# The Parallel Evolution of Language and Auditory Verbal Hallucinations

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Crow (2000, 2004, 2008a) and Berlim et al. (2003) have postulated that schizophrenia developed in a parallel manner to human language faculty evolution since only *Homo sapiens sapiens* suffers from the disease and its nuclear symptoms, such as auditory verbal hallucinations, are directly related to the human language faculty. The World Health Organization's *Ten Country Study* shows that the index for schizophrenia's nuclear symptoms in human populations is as universal as language use (Crow, 2000)apparently genetic in origin, persists in spite of a substantial fecundity disadvantage. The hypothesis is proposed that the predisposition to schizophrenia is a component of Homo sapiens-specific variation associated with the capacity for language. A genetic change (the `speciation event', predicted to be related to the Xq21.3 to Yp chromosomal transposition that separates Homo sapiens from the great apes. Crow (2000, 2008a) notes that the lack of extrinsic causes and the consistent character of schizophrenia's onset suggest that the disease's cause is intrinsic. But schizophrenia also carries a strong evolutionary disadvantage. Schizophrenics are much less likely to have children (Crow, 2000)apparently genetic in origin, persists in spite of a substantial fecundity disadvantage. The hypothesis is proposed that the predisposition to schizophrenia is a component of Homo sapiens-specific variation associated with the capacity for language. A genetic change (the `speciation event', predicted to be related to the Xq21.3 to Yp chromosomal transposition that separates Homo sapiens from the great apes. This disadvantage in fecundity indicates that it is difficult for schizophrenics to pass on to the next generation whatever gene predisposes them to the disease. These data give us the central paradox of schizophrenia: Why does schizophrenia persist and survive if it is a genetic disease and if schizophrenics rarely pass their genes to the following generation? Huxley et al. (1964) proposed that it would be necessary for there to be an advantage present in the genes the predispose people to schizophrenia to even out the disadvantages, i.e., the symptoms, associated with the disease. Crow (2000, 2008a) reasons that human language faculty is precisely the advantageous phenotype that permits the persistence of schizophrenia's nuclear symptoms. That seems reasonable since it is almost impossible to conceive these symptoms without the structure of language. For example, auditory verbal hallucinations (AVH) are one of the disease's nuclear symptoms. A person who experiences them hears imaginary voices and interprets them as if they were the speech of real voices. Such a description does not mean

anything without reference to language. As we will see, AVH, in fact, facilitate the development and evolution of the human language faculty.

This paper examines what AVH indicate about the neural processing of language and its origin and postulates that AVH are an antiquated form of syntactic neural processing which exemplifies Crow's hypothesis (2004) and (2 that AVH are a syntactic deficit. Moreover, the paper compares various neuroimaging studies to shed light on the difference between healthy language processing and schizophrenic language processing. Anthropological data are reviewed to help narrow down when modern syntactic neural processing developed. It is postulated that (i) AVH developed in a parallel manner to human language faculty; (ii) AVH represent an antiquated form of syntactic neural processing; (iii) the sub species, Homo sapiens sapiens, has replaced AVH-prone models of language processing with cerebral torque (Crow, 2008b); and (iv) the new model for syntactic neural processing is the result of systematic, intrinsic developmental cerebral asymmetries that regulate syntactic processing.

The next section discusses the cerebral anatomy of our subspecies, outlines a model for neural language processing referred to as cerebral torque (Crow, 2008b), and demonstrates how this model fits into modern linguistic theory. The following section describes how the schizophrenic brain differs anatomically from healthy human brains and purposes a model for the neural processing of AVH supported by neuroimaging research. The subsequent section shows how these different models of neural language processing could have evolved by comparing the neuroanatomy of various species to that of *Homo sapiens sapiens* and by introducing Jaynes' concept of the bicameral mind (1976). Finally, the conclusion discusses how Crow's hypotheses that schizophrenia is a result of the evolution of the human language faculty and that AVH are a syntactic deficit are supported by research in other fields and speculates how the shift from AVH-prone neural language processing to modern neural language processing could have occurred.

**Cerebral Anatomy of Homo sapiens sapiens and Cerebral Torque**

Cerebral torque is based on data that demonstrate the healthy human brain's right hemisphere has more mass anteriorly while the left hemisphere has more mass posteriorly. This results in a convergence of neural connections from the right to the left hemisphere anteriorly and from the left to the right hemisphere posteriorly (Crow, 2000, 2004, 2008a, 2008b, 2010). That is, in human beings, the right frontal lobe is typically larger than the left frontal lobe while the left posterior temporal lobe is typically larger than the right posterior temporal lobe (Steinmann & Mulert, 2012). Therefore, the connections between the frontal lobe and the temporal lobe, the *arcuate fasciculus*, expand posteriorly in the left hemisphere while the right hemisphere's *arcuate fasciculus* expands anteriorly. Likewise, the fibers of the corpus callosum that connect the inferior frontal gyrus (IFG) expand in the right hemisphere and the fibers that connect the superior temporal gyrus (STG) expand in the left hemisphere.

Different types of aphasia research show that these asymmetric cerebral areas have distinct linguistic roles. This specialization characterizes the hemispheric lateralization of linguistic functions (Crow, 2008b, 2010). Cerebral lateralization manifests itself in manual dexterity in *Homo sapiens sapiens* and ambidexterity correlates with linguistic deficits (Leask & Crow, 2001; Peters, Reimers, & Manning,

2006). De Saussure (1959) maintained that language consists of a signifier and a signified and that these two roles are processed inversely between production of language and perception of language. In other words, language production is the process of codifying meaning into a set of symbols while language perception is the process of decoding a set of symbols into meaning. More recently, Chomsky (1995) captured this paradigm with the minimalist distinction between performance systems: the *articulatory-perceptual* and the *conceptual-intentional*. Within this paradigm, the *articulatory* is the physiological system used to produce language symbols and the *perceptual* is the physiological system used to perceive language symbols while the *intentional* is the physiological system that produces language meaning and the *conceptual* is the physiological system that perceives language meaning. That is, the productive signifier is *articulatory*, the perceptive signifier is *perceptual*, the productive signified is *intentional*, and the perceptive signified is *conceptual*. Crow (2004, 2008a, 2008b, 2010) argues that the right anterior quadrant processes the *intentional*, the left anterior quadrant processes the *articulatory*, the left posterior quadrant processes the *perceptual* and the right posterior quadrant processes the *conceptual*.

Cerebral torque facilitates the counterclockwise neural processing of these areas through the connective fibers of the *corpus callosum* and the *arcuate fasciculus* (Crow, 2008b). This model parallels anatomical data when one notes that linguistic production consists of specifying the intention of the speaker (which can be any thought that the speaker wishes to share) codifying it to phonetic form articulation, and transmitting it to the listener. Meanwhile, linguistic perception consists of specifying the phonetic form perception of the speaker (which can be any perceivable signal) and decoding it so that the listener can internalize it. It is reasonable to think that something as open-ended as any thought that the speaker wishes to share or any perceivable signal used to convey a thought would require more neurons and more connections than something more specific such as the precise signal used to convey an idea or thhhe precise thought conveyed thru a signal. Therefore, human cerebral anatomy reflects the need to have more connections (and, therefore, greater mass) in the right anterior quadrant and the left posterior quadrant for language processing. Cerebral torque reflects the logical direction of linguistic processing and controls deictic reference which distinguishes *I* from *you* in language acts. In other words, cerebral torque establishes the identity of a speaker as either internally oneself or externally someone else. Formal syntactic theory refers to this distinction as different indexes. In AVH, the indexes refer to pronouns that are perceived as different entities even though they are the same entity. That is, cerebral anatomy and cerebral torque reflect the logistics of most language tasks which require the brain to make a clear distinction between whom the speaker is and whom the listener is. This distinction is what is disrupted in AVH and other symptoms of schizophrenia.

**The Schizophrenic Brain and AVH**

In general, schizophrenia's nuclear symptoms occur when lateralized processing (cerebral torque) breaks down. That is, if the association between the signifier and the signified is arbitrary as de Saussure (1959) argued, schizophrenia occurs when such associations do not coincide with normal language due to a lack of cerebral lateralization (Ketteler & Ketteler, 2010). The fact that it is more likely for preschizophrenic children to be ambidextrous than

other children (Crow, 2000)apparently genetic in origin, persists in spite of a substantial fecundity disadvantage. The hypothesis is proposed that the predisposition to schizophrenia is a component of Homo sapiens-specific variation associated with the capacity for language. A genetic change (the `speciation event', predicted to be related to the Xq21.3 to Yp chromosomal transposition that separates Homo sapiens from the great apes is visible evidence of this. There are also anatomical differences between healthy brains and schizophrenic brains resulting in a loss or inversion of cerebral asymmetry. A loss of neural mass in the left temporal lobe characterizes the schizophrenic brain (Crow, Ball, Bloom, Brown, & Bruton, 1989; Highley, McDonald, Walker, Esiri, & Crow, 1999). Naturally, it is also common to encounter changes in the STG relatively selective to the left hemisphere in the schizophrenic brain (Crow, 2000)apparently genetic in origin, persists in spite of a substantial fecundity disadvantage. The hypothesis is proposed that the predisposition to schizophrenia is a component of Homo sapiens-specific variation associated with the capacity for language. A genetic change (the `speciation event', predicted to be related to the Xq21.3 to Yp chromosomal transposition that separates Homo sapiens from the great apes. An MRI study of first-episode psychosis showed a loss or inversion of asymmetry in the *planum temporale*, an area close to Wernicke's area, in some schizophrenics (Crow, 2000)apparently genetic in origin, persists in spite of a substantial fecundity disadvantage. The hypothesis is proposed that the predisposition to schizophrenia is a component of Homo sapiens-specific variation associated with the capacity for language. A genetic change (the `speciation event', predicted to be related to the Xq21.3 to Yp chromosomal transposition that separates Homo sapiens from the great apes. This indicates that the left *arcuate fasciculus* may not expand as much in the schizophrenic brain. And if inversion occurs, this would indicate that the right *arcuate fasciculus* would not expand much either. If the bundles linking the right IFG to the right STG expand less anteriorly and the bundles linking the left STG to the right STG expand less from right to left, the right STG might misinterpret signals sent from the right IFG as signals from the left STG. This sort of disruption of cerebral torque would explain AVH.

The schizophrenic brain's anatomical changes reflect an alteration in the connectivity of neurons affecting cerebral torque and language processing. Froud et al. (2010) used magnetoencephalography (MEG) to show that the schizophrenic brain did not maintain the neuronal activation pattern characteristic of the healthy brain nor any other consist pattern between subjects during a semantic priming task. This might be due to individual growth and development patterns in the brain for each schizophrenic subject which would theoretically manifest different symptoms. AVH are the Chomskyan *intentional/articulatory* system misinterpreted as the Chomskyan *perceptual/conceptual* system. It follows that signals from the right IFG would be misinterpreted as signals from the left STG. This misinterpretation could be responsible for the dissolution of indexes described by Crow (2004)and (2 and would indicate that AVH is a syntactic disorder or deficit. An fMRI study of the subjective reality of AVH correlated positively with simultaneous activation in the IFG and the right posterior temporal lobe, the right ventral striatum, the auditory cortex, and other regions (Raij et al., 2009). The right IFG (the Chomskyan *intentional*) and the right STG (the Chomskyan *conceptual*) appear to play an important role in AVH, specifically as

the symptom becomes more vivid. It is also important to note that while MEG imaging is not as spatially sensitive as fMRI imaging, fMRI imaging is not as temporally sensitive as MEG imaging. It is possible that in Raij et al,'s (2009) study the right IFG activated milliseconds before the right posterior temporal gyrus.

Wilson et al. (2011) used Diffusion Tensor Imaging (DTI) to study patients of primary progressive aphasia, a degenerative aphasia, and found that only the left Superior Longitudinal Fasciculus (SLF) affected their syntactic processing. In other words, aphasia in the right SLF did not affect the syntactic processing for these patients who do not experience AVH. I propose that STG anatomic inversion leads to more symmetrical fiber bundles, which leads to signals from the right hemisphere being misinterpreted as signals from the left hemisphere. That is, right hemisphere signals in the right ventral striatum being misinterpreted as syntactically valid signals from the left STG.[1] It follows that there must be some developmental mechanism that consistently results in abnormal neuroanatomy which starts to manifest clinical symptoms in late adolescence/early adulthood. Schizophrenia and AVH, therefore, allow for research on a unique type of incomplete degenerative linguistic neural processing. The next section addresses the possibility that this type of neural processing represents antiquated neurodevelopment resulting in antiquated neural processing.

**Human Brain Evolution and the Bicameral Mind**

As stated earlier, hand preferences are evidence of cerebral lateralization while ambidexterity is associated with linguistic deficits. Research on simians and primates demonstrates that these species do not favor one hand or the other and are ambidextrous (Crow, 2004; Uomini, 2009). Analogically, these species do not have the cerebral asymmetry that characterizes our species, which represents a discontinuum in the simian phylogenetic tree (Crow, 2004; Uomini, 2009). Uomini (2009) notes that anthropological evidence of manual tools indicates that there was not a profound manual preference for using tools until Neanderthals started to exhibit the trait. We can therefore assume that species in the *Homo* genus started having systemic cerebral asymmetry close to the separation of *Homo neanderthalensis* and our ancestors, which also coincides with the most recent genetic data. Likewise, we can assume that these species started to have a mind that started to resemble ours, with all of its benefits and flaws. However, the fact that language evolution is limited by the preexisting capacities of the population's individuals indicates that any systemic changes to the cerebral circuits of our ancestors already existed when language came about (Schoenemann, 2009). Therefore, a comparison of the cerebral systems of *Homo sapiens sapiens* that pertain to language with the homological systems of other simians sheds light on cerebral and language evolution.

As expected, homologues to the IFG and the STG exist in primates. According to Schoenemann (2009), the function of these regions in other primates is unclear. It does seem that they are relatively larger in *Homo sapiens sapiens* than in certain types of monkeys. However, the *arcuate fasciculus's* homologue connects the homologue of the IFG to an adjacent region of the STG's homologue in macaque monkeys. Moreover, only chimpanzees and human beings have obvious connections between the IFG and mid-temporal regions important for semantics (Schoenemann, 2009). So the human *arcuate fasciculus* seems to have evolved slowly for some time. Additionally, the prefrontal cortex of

*Homo sapiens sapiens* is twice as large as would be expected for a primate of our size (Schoenemann, 2009). Brodmann's area 13 (BA 13) and Brodmann's area 10 (BA 10) manifest opposite evolutionary patterns in humans. BA 13, which processes information relevant to emotional aspects of social interaction, are only 50% larger than the homologues of others simians while the whole brain is 3 times larger (Schoenemann, 2009). To the contrary, BA 10, which is required for semantic selection tasks, is approximately 6.6 times larger than the homologue of other simians (Schoenemann, 2009). This comparison demonstrates that the cerebral areas associated with linguistic functions have developed more than some cerebral areas associated with other functions.

The breakdown of the bicameral mind postulated by Jaynes (1976) suggests that the intrinsic developmental advantage that creates cerebral asymmetry and cerebral torque evolved as the final innovation in the linguistic development of the human brain. The bicameral mind is based on the idea that, before the second millennium B.C., human behavior was divided in two parts without explicit access to conscious mechanisms: an executive and a follower (Jaynes, 1976). While I question Jaynes' exact chronology, I assume as he does that the bicameral mind was one of the last phases in human language faculty evolution. According to this theory, human beings were much more prone to AVH before the breakdown of the bicameral mind. The difference between the bicameral mind and the modern mind is that the modern mind has a higher stress-threshold for AVH than the bicameral mind (Jaynes, 1976). A clear developmental difference can be observed between the childhood mind with imaginary friends and the adult mind without imaginary friends. The schizophrenic mind, therefore, has a stress-threshold lower than the modern mind but somewhat higher than the bicameral mind. Developmental malfunction of regional cerebral growth resulting in abnormal cerebral torque might be the result of stress. Kennedy and Adolphs (2011) demonstrate that urban stress provokes schizophrenic symptoms. This suggests that only a species with sufficiently complex social structures can develop the neural processing necessary for AVH. A combination of Crow's and Jaynes's theories suggests that AVH are an artifact of incomplete cerebral development resulting in incomplete passage from the bicameral mind to the childhood mind to the modern adult mind.

**Conclusions**

The hypothesis developed here is that developmental variables required for cerebral torque were our ancestors' last evolutionary changes regarding the human language faculty. These changes were an evolutionary advantage that made the breakdown of the bicameral mind and the modern human mind possible resulting in modern human language faculty lacking AVH. Since AVH manifest some syntactic deficits, AVH shed light on primitive syntactic neural processing. In other words, Crow's and Jaynes' theories can be reconciled in a way that interprets them as an antiquated form of syntactic neural processing. This indicates that AVH research can be a very useful to the field of language evolution. The human language faculty is unique because it is recursive (Fitch, Hauser, & Chomsky, 2005; Hauser, Chomsky, & Fitch, 2002). The human language faculty's recursive nature takes its highest form in syntax. AVH research is an important relic of syntactic development, specifically as it pertains to first and second person indexes and deictic reference. Since urban stress is linked to schizophrenic

symptoms, it seems likely that modern syntactic neural processing existed when agricultural methods were developed that support relatively large, urbanized societies which, in turn, would trigger more of the stress that results in schizophrenic symptom. In other words, urban stress leads to primitive syntactic neural processing. Modern syntactic neural processing and the modern human mind could not have evolved until environmental pressures arose to force their creation and the breakdown of the bicameral mind. Competition between humans and Neanderthals could have provided the requisite pressure. Some linguistic capabilities must have existed before the modern human genome spilt from Neanderthals. It seems likely that the communicative capacities of language would have been a huge evolutionary advantage. If one species was able to break the cycle of consist AVH, it would have thrived. Our ancestors somehow manage to start using higher order cognition. This required the proper indexing of our own thoughts in an organized fashion similar to language. AVH must have evolved in a parallel manner to language since the first beings that had something to say first had to have something to say to themselves. AVH are thus a relic of this.

## Notes

1 An alternative analysis might suggest that STG anatomic inversion results in much more rapid counterclockwise cerebral torque but the MEG data suggest otherwise. Nevertheless, the clinical symptoms of patients from this study were mild.

## References

Berlim, M. T., Mattevi, B. S., Belmonte-de-Abreu, P., & Crow, T. J. (2003). The Etiology of Schizophrenia and the Origin of Language: Overview of a Theory. *Comprehensive Psychiatry*, *44*(1), 7–14. doi:10.1053/comp.2003.50003

Chomsky, N. (1995). *The Minimalist Program*. Cambridge, Mass.: The MIT Press.

Crow, T. J. (2000). Schizophrenia as the Price that Homo sapiens Pays for Language: a Resolution of the Central Paradox in the Origin of the Species. *Brain Research Reviews*, *31*(2-3), 118–129. doi:10.1016/S0165-0173(99)00029-6

Crow, T. J. (2004). Auditory Hallucinations as Primary Disorders of Syntax: An Evolutionary Theory of the Origins of Language. *Cognitive Neuropsychiatry*, *9*(1/2), 125–145. doi:10.1080/13546800344000192

Crow, T. J. (2008b). Chapter 29 - The Torque Defines the Four Quadrants of the Human Language Circuit and the Nuclear Symptoms of Schizophrenia Identify their Component Functions. *Handbook of the Neuroscience of Language* (pp. 299–308). San Diego: Elsevier. Retrieved from http://www.sciencedirect.com/science/article/pii/B978008045352100029X

Crow, T. J. (2008a). The Big Bang Theory of the Origin of Psychosis and the Faculty of Language. *Schizophrenia Research*, *102*(1-3), 31–52. doi:10.1016/j.schres.2008.03.010

Crow, T. J. (2010). The Nuclear Symptoms of Schizophrenia Reveal the Four Quadrant Structure of Language and its Deictic Frame. *Journal of Neurolinguistics*, *23*(1), 1–9. doi:10.1016/j.jneuroling.2009.08.005

Crow, T. J., Ball, J., Bloom, S. R., Brown, R., & Bruton, C. J. (1989). Schizophrenia as an Anomaly of Development of Cerebral Asymmetry. A postmortem study and a proposal concerning the genetic basis of the disease. *Archives of general psychiatry*, *46*(12), 1145–50.

de Saussure, F. (1959). *Course in General Linguistics.* New York: Philosophical Library.

Fitch, W. T., Hauser, M. D., & Chomsky, N. (2005). The Evolution of the Language Faculty: Clarifications and Implications. *Cognition*, *97*(2), 179–210.

Froud, K., Titone, D., Marantz, A., & Levy, D. L. (2010). Brain/Behavior Asymmetry in Schizophrenia: A MEG Study of Cross-modal Semantic Priming. *Journal of Neurolinguistics*, *23*(3), 223–239. doi:10.1016/j.jneuroling.2009.03.001

Hauser, M., Chomsky, N., & Fitch, W. (2002). The Faculty of Language: What is it, Who has it, and How did it Evolve? *Science (New York, N.Y.)*, *298*(5598), 1569–79.

Highley, J. R., McDonald, B., Walker, M. A., Esiri, M. M., & Crow, T. J. (1999). The Temporal Lobes, Asymmetry and Schizophrenia: a Post Mortem Stereologi-

cal Study of Tissue. *British Journal of Psychiatry*, *175*, 127–134.

Huxley, J., Mayr, E., Osmond, H., & Hoffer, A. (1964). Schizophrenia as a Genetic Morphism. *Nature*, *204*(4955), 220–221. doi:10.1038/204220a0

Jaynes, J. (1976). *The Origin of Consciousness in the Breakdown of the Bicameral Mind*. Boston: Houghton Mifflin.

Kennedy, D., & Adolphs, R. (2011). Stress and the City. *Nature*, *474*(7352), 452–453.

Ketteler, D., & Ketteler, S. (2010). Is Schizophrenia "the Price that Homo sapiens Pays for Language"? Subcortical Language Processing as the Missing Link between Evolution and Language Disorder in Psychosis – A Neurolinguistic Approach. *Journal of Neurolinguistics*, *23*(4), 342–353. doi:10.1016/j.jneuroling.2010.04.003

Leask, S., & Crow, T. J. (2001). Word Acquisition Reflects Lateralization of Hand Skill. *Trends in cognitive sciences*, *5*(12), 513–516.

Peters, M., Reimers, S., & Manning, J. T. (2006). Hand Preference for Writing and Associations with Selected Demographic and Behavioral Variables in 255,100 Subjects: The BBC Internet Study. *Brain and Cognition*, *62*(2), 177–189.

Raij, T. T., Valkonen-Korhonen, M., Holi, M., Therman, S., Lehtonen, J., & Hari, R. (2009). Reality of Auditory Verbal Hallucinations. *Brain*, *132*(11), 2994–3001. doi:10.1093/brain/awp186

Schoenemann, P. T. (2009). Evolution of Brain and Language. *Language Learning*, *59*(Supplement 1), 162–186. doi:10.1111/j.1467-9922.2009.00539.x

Steinmann, S., & Mulert, C. (2012). Functional Relevance of Interhemispheric Fiber Tracts in Speech Processing. *Journal of Neurolinguistics*, *25*(1), 1–12.

Uomini, N. (2009). The Prehistory of Handedness: Archaeological Data and Comparative Ethology. *Journal of Human Evolution*, *57*(4), 411–419.

Wilson, S., Galantucci, S., Tartaglia, M., Rising, K., Patterson, D., Henry, M., Ogar, J., et al. (2011). Syntactic Processing Depends on Dorsal Language Tracts. *Neuron*, *72*(2), 397–403.