Asymmetry of Chimpanzee Planum Temporale: Humanlike Pattern of Wernicke's Brain Language Area Homolog

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The anatomic pattern and left hemisphere size predominance of the planum temporale, a language area of the human brain, are also present in chimpanzees (*Pan troglodytes*). The left planum temporale was significantly larger in 94 percent (17 of 18) of chimpanzee brains examined. It is widely accepted that the planum temporale is a key component of Wernicke's receptive language area, which is also implicated in human communication-related disorders such as schizophrenia and in normal variations such as musical talent. However, anatomic hemispheric asymmetry of this cerebrocortical site is clearly not unique to humans, as is currently thought. The evolutionary origin of human language may have been founded on this basal anatomic substrate, which was already lateralized to the left hemisphere in the common ancestor of chimpanzees and humans 8 million years ago.

The planum temporale (PT) is a key site within Wernicke's posterior receptive language area in the left hemisphere of the human brain and is thought to be an epicenter within a dispersed mosaic of language-related regions in the cerebral cortex (*1*). The left hemisphere predominance of the PT is more pronounced than any other human brain asymmetry. Further, it is currently widely accepted that asymmetry of this brain region is unique to humans (*2*, *3*). Although the PT is a major component of the auditory association cortex, it appears to be equipotential with regard to its role in production and comprehension of both spoken (vocal-auditory) and signed (gestural-visual) human languages (*3*). The landmark study by Geschwind and Levitsky (*2*) of the PT gave rise to a plethora of interest in this region of the cerebral cortex.

Here we report a morphologic pattern with left hemisphere PT predominance in the temporal lobe of our closest living relatives, chimpanzees (*Pan troglodytes*), which parallels that of humans. The presence of a similar pattern of neural asymmetry in chimpanzees may offer new insight into both the organization of human cortical language areas and the nature of their basal design, origin, and subsequent incorporation during early human evolution.

Since an early synthesis by Geschwind and Galaburda (*4*) it has been widely accepted that anatomic asymmetries of the PT in humans underlie well-established functional asymmetries within this and surrounding left hemisphere perisylvian language regions (*5*). Further, many studies have linked the PT to a melange of behaviors and disorders, other than language, that are also purportedly unique to humans. For example, divergent anatomic asymmetries of the PT are considered to be associated with normal human variation such as musical talent (*6*), development (*7*), handedness (*8*), sexual dimorphism (*9*), and communication-related disorders such as schizophrenia (*10*).

To date, no study has directly demonstrated PT asymmetry in any nonhuman primate, although two early German studies (*11*, *12*) were cited (*2*) as having reported a lack of PT asymmetry in apes. However, in most humans that are left hemisphere–dominant for language, that portion of the sylvian fissure (SF) associated with the larger left PT extends more posteriorly than on the right (*2*). On the basis of on this anatomic association, SF asymmetry has also been assessed in some nonhuman primates. For example, LeMay and Geschwind (*13*) showed that orangutans (*Pongo pygmaeus*) in particular, and less so gorillas (*Gorilla gorilla*), showed SF asymmetry whereas lesser apes did not. Likewise, Yeni-Komshian and Benson reported a longer left SF in chimpanzees (*14*). Conversely, SF asymmetry has not been demonstrated consistently in Old World monkeys (*13*–*15*), and the PT has even been reported to be absent (*11*). In human fossil endocasts, perisylvian asymmetries have been shown to be present in *Australopithecus*, *Homo habilis*, *Homo erectus*, and *Homo sapiens neandertalensis* (*16*). Clearly, because of the presence of a similar pattern of left hemisphere predominance in most hominoids, the presence of SF asymmetries in fossil humans cannot be used to support the notion that they may have had a capacity for language or speech.

We ascertained the borders of the PT in 18 chimpanzee brains (*17*) by using anatomic landmarks determined for humans (*18*). Although the PT of three brains in this sample could not be directly quantified because of their friable condition, a visual appraisal of PT asymmetry was conducted and calipers were used to measure its lateral extent (*17*). The new and relatively simple method used for this study was devised to measure the cortical surface area of the PT and, more important, to include often neglected elements such as three-dimensional (3D) contours and tissue buried within sulci (*19*). Our initial approach, which used high-resolution magnetic resonance imaging with both 2D linear sagittal measures and 3D planar reconstructions, was unable to delineate the often subtle anatomic landmarks that were otherwise immediately apparent upon direct observation of cadaveric tissues with an operating microscope.

Although a previous report (*14*) stated that PT and Heschl's gyrus landmarks were poorly developed and difficult to identify in chimpanzees, we did not find this to be the case. In fact, we determined these structures to be robustly represented (Fig. 1). Homology of the chimpanzee and human PT is supported at several levels because this area has been shown to share anatomic, positional, cytoarchitectonic, and connectional characteristics in both (*20*). In addition, many studies in nonhominoid primates such as Old World monkeys support homology of this region. It seems reasonable to conclude that the PT of great apes deviates little from this well-established basal primate pattern.

In this sample of 18 chimpanzee brains, the PT was larger in the left hemisphere in 17 brains, or 94%. In the 15 brains where PT surface area was quantified, the left PT was significantly ($P < 0.001$) larger than the right PT (Fig. 2 and Table 1). The right PT contained a separate pedicled gyrus in two brains. Conversely, the left PT had a separate, strongly represented, pedicled gyrus in five brains. Furthermore, the left PT appeared better developed than the right and in five brains showed a pronounced globular swelling that projected superiorly. The chimpanzee Heschl's gyrus homolog also showed evidence of a strongly excavated middle Heschl's sulcus, within the confines of a single gyral pedicle, predominant-

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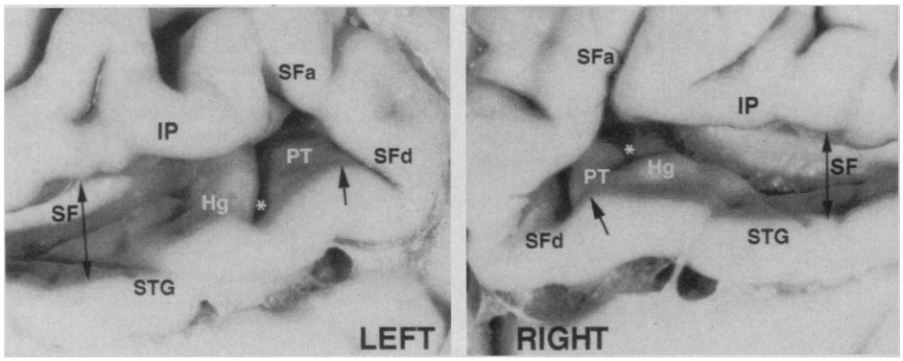

Fig. 1. With the SF spread open widely, Heschl's gyrus and sulcus (indicated by Hg and an asterisk, respectively) and the PT on the left and right posterior superior temporal gyri (STG) of the chimpanzee brain are clearly seen. SFa, ascending limb of the SF; SFd, descending limb of the SF; IP, inferior parietal lobule. Single-headed arrows demarcate the ridge at the point of diversion of the posterior PT and the SFd (*19*).

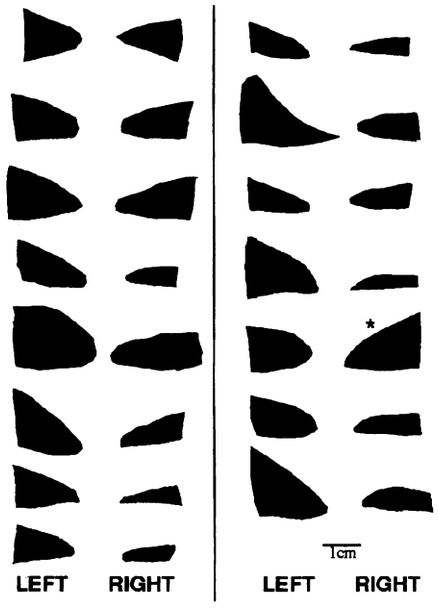

Fig. 2. Cortical surface area of the PT in left and right hemispheres of 15 chimpanzee brains, as measured by black plastic templates (*19*). Asterisk indicates right greater than left.

ly in the right hemisphere.

We have demonstrated that the PT homolog of chimpanzees is similar in anatomic pattern and left hemisphere size predominance to the PT of humans. In humans, this region is widely believed to be a key functional substrate for language and other communication-related tasks (*1–10*). Because chimpanzees do not possess either a communicative capacity remotely similar to that of human language or presumably any of the other functions and pathologies attributed to this region in humans, an alternate interpretation of the significance of this anatomical pattern is clearly necessary. In fact, there are several ways in which this finding might be interpreted. The most parsimonious assumption that may be made, however, is that the PT was already lateralized anatomically to the left hemisphere in the common ancestor of chimpanzees and humans about 8 million years ago. Within this evolutionary scenario, however, several distinct evolutionary hypotheses are embedded.

First, that the asymmetric PT in the common ancestor was unrelated to language or communicative functions but later became coapted to subserve the unique form of human language. Conversely, the PT did not evolve a functional role in communication-related tasks in the chimpanzee lineage and is currently involved with some other function.

Second, that the ancestral, asymmetric PT was involved with communication-related functions, which then followed disparate evolutionary trajectories during the subsequent differentiation of the chimpanzee and human lineages. Because both of these discrete functional trajectories were founded on a communication-related basal neural framework, they gave rise to the unique and distinct forms of human and chimpanzee "language" over the subsequent 8 million years. Within this hypothetical framework, chimpanzees would possess the neural substrate for "chimpanzee language," which may be mediated through use of a subtle "gestural-visual" mode we have yet to understand better (*21*). Many studies have supported this speculative notion based on the extraordinary and diverse cognitive abilities and purported prelinguistic capacity of chimpanzees (*22*).

Third, it may be that the PT was never, and currently is not, related directly to language or communicative functions in either humans (*23*) or chimpanzees. Instead, the PT may be involved with yet to be understood or tangential functions that are also localized to the PT in the left hemisphere and that may even be common to both species. This latter interpretation would characterize the PT in humans, a brain region that current dogma mandates to be a key substrate for language and other related functions, as an epiphenomenon.

It is less likely that the PT was symmetric in the common ancestor of humans and chimpanzees and then became lateralized to the left hemisphere in both lineages independently, because this would involve homoplasy, that is, separate evolutionary processes acting in parallel. Furthermore, evidence from SF length in another living hominoid species, the orangutan, which may have shared a common ancestor with humans around 12 million years ago, also indicates that the PT was already asymmetric and lateralized to the left hemisphere at this much earlier time point (*14*). For this reason, it would be instructive to further characterize this region in the closely related bonobo (*Pan paniscus*) as well as the other great apes and lesser apes.

Regardless of its putative functional role in communication or language tasks, the anatomic substrate of the PT appears to have had a long evolutionary history within the cerebral cortex of at least hominoid primates. Whether the PT represents the functional substrate of a species-specific communication-related behavior in chimpanzees is currently not known. It has been suggested, however, that cognitive and communicative abilities may have coevolved during hominid evolution (*24*). Within this theoretical framework, it seems reasonable to hypothesize that the PT (which was already asymmetric and likely

Table 1. Surface area of the PT. The PT was significantly larger in the left hemisphere in 14 of 15 brains quantified. Asterisk indicates right greater than left.

Specimen no.	Left PT (mm^2)	Right PT (mm^2)
JH7	128.7	111.9
YN95-115	146.6	126.3
JM 2-5	177.8	157.3
JH1	134.6	55.8
YN95-4	279.6	178.0
JH8	177.2	87.1
YN92-115	119.3	53.5
YN88-256	102.4	47.9
J961	131.1	78.3
JH5	231.7	90.3
YN80-7	90.6	80.0
YN97-139	189.5	53.5
YN94-225	146.1	192.5*
F6-10	88.4	55.6
YN77-111	215.7	91.3
Average	157.3	97.3
SD	54.6	46.9

modally equipotential in the common ancestor) further evolved independently to subserve the species-specific repertoires that characterize human and chimpanzee communication and cognition.

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17. Eleven chimpanzee brains from the collection of Dr. Ralph Holloway and four from the Yerkes Primate Center (YN97-139, YN92-115, YN88-256, and YN77-117) were used (thanks to Daniel Anderson, Jeremy Dahl, and Harold McClure; and to James Rilling, Department of Anthropology, Emory University). The cortical surface area of the PT in these 15 brains was quantified as described below (*21*). Three brains from the Smithsonian Institution were also used (SI-292178, SI-225776, and SI-292176) (thanks to Dr. Richard W. Thorington Jr., Linda K. Gordon, and Jeremy Jacobs, Department of Vertebrate Zoology.) Two of us (P.J.G. and A.R.B.) first noted marked asymmetry of the PT and Heschl's gyrus in these three brains during a magnetic resonance imaging study. Because of the friable nature of these three brains, the SF could not be spread widely; thus, the lateral margin of the PT was quantified with calipers. Results showed that the PT was 51 ± 40% larger on the left than on the right (49, 12, and 91% greater on the left than on the right, respectively).
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19. The quantitative method employed here, which accounted for the 3D complexity of the PT unlike methods used previously, involved the following. (i) After wide spreading of the SF to obtain an unobstructed view of the PT, and with the use of an operating microscope at magnification ×4 to ×10, three of us (P.J.G., R.L.H., and D.C.B.) identified the suite of defined structures that demarcate the borders of the PT (*20*). In brief, these are as follows. Anterior: Heschl's sulcus, which forms the posterior border of Heschl's gyrus. In the event of a secondary posterior portion of Heschl's gyrus, the criterion used to include this within the PT was if it comprised a distinct retroinsular pedicle of origin. Lateral: superolateral margin of the superior temporal convolutions. Posterior: the termination of the horizontal portion of the SF. A distinct ridge, formed at the point of diversion of the horizontal and descending limbs of the SF was apparent (arrows in Fig. 1). Medial: the retroinsular point forming the apex of the approximately triangular PT. (ii) A sheet of thin black plastic was formed to fit precisely within the defined margins of the PT. The thin flexible plastic conformed to the 3D complexity of the PT and was able to be inserted within Heschl's sulcus even in cases where this structure was deeply invaginated (asterisk in Fig. 1). (iii) The black plastic templates were attached to white cards and scanned at 150 dots per inch into TIFF files. Their area was then determined in millimeters with the use of SigmaScanPro. Statistical analysis (Statistical Package for the Social Sciences) included analysis of variance and paired *t* tests.
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Stabilization of Dendritic Arbor Structure in Vivo by CaMKII

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Calcium-calmodulin–dependent protein kinase II (CaMKII) promotes the maturation of retinotectal glutamatergic synapses in *Xenopus*. Whether CaMKII activity also controls morphological maturation of optic tectal neurons was tested using in vivo time-lapse imaging of single neurons over periods of up to 5 days. Dendritic arbor elaboration slows with maturation, in correlation with the onset of CaMKII expression. Elevating CaMKII activity in young neurons by viral expression of constitutively active CaMKII slowed dendritic growth to a rate comparable to that of mature neurons. CaMKII overexpression stabilized dendritic structure in more mature neurons, whereas CaMKII inhibition increased their dendritic growth. Thus, endogenous CaMKII activity limits dendritic growth and stabilizes dendrites, and it may act as an activity-dependent mediator of neuronal maturation.

During brain development, neurons elaborate complex dendritic arbors. This process is controlled by mechanisms that promote and limit neuronal growth (*1*). Because neuronal activity and the resultant calcium influx can decrease neurite extension (*2*), activity may control dendritic growth by a calcium-mediated mechanism.

Calcium-sensitive enzymes such as CaMKII can influence both neuronal growth (*3*) and synaptic efficacy (*4*); however, it is not clear whether these effects are coordinated. Because CaMKII is concentrated in postsynaptic densities (*5*), with a wide range of substrates including transmitter receptors, channel proteins, and cytoskeletal proteins (*6*), it could transduce input activity into coordinated changes in both neuronal growth and synaptic strength. CaMKII expression and subcellular localization are developmentally regulated (*7*, *8*). Postsynaptic elevation of CaMKII activity influences development of presynaptic retinotectal axons (*9*) and maturation of retinotectal synaptic responses (*10*). These findings suggest that CaMKII may coordinate the development of synaptic physiology and neuronal morphology.

CaMKII immunoreactivity is distributed in a rostrocaudal gradient in the optic tectum (Fig. 1). A crescent-shaped proliferative zone in the caudomedial region of the optic tectum of *Xenopus laevis* tadpoles con-

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