusion syndrome. Birth weights and gestation length were determined by parental report and corroborated with medical records when medical records were available.

## MRI Protocol and Measurement Methods

**MRI Acquisition—**Volumetric 1.5 mm thick axial images were acquired using a GE 1.5 T Signa scanner. Scan parameters consisted of a repetition time of 24 ms, an echo time of 5 ms, flip angle of 45°, a 24 cm field of view and a 192 × 256 matrix. These images were acquired at the National Institutes of Health.

**Image Processing—**Brain structure data collection for this study was performed at the University of Florida Mcknight Brain Institute. The images were reformatted into 1 mm thick sagittal sections to correct for tip in the coronal, axial and sagittal planes of section. Parameter files were created that stored the distance between the anterior commissure and borders of the brain. Talairach coordinates were used to identify the same medial to lateral locations in each brain. These coordinates are reliable for sagittal positions. The Talairach system standardizes positions by relating them to a brain atlas where the horizontal plane intersects the anterior and posterior commissure. The images were not warped or altered during the reformatting process. Each image was assigned a new random number to ensure that raters were blind to the zygosity and pairing of the twins.

**Data Collection Procedures—**Surface area measurements were obtained for the PT (Leonard, 2001). The Sylvian fissure is surrounded by horizontal and vertical planes of cortical tissue. The PT is defined by the horizontal bank, which extends from Heschl's sulcus to the origin of the vertical bank or posterior ascending ramus (PP). The PP, also called the planum parietale, rises from the termination of the PT into the parietal cortex. The surface area of the PT and PP was measured between 46 and 56 cm lateral to the midline in sagittal sections. Asymmetry of the planum is most dramatic in this lateral region of the PT (Best and Demb, 1999). These coordinates were also chosen in order to replicate the methods of previous magnetic resonance imaging (MRI) studies showing cognitive associations with PT measures (Foundas *et al*., 1994; Leonard *et al*., 1996; Gauger *et al*., 1997; Eckert *et al*., 2001). Raters were blind to zygosity and twin pairs. Intra-rater reliability was 0.94. Inter-rater reliability was 0.87.

In addition, the morphology of the left and right Sylvian fissure was categorized according to Witelson and Kigar classification criteria (Witelson and Kigar, 1992). Witelson and Kigar classify Sylvian fissure morphology into three categories based on the orientation of the Sylvian fissure, posterior to Heschl's gyrus. The horizontal and vertical type (HV) is defined by the presence of both a horizontal and a vertical branch of the Sylvian fissure. The horizontal type (H) is determined by the absence of a vertical branch. The vertical type (V) does not exhibit a horizontal branch. The V type is more frequently found in the right hemisphere when there is a very small PT. Intra-rater classification reliability was 0.94 and 90% of cases were consistently classified between raters.

A non-language-related brain asymmetry was also examined. Surface area measurements of the central sulcus (CS) were made in sagittal sections that included the hand bump (Penfield and Boldrey, 1937; Kim *et al*., 1993; Yousry *et al*., 1997). The boundaries for this region were Talairach coordinates 32–55 mm. The anterior bank of the CS was measured from the base of the CS to the peak of the posterior segment of the precentral gyrus. The posterior bank of the

CS was measured from the base of the CS to the peak of the anterior segment to the postcentral gyrus. The anterior and posterior bank measurements are highly correlated and because there is no clear anatomical boundary between motor and sensory cortex within the CS, the anterior and posterior measures were added together. Intra-rater reliability was 0.97 and inter-rater reliability was 0.90.

Total cerebral volume (TCV) was also measured to determine if co-twin relations in brain asymmetries were driven by brain size. Quantitative analysis of TCV was performed using the Montreal Neurological Institute ANIMAL and INSECT methods. These methods produce brain volumes using both a voxel-intensity-based artificial neural network technique and information from a non-linear registration-based regional segmentation approach (Collins *et al*., 1999).

**Statistical Analyses—**Chi-square, Pearson correlations and t-tests were performed to determine if variables such as writing hand, birth order and SES might confound the interpretation of the co-twin results between zygosity groups. Pearson correlations were used to examine the co-twin anatomical relations. Results from all the exploratory demographic analyses are presented and should be viewed with caution due to the possibility for type 1 error.

## Results

### Writing Hand and Sylvian Fissure Morphology

All left-handed DZ ($n = 2$) and MZ ($n = 6$) participants were second born [$\chi^2(1,37) = 31.8$, $P < 0.0001$] and had co-twins who were right-handed. MZ twins discordant for handedness had a shorter gestation than MZ twins concordant for writing hand [$t(1,25) = 2.24$, $P < 0.05$].

Partial correlations, controlling for twin pair, were performed to examine the relation of the planum measures to writing hand. Table 2 shows that writing hand was related to PT asymmetry in MZ and DZ twins. This finding was due to the high prevalence of a right hemisphere type V Sylvian fissure morphology in participants with a left writing hand. Six of eight left-hand writers had a type V compared to 4 of 68 right-hand writers [controlling for twin pair: partial $r(1,73) = 0.619$, $P < 0.001$].

The Witelson and Kigar classification method was also used to determine if the left and right Sylvian fissures of one twin exhibited the opposite hemispheric morphology of the co-twin. There were no hemispheric reversals of Sylvian fissure morphology in twins discordant for writing hand. One pair of right-handed MZ twins exhibited reversals of Sylvian fissure morphology. The morphology of surrounding perisylvian cortex of one twin was not, however, a reversed mirror image of the co-twin's morphology (Fig. 1).

MZ twins were more likely than DZ twins to be concordant for right hemisphere [$\chi^2(1,38) = 3.79$, $P < 0.10$] and bilateral Sylvian fissure morphology [$\chi^2(1,38) = 4.79$, $P < 0.05$]. There was not a significant difference between the percentage of MZ twins (19%) and DZ twins (33%) that were discordant for left hemisphere Sylvian fissure morphology [$\chi^2(1,38) = 1.03$, n.s.].

### Neuroanatomical Concordance

Pearson correlations between co-twin anatomical measures are presented in Table 3. As expected, total cerebral volume was significantly correlated between MZ and DZ co-twins. The planar asymmetry correlation coefficient approached significance in the MZ twins. Power analysis for the planar asymmetry relation in MZ twins indicated there was low power (PT asymmetry $\beta = 0.34$).

### Demographic and Perinatal Factors Affecting Planum Concordance

MZ twin pairs discordant for writing hand exhibited significantly greater absolute differences in PT asymmetry [$t(24) = 2.08$, $P < 0.05$]. Figure 2 shows the planar asymmetry relation between MZ co-twins, coded by writing hand discordance. Excluding twins discordant for writing hand improved the planar asymmetry correlation between MZ co-twins [$r(20) = 0.377$, n.s.], but not the significance value.

MZ twin pairs with birth weight differences >20% exhibited greater absolute differences in PT asymmetry [$t(25) = 2.67$, $P < 0.05$]. Figure 3 shows the planar asymmetry relation between MZ co-twins, coded by birth weight differences. Excluding twins with birth weight differences (>20%) improved the planar asymmetry correlation between MZ co-twins [$r(21) = 0.437$, $P < 0.05$]. The increase in planar asymmetry relation was due to the exclusion of twins with birth weight differences (>20%) that exhibited discrepant left Sylvian fissure morphology.

MZ twin pairs with birth weight differences >20% also exhibited the greatest differences in TCV [$t(25) = 2.70$, $P < 05$]. Figure 4 shows that MZ twins with the greatest differences in TCV had the greatest differences in planar asymmetry [$r(26) = 0.612$, $P < 0.001$]. Figure 4 also shows that this relation was due, in part, to twins with birth weight differences (>20%). Discordance for central sulcus asymmetry was not explained, however, by birth weight differences [$t(25) = 0.48$, n.s.].

## Discussion

The gyral and sulcal features of monozygotic twins are surprisingly dissimilar. Monozygotic twin discordance in brain structure appears to be due, in part, to intrauterine events that could lead to divergent morphological development. Weak relations for co-twin PT measures were found for MZ and DZ twins. There was an increase in MZ twin planum similarity when twins with birth weight differences (>20%) were excluded from the analyses. These findings suggest that perinatal pathological events can disrupt concordant expression of brain morphology in MZ twins.

### The Twin Design and Perinatal Events

The twin paradigm for estimating genetic effects for various phenotypes has received criticism for over 50 years (Price, 1950). Genetic estimates may be inflated for some traits mediated by parental care (Saudino *et al*., 2000), but it is also likely that genetic effects for other traits, such as brain structure, are underestimated. Although MZ twins are frequently described as identical, antenatal environmental events and post-zygotic genetic effects produce discordance in MZ twins (Martin *et al*., 1997). Teratogens, infection, length of delivery, post-zygotic non-disjunction, differential imprinting, skewed X-inactivation and major gene malformations have been related to phenotypic differences in twins. These are some reasons power analyses indicate that at least 200 twin pairs are necessary to estimate a highly heritable quantitative phenotype (Martin *et al*., 1978).

The uniqueness of twinning is another criticism. Events related to placentation are unique for twins and limit strict comparisons to singleton development. TTTS is an example of a developmental problem largely specific to monochorionic twins (Gaziano *et al*., 2000). In TTTS, the arterial vasculature of one twin is shared with the venous vasculature of the other twin. This causes one twin to transfuse the co-twin, leading to hypoxia, reduced concentrations of essential amino acids, growth retardation (Gall, 1996) and depressed IQ (Munsinger, 1977) in the donor twin and hyperperfusion of the recipient twin. TTTS could alter normal brain development for the donor or recipient twin.

The increased frequency of perinatal risk factors in monozygotic monochorionic twins has led some to suggest that heritability estimates should be based on comparisons of MZ dichorionic twins to DZ twins (Corey *et al*., 1979). The relation between twin birth weight difference (>20%) and differences in planar asymmetry supports this point. Valid estimates of brain structure heritability must take into account intrauterine events unique to twins and chorionic status in particular.

Alternatively, MZ twins provide a model for identifying environmental variables that influence human cortical development. For example, maternal drug use has been related to discordance for congenital structural anomalies in a pair of MZ twins (Reitnauer *et al*., 1997). Examining the relation between brain structure concordance and the timing of pathological events could provide evidence as to when particular neuroanatomical features are most susceptible to perinatal insults.

## Comparison to Other Imaging Twin Studies

The heritability findings for planar asymmetry are similar to findings from other twin studies for gyral and sulcal topography. It has been estimated (Bartley *et al*., 1997) that 7–17% of gyral patterning was due to genetic influences in 10 MZ and nine DZ adult twin pairs. In another small study, low heritability estimates were also reported for temporal lobe regions (Tramo *et al*., 1998). Figure 1 illustrates these findings. Even MZ twins that exhibit the same Sylvian fissure morphology demonstrate differences in tertiary gyri and sulci.

Planar asymmetry discordance and the high frequency of left hemisphere Sylvian fissure discordance in twins exhibiting birth weight differences (>20%) suggest that Sylvian fissure morphology can be affected by perinatal events. In addition, Ajayi-Obe and coworkers found that a group of infants born prematurely exhibited less gyral and sulcal complexity than full term infants, despite their similar cerebral volumes and age corrected for prematurity (Ajayi-Obe *et al*., 2000). The association of perinatal events with cortical development helps to explain the low heritability estimates reported by previous small sample twin studies.

Only one other study has examined PT concordance in twins (Steinmetz *et al*., 1995). They did not find significant concordance for planar asymmetry in 20 monozygotic twin pairs. Steinmetz *et al*. did report that twins discordant for handedness exhibited large discrepancies in planar asymmetry. All but one of the left-handers in the Steinmetz *et al*. study exhibited symmetry or reversed planar asymmetry. In contrast, the left-handed twins in this study exhibited leftward asymmetry. These left-hand writers were all second-born and more likely to be born prematurely. Hopkins *et al.* have shown that chimpanzee handedness is heritable (Hopkins *et al*., 2001), but the degree of heritability is modified by offspring parity (developmental instability). An uncoupling of handedness and planar asymmetry in this study could have occurred because of pathological events. There are too few cases, however, to discount a more stochastic explanation for the handedness findings.

## Anatomical Specificity

Birth weight differences accounted for differences in MZ twin Sylvian fissure morphology, planar asymmetry and total cerebral volume. There was not a similar explanation for discordance in central sulcus asymmetry, however. Variation in central sulcus asymmetry was also unrelated between co-twins. This replicated finding (Bonan *et al*., 1998) suggests there are not strong genetic effects on central sulcus asymmetry. The modulating effects of perinatal events on development may be most easily seen for genetically mediated phenotypes.

If TTTS explains the birth weight differences, then the timing of TTTS inf luences could provide insight into planum development. The development of TTTS is not well understood,

however. The influences of TTTS may be greatest after 20 weeks gestation, when placental vascular patterns are stabilized (Sebire *et al*., 2001). This timing appears to coincide with the period of gestation when the Sylvian fissure is developing and planar asymmetry is first seen (Wada *et al*., 1975; Chi *et al*., 1977; Bernard *et al*., 1988).

## Developing Asymmetry

Individuals with situs inversus (reversed asymmetry of visceral organs) have been studied to determine if the same mechanisms for visceral organ asymmetry affect cerebral asymmetry. Three cases of situs inversus exhibited reversed frontal and occipital petalia (Kennedy *et al*., 1999). Other evidence of anomalous laterality was not found. All three subjects were right-handed, had a left hemisphere language dominance and 2/3 had leftward planar asymmetry. Although one case of right hemisphere aphasia has been reported in a stroke patient with situs inversus (Cohen *et al*., 1993), most situs inversus studies do not report an increased incidence of left-handedness or anomalous language organization (Woods, 1986; Tanaka *et al*., 1999). These studies do not support the idea that mechanisms affecting visceral organ asymmetries are related to cortical asymmetries. Perhaps similar, but different genetic mechanisms direct the development of cerebral asymmetry than for visceral organ asymmetry (Alexander and Annett, 1996).

Although little is known about the events that produce cerebral asymmetry, considerable progress has been made in understanding the cascade of events leading to asymmetric development of visceral organs. Visceral organ asymmetry is dependent on asymmetric expression of key regulatory proteins. Activin inhibits the expression of sonic hedgehog (*Shh*) on the right, but not left side of the primitive streak (Levin *et al*., 1995). Left-sided expression of *Shh* leads to the expression of transforming growth factor beta (TGF-β) by *Nodal*, which has downstream effects on the expression of *Pitx2 (*Levin *et al*., 1995). Asymmetric expression of *Pitx2* leads to asymmetric development of the heart, lungs, pituitary and pineal body (Lin *et al*., 1999; Liang *et al*., 2000). The conservation of these molecular events across frogs, chickens and mice is strong evidence that similar mechanisms could regulate human asymmetries.

Discordant asymmetry may occur in twins because of placentation and orientation of embryos. Obliquely conjoined twins provide support for this hypothesis. In conjoined twins, the twin on the right frequently exhibits laterality deficits (Levin *et al*., 1996). Levin suggests the parallel orientation of the two primitive streaks allows activin on the right side of the left embryo to inhibit *Shh* on the left side of the right embryo. This mechanism could explain mirrored lateralization for brain function in MZ twins (Sommer *et al*., 1999), but only for monochorionic monoamniotic MZ twins who make up <1% of twins (Hill *et al*., 1996).

## The Epigenesis of Planum Temporale Asymmetry

The cascade of regulatory events guiding left and right plana development could begin around 21 weeks when the Sylvian fissure develops (Bernard *et al*., 1988) and taper off around 29–31 weeks when planar asymmetry is first discernible (Wada *et al*., 1975; Chi *et al*., 1977). The weak heritability estimates in this study suggest that large numbers of families would be necessary to find genetic linkage for planum development. Identification of genetic expression patterns regulating planum development may require microarray studies of post-mortem human or chimpanzee fetal brains.

A large number of factors, including nutrition, probably affect genomic to synaptic levels of planum development. For example, vitamin A deficient quail embryos exhibit cardiac situs inversus. Administration of retinoic acid rescues the expression of *nodal* and *Pitx2*, and produces normal cardiac development (Zile *et al*., 2000). Teratogens might also affect planar

asymmetry. Prenatal alcohol exposure produces neuronal migration errors in rat cortex (Hirai *et al*., 1999) and has been related to changes in white and grey matter in the left hemisphere temporal-parietal region of children (Sowell *et al*., 2001).

## Summary

This study supports suggestions that PT development is mediated by genetic and experiential factors (Habib and Galaburda, 1986). Monozygotic twins exhibit weak concordance for planar asymmetry, due in part to modulating factors related to birth weight differences and writing hand discordance. These findings suggest that intrauterine events can alter the direction of brain development between twins and highlight the importance of having large twin pair samples when estimating neuroanatomical heritability. On the other hand, MZ twins may be a good model for identifying specific perinatal events and the timing of those events that negatively affect brain development and function.

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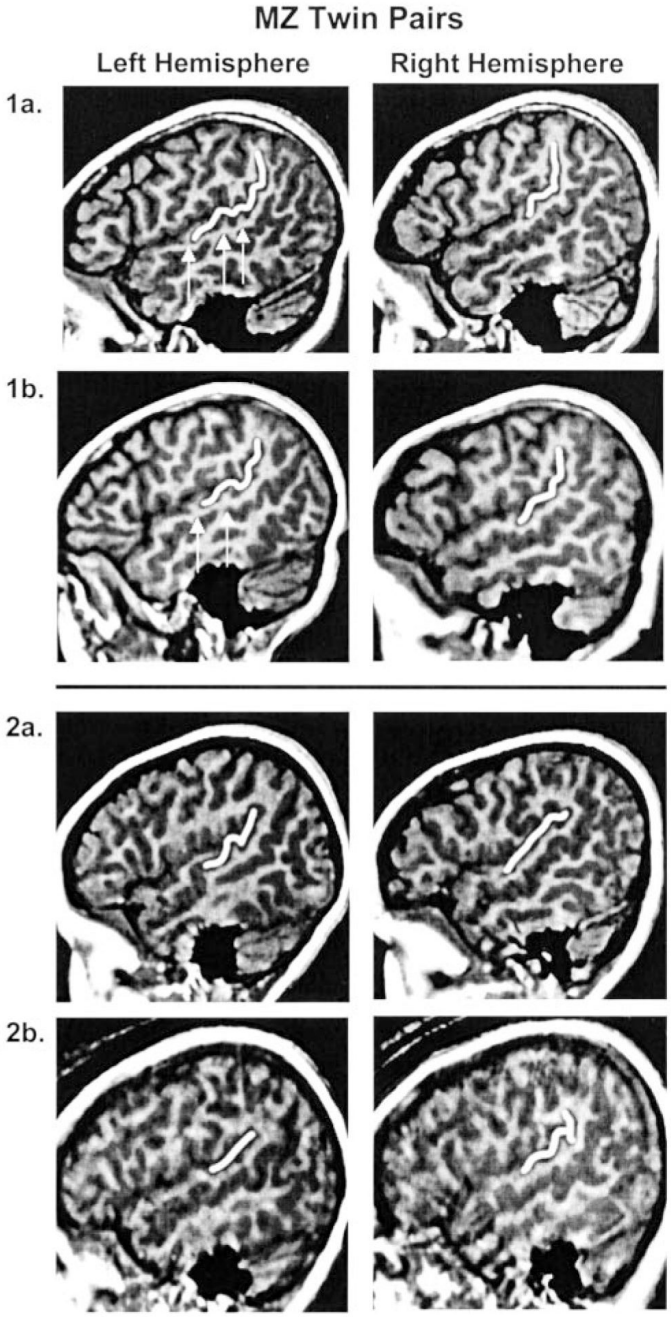


**Figure 1.**
Sagittal images of MZ twin pairs. Twin pair 1a,b exhibit similar Sylvian fissure morphology. Also note the similarity in brain shape, but variation in minor gyri and sulci (arrows). Twin pair 2a,b exhibit hemispheric reversals in Witelson and Kigar Sylvian fissure classification, but they are not mirror image reversals. Images created at Talairach position 50 for the right hemisphere and 47 for the left hemisphere. The PT and PP are outlined in each image.

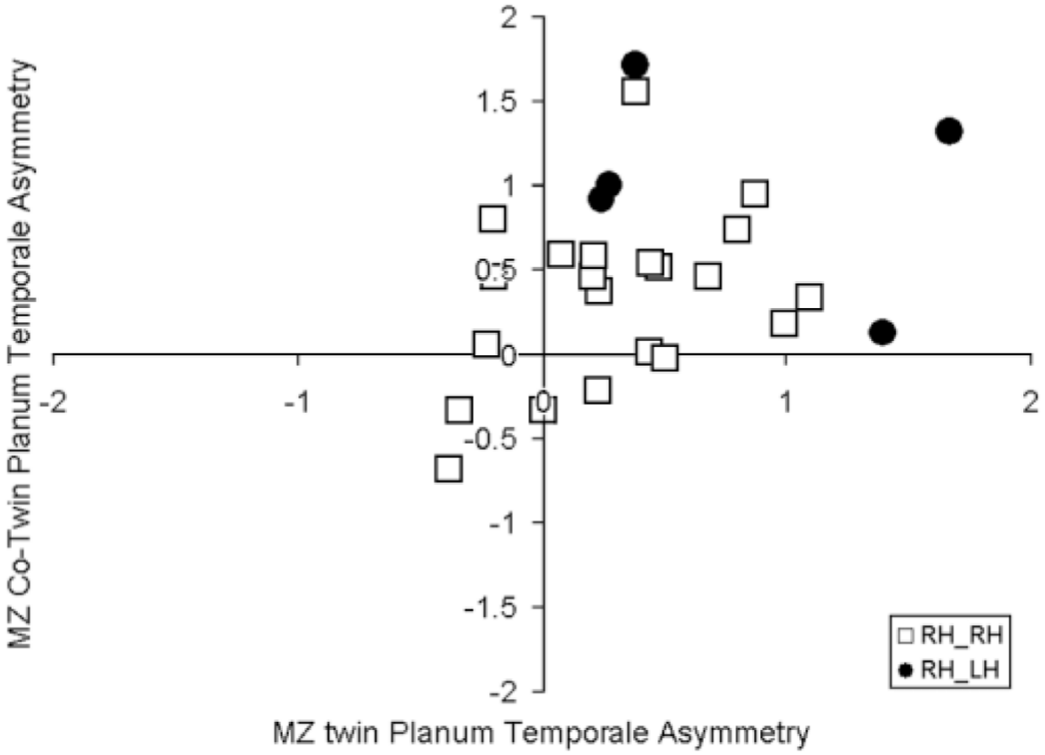


**Figure 2.**
PT asymmetry relation between MZ twins by writing hand concordance (RH_RH = right-handed twin 1 and right-handed twin 2; RH_LH = right-handed twin 1 and left-handed twin 2).

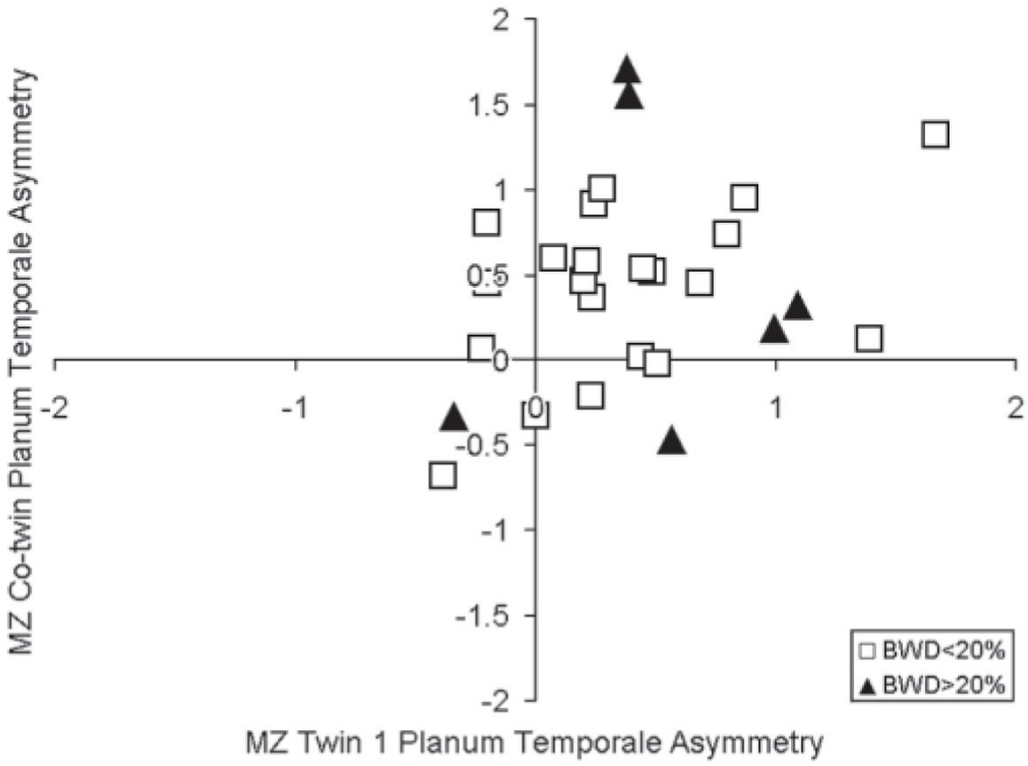


**Figure 3.**
PTA relation between MZ twins by writing birth weight differences greater than 20% (BWD > 20%).

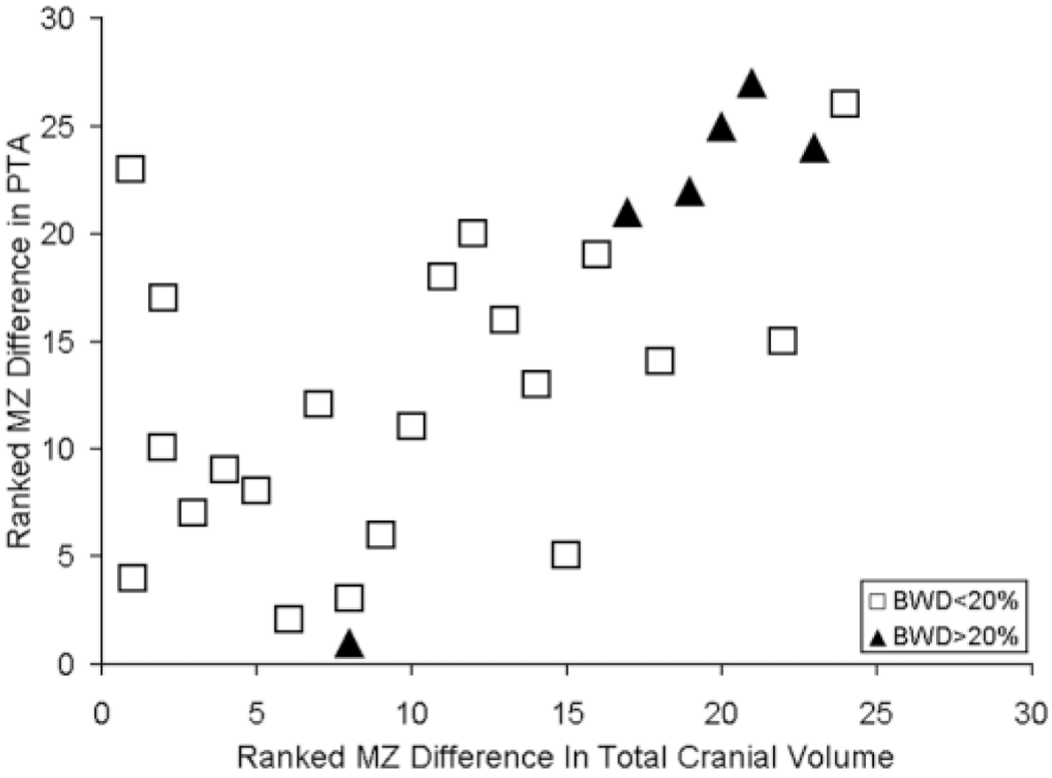


**Figure 4.**
MZ twins exhibiting the largest PT asymmetry differences also exhibit the largest total cerebral volume differences in the group.

**Table 1**

Demographic characteristics of the MZ and DZ twins

| | MZ twins | DZ twins |
|---|---|---|
| Age (years) | 11.9 (2.7) | 9.89 (2.8) |
| SES (Hollingshead) | 42.9 (16.1) | 34.9 (13.1) |
| Writing hand (% right) | 83% (41/47) | 92% (22/24) |
| Writing hand concordance | 74% (17/23) | 83% (10/12) |

Writing hand information missing for one MZ twin.

**Table 2**

Writing hand predicts to planum measures

| | LPT | RPT | PTA | LPP | RPP | PPA |
|---|---|---|---|---|---|---|
| DZ | 0.259 | −0.273 | 0.509[a] | −0.242 | 0.166 | −0.334 |
| MZ | 0.175 | −0.357[a] | 0.478[b] | −0.084 | 0.281[a] | −0.203 |

[a] $P < 0.05$;

[b] $P < 0.005$; L, left hemisphere; R, right hemisphere; A, asymmetry.

**Table 3**

PTA ICC for DZ and MZ twins

| | LPT | RPT | PTA | LPP | RPP | PPA | LCS | RCS | CSA | TCV |
|---|---|---|---|---|---|---|---|---|---|---|
| DZ twins | −0.171 | 0.105 | −0.235 | −0.052 | 0.366 | −0.193 | 0.225 | 0.184 | −0.343 | 0.571[b] |
| MZ twins | 0.218 | 0.161 | 0.303 | 0.133 | −0.149 | 0.162 | 0.331[a] | 0.563[c] | 0.098 | 0.944[d] |

[a] $P < 0.10$;

[b] $P < 0.05$;

[c] $P < 0.005$;

[d] $P < 0.001$; L, left hemisphere; R, right hemisphere; A, asymmetry.