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LANGUAGE UNBOUND: GENOMIC CONFLICT AND PSYCHOSIS

IN THE ORIGIN OF MODERN HUMANS

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Words are the physicians of the mind diseased.
-Aeschylus, *Prometheus Bound*

INTRODUCTION

The question of the origin of modern humans has inspired more scenarios, stories, and research than perhaps any other in biology and the humanities. In one of the first such stories, Plato describes how Epimetheus the Titan distributed abilities to each kind of animal, but used them up before reaching humans. His brother Prometheus, seeing that humans had nothing enabling them to survive, stole technology and fire, and knowledge and philosophy, from the gods and bequeathed these skills and abilities upon them. As eating from the tree of knowledge prompted the Biblical God to banish Adam and Eve, so Zeus punished Prometheus by binding him to a rock, and so humans have paid dearly for their gifts of cognition ever since they were bestowed.

In this paper I will seek to bring the Prometheus myth and metaphor up to date, with a focus on language, the gift most uniquely human. Analysis of the evolution of human language brings together three of the greatest unknowns in biology: the brain, the genome, and the evolution of modern humans. It has thus generated a vast literature, a verbiage so extensive that it tends to obscure the paucity of facts. Moreover, the facts that do exist reside in diverse, specialized disciplines from genetics to phylogeny, paleontology, anatomy, neuroscience, psychology, psychiatry and linguistics. My goal is to integrate across these disciplines using the only tool that unites them, evolutionary biology. Language, and humans, are social, so my conceptual monkey-wrench of choice for such a construction is theory for the evolution of social behavior (Alexander 1980, 1987), the only science that addresses how human sociality, and its genetic underpinnings, change under Darwinian selection.

I begin with a few basics about the brain, language, and how natural selection works at different levels from genes to groups. Next, I explain how the brain and language can be studied using the three main approaches for analyzing the adaptive significance of traits: functional design, measurement of selection, and the comparative method. I then apply the comparative method to a new form of diversity: autistic and psychotic-affective spectrum conditions, the main generalized ‘mutations’ of human sociality and language. Our goal here is to understand how human language and communication have evolved by analyzing how these adaptive systems can be perturbed. The nature of such perturbations provides insights into our cognitive and emotional architecture,

just as mutations in a single gene provide insight into its functions in physiology and development.

Virtually all previous studies of language evolution have focused on cooperative and beneficial aspects of human communication, such as coordination of activities, pedagogy, or impressing a potential mate with syntactic and emotive prowess. This perspective is incomplete, because human social interaction is always permeated by complex mixtures of cooperation and conflict, which follow inevitably from asymmetries in genetic relatedness (Alexander 1980, 1987; Haig 2006a). In the fourth section of this chapter, I thus explain the potential roles of conflicts – especially genomic conflicts – in the evolution of language. Finally, I end the chapter by linking evolutionary genomics with psychiatry and neuroscience, to develop a perspective for understanding the selective pressures involved in the origin of modern humans. We will also unbind Prometheus, and discuss new approaches to free humans from the disorders of our evolutionary legacy.

WHAT, AND WHERE, IS HUMAN LINGUISTIC COMMUNICATION?

Human linguistic communication involves activation of both the left and right hemispheres of the brain, plus a complex system of over 100 muscles for articulation, plus a suite of ancillary movements involving manual gestures and facial-expression changes around the eyes and mouth (Galantucci et al. 2006; Lieberman 2007). Crow (2004) and Mitchell and Crow (2005) have described a simple model of the brain as a 'four-chambered organ' in how it processes and produces language via the activation of heteromodal association cortex, the 'thinking' regions of the neocortex that integrate sensory data and motor feedback with thought and memory (Figure 1).

The left hemisphere harbors Broca's area, the locus of encoding and producing speech, which translates 'thoughts' or 'inner speech' into linear strings of neural commands to move specific muscles in specific ways. Also on the left, nearer the back, is Wernicke's area, most simply described as the brain region for decoding the literal denotations of speech by others.

On the right, we have a posterior region 'for' establishing the meanings of heard speech and accompanying movements – that is, the prosody and pragmatics (non-literal meanings) of language that are inferred more or less automatically from tone, inflection, and other clues that overlay literal word-for-word interpretation. Here lies irony, sarcasm, humor – and much of emotion. The right frontal region is an analogue of Broca's area, but for generating the thoughts and intentions that precede speech. In this area, discourse plans are

generated via some form of spatial recombination of components, and then transferred across the corpus callosum.

The two hemispheres thus process forms of information more or less separately, but they must still regulate unitary behavior.

For instance, a husband may tell his wife that he is going to be working late. Her left brain hears that he is going to be "working late" and accepts that on face value. Her right hemisphere, however, hears the melody of his voice, notes the changes in his face and body language as he talks and decides that he is up to something that does not involve work. How she reacts will in turn depend upon which half of her brain prevails as well as on her past experience with her husband and his late night sojourns. In any case, she is in conflict. (Joseph 1992)

Like any discussion involving the brain, this is all a considerable oversimplification. I refer the reader to Cook (2002) and Mitchell and Crow (2005) for subtleties and evidence regarding differential linguistic functions of the two hemispheres. Our main message so far is that left-brain language areas are usually relatively specialized as a system for the rapid, temporal, linear functions of encoding and decoding, while the right-brain areas specialize for processing of spatial, multidimensional information involving emotions, intentions, metaphors, meanings and one of their external manifestations, sociality. This conceptual, neurological model of language functions has been applied to the components of linguistic discourse by Cook (2002) (Table 1).

The actual neurological mechanisms used in processing and producing language appear simpler than one might think. Thus, Ivry and Robertson (1998) provide the rather wooly concepts such as ‘coding’ and ‘meaning’ with a solid neurophysiological basis, in showing how the left hemisphere is relatively specialized for higher-frequency, more-local forms of information processing than is the right. Similarly, the well-supported ‘motor theory of language’ posits that we decode speech in part by activating the same premotor neural circuits that we would use to make the very sounds we hear – a reversible sound-to-neuron translation system (Galantucci et al. 2006). Perception and production are entwined more generally in the human mirror neuron systems, whereby we interpret hand movements, sounds, and facial expression via activation of the premotor neural pathways that we would use to generate them ourselves (Iocaboni and Dapretto 2006). Effective human discourse thus relies on forms of social-emotional resonance, mediated by the left and right brains working in concert. Such mechanisms take on vital importance in understanding how language could have evolved step by step (e. g., Arbib 2005), and in

understanding how sociality and language can go awry in human neurodevelopment.

HOW, WHY, AND WHERE DOES LANGUAGE EVOLVE?

We have described human language and its neural instantiations. Now let us wrap this all together, call it 'a trait', and see how it should evolve, from first principles. I am interested not in imagined prehistorical sequences, but in how basic social-evolution theory can help to explain the functions of language, simple or complex, in human interaction. Towards this end we will take a series of small steps down an evolutionary garden path.

(1) Humans are expected to behave so as to maximize their inclusive fitness, barring errors or rapid environmental change.

This is simply how natural selection works, on all organisms, all the time. Alexander (1980, 1989) expands upon the caveats involved.

(2) The best way to maximize one's inclusive fitness is often to alter the behavior of other humans, given that humans are extremely social, inter-dependent animals that exhibit pervasive confluences and conflicts of interest.

My inclusive fitness is not my brother's inclusive fitness, nor my mother's or children's – we are family but related only by one-half for autosomal genes, the bulk of our genomes. Worse, non-relatives are motivated to mutual aid only by reciprocity and larger-scale common interests. Human interaction and human history are thus litanies of shifting conflict and cooperation, between and within individuals, families, cultures, and other groups, over resources historically linked to reproduction.

How do we alter the behavior of others? There are only three ways: 'persuasion' (negotiating and providing mutualistic benefits), 'coercion' (imposing costs on others, or threatening to do so), and force (taking control of others' behavior away, or threatening such action) (Brown et al. 1997). These methods are used from playground to boudoir to battlefield, and their deployment as alternatives depends critically on the presence and form of asymmetries in physical power, resources and information.

(3) One of the best ways to alter the behavior and thought of other humans, compared to other modalities or actions, is via language and its facial-gestural trappings.

Language offers us the ability to convince, persuade or coerce other humans with logic, to use emotional prosody for persuasion or coercion, and to lie. Indeed, I would suggest that language and emotion evolved in large part for verbally 'manipulating', in a more or less non-pejorative sense, the thoughts and behavior of others. Talk may be energetically cheap, but it can be very powerful and thus very expensive, or profitable, socially. Such large gains and losses may be possible because language is the medium of information, information is power, and power in the control of resources and other humans is the most general and flexible of all avenues to reproductive success.

Studies focusing on the human 'social brain' and 'Machiavellian intelligence' have addressed the niceties of selection for social skills in primate and human evolution (Whiten & Byrne 1997), but they have only begun to interface with studies of language evolution. The common currencies between these fields are neurological, developmental, and genetic. For example, the superior temporal gyrus subserves both language processing and social cognition (Bigler et al. 2007), social skills and language develop in concert throughout early childhood (Bloom 2004), and psychiatric disorders of the social brain virtually always involve genetically-based alterations of language (Delisi 2001; Seung 2007). Is there recent and ongoing selection in human evolution for social skills, and for aspects of language? As discussed below, we must uncover the genes involved to find out for sure – and we can.

(4) Humans are expected to be unaware of, and deny, that their behavior is selfish, nepotistic, or manipulative of others. As a result, much of our most fitness-salient thought, language and behavior should be unconscious, repressed, projected, rationalized, deluded, automatic, or self-deceptive – with denial or the social emotions of shame, embarrassment, and admitted guilt quickly deflecting any suggested or actual culpability.

Alexander (1989) made this essential point, which goes to the psychoanalytic core of human consciousness and behavior. We thus admire but eschew Machiavelli, we esteem altruistic humans who perform heroically for stranger or nation – and we venerate the various gods who sacrifice themselves for us. We also make moral decisions, but cannot provide them any coherent justification because they 'are not open to conscious introspection' (Hauser et al. 2007). Civilized behavior may thus result from repression of Freud's 'sex instinct', modernized in terms of maximizing inclusive fitness.

Robust empirical analysis of such psychological tendencies as delusion and denial is fiendishly difficult, but modern neuroscience can tap the unconscious and offer clues (Trivers 2000; Stein et al. 2006). For example, some patients with right (but not left) hemisphere strokes, leading to left-side paralysis, will

vehemently deny their obvious inability to move their left arm, offering instead rationalizations such as fatigue (Ramachandran 1996). This and other evidence suggests that the voluble left hemisphere serves as a cognitive 'spin-doctor' that maintains (self-serving) conceptual and world-view consistency, while the mute right hemisphere serves us as 'anomaly detector' or 'devil's advocate', prompting cognitive change should the weight of evidence contrary to left-hemisphere's beliefs become too great (Ramachandran 1996). The relationship between left and right hemisphere is also indicated by their severance: cutting the corpus callosum (to control intractable epilepsy) results in complete loss of speech for days, weeks, or months in most patients, but right-hemisphere damage does not cause loss of speech. The implication is that the left hemisphere normally awaits cognitive input from the right hemisphere before initiating speech (Cook 2002), as also suggested by Crow's model of the four-chambered brain. To the extent that consciousness (whatever that is) is associated with language and speech production, it is predominantly a left-hemisphere function – but this extent remains quite unknown, and the right-hemisphere also mediates perceptions that we would consider as conscious (Joseph 1992).

The garden path has led us back to the brain. If you politely followed the entire route, you might agree that the lateralized social and linguistic brain is an astoundingly-complex parallel processer designed to maximize inclusive fitness, without being aware, or admitting, that it does precisely this. Now – maximizing inclusive fitness is an inherently social enterprise, so we must discuss next the contexts of social and language evolution, the arenas of conflict and cooperation that generate variation in the reproduction of alleles and their bearers. There are three such arenas: within family, within and between group, and within individual.

Within-family conflicts

Children develop in the womb nourished from their mother-invading placenta, then from breast, hand, and crying or babbling mouth. Their linguistic minds develop mainly through interactions with their mother and other family members, via the simple, exaggerated language of 'motherese' and pretend, scenario-building play with self, mother, toys and peers (Vygotsky 1962; Bloom 2004; Falk 2004). Childhood is also the main arena for two forms of social strife: parent-offspring conflict, and conflict that involves genomic imprinting. Put most simply, the child's non-imprinted autosomal genes, and the child's paternally-expressed imprinted genes, have been selected for expression and activity that provides more in the way of developmental-reproductive resources to the child than the mother's genes, or the child's maternally-expressed imprinted genes, have been selected to provide (Haig 2006a). To the extent

that language mediates the transfer of resources within families, it should be a key weapon in both forms of conflict (Crespi 2007). Indeed, according to Vygotsky (1962),

'During this stage, the child and adult are constantly issuing instructions or asking questions of each other so that, for the child, the whole process of speaking becomes bound up with attempts by the two parties to control each other's actions'.

For children, suckling, crying, cooing, babbling, persuasive requests, charm, and smiles, and coercive tantrums, arguments, and refusals, stock the social armory (Badcock 1989; Isles et al. 2006; Locke 2006). Mutual dependency and coincident fitness interests temper these battles and reduce associated costs, making many conflicts subtle unless development is perturbed.

Conflicts within and between groups

For our growing child, within and between group conflicts come into play with sexual and social maturity. Sexual selection and sexual conflicts within local groups suffuse adolescence and young adulthood; the former has been postulated as a driving force in the evolution of language (Locke and Bogin 2006), and both processes should contribute to the well-documented sex differences in verbal abilities, with females superior.

Alexander (1989) describes evidence for the pervasiveness of group against group conflicts in human evolution, Bowles (2006) lends population-genetic rigor to the efficacy of this level of selection in humans, and Lahti & Weinstein (2005) explain how the tension between within-group cooperation and within-group conflict should shift in relation to the strength of external threats. Many group-level traits in humans, such as religion, local linguistic distinctiveness, and group-competitive team sport, can best be interpreted in the context of this selective arena and level (Alexander 1989; Nettle & Dunbar 1997). To the extent that group against group conflicts have driven the evolution of the human psyche (Alexander 1989) they must also have mediated the evolution of language, perhaps as the most effective possible means of coordinating within-group cooperation under this lethal selective pressure. Group cohesion should also be greatly strengthened by shared delusions, such as religious and nationalistic beliefs of own-group supremacy and righteousness – righteousness raised from the individual level to that of groups and gods.

Hypotheses regarding the roles of sexual selection and sexual conflict in the evolution of language can be evaluated via joint analysis of sex differences in linguistic abilities and the genetic basis of such abilities. Of particular interest is

whether language-related traits are X-linked, and how gene expression levels of X-linked genes covary with verbal skills. For example, the corpus callosum, which strongly mediates linguistic abilities (e. g., Dougherty et al. 2007), is also sexually-dimorphic in humans – and callosal disorders (such as its absence or reduction) show a strong enrichment to the X chromosome (Crespi, unpublished data), as do genes whose mutations influence general intelligence (Skuse 2005).

Conflict within individuals

So-called individuals are divisible genetically, because they bear sets of genes with different routes for maximizing their frequency in the next generation, via divergent effects on their bearers. These genetic 'factions' (Haig 2006a), which are more or less in conflict depending on their patterns of relatedness and inheritance, include autosomal genes, sex-linked genes, mitochondrial genes, genes in strong linkage disequilibrium, and paternally vs. maternally-inherited genes subject to silencing by genomic imprints. Of these, imprinted genes are one of the best understood, and with sex-linked genes they are also most motivated by selection to influence human cognition, emotionality, and language development. Genes are most often imprinted in the placenta, but the brain runs a close second (Isles et al. 2006), presumably because these two organs directly mediate the transfer of fitness-limiting resources in networks of kin.

The effects of imprinted genes are usually unseen, because they engage in dynamically-balanced, phenotypic 'tugs-of-war', as between mother and placenta in fetal growth (e. g., Cattanach et al. 2006). In pathology such conflictual systems are revealed, as one party stumbles and loses ground, if not the tug of war itself, due to mutation or epimutation. The major disorders of the human 'social placenta', including gestational diabetes, fetal growth restriction, hydatidiform moles, and pre-eclampsia, are mediated in considerable part by imbalances in imprinted gene expression (e. g., Oudejans et al. 2004). And so, I argue below, are the main disorders of the social brain. But to consider this, we first need some tools to dissect the human brain and language, and to uncover the selective pressures under which they evolve.

HOW DO WE STUDY THE EVOLUTION OF LANGAUGE?

Many studies of the 'evolution' of language have used arguments from plausability, with a loose rubric of descent with modification as their main evolutionary tool. Such weak inference appears difficult to avoid, given that we seek to understand a revolutionary, ~50,000-years-past transition in an organ that we do not understand. I will suggest here that recent, converging evidence

from three disciplines, neuroscience, genomics, and psychiatry, is poised to loosen, and ultimately remove, this veil of ignorance and speculation.

Tinbergen describes four methods for analyzing traits in biology, posed as questions: (1) adaptive function and (2) phylogeny are the two evolutionary, ultimate questions, and (3) ontogeny and (4) mechanism, are the two proximate ones. We will address his evolutionary, ultimate questions with three approaches for analyzing the adaptive significance of human language and communication: functional design, measurement of selection, and the comparative method.

Functional design

Functional design refers to what a trait or form of a trait is 'for' - how it enhances performance at some task. Especially for complex traits, analyses of functional design benefit greatly from understanding how the trait 'works' and how its components function together.

For human language and human communication, the burgeoning field of brain imaging, especially functional MRI, is telling us how the brain works - for example, that the medial prefrontal cortex is for theory of mind and empathy, and that the orbitofrontal cortex is for regulating impulse and socially-appropriate behavior (e. g., Saxe 2006). Analyses of activation patterns in normal brains engaging in various tasks is finally lighting and mapping the mind's former heart of darkness, and telling us that the brain is both highly modular and tightly integrated. As we decided above, it is 'for' maximizing inclusive fitness, and so we find regions like the insula that mediate both visceral disgust and ingroup-outgroup judgements (Harris and Fiske 2006), the fusiform gyrus specialized for recognizing faces (Gobbini and Haxby 2006), the ventromedial prefrontal cortex for solving moral dilemmas (Koenigs et al. 2007), and a suite of interconnected regions from amygdalae to frontal lobes comprising the human 'social brain' (Saxe 2006).

These natural-history studies dovetail with older work on the effects of damage to specific brain regions, such as lesions in Broca's or Wernicke's areas causing forms of aphasia, or impairments in understanding emotional prosody of speech following right-hemisphere damage or sectioning of the corpus callosum. What is perhaps most exciting is the nascent integration of genetics with functional imaging, which has shown that brain activation patterns depend on genotype, for such genes as the serotonin transporter SLC6A4 (Rao et al. 2007) and the dysbindin gene DTNBP1 (Fallgatter et al. 2006). Such studies provide a strong, integrative link between genes and brain function - a link that we might prefer to

deny or explain away, but that can some day tell us how we became human, once we understand how genes for our brain evolved.

Measurement of selection

Functional design tells us about performance, but fitness is the currency of evolution, especially given the ubiquity of pleiotropy and tradeoffs. Fitness variation is the outcome of selection, the statistical relation between a trait and some measure of reproductive success. So how can we possibly measure selection, the driving force of evolution, on perhaps the most complex structure in the universe, with its 40-100 billion neurons each with 10,000-100,000 synapses (Rapoport 1999)? What's worse, the key selective events took place tens of thousands of years ago, and evolutionary psychologists struggle to measure selection even in extant populations.

There is a way, albeit indirect. We find genes 'for' brain size, structure, laterality, language, spatial skills, and mood - genes whose variants can reliably be associated with variation in neurophysiological, neuroanatomical, cognitive, and emotional traits. Then we use genetic-variation data from extant humans, and from other primates, to test for the presence and form of selection on these genes - especially 'positive selection', the signature of adaptive directional change in nucleotide sequence.

FOXP2 is perhaps the best-known such gene: it was subject to positive selection in the human lineage roughly 50,000 years ago leading to two key amino acid changes (Zhang et al. 2002). The gene is highly expressed in language regions of the brain (Vargha-Khadem et al. 2005), its extant mutations have been associated with impaired language and articulation of speech, autism, and schizophrenia with auditory hallucinations (see Vargha-Khadem et al. 2005; Crespi 2007), and it may be subject to genomic-imprinting effects (Feuk et al. 2006). There are other such genes. For example, higher expression of the X-linked, non-inactivated gene GTPBP6 is strongly associated with lower verbal skills in Klinefelter syndrome subjects (XXY males)(Vawter et al. 2007), and this gene has apparently been subject to positive selection in the human lineage (Crespi and Summers, unpublished). Genetic variation in the EFHC2 gene, also X-linked, explains over 13% of the variation in recognition of fear from faces - a social-emotional trait - in Turner syndrome (XO) females, and the better-recognizing haplotype appears to have been selected for in recent human evolution (Weiss et al. 2007). And our functionally-imaged genes SLC6A4 and DTNBP1 both show strong evidence of recent positive selection in humans (Voight et al. 2006); DTNBP1 also shows associations of some alleles and haplotypes with schizophrenia risk and general intelligence (Zinkstok et al. 2007), and SLC6A4 harbors variants associated with schizophrenia risk (Fan

and Sklar 2005), autism (Brune et al. 2006), and major depression (Vergne and Nemeroff 2006).

We are just beginning to close the loops between brain function and genetic variation, between genetic variants and positive selection during recent human evolution - and between genes and psychiatric disorders of the social brain. To understand language evolution, we need more of the genes underlying the primary human disorders of language: autism, schizophrenia, specific language impairment, and dyslexia, and genes 'for' lateralization and language ability in non-clinical populations. One such gene has recently been uncovered: haplotypes of the LRRTM1 gene on chromosome 2 are associated both with schizophrenia risk, and with handedness in dyslexics (Francks et al. 2007). This gene is of special interest because it is imprinted, with expression only from the paternal allele, and it has apparently been subject to positive selection in recent human evolution (Voight et al. 2006).

Measuring positive selection on human genes, and linking genetic variants to cognitive and emotion phenotypes, both have severe limits. Some selected variants will be virtually fixed in humans - for example, the FOXP2 functional mutations causing major speech and language impairment are found almost exclusively in a single extended family in London. In other cases, signatures of selection may be erased by recombination of extended haplotypes bearing selected alleles. And there are over 30,000 genes in humans, a very large fraction of which are expressed in the brain. So bottom-up from genes to language will take awhile. What is top down?

Comparative method

An eagle ate the liver of Prometheus each day as he stood chained, paying the price for inspiring humanity with the skills and abilities of gods. Humans likewise pay a huge cost in suffering for their evolutionary legacy of complex social and technical cognition. The cost comes due when some combination of genetic, epigenetic and environmental factors causes neurodevelopment to go wrong. 'Going wrong' is a vague and relative term, but precisely how development is perturbed can provide vital cues to understanding human cognition and emotion.

Marcus and Rabagliati (2006) discuss how we can use human developmental disorders to understand the nature and origins of language, especially its modularity. By their exposition, impairments of particular aspects of language should correlate with impairments in particular ancestral cognitive structures. For example, studies of autistic children show that humans can learn the meanings of words (or how to converse) either naturally, via social-cognitive

mechanisms, or via a general capacity for logic - brute intellectual force - when social cognition is underdeveloped (Grandin 1995; Marcus and Rabagliati 2006).

We can generalize their approach, and consider neurodevelopmental disorders as relatively-generalized 'mutations' of the mind - though not so much mutations as naturally-assorting, cognitive-emotional variation that grades smoothly into normality. Indeed, both autism and schizophrenia are usually discussed as discrete conditions, but all of their core phenotypes represent just tails on smooth continua of personality and behavior (Claridge 1997; Happé et al. 2006). Such conditions are each also convergent, in that a very wide range of developmental perturbations can result in relatively-small, circumscribed sets of psychological traits - their formal psychiatric-diagnostic criteria.

We will consider such convergent neurodevelopmental disorders, especially autistic spectrum conditions and what I call psychotic-affective spectrum conditions, as taxa for comparative-evolutionary study. We will thus compare them, to uncover just how and why they show particular patterns of similarities and differences. As the comparative method in evolutionary ecology allows us to infer selection, the comparative method in psychiatry should, in theory, reveal aspects of human cognitive architecture, and especially language, built by evolution. These are the outcomes of selection for performance in particular mental domains, and the results of maximizing inclusive fitness, for genes and humans that cooperate and compete.

THE AUTISTIC SPECTRUM

Autism is a spectrum of conditions (Table 2), all of which involve some combination of impairments in social interaction, language and communication, as well as repetitive, stereotyped behaviors (Figure 2) (Happe et al. 2006). As regards language the variation is extensive, ranging from mutism in roughly 40% of cases of infantile, ‘Kanner’ autism, to well-developed literal verbal skills in Asperger syndrome, though pragmatic, social-emotional verbal skills remain underdeveloped (Seung 2007). Autism is also highly heritable, with a risk to unborn siblings of autistics 25-100 times higher than in the general population, but its heritability is largely a function of component phenotypes, which are only loosely associated (Happe et al. 2006).

Autistic spectrum conditions are normally considered in terms of disability, in part because some degree of mental retardation is so common. The relative weaknesses found in autism can indeed be problematic for social functioning, as they center around ‘mentalistic’ skills of language, imagination and emotion used in social interactions – most importantly, skills used in inferring the

motivations, intentions and thoughts of other humans (Box 1). Deficits in mentalism are grounded in egocentrism – hence the very term 'autistic', for self-oriented. Such egocentrism applies most closely to Asperger syndrome, a condition characterized by extremely self-centered behavior and specific reductions in social cooperation and reciprocity (Frith 2004).

In addition to these relative weakneses, autism also exhibits a pattern of relative cognitive strengths (Box 1). These strengths center around perceptual, spatial and mechanistic skills, and indeed Baron-Cohen et al. (2001) have found that 'autistic conditions are associated with scientific skills' in non-clinical populations, and Wheelright and Baron-Cohen (2001) reported a familial association of autism with engineering. This is the world of non-human 'things': tools, systems, and non-human animals, where activities and actions are much more predictable and can often be controlled. This is also a world where language is relatively literal and mechanical, or non-existent. Chen et al. (2007) describe how this apparently-disparate pattern of enhanced spatial skills, and reduced linguistic and mentalistic skills, may be jointly mediated by the egocentric cognition characteristic of autism and Asperger syndrome.

HOW AND WHY IS LANGUAGE AFFECTED IN AUTISM?

In his original description of autistic children, Kanner noted mutism, unresponsiveness to questions and lack of drive to communicate with language or gesture, pronoun reversal (especially discussion of the self in the third person), echolalia (repetition of heard speech), and a linguistic focus on one's own specific, often-obsessive interests. These are all deficits in the sociality of language, and they also include the pragmatics of subtle social meaning (Rapin and Dunn 2003). By contrast, literal verbal processing and memory – the mechanical syntax and phonology of language, are relatively preserved, or even enhanced in such skills as hyperlexic reading (Newman et al. 2007).

For Asperger syndrome, Ellis and Gunter (1999) and Gunter et al. (2002) characterize this general pattern of strengths and deficits, for language and other traits, as indicating relative right-hemisphere impairment, and reduced inter-hemispheric connectivity. This inference certainly fits with the pattern of relative social weaknesses in autism, and its underlying cause apparently involves accelerated early brain growth and reversed lateralization in many cases (Flagg et al. 2005; Herbert et al. 2005), although the actual mechanisms and connections remain unclear. The ultimate result is that autistics tend to use speech primarily as a mechanical tool for serving their self-interest, and they think less in words and inner speech but more in mental pictures (Grandin 1995; Whitehouse et al. 2006). Literal and pragmatic speech are thus partly dissociable, as are thinking in words compared to images. In autism, complex

language can be acquired, but not through the usual route of Vygotsky's (1962) developmental pathways from external social interactions and relationships to private speech, inner speech and thought in words. The essence of the autistic spectrum is that the child's assimilation of social interactions, the process that drives this process of enculturation, mental development, and language, is underdeveloped to a greater or lesser degree (Badcock and Crespi 2006).

THE PSYCHOTIC-AFFECTIVE SPECTRUM

Psychosis is literally a disordering of the psyche, the Greek 'soul'. In schizophrenia, such disordering commonly involves delusions and auditory hallucinations, loss of coherence and logic in thought and discourse, and emotionality ('affect') externally-reduced or inappropriate to social context (Tamminga & Holcomb, 2005). Auditory hallucinations, a primary symptom found in over 60% of persons diagnosed with schizophrenia, are also common in bipolar disorder, which involves cycling between manic and depressive states (Baethge et al., 2005), and in major depression. Bipolar disorder and major depression commonly involve other psychotic symptoms such as delusions, as well as symptoms related to dysregulated emotionality (Boks et al. 2007a). Schizophrenia, bipolar disorder, major depression, and related conditions (Table 3) thus exhibit broad phenotypic overlap (Figure 3), and they also overlap in their polygenic underpinnings (Craddock & Forty, 2006; Blackwood et al. 2007). Like the autistic spectrum, psychotic-affective spectrum conditions involve a pattern of relative strengths and weaknesses with regard to cognition, emotion, and aspects of language (Box 2).

Most research to date has focused on schizophrenia. Specific symptoms of this condition, such as auditory hallucinations, delusions, and dysregulated affect, are also common in non-clinical settings (Claridge 1997; Bentall 2003), and some schizotypical traits such as belief in supernatural beings and other aspects of 'magical ideation' are taken for granted, and promulgated, in modern society.

Schizophrenia exhibits a lifetime prevalence of about 1% (Tamminga & Holcomb 2005), across virtually all cultures and racial groups, and it is considered unique to humans, in contrast to other major psychiatric conditions which appear to exhibit approximate non-human homologues (Crow 1997; Horrobin 1998). This uniqueness derives from the observation that the 'nuclear', or 'first-rank' symptoms of schizophrenia involve language and its relations with thought, and they also involve the most recently-evolved and expanded regions and features of the human brain - including strong lateralization of cognitive, emotional and linguistic functions to the left and right hemispheres. One of the

most-consistent findings in the schizophrenia literature is that structural and functional brain asymmetry is reduced compared to controls, for a variety of cognitive and emotional traits, but most-notably for language (e. g., Sommer et al. 2001; Spaniel et al. 2007).

HOW AND WHY IS LANGUAGE AFFECTED IN SCHIZOPHRENIA?

Speech in schizophrenia can be characterized as language expanded pathologically in all possible directions, with the discourse of any schizophrenic individual inhabiting some region of a chaotic linguistic landscape. Symptoms include poverty of speech, continual fast speech, distraction and derailment, incoherence, loss of logic, invention of new words, use of real words in new ways (e. g., 'handshoe' for glove), and choice of words by sound rather than meaning ('clanging') (McKenna & Oh 2005). That said, linguists inform us that schizophrenic speech is only quantitatively, but not qualitatively, different from speech in normal populations (Covington et al. 2005). Thought, the self-other distinction, and emotion exhibit comparable bedlam in schizophrenia: for example, thoughts may be removed from one's head, inserted from outside, or broadcast to others, feelings, actions or thoughts may be controlled by others, and auditory hallucination, the most-common core phenotype of schizophrenia, involves hearing one's thoughts spoken aloud, voices discussing ones-self in the third person, running commentary on one's behavior, or commands to engage in specific acts (Crow 1997).

What can such unfathomable phenotypes tell us about the evolution of language? Crow (1997, 2004) interprets all of these symptoms in terms of consequences of failure to establish left-hemisphere dominance for speech, such that the four-chambered brain dysfunctions in direction and strength of mental flow. In turn, reduced hemispheric dominance derives from delayed development, especially of the later-maturing left hemisphere, during gestation and childhood. Impaired or reduced left-hemisphere language function in schizophrenia and schizotypy may then result in greater reliance on right-hemisphere processing for some components of thought and language (Fisher et al., 2004; Mohr et al., 2005). A key consequence of such a shift may be more 'coarse' semantic processing, generation of more-distant associations between events and thoughts, overestimation of meaningfulness of coincidences, increased magical ideation, and at the extreme, hallucination, delusion, paranoia, and other symptoms of schizophrenia (Claridge 1997; Leonhard & Brugger, 1998; Pizzagalli et al., 2000; Brugger, 2001; Mohr et al., 2005). The hypothesis also provides a simple explanation for the links between creativity and schizotypy as a cognitive style that involves more-distant and more-novel associations between aspects of thought and language (Gianotti et al., 2001; Brugger, 2001; Barrantes-Vidal, 2004).

The links of imagination and creativity, especially verbal creativity, with the psychotic-affective spectrum (Claridge et al., 1990; Nettle 2001) strongly contrast with the lower levels of pretend play and symbolic creativity in autistics (Blanc et al., 2005), their reduced use of inner speech (Whitehouse et al. 2006) and their use of literal rather than figurative or metaphorical language. Indeed, to the extent that thought in words involves play and imagination as social-scenario building (Alexander 1989; Knight 2000), it may be underdeveloped in autism and hyper-developed, as well as selectively dysfunctional, in schizophrenia. Whereas in autism the left hemisphere may thus contribute disproportionately much to language functions, in schizotypy and schizophrenia we apparently see the reverse – reduced skill with syntax and phonology (DeLisi 2001) but increased contribution of right-hemisphere social-linguistic non-literal meanings and emotion to discourse and thought, even though meanings are misinterpreted through some combination of delusion, rationalization and confabulation (Arbib and Mundhenk 2005).

We can now revisit Table 1 and imagine a continuum between autistic-spectrum and psychotic-affective spectrum conditions, mediated in part by lateralized brain structure and function being altered during neurodevelopment in the two directions possible. This is a considerable oversimplification but at least a potentially-useful framework, amenable to falsification. Perhaps the most compelling evidence to date is the cognitive similarities between dyslexia (reading impairment) and schizophrenia (Condray 2005), and the virtual restriction of hyperlexia (fast, precocious, untaught reading) to autistics (Newman et al. 2007). Convergent evidence also comes from the two main forms of sex-chromosome aneuploidy in humans. Turner syndrome (XO females) involves well-developed literal verbal skills (including hyperlexic reading), poor visual-spatial skills, and a high incidence of autism – all suggesting relative right-hemisphere weaknesses (Temple and Carney 1996; Crow 1997; Skuse 2005). By contrast, Klinefelter syndrome subjects (XXY males) exhibit poor verbal skills, relatively-preserved visual-spatial skills, and a notably-high risk of both dyslexia and schizophrenia (Geschwind et al. 2000; Condray 2005; Crow 1997; Boks et al. 2007b) – consistent with relative left-hemisphere dysfunction, as in schizophrenia itself (Crow 1997, 2004). These findings also fit with the emergence of a cognitive trade-off between verbal skills and spatial skills, when a usually-overriding factor for highly-variable general intelligence is factored out (Ando et al. 2001; Johnson and Bouchard 2007).

Finally, a central phenotype of schizophrenia, auditory verbal hallucination, can also be understood in terms of dysfunctional mentalizing (Box 2), which takes us beyond the simple neurological level of impaired self-monitoring of speech. Vygotsky (1962) described a comprehensive theory for the development of

human language and thought, whereby both develop from birth through adolescence via external social interactions and relationships becoming internalized in the brain. Language in particular develops from 'private speech' in preschoolers – talking out loud to and with ones-self in social dialogues, commentaries and commands, usually with teletubbies or equivalent as avatars of social-emotional-verbal play. As the child develops, private speech becomes truly private – in the brain alone as inner speech, but with the same forms of social dialogue, commentary and command. Jones and Fernyhough (2007) point out that auditory hallucinations in schizophrenia exhibit precisely the same manifestations as private speech in young children, thus providing the first coherent explanation for their social forms and contents, and for the subvocalizations that accompany them in schizophrenia. Moreover, Bentall (2003, page 354) describes how such hallucinations often involve the voices of 'significant family members', and Birchwood et al. (2004) describe them as operating 'like external social relationships'. By implication, we all have voices of sorts in our heads, but after early childhood we do not hear them as such – they only emerge as our 'own' thoughts after our minds have developed to full self-consciousness, and after some sort of neural consensus has been reached (Haig 2006b).

This integration of child development with psychopathology dovetails with the highly-speculative psycho-historical hypothesis of Jaynes (1976), that the right hemisphere of humans routinely perceived auditory hallucinations, interpreted as voices from gods, during prehistory from about 10,000 to about 1000 years ago, when true self-consciousness evolved. Does child development recapitulate this process? Functional imaging studies of children that test for spontaneous, auditory-cortex activity during silence (Hunter et al. 2006), may provide clues. Jaynes suggested that symptoms of schizophrenia represent vestiges of the bicameral (two-chambered) hallucinating mind. By contrast, the inner speech that fuels hallucination in schizophrenia is reduced in autism (Whitehouse et al. 2006), as is the sense of self-consciousness and personal agency (Toichi et al. 2002).

Our consideration of autistic and psychotic-affective spectrum conditions suggests that human mind and language exhibit a psychological architecture that stretches along a continuum from mentalistic and hyper-social to mechanistic and purely-egoistical cognition (Badcock 2004) – from a world of people to a world of things, with so-called normality at the center exhibiting a balance between the two. The autistic and psychotic-affective spectra may thus be considered as complex and diverse but also diametrical conditions, with diametric profiles of cognitive strengths and weaknesses, as described in Boxes 1 and 2. The etiologies of these conditions are thus presumably mediated by some partially-shared set of genetic, epigenetic and environmental factors that

influence the development of brain and sociality, and can be perturbed in two main directions (Crespi and Badcock 2007; Crespi 2008). We have seen such diametric causes before, underlying the primary dimensions of human genetics and behavior. They are two: paternal vs. maternal and male vs. female.

GENOMIC CONFLICTS

Development is a trajectory maintained in dynamic balance by forces of homeostasis and canalization. In many situations, the trajectories bifurcate, leading to two more or less distinct forms, such as male and female mammals that diverge as embryos under the cascading effects of genes on the Y and X chromosomes (Davies & Wilkinson 2006). In other situations, divergent genomic interests create a dynamic balance where a single course is followed unless development is perturbed. One example is placental development, where a maternal-fetal tug-of-war, mediated in part by imprinted genes, creates conditions for diametric pathologies such as fetal growth restriction vs. overgrowth, as seen in Silver-Russell vs Beckwith-Wiedemann syndromes (Monk & Moore 2004).

In sexual differentiation, and in genomic imprinting, we see forms of divergent selection at work, generating sets of phenotypes related to sex, growth, development – and language. For example, females exhibit a well-documented superiority to males in verbal skills, apparently in part due to their lower degree of lateralization and relatively large corpus callosum; by contrast, males show relative strengths in some visual-spatial skills (Geary 1998). Females are also, on average, more empathic, and males more systematic (Baron-Cohen 2003, 2006), and these differences are only controversial if ignorance tars them with the brush of determinism or ethics – we could always change the environment and find quite different patterns, or no sex differences, even if the genetics remained the same.

It is my main thesis here that the genomic axes of imprinting and sex mediate in some measure the development of autism and psychotic-affective spectrum conditions, and that liability to these conditions evolved with the origin of modern humans, and hyper-sociality driven by language. We will evaluate these hypotheses with several lines of evidence.

Imprinted-gene conflicts

A role for dysregulated genomic imprinting in autistic and psychotic-affective spectrum conditions is supported by several lines of evidence, including: (1) strong parent of origin effects in the genomic bases of both sets of conditions, (2) high rates of autism in cytogenetic disorders involving imbalance towards

paternally-expressed imprinted genes, such as Angelman syndrome and Rett syndrome, while the opposite imbalance involves high rates of psychosis, as in Prader-Willi syndrome, and (3) data from genome scan and genetic-association studies that implicates imprinted genes in the development of autism, Rett syndrome, schizophrenia, and bipolar disorder (Badcock & Crespi 2006; Crespi & Badcock 2007; Crespi 2008). Similarly, the high rates of psychotic-spectrum conditions in Klinefelter syndrome, and autism in Turner syndrome (where the X is maternaly-inherited), can be explained under Haig's (2006a) hypothesis that X-chromosome genes are selected for benefits to matrilineal interests, as are maternally-expressed imprinted genes on autosomes.

At the phenotypic level, a bias towards paternal-gene expression should result in more 'selfish' phenotypes (especially in interactions with mother), as seen most clearly in Asperger syndrome and 'high-functioning' autism where pathological effects of disrupted development are relatively small. Benefits to mothers and matrilines from psychotic-affective traits are less obvious, but can potentially be understood in terms of small deviations towards enhanced mentalistic skills in 'healthy' positive schizotypy, which can involve higher verbal fluency, increased 'openness' to the environment, and better-developed empathy, altruism, and spirituality (Crespi & Badcock 2007) – precisely the traits of a child who will never see the inside of a psychiatry clinic. But the ultimate currency and evidence is reproductive, and there is evidence from six studies for increased fecundity in first-order relatives of schizophrenics, especially on the maternal line (reviewed in Crespi & Badcock 2007). Mothers with more-autistic offspring should tend to have fewer children, due to their increased costs. This prediction is obvious for cases of Kanner autism due to its high level of impairment at an early age, but cases involving high-functioning autism or Asperger syndrome should provide useful tests.

Sexual differentiation and conflict

What of sex? Our second line of evidence derives from Baron-Cohen (Baron-Cohen et al. 2005), who has championed the 'extreme male brain' theory of autism, positing that this condition is due predominantly to an overdose of testosterone in the womb. Baron-Cohen has marshalled an impressive body of evidence showing parallels between males and autistics for cognitive traits, such as high systematizing and low empathizing, and some aspects of neuroanatomy such as reduced large-scale connectivity. However, there is an absence of evidence for higher fetal testosterone in autism, and his evidence is also consistent with a paternal bias for imprinted genes, given the similarities (though not identity) of the paternal vs. maternal cognitive axis with that of males vs. females (Badcock and Crespi 2006), such as the higher cost of rearing males (Gibson and Mace 2003). Baron-Cohen (2003, page 173) also

discussed the 'extreme female brain' as exhibiting high empathy and low systematizing ability, but he dismissed its role in psychopathology on the presumption that hyper-developed theory of mind skills would be accurate and adaptive rather than pathologically overdeveloped.

Now let us look at sex and imprinting effects together, as they must occur in nature. The so-called 'male brain' appears relatively similar, neuroanatomically and cognitively, to a brain biased towards increased influence of paternally-expressed imprinted genes (Crespi & Badcock 2007). Conversely, a 'more-female' brain is similar to a brain developing under a relatively-strong influence of maternally-expressed imprinted genes. Sex ratios in autism are highly male-biased at the 'mild' end of these conditions, but in severe autism the sex ratio is near equality. Similarly, schizophrenia is relatively mild, with a later onset as well, in females than in males. The most severe neurological and cognitive impairments are found, in both conditions, where the direction of genomic-imprinting dysregulation opposes the sex difference: in females with autism, and in males with schizophrenia (Crespi & Badcock 2007). This hypothesis may also help to explain such patterns as the hypo-gonadism found in males with Klinefelter and Prader-Willi syndromes, the relatively female-like neuroanatomy and hormonal profile of male schizophrenics (Mendrek 2007), a role for imprinting effects in sexual preference (Green & Keverne, 2000; Mustanski et al., 2005), and Freud's (1911) contention that paranoid schizophrenia in males is underlain by repressed homosexuality. Sexual conflict, with alleles differentially favoring one sex over another (Chapman 2006), represents a third force, in addition to sexual differentiation and imprinted-gene conflict, potentially mediating these effects – which we will not understand until we have dissected the genes and mechanisms involved.

The origin of modern humans

We have two human sexes, and two main disordered spectra of the social-linguistic and technical brain – how did we get this way? Our final line of evidence seeks to connect psychosis, autism and language with the origin of modern humans. The only real connection, aside from untestable speculation, is genetic: what genes made us human, how did they evolve, and how do they relate to disorders of sociality? By my reckoning, there are three main dimensions of recent human evolution: (1) language, and thinking in words and abstract concepts, (2) emotionality, which became enhanced and encephalized as a social tool for maximizing inclusive fitness by subtle persuasion and coercion, and (3) technical skills, forged by systematic causal thinking and fine-motor abilities (Wolpert 2003). Expansion of each of these dimensions presumably created novel scope for forms of psychiatric dysregulation, but only

the first is uniquely human, emerging from Crow's four-chambered brain and potentiating schizophrenia.

Now - a considerable suite of genes are known or suspected to underly schizophrenia – have they been subject to recent positive selection, with schizophrenia as a maladaptive byproduct? The short answer is, apparently, yes – many genes that influence the risk of developing schizophrenia show signatures of recent positive selection in the human lineage, including DTNBP1, FOXP2 and MCPH1 (Crespi 2006; Voight et al. 2006; Lencz et al. 2007) and data from the first-generation human HapMap shows an enriched signal of selection for schizophrenia genes (Crespi et al. 2007). The long and real answer must address the question of how schizophrenia coevolved with human cognition, emotionality, and language. This answer awaits studies that deeply integrate genomics with neuroscience and psychiatry, in the context of evolutionary theory.

CONCLUSIONS

Myth does not mean something untrue, but a concentration of truths.
- Doris Lessing, *African Laughter*

Hercules rescued Prometheus from his bonds, during the course of his twelve labors. Jesus likewise rose from the dead and ascended to heaven, suggesting that altruism has its own special and personal rewards. We poor humans will only be free from soul-wrenching autistic and psychotic-affective disorders once we have dissected their evolutionary-genetic and epigenetic bases, and developed prenatal tests and preventatives. During this labor, we should also uncover genes underlying the evolution of language, intelligence, emotion, and technical skills, and elucidate how their variants are subject to tradeoffs, pleiotropic effects and dysregulation.

I have argued here that an important cause of disordered language, cognition and emotion is conflict, expressed at multiple levels from different human groups, to families, to mother and child, and to genes that harbor divergent interests within individuals. At each of these levels, the nexus of conflict within and between groups of people or genes is divergent avenues of maximizing inclusive fitness, which lead to exceptionally strong selection, tugs of war, and imbalances of power (Alexander 1989). Balancing this conflict are the confluences of interest that emerge from genic cooperation, mother's love for child, and love of God – who, like our circle of kin, created us in body and psyche and promises immortality, and who we serve to give life its meaning. In the beginning was the Word, and the Word was God – as are we, modern humans.

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Glossary

Asperger Syndrome: Idiopathic (with unknown cause) autistic condition that involves specific deficits in social reciprocity but no language delay or mental retardation

Autism: Idiopathic condition defined by deficits in language, communication, and social reciprocity, and by the presence of restrictive interests and repetitive behavior

Autistic Spectrum Conditions: Autism and Asperger syndrome, which grade into normality, as well as genomically-based neurological conditions that involve high rates of autism, such as Fragile X syndrome, Rett syndrome, Angelman syndrome and other conditions in Table 2

Broca's Area: Region of the left frontal lobe of the brain that is specialized for speech production

Corpus Callosum: Large bundle of nerve fibers that connects the left and right hemispheres of the brain

Dyslexia: Learning disability manifested as impairments in reading and spelling

Genomic Conflict: Presence in the same genome of genes that maximize their replication via different, conflicting effects on growth, development and behavior (e. g., conflict between Y-linked genes and autosomes over offspring sex ratio, or between paternally-imprinted and maternally-imprinted genes over growth)

Genomic Imprinting: Silencing of a gene in an individual depending upon whether the gene was inherited from the father or the mother. According to Haig's kinship theory of imprinting, paternally-silenced (maternally-expressed) genes are expected to restrict the 'selfish' interests of offspring, and paternally-expressed genes are expected to enhance such interests

Hyperlexia: Spontaneous, precocious mastery of single-word reading, which often also involves impairments in comprehension of the meaning of written material

Klinefelter Syndrome: Syndrome due to one or more extra X chromosomes in males, usually XXY. This condition involves poor verbal skills, spared visual-spatial skills, and a high incidence of psychotic-affective spectrum conditions

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Glossary (continued)

Positive Selection: Selection 'for' specific alleles or haplotypes (contiguous blocks of DNA with the same alleles at polymorphic sites), as indicated by high rates of amino acid substitution or by the presence of haplotypes that are unexpectedly large and have thus recently risen to a relatively-high frequency in a population

Psychosis: mental state characterized by loss of contact with objective reality, which often involves paranoid or grandiose delusions, hallucinations, or disorganized thinking. Psychosis is common in schizophrenia and not uncommon in bipolar disorder and major depression

Psychotic-Affective Spectrum Conditions: A suite of genetically-related and phenotypically-related idiopathic psychiatric conditions that includes schizophrenia, schizotypal personality disorder, bipolar disorder, major depression, anxiety disorders, and panic attacks, as well as genomic conditions such as Klinefelter syndrome, Velocardiofacial syndrome, Prader-Willi syndrome and other conditions in Table 3. 'Psychotic' refers to cognitive (thought) symptoms, and 'affective' refers to mood (emotional) symptoms

Schizophrenia: Set of related psychiatric disorders characterized by psychosis or dysregulated affect, such as 'flat' (lack of) affect or affect incongruent with environmental conditions

Social Brain: Distributed, integrated neural systems for the acquisition and processing of social information; also refers to the idea that the human brain evolved in the context of strong selection from fitness-mediating effects of complex social interactions

Turner Syndrome: Syndrome due to full or partial loss of an X chromosome in females, such that females are mainly XO. This syndrome involves good verbal skills but impaired visual-spatial skills, and a high incidence of autism in females with the intact X inherited from their mother

Wernicke's Area: Area of the left hemisphere involved in the comprehension of spoken language

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Table 1. Cook (2002) describes how the left and right hemispheres of the brain are more or less specialized for mediating different components of language, from its smallest parts to its largest, conversation or discourse.

LEVEL OF LINGUISTIC COMPLEXITY	HEMISPHERE OF THE BRAIN	
	LEFT	RIGHT
Phoneme	Auditory segmentation	Intonational decoding
Word	Denotation	Connotation
	Close associations	Distant Associations
Noun-adjective Phrase	Literal meanings	Metaphorical meanings
Sentence	Literal meanings	Emotional implications
Paragraph	Explicit event-by-event meanings	Implicit meanings
Discourse	Sequential	Contextual

Table 2. The autistic spectrum encompasses a suite of conditions. These conditions include autism (Kanner autism), and syndromes or conditions that overlap strongly with autism in terms of their phenotypic expressions, for multiple traits, in at least a substantial proportion of subjects.

Condition	Selected recent citation
Kanner (infantile) autism	Happé et al. 2006
Asperger syndrome	Frith 2004
Rett syndrome	LaSalle et al. 2005
Fragile X syndrome	Belmonte & Bourgeron 2006
Angelman syndrome	Cohen et al. 2005
Tourette's syndrome	Canitano & Vivanti 2007
Turner syndrome	Skuse 2005
Smith-Lemli-Opitz syndrome	Cohen et al. 2005
Specific language impairment	Conti-Ramsden et al. 2006
Hyperlexia	Newman et al. 2007

Table 3. The psychotic-affective spectrum involves a suite of broadly-overlapping conditions. The best-known conditions include schizophrenia, bipolar disorder, and major depression. The other conditions overlap strongly with these three in terms of their phenotypic expression, for a substantial proportion of subjects. For example, Klinefelter syndrome, velocardiofacial syndrome, and Prader-Willi syndrome involve notably-elevated rates of psychosis, and dyslexia and schizophrenia share a suite of neuroanatomical and cognitive features. Autistic behavior has been described for velocardiofacial syndrome and Prader-Willi syndrome, but it apparently reflects a personality premorbid for schizophrenia or aspects of negative schizotypy (Crespi and Badcock 2007; Eliez 2007) and it is not underlain by autistic-spectrum neurological or physiological traits, or overlap in genetic underpinnings. This spectrum also includes panic disorder, delusional disorder, and anxiety disorders.

Condition	Selected recent citation
Schizophrenia	Tamminga & Holcomb 2005
Bipolar disorder	Craddock & Forty 2006
Major depression	Craddock & Forty 2006
Schizotypal personality disorder	Claridge 1997
Klinefelter syndrome	Boks et al. 2007b
Velocardiofacial syndrome	Feinstein et al. 2002
Prader-Willi syndrome	Soni et al. 2007
Metachromatic leukodystrophy	Black et al. 2003
Dyslexia	Condray 2005

Box 1. People with autistic spectrum conditions, especially autism and Asperger syndrome, exhibit relative strengths and weaknesses in aspects of cognition, emotion, and language. See Baron-Cohen (2003, 2006), Mottron et al. (2006) and Crespi and Badcock (2007) for details.

Relative Strengths

Mechanistic cognition involving understanding or engagement of 'systems', 'folk physics' and how things work

Encoding and decoding skills for language and other tasks

Some visual-spatial skills involving multidimensional spatial problem-solving, such as block design and embedded figures tests

Perception of local vs global features of environment, and 'bottom-up' processing of information

Inability to deceive

Special abilities and savant skills in about 10% of subjects, including calender calculation, list memory, music memory, 3-D drawing, arithmetic computation, perfect pitch, hyperlexia (precocious, untaught high-speed reading)

Relative Weaknesses

Mentalistic, theory-of-mind skills, such as interpreting gaze, inferring intentions, sharing attention, and understanding false beliefs

Pragmatics of language, such as non-literal meanings, metaphors, emotions, humor, irony

Expression of social emotions such as shame, embarrassment, guilt, contempt

Executive functioning, central coherence

Pretend play, imagination, abstraction, inner speech

Box 2. Individuals with psychotic-affective spectrum conditions exhibit relative strengths and weaknesses in aspects of cognition, emotion, and language. The strengths are found primarily in individuals exhibiting mild, non-clinical manifestations of these conditions – in the conditions themselves, the 'strengths' are hyper-developed and dysfunctional, as shown in parentheses. The evidence regarding strengths is also relatively sparse, because most research on schizophrenia and schizotypy focuses of characterizing deficits in clinical populations with a high incidence of pathology. See Crespi and Badcock (2007) for details, and Kravariti et al. (2006) in particular for data on verbal and visual-spatial abilities in schizophrenia and schizotypy.

Relative Strengths (pathological over-development shown in parentheses)

Mentalistic cognition involving application of 'folk psychology'

Perception of global vs local features of environment, 'top-down' processing

Sensitivity to gaze, inferring intentions, shared attention, personal agency, deception (over-interpretation of intention, paranoia, erotomania, delusions of conspiracy, megalomania, self-deception)

Pragmatics of language, such as non-literal meanings, metaphors, emotions, humor, irony (misinterpreted language in psychosis, dysregulated or 'flat' affect)

Understanding and expression of social emotions such as shame, embarrassment, guilt, contempt (emotions typically expressed by voices in auditory hallucination, and in depression)

Pretend play, imagination, verbal creativity, inner speech (magical ideation, auditory hallucination, thought insertion, thought disorder, disorganized speech)

Weaknesses

Mechanistic cognition; rapid decoding and encoding skills, such as reading

Some visual-spatial skills involving 2D and 3D spatial problem-solving, which have been characterized as 'trait markers' for schizophrenia

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Figure 1. Crow (2004) considers the human linguistic brain to comprise four ‘chambers’ of heteromodal association neocortex (neocortical regions used for ‘thinking’): Broca’s area for speech encoding and production, Wernicke’s area for decoding of literal speech, posterior right occipital regions for inferring and deducing non-literal meanings, and anterior right frontal regions for initiating transitions from thought to speech. Normally, these chambers exhibit separate but integrated functions. In schizophrenia this functional distinctiveness is presumed to break down, in association with neurodevelopmentally-reduced levels of cerebral asymmetry, to produce the disordering of language that characterizes psychosis. This model of the brain in language was developed via consideration of the ‘first-rank’ symptoms of schizophrenia, and the neuroanatomical changes wrought by the evolution of modern humans.

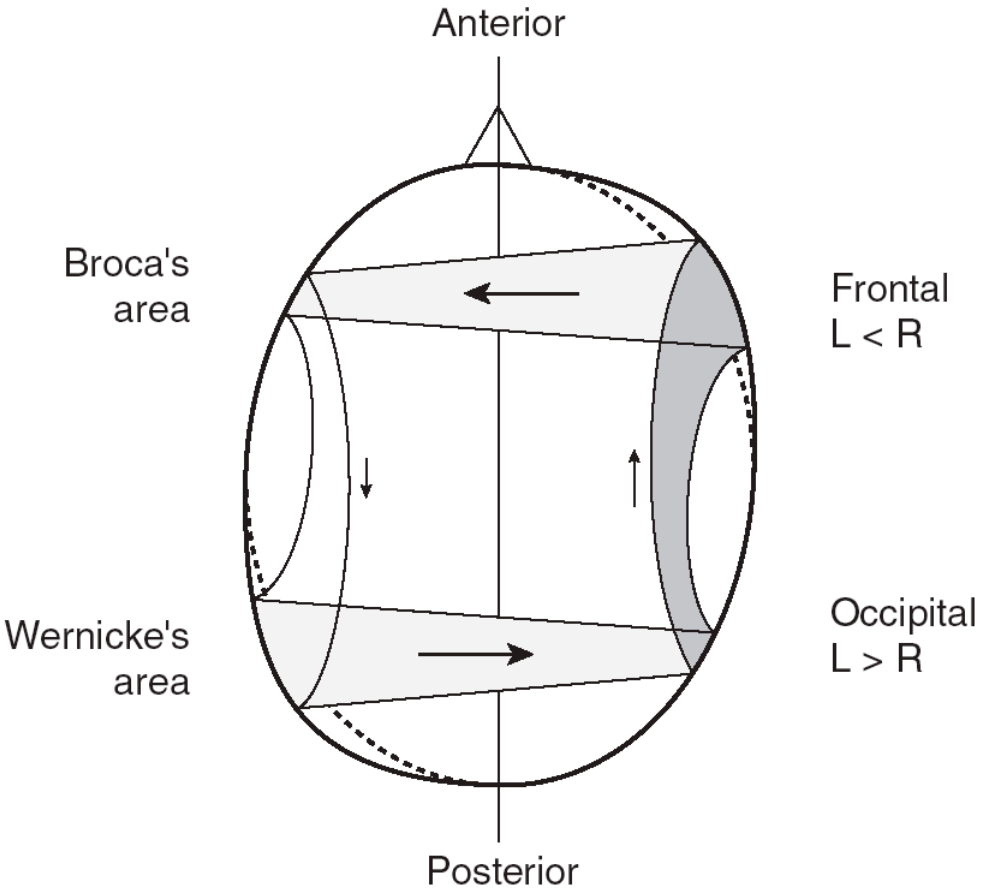

Figure 2. The autistic spectrum can be visualized in terms of three suites of traits that partially overlap in their phenotypic expression and genetic underpinnings. These three suites of traits make up the DSM-IV criteria for diagnosis of autism. At the core of these criteria we find a reduction in mentalistic cognition, which is mediated in part by effects on the development of language.

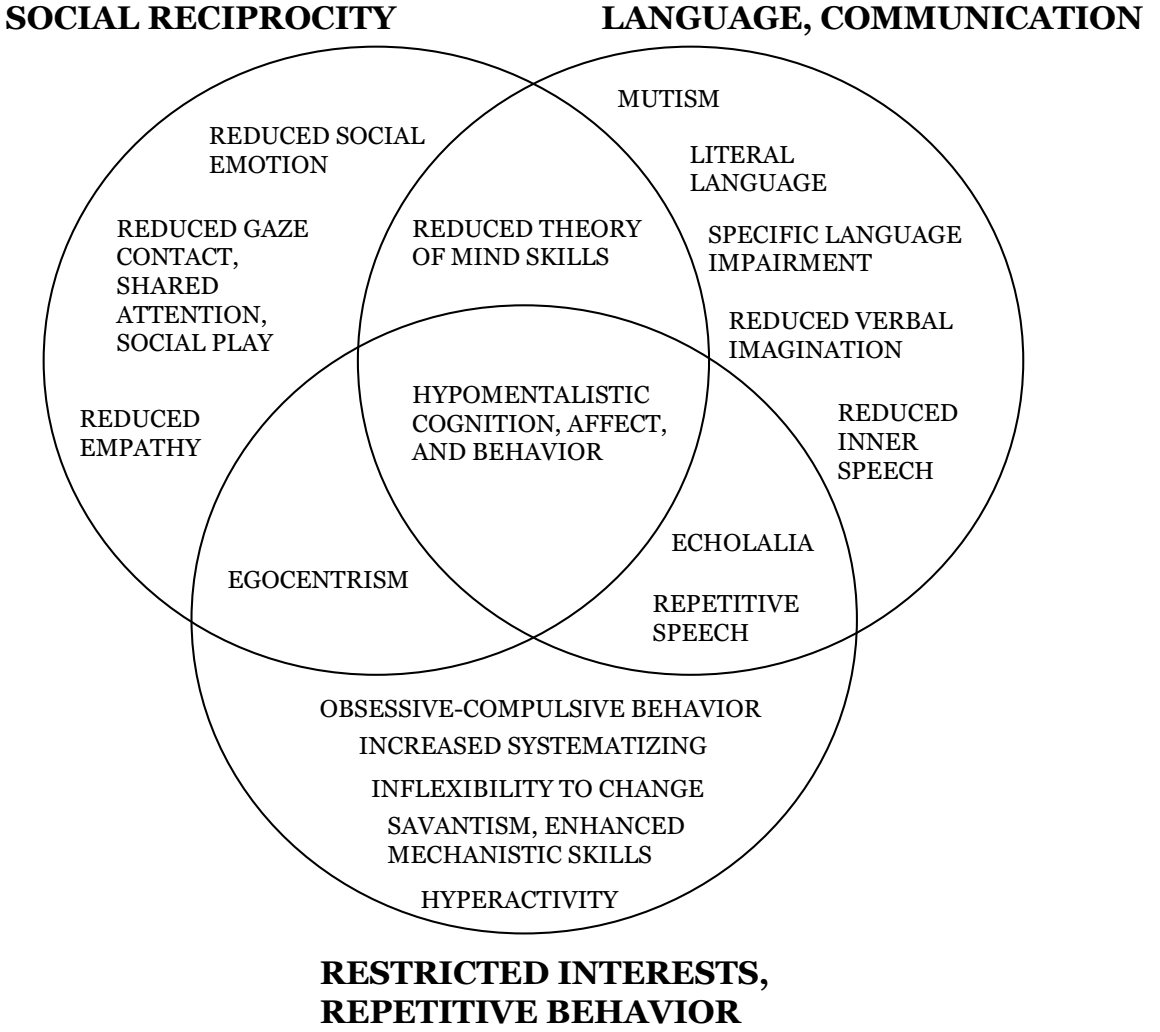

Figure 3. The psychotic-affective spectrum can be visualized in terms of three main conditions, schizophrenia, bipolar disorder, and major depression, that exhibit partial overlap in their phenotypic expression and genetic underpinnings. These three conditions have historically been considered as more or less separate, but recent genetic studies, and consideration of intermediate conditions, have demonstrated that they partially share a broad range of features and risk factors. At the core of the three conditions we find hyper-development in aspects of mentalistic cognition and emotion, which is mediated in part by variation in the development of language.