Sexual Dimorphism in the Human Corpus Callosum: Its Evolutionary and Clinical Implications

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I am very grateful for the opportunity to provide a contribution to this Festschrift for my friend and colleague Dr. Tobias. Along with Dr. Dart (and Alun Hughes), Phillip Tobias has had a tremendous effect on my career, through my return to paleoneurology in 1969 after having dismissed it as a hopeless venture in my 1964 doctoral dissertation! (For that, I am occasionally vague about the extent of my thanks... or whom to blame!)

In 1982 I had the opportunity to revisit the University of the Witwatersrand, and there I received the customary excellent hospitality and help in my studies. At that time I tried to convince Dr. Tobias that, given the Anatomy Department's excellent records and procedures, it would be very helpful to have an ongoing project devoted to studying sexual dimorphism of the corpus callosum, as Dr. de Lacoste-Utamsing and I had published our findings in Science in 1982 on a very small sample. Dr. Tobias got things rolling, and with the aid of Dr. Wium I received some black-and-white photographs, without sexual identification, of mid-sagittally sectioned human brains.

The following report of my studies of these materials so kindly given to me by Dr. Tobias and Dr. Wium should perhaps be thought of as a joint paper—although I would not force Dr. Tobias to attach his imprimatur to these findings or speculations.

INTRODUCTION

It is well-appreciated that structures of the nervous system subserving reproduction are probably sexually dimorphic in a wide range of animals. The hypothalamus has been the structure most heavily implicated, in particular the preoptic nuclei (for reviews on the human hypothalamus, see Swaab and Hofman, 1984; Swaab and Fliers, 1985). The thought that sexual dimorphism might include other regions of the brain such as the cerebral cortex (in addition to total brain weight), even in humans, has probably been more common than the literature suggests. This stems from the sexist struggles and pronouncements of the past, which were particularly vociferous during the latter half of the 19th century. Certainly, behavioral differences between the sexes have been suspected from time immemorial.

The first reliable account of sexual dimorphism in midline structures appears to have been suggested by Papez (1927) in his description of Helen Gardner's brain. Unfortunately, the
original measurements were not published, precluding use of statistical techniques developed since then to quantify the differences. Before this, Bean (1906) had suggested some sexual dimorphism as well as strong ethnic brain differences, but I find this account unreliable. Reviewing the topic a few years later, Mall (1909) found little of substance aside from brain weight to demonstrate such dimorphic differences.

**Corpus Callosum**

Recent studies of the human corpus callosum have indicated that this system of fibers interconnecting left and right cerebral hemispheres is relatively larger in females, particularly in the splenial [posterior] portion (de Lacoste-Utamsing and Holloway, 1982; Holloway and de Lacoste, 1986). Furthermore, this dimorphism is apparent by gestational age (GA) 26 weeks (de Lacoste et al., 1986). Associated and confirmatory evidence regarding parietal/frontal (P/F) lobe ratios suggests that females have higher P/F ratios than males, particularly on the left side (Baack et al., 1982). Kimura (1980, 1983) and Mateer et al. (1982) suggested that the posterior parietal lobes are less asymmetrical in females; Kimura and Harshman (1984) reported a sex-related difference for anterior vs. posterior lesions or cerebral insults and their effects on cognitive behavior. These later findings indicated that, compared with males, females have less lateralization of the posterior half of the cerebral cortex but more lateralization of their frontal lobes. Thus, both right- and left-sided damage or lesions in Broca's area appears more damaging in females, whereas damage to Wernicke's area is more detrimental to visuospatial cognitive tasks in males.

Reviews by Harris (1978), McGlone (1980), and Witelson (1982) indicated gender-related differences in cognitive tasks and both susceptibility to and recovery from damage to cerebral structures involved in motor and receptive speech-processing, and the extensive review by Hall (1984) revealed differences in nonverbal performance. More recently, although admitting the possibility of sex-related differences in some areas of cognitive behavior, Witelson (1985) claimed that differences in the corpus callosum correlate with handedness, not gender: left-handers were claimed to have larger corpora callosa. [For additional reviews on sex-related differences, asymmetries, and associated behavior, see Witelson, 1976, 1982].

Most recently, reviewing the question of sexual dimorphism and the corpus callosum, Witelson and Kigar (1987) concluded that few, if any, independent studies concurred with the 1982 report by de Lacoste-Utamsing and Holloway. As will be seen, this and other studies either ignored the size of the brain or measured it incorrectly. Indeed, since Holloway and de LaCoste's replication study (1986)—which was not mentioned by Witelson and Kigar—several reports have commented that no statistically significant differences can be found between male and female corpora callosa (reviewed by Peters, 1988). Weber and Weis (1986), who measured the brains obtained at autopsy of 18 males and 18 females, found no significant sexual dimorphic differences. The average age was 74.7 years, which is quite high, and brain weights were 1029.5 cc for males and 890.3 cc for females, values that are quite dimorphic and certainly low. The posterior fifth of the corpus callosum, containing mainly the splenial portion, averaged 164.5 and 162.4 mm² for males and females, respectively, and the entire corpus callosum averaged 639.5 and 613.3 mm²—with SDs in the range of 105.3 mm²! Weber and Weis did not provide relative figures, so one cannot determine the ratios for males/females when corrected for brain size. Most noteworthy was the finding that, despite the difference between average brain sizes, mean measures of the corpus callosum were almost equal, a finding consistently replicated in several studies in addition to the original ones.

**Splenium**

Wium (1984) reported finding absolutely larger splenia in mid-sagittal sections of brain in females than in males but unfortunately did not present the data. Interestingly, this investigator found that the splenial portion of male corpora callosa had more fibers [this is discussed later]. Bell and Variend (1985) claimed a lack of sexual dimorphism in the corpus callosum of 40 brains from autopsies of children from birth to age 14 years, and reported find-
ings different from those in fetal brains studied by Holloway and de LaCoste (1986). They did not report brain weights, also, their sample sizes within age groups were very small.

Other studies have used nuclear magnetic resonance (NMR) for imaging, which does not provide brain weight. Yoshii et al. (1986) studied 14 normal males and 19 females aged 24–82 years. Their abstract indicates that no sex-related differences were found, although a blinded rating revealed the female splenium to be more bulbous (p = 0.025), and there was no significant effect of handedness. Clearly, relative values were not studied in this sample, thus leaving open the question of relative sexual dimorphism.

Bleier et al. (1966) found no dimorphic differences in their NMR study but did not present their data. Byne et al. (1988) gave some of the data on which they based their conclusions: mean area of the corpus callosum (CC) was 601 mm² in the 22 women and only 519 in the 15 men, a substantial difference whether significant or not; and the posterior one-fifth [splenium] was 168 mm² for the women and 160 mm² for the men. Interestingly, these authors found “a marginally significant main effect of sex for minimum width only, with the width being smaller in men, F[1, 33] = 4.45, p = 04.” Analysis of variance of other callosal measurements did not indicate any significant effect of gender. Brain sizes were not stated, and one can only wonder how much larger the male brains were in view of the females’ larger callosal measures.

Oppenheim et al. (1987), also, found no significant differences in NMR studies of 40 males and 40 female brains. Mean splenial area, as a percentage of total corpus calloso area, was 30.4% for males and 31.2% for females. Brain size was not studied, and original areal measures were not included in the tables. Most recently, Weis et al. (1988) claimed no sexual dimorphism in their NMR study—but, again, brain size was not stated. For males and females respectively, the average corpus calloso area was 669.9 and 665.2 mm², and the splenial one-fifth area measured 191.5 and 199.9 mm²; similarly, values for critical splenial dimensions were larger in the females (see Table 2, p. 414, in their report).

Kertesz et al. (1987), who studied the brains of 51 men and 53 women, claimed it was not possible to sex them beyond chance on the basis of callosal measurements which certainly appear very similar [mean callosal means, m/f, were 724 and 716 mm²]. But the ratios of callosal area to either sagittal or axial brain section were higher for females, and significantly so in a horizontal brain area. Unfortunately, these authors confused an NMR cross-sectional area with brain weight [or size], and failed to control for cranial shape (e.g., cephalic index). Given this poor methodology, it is not surprising they claimed no correlation between callosal and brain areas, a finding at variance with all other published reports, the independent study described here, and three of my current studies; their ratio data was thus rendered meaningless. Also, the authors dismissed as insignificant their subjective ratings of splenial vs genu size that favored females. In sum, this was a poorly devised set of comparisons with no true size control variable, and findings that actually point to a relative large CC in females. As an aside, Kertesz et al.’s data did not support Witelson’s claim (1985) of a relationship to handedness.

Demeter et al. (1988) published findings indicating no significant sexual brain dimorphism in their study of autopsy records for 22 males and 12 females. Mean measurements for males/females were: splenial width, 11.8 and 11.6 mm; posterior one-fifth of the CC, 165 mm² for both; and total CC area, 627 and 582 mm² (a real difference). These authors measured but did not present brain size explicitly, but their Figure 4 (p. 222) shows a very large dimorphism—up to 1700 grams for the males (>1400 g in 9 of them) and with no overlapping of weights for females. Further, brain size was ignored as a corrective factor on the assumption that this does not correlate with the corpus callosum!—an assumption that is erroneous (see Bell and Variend, 1985).

**BRAIN SIZE**

It is obvious that virtually all of these studies failed to take brain size into account adequately, ignoring the ratio data [and ANOVA] and statistical analyses detailed in the report by Holloway and de Lacoste (1986). At the same
time, all have indicated that measurements of the female corpus callosum are equal to or are just above or below those of the male CC. Consequently, t tests would not demonstrate statistically significant differences—except in relation to brain size, which is highly dimorphic. I thus came to the conclusion that an independent investigation, properly designed and carefully conducted, was needed to document or disprove such findings. In performing such a study I sought to overcome the drawbacks of small samples [e.g., de Lacoste-Utamsing and Holloway, 1982; Witelson, 1985; Holloway and de Lacoste, 1986; Demeter et al., 1988], failure to correct for the larger male brain, and the known overlap in female—male behavior. Also, the proposed study seemed clinically important, as sexual dimorphism of the human corpus callosum could have significant implications in relation to the effects and treatment of brain-damage.

THE INDEPENDENT STUDY OF HUMAN CORPORAL CALLOSA

I received black-and-white photographs, measuring 8 × 10 in, of 24 midsagittally sectioned human brains, one photograph of each half with a 1.0-mm scale on the midsagittal plane. Information regarding ethnicity, gender, age, and brain weight, was not opened until measurements on the photos were complete.

I selected the better print [R or L hemisphere] of each brain. After exclusion of cases in which the fourth cerebral aqueduct had not been accurately sectioned, the corpus callosum appeared damaged, or the structure appeared ambiguous or partial, there were 22 cases for which the CC outline was traceable without ambiguity. This outline, drawn in black ink and traced onto bond paper, included the whole of the CC and hemispheric outline but excluded the cerebellum and brainstem. The cerebral outline was traced as a size control, because of the absence of many brain weights. [As the cerebral cortex accounts for some 76% of brain volume in humans, there is fairly close correlation between midsagittal cerebral area and total brain volume.]

Measurements were made on the tracings with a Tamaya Planix-3 digital planimeter. Total CC area and the area of the cerebral outline were measured ×3 and averaged. The AP length of the CC was divided into fifths, and the posterior fifth [almost exclusively splenium] was drawn ×3. Finally, a vernier caliper was used to measure the dorsoventral distance (DVD) of the posterior portion of the splenium at its widest, to the nearest 0.1 mm. As brain size, gender, and CC size correlate, correction was made for brain size to establish sex, as follows: relative CC area = (CC area/total cerebral area) × 100; relative splenial area = (posterior one-fifth/CC area) × 100; and relative splenial distance = (dorsoventral splenial width × 100)/total cerebral area.

Statistical analyses relied on Statgraphics or SPSSX packages, with t tests on grouped data, ANOVA analyses [hierarchical, classical, and regression] controlling for brain size as measured by total cerebral area, and univariate statistics for means, SDs, SEs, skewness and kurtosis, and with sign and rank order tests assuming lack of normality in mean values.

As expected, total cerebral area was larger in males than females [Table 1], but the difference was not significant; however, absolute values for total CC area and splenial area were larger, and splenial DVD was significantly so, for females. The findings thus far paralleled those in most of the studies discussed earlier. Interestingly, the relative area and relative DVD were significantly larger in females, whereas their relative splenial area [splenial area/total CC area] was larger but not significantly so, and dividing the DVD by an exponential value 0.33 of CC area [i.e., to approximate linearity] yielded a ratio not significantly higher in females. On ANOVA multivariate analysis, for the CC area both the covariate [total area] and sex effects were significant [Table 2], although for the splenial area only the latter was significant. The same result was obtained for DVD, but the relative measure of this [DVD/CC area 0.33] showed no significant effects by the covariate, total area, or sex effect, although the last-named had the higher F [3.98, against 2.548 for size] and p = 0.060.

SIGNIFICANCE OF THE SEXUAL DIMORPHISM

The findings in the independent study supported those in earlier investigations by my
TABLE 1. Basic Statistics* on Raw and Corrected (Ratio) Variables in Human Brain, in 22 Cases (13 m, 9 f)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Sex</th>
<th>Mean</th>
<th>SD</th>
<th>SE</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total corpus callosal (CC) area</td>
<td>M</td>
<td>7.0308</td>
<td>1.414</td>
<td>.392</td>
<td>-0.99</td>
<td>.341</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>7.6556</td>
<td>1.506</td>
<td>.502</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Splenial area</td>
<td>M</td>
<td>1.3769</td>
<td>.327</td>
<td>.091</td>
<td>-1.61</td>
<td>.124</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>1.5889</td>
<td>.267</td>
<td>.089</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dorsoventral distance [DVD]</td>
<td>M</td>
<td>1.2323</td>
<td>.161</td>
<td>.045</td>
<td>-2.77</td>
<td>.012</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>1.4311</td>
<td>.172</td>
<td>.057</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total cortical area</td>
<td>M</td>
<td>117.48</td>
<td>11.989</td>
<td>3.325</td>
<td>1.28</td>
<td>.217</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>110.78</td>
<td>12.314</td>
<td>4.105</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Relative CC area (CC/total area)</td>
<td>M</td>
<td>5.97</td>
<td>.916</td>
<td>.254</td>
<td>-2.41</td>
<td>.026</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>6.87</td>
<td>.780</td>
<td>.260</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Relative splenial area (splenial/total CC area)</td>
<td>M</td>
<td>19.895</td>
<td>4.945</td>
<td>1.371</td>
<td>-.70</td>
<td>.494</td>
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<tr>
<td></td>
<td>F</td>
<td>21.322</td>
<td>4.377</td>
<td>1.459</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Relative DVD [(DVD × 100)/total area]</td>
<td>M</td>
<td>1.054</td>
<td>.132</td>
<td>.037</td>
<td>-3.35</td>
<td>.003</td>
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<tr>
<td></td>
<td>F</td>
<td>1.307</td>
<td>.223</td>
<td>.074</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DVD corrected [DVD/CC²]</td>
<td>M</td>
<td>64.905</td>
<td>6.28</td>
<td>1.742</td>
<td>-2.36</td>
<td>.029</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>73.860</td>
<td>11.508</td>
<td>3.836</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Relative splenial area, corrected</td>
<td>M</td>
<td>17.173</td>
<td>5.169</td>
<td>1.434</td>
<td>-1.10</td>
<td>.286</td>
</tr>
<tr>
<td>(splenial/CC area)/total area</td>
<td>F</td>
<td>19.620</td>
<td>5.096</td>
<td>1.699</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Areas in cm²; distances in cm. [Note negative t-values, and italicized p-values.]

colleague and me (de LaCoste-Utamsing and Holloway, 1982; Holloway and de Lacoste, 1986) that, in general, the relative area of the corpus callosum, the area of the splenium, and their dorsoventral distance, are larger in female and for the most part significantly so. The mixed findings were not surprising, given the smallness of the sample and the rather crude measuring techniques; but, even discounting the lack of significance in some ratios, absolute values for females were larger except for brain size. This concurs with my three other samples, two of roughly 50 cases each from New York hospitals and another, smaller, one from Australia, which show significantly larger corpora callosa and attendant variates in females. Furthermore, when the samples are standardized (mean = 0; SD = 1.0) and combined, differences in the relative values are significantly different (unpublished data).

TABLE 2. ANOVA (Multivariate) Analyses of Variance, Controlling for Brain Size, in 22 Cases (13 m, 9 f)

<table>
<thead>
<tr>
<th>Variables</th>
<th>Mean square</th>
<th>F</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>CC area:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cov. = total area</td>
<td>16.837</td>
<td>15.787</td>
<td>.001</td>
</tr>
<tr>
<td>Main effect = sex</td>
<td>7.125</td>
<td>6.680</td>
<td>.018</td>
</tr>
<tr>
<td>Splenial area</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cov. = total area</td>
<td>.172</td>
<td>2.140</td>
<td>.160</td>
</tr>
<tr>
<td>Main effect = sex</td>
<td>.393</td>
<td>4.888</td>
<td>.039</td>
</tr>
<tr>
<td>Dorsoventral distance:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cov. = total area</td>
<td>.000</td>
<td>.011</td>
<td>.918</td>
</tr>
<tr>
<td>Main effect = sex</td>
<td>.223</td>
<td>7.886</td>
<td>.011</td>
</tr>
<tr>
<td>Relative dorsoventral distance:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cov. = total area</td>
<td>195.532</td>
<td>2.548</td>
<td>.127</td>
</tr>
<tr>
<td>Main effect = sex</td>
<td>305.771</td>
<td>3.985</td>
<td>.060</td>
</tr>
</tbody>
</table>
It has been difficult to gather these data, and they have given rise to the same reactions of incredulity as we experienced back in 1981–1982—as if the whole were a vast sexist plot (in particular, see the discussion in Bleier et al., 1986, and Byrne et al., 1988, for such innuendoes). All of the data are from cases culled to exclude neurologic and neuroanatomic disorder as discerned from the autopsy. Some cases could not be used, because of poor sectioning (parasagittal, not midsagittal through the 4th ventricle), damage to the corpus callosum, or its ambiguous tracing. In some of the NMR studies discussed above, the CC was not measured blind; i.e., gender could have been deduced from the cranial outline.

A further problem is that nearly all medical institutions follow autopsy procedures that demand coronal sectioning of the brain, and it is difficult (sometimes impossible) to convince medical personnel and pathology staff that one can gain twice as much information through sagittal sectioning. Indeed, despite Geschwind’s many provocative contributions (e.g., Geschwind and Galaburda, 1984) one still cannot find reliable autopsy data of the weight for each cerebral hemisphere, nor is handedness determined as routine and included as an integral part of the autopsy report. A typical result of this lack of foresight is the lack of independent confirmation or rejection, so far, of the important but ambiguous data published by Witelson (1985) concerning the corpus callosum and handedness: Nasrallah et al. (1986) apparently did not confirm her findings in their study of schizophrenics with normal controls, and neither did Kertesz et al. (1987).

NMR shows the outline of the corpus callosum in great detail; it permits immediate tracing or digitizing, procedures that can be applied for both healthy persons and patients, with the potential for yielding a tremendous amount of useful behavioral data. The drawback is NMR’s inability to compute brain size, which is essential to an understanding of these dimorphic differences. Studies that neglect head shape and rely on a single axial plane are useless for this purpose.

**Why the Dimorphism?**

The reasons for dimorphism of the corpus callosum are unclear, although it is most probable that some hormonal-target-tissue interaction is responsible for its development (reviewed by Arnold and Gorski, 1984; Juraska, 1986; Toran-Allerand, 1986).

There seem to have been no reliable histologic studies other than in rats that have looked for sexually dimorphic features in the number, density, or ratio of myelinated/unmyelinated fibers in the corpus callosum. If indeed the posterior brain is more lateralized in males, one might expect fewer crossing fibers between the parietal lobes per volume of cerebral cortex, and thus relatively fewer fibers in the corpus callosum (i.e., per unit of parietal cortex connected) in males. In fact, however, Wium (1984) reported finding more nerve fibers per unit sectional area in the male splenium, and more spaces containing no nerve fibers in females. Wium unfortunately presented no data, and so far as I know this intriguing finding, of potentially immense clinical value, has not been replicated.

Juraska and Kopcik’s study (1988) of the rearing of rats is very relevant. These authors found that rats raised in isolated environments did not have dimorphic callosa, and that enriched rearing appeared to increase the posterior third in both sexes. However, ultrastructural analyses showed that in either situation the females had significantly more unmyelinated axons, and that in enriched environments the number of myelinated fibers was significantly greater in females than in males. It thus appears that both gender and environment can affect the ultrastructural level of the corpus callosum in rats, despite the contrary findings in humans (Wium, 1984).

Dimorphism of the corpus callosum at a gestational age of 26 weeks GA (de Lacoste et al., 1986) makes cultural influences an unlikely source of such differences, although postnatal cultural/biological interactions may heighten or lessen expectations and behavioral scores on verbal and nonverbal cognitive tests. Halpern (1986) argued that this sex-related dimorphism is meaningless, giving data from South Africa that suggested, but did not prove, its freedom from ethnic effects: it was also present in a small sample of Australian Aborigines. I have not detected the dimorphism in either Pan or Macaca [unpublished studies, but see de LaCoste and Woodward (1988) who found it on combining primate species], suggesting the in-
triguing possibility that *Homo* is possibly species-specific for this.

Dimorphism of the corpus callosum could be an evolutionary residuum [Holloway, 1983] from an earlier stage in hominid evolution; but this of course is purely speculative. Such a model assumes greater degrees of, and selection pressures for, sexual division of labor, relating to a more complementary hominid social behavioral *strategy* to support major changes in growth of the offspring, e.g., more learning, later development and maturation, and prolonged dependency of infants and children. I postulate that females were more skilled in social relationships and communication and that men were perhaps advantaged in visuospatial integrative tasks. The differences might have been driven, with positive and negative feedback [Holloway, 1967, 1981]. These would not have been bimodal patterns; there would have been considerable overlap, as in today's verbal and nonverbal scorings and cultural contexts [Maccoby and Jacklin, 1974; Hall, 1984; Halpern, 1986] and measures of the corpus callosum.

Despite the overlap of areal and other size measures and of cognitive competencies there is significant sexual dimorphism in the human brain, and purely environmental *role-expectancy* cannot explain the structural and developmental differences [de Lacoste et al., 1986]. Studies of large numbers of autopsy specimens of normal brain, with neuropathologic effects excluded, showed significantly higher relative brain weights for females once body size was taken into account [Holloway, 1980]. The difference was slight, but it became considerably larger when values for males and females were also corrected for body fat and the brain was weighed as a proportion of lean body mass; then, the relative brain size of women became even larger, with the difference still significant.

**CONCLUSION**

In addition to considering female reproductive functions as reflecting a high evolutionary investment, we may deduce that the female brain represents an equally strong investment in developing an organ of greater social intelligence.

Unfortunately, the origins of this dimorphism will probably always be beyond our empirical grasp. It is unlikely that Plio/Pleistocene soft tissues such as brain will be found, and even the finest of the rare brain endocasts yield no evidence. At most, complete fossil endocasts might provide both a reliable sex identification and cerebral asymmetries stronger in one sex, as was described by Bears et al. [1986] for modern humans. One can but hope.

Meanwhile, I believe the most useful measures to be the ending of sexist rhetoric and the expansion of careful, conscientious exploration of both phenotypic and genotypic variation in the human brain, between the sexes, and between and among as many ethnic groups as we can study. The results could help us to a better understanding of our past and our present, and provide a rational basis for aiding those afflicted with nervous and neurologic disorders, cerebral catastrophe, the ravages of environmental insults, and aging.

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