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Language Evolution: Loquacious Monkey Brains?

Theories about the evolutionary path that led to the neuronal network underlying human language have long generated much heat without light. Using PET imaging, a recent study suggests that monkeys share a circuit for vocal perception with humans, adding some empirical data to the debate.

Asif A. Ghazanfar[1] and Cory T. Miller[2]

Humans have long had a fascination with the utterances of other animals and how their vocal signals may or may not relate to our speech. Even the daring adventurer and master linguist, Sir Richard Burton (1821–1890), could not resist investigating whether monkeys communicated using speech-like vocalizations [1]. Having collected about 40 monkeys in his house, he compiled a 'monkey dictionary' and soon learned to mimic some of their sounds. He was convinced that they monkeys understood him. Sadly, the results of his study perished in a fire. The interest in monkey calls and its relation to human communication, however, continues to burn unabated.

Determining the substrates required for the evolution of human speech and language is a difficult task as most traits thought to give rise to the unique aspects of human communication — the vocal production apparatus and the brain — do not fossilize. Thus, we are only left with one robust method of inquiry: comparing our behavior and brain with those of other extant primates. Primatologists continue to go back and forth debating whether the calls of nonhuman primates are in some ways like human speech, for example, in having external referents [2], whether they are mostly just your typical animal signals [3], or whether their capacities are mostly similar to those of humans and only lack the recursive structure of language [4]. Thus, at the behavioral level, there is much healthy debate and we continue to learn more and more about the form and function of primate vocal signals.

Oddly enough, popular speculation about the underlying neural mechanisms of speech/language evolution is frequently driven by the notion that the human vocal communication, particularly language, did not evolve via an ancestral 'vocal' pathway, but primarily through a 'gestural' pathway (for example [5]). Much of the support for this argument is derived from a set of assumptions about the role of 'mirror' neurons, a small population of neurons in the ventral premotor (PMv) cortex of monkeys that responds both to the sight and execution of an action [6] and is thought to be a homolog of Broca's area in humans.

Beyond this idea, others invoke a sort of spontaneous neural transformation in the ancestral primate brain that gives rise to human language circuits, here epitomized by a quote from Steven Pinker [7]: "language could have arisen, and probably did arise, … by a revamping of primate brain circuits that originally had *no role in primate vocal communication*" (italics added). For the most part, these arguments ignore much of what we know about the processing of vocalizations in the primate brain and their similarities with human speech processing [8]. Nevertheless, one straightforward,

initial test for the existence of homologies between monkey and human neural circuits for communication would be to show that the circuits activated by species-specific calls in monkeys are the same as those human neural circuits activated by speech.

Gil-da-Costa *et al.* [9] have made an effort to bridge this gap between speculation and empirical data. In their recent study, rhesus monkeys listened to two of their species-typical vocalizations — coos and screams — and a set of non-biological sounds. After a listening session, their brain activity was mapped using positron emission tomography (PET). The key to the study is that the non-biological sounds were, statistically speaking, matched in their low-level acoustic features to the two call types. Thus, any cortical region that responded more to the vocalizations than to the other sounds was considered to be a 'vocalization-specific' area.

Two such areas (among others) included the posterior part of the superior temporal gyrus and the PMv. Given that these regions are located in the posterior and anterior parts of the Sylvian fissure, their activation by monkey calls naturally bring to mind parallels with the human perisylvian area, classically defined as containing the core circuit for human language [10]. Indeed, the authors [9] conclude that "…species-specific vocalizations in nonhuman primates seem to be processed by homologs of the same core regions that process spoken language in humans".

These are bold claims — perhaps too bold. The limitations of the experimental design and the inconsistencies with other published work suggest that a more cautious interpretation is warranted. Putting aside the pain-staking efforts needed for establishing homologous cortical areas between monkeys and humans [11], a typical design for any study assessing the 'species-specificity' of neural responses would be to compare responses to conspecific signals with those of heterospecific signals and/or other behaviorally relevant sounds (for example [12,13]). We do not know whether the cortical areas activated in the Gil-da-Costa *et al.* [9] study are especially sensitive to monkey calls relative to other salient sounds. By using only two rhesus monkey calls, there is the distinct possibility that other call types (there are ~12–15 calls in their repertoire) might not activate these areas or that other biologically relevant or familiar sounds might activate them at a greater or equal level.

Doubtless, because of their experimental design, their data are at odds with previous imaging and anatomical data in primates, including humans. For instance, when neural activity was averaged across subjects, Gil-da-Costa *et al.* [9] found greater vocalization-related activity in the right hemisphere. In contrast, numerous behavioral studies (for example [14,15]), a lesion study [16] and a PET imaging study [13] all found that conspecific vocal processing by macaques, like humans, is biased towards the left hemisphere.

The study by Poremba *et al.* [13] is particularly noteworthy here. They used PET imaging to examine the patterns of neural activity induced in the rhesus monkey brain using a large set of monkey calls, human speech, phase-scrambled vocalizations and many other sounds. Under these stimulus conditions, the anterior portion of the left temporal lobe shows a bias towards conspecifics calls when compared to other sounds. Interestingly, like Gil-da-Costa *et al.* [9], they found a right hemisphere bias in the posterior part of the superior temporal gyrus; however, this bias was not specific to any particular class of sounds.

Interpreting auditory activity induced by vocalizations in the monkey PMv as evidence for a Broca's area homolog is even more problematic. Like the human inferior frontal gyrus, monkey PMv neurons respond to a number of different stimulus classes in the visual, somatosensory, and auditory modalities. For example, PMv neurons respond to the sights and sounds of salient activities such as the ripping of paper and cracking open peanuts [17]. In light of these data and the extensive connections between the frontal and auditory cortices [18], it is not too surprising that they respond to another class of behaviorally relevant sounds: vocalizations.

The best evidence that monkey PMv is related to Broca's area would be a demonstration that it is involved in the production of vocalizations. Unfortunately, data addressing this issue are limited and largely inconclusive for both monkeys and humans. For instance, experimental lesions of the same two areas described by Gil-da-Costa *et al.* [9] as Broca's and Wernicke's did not lead to a disruption of monkey vocal production (in contrast to lesions of the anterior cingulate cortex) [19]. In humans, only patients with large-scale lesions in and around Broca's area have a disruption in speech production. Without recording neural activity from this area during vocal behavior by monkeys, it is impossible to ascertain the extent to which any area within the frontal cortex is functionally homologous to a language processing area in humans.

In the final analysis, the study by Gil-da-Costa *et al.* [9], though not conclusive, certainly sets up fertile ground for rigorous comparative investigations of the neural evolution of speech and language. One way forward would be for neurobiologists working with nonhuman primates to abandon the idea that Broca's and Wernicke's are monolithic areas [10] and incorporate what we now know from recent human studies: speech and language processing are dynamic and distributed across multiple regions within and beyond the perisylvian region [20]. Indeed, Gil-da-Costa *et al.* [9], report (but do not emphasize) vocalization-induced activity in many regions beyond posterior auditory cortex and PMv, forming a larger network for vocal processing. While not much of a sound-bite and more complicated, such a large-scale network is far more realistic.

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Bacterial Division: Another Way to Box in the Ring

Proper placement of the cell division site in some rod-shaped bacteria requires two different negative regulatory systems, nucleoid occlusion and the Min proteins. *Caulobacter crescentus* lacks these systems, but recent work has uncovered a novel regulator that achieves the same goals.

William Margolin

Cells that divide by binary fission need to place their division plane between segregated daughter chromosomes to ensure partitioning of the cytoplasm and genetic material. In bacteria, this division plane is defined by the presence of the Z ring, which is composed of polymers of FtsZ, a tubulin homolog. Like tubulin, FtsZ assembly is GTP-dependent and its assembly is modulated by a number of regulatory proteins. By a process that is as yet unknown, the Z ring recruits a number of other proteins and then contracts along with the cell wall, ultimately splitting the cell in two [1].

In rod shaped bacteria such as *Escherichia coli* or *Bacillus subtilis*, two negative spatial regulatory systems can largely explain the precision and robustness of Z ring targeting to the cell centre [2]. The first, nucleoid occlusion, prevents Z rings from assembling over the unpartitioned bulk chromosome, or nucleoid [3]. In *E. coli* or *B. subtilis*, a single DNA-binding protein localizes throughout the nucleoid and mediates nucleoid occlusion by inhibiting local assembly of Z rings [4,5]. During the process of chromosome segregation, Z rings are finally permitted to form between the partitioned nucleoids. Despite its important role in limiting where the Z ring can form, nucleoid occlusion is not necessary to position Z rings in these organisms, because inactivation of the nucleoid occlusion proteins has little effect on its own. However, concomitant inactivation of the second spatial regulator — the Min system — prevents Z rings from forming at division sites, and is ultimately lethal.

In *E. coli*, the Min system consists of three proteins, MinC, MinD and MinE, which shuttle from one cell pole to the other. MinD is a member of a large family of bacterial ATPases with deviant Walker A motifs, which also includes the ParA partitioning proteins [6]. In its ATP-bound form, MinD binds to the cytoplasmic membrane via an amphipathic helix. Binding of MinE protein to MinD stimulates hydrolysis of the ATP, causing release of MinD from the membrane and its movement through the cytoplasm [7]. After nucleotide exchange, MinD-ATP rebinds the membrane, but because of the high level of MinE at the most recently occupied cell pole, most MinD-ATP binds at the opposite cell pole. This process then repeats, resulting in a full oscillation period of about a minute. Because of its relatively long dwell times at the membrane, the average concentration of MinD over time is considerably higher at cell poles than near the cell centre [8].