## LANGUAGE ORIGINS IN LIGHT OF NEURO-ATYPICAL COGNITION AND SPEECH PROFILES

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Crow's (1997) suggestion that 'schizophrenia is the prize that Homo Sapiens pays for language' first linked schizophrenia (SZ) to language evolution. The received view that language is a mere expression of thought and that cognition has primacy over communication, on the other hand, is shared by both lay people and scholars as different as Crow, Frith, Hurford and Fodor. Yet both SZ and ASD may suggest the opposite, as we argue here in line with the un-Cartesian program (Hinzen & Sheehan, 2013). Speech processing as such presents with abnormalities in either condition, which can be derived neither from an intangible thought process separated from language, nor from non-speech related distal problems. These abnormalities include: auditory verbal hallucinations (AVHs) in SZ; the lack of a selective attentional bias for speech over non-speech from birth in ASD (Blasi et al., 2015) many 'first rank symptoms' (thought broadcasting, thought withdrawal, etc.) in SZ; and the reversal of the typical comprehension over production advantage in development in ASD. These abnormalities of speech processing, which go along with atypical cognitive profiles, would be a coincidence according to the received view. If speech processing, instead, differs from thought only insofar as thought (as normally processed) is non-overt essentially, abnormalities in thought can follow from abnormalities in speech processing. Cognition and communication would go hand in hand, integrated but distinguishable in normalcy, and disintegrated in different ways in SZ and ASD.

Sociality is (profoundly) impeded in both SZ and ASD; and also speech, albeit in different ways. Could neuro-atypical speech in both conditions be derived from a social impairment? We suggest it is the other way round. Apart

from the telling case of speechless children with ASD, who are the most impaired socially and cognitively, there are general problems with person deixis in both conditions. But deixis is inherent to and originates in speech. In ASD, the 'total feedback' characteristic of speech is difficult to reach in that 'the so-called internalization of communicative behavior that constitutes a major portion of 'thinking' (Hockett, 1960) is overtly precarious (preference for self-reference with proper names or in 3<sup>rd</sup> person; 1<sup>st</sup>-2<sup>nd</sup> person pronoun reversal). In SZ, the deictic frame reached in development in which the world ('it'), sociality ('you') and deictic center (I') are triangulated, breaks down. AVHs concur with a loss of the deictic anchoring of thought and speech, in that thought becomes speech directed at or about the patient. The speech element cannot be subtracted from AVH without losing its substance. It also pervades the rest of reality distortion symptoms to the extent that these, too, adopt the form of an interaction of the self with others. Rational thought may well find its foundation in a healthy speech system deployed in social interaction. A speechless Language of Thought (Fodor 1975) equivalent to its human-specific form has not been documented in any species. Sociality, communication (i.e. speech and co-speech gestures) and thought profiles in humans are inherent facets of natural language, which must inform language evolution.

The role of speech in cognition is also supported by the leading role that speech as such plays in the induction of hemispheric specialization in humans, which is considered of high cognitive import (Hervé et al., 2013) and is abnormal both in SZ and ASD. Brain correlates for either are consistent with the view argued for here but pose a new question: how is it that not all the abnormalities in speech processing (aphasias eg.) give rise to thought disturbances? Our tentative answer targets the insula (Klein et al., 2013), a highly integrative (interoception and exteroception) and phylogenetically novel (present in great apes) cortical structure that is involved both in speech processing (production and perception) and in the dopamine sensitive salience network (with the cingulate), which is differently abnormal in SZ and ASD.

## References

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