cognitive-affective competencies. There is now good evidence that humans process information through at least two pathways; one is the route that Burns focuses on. It is relatively slow and cortically focused, and it involves symbolic processing. The other route is far older. It operates through thalamic and amygdala tracts (LeDoux 1998), is preconscious and is the basis for automatic processing (Bargh & Chartland 1999) and emotional reasoning (Haidt 2001). This limbic-based system is key to rapid judgments of threat versus safeness, and can then recruit cognitive processes "to fit" and explain an emotion (Gilbert 1989; 1992; Haidt 2001). One cannot rule out the possibility therefore that (gene-linked) problems in these systems (e.g., people are shifted to threat- rather than safeness-processing in early life and have difficulties in feeling safe when with others) have effects on subsequent maturation of frontal cortical processing systems. There is increasing evidence that early experience does influence cortical maturation and the regulating pathways between cortex and limbic system (Saplosky 2000; Schore 2001), with genes also influencing the specific, emergent choreographies of experience with neuronal networking. Early vulnerability may emerge from a lack of felt safeness, marked by low positive affect, poor sooth-ability, and social disengagement; and later, low social confidence and fear of others. Threat processing systems (rather than safeness creation and affiliation) seem to dominate signal interpretation and role formation (Gilbert 1993). This is supported in Burns' review on the link between amygdala processing and frontal cortex regulation. Phenotypic variations in threat sensitivity (related partly to sensitivities in the amydala), may exist within the general population, but such sensitivities may become more extreme because of the further impact of traum, threat, and lack of safeness. Our abilities to learn about others, mature a theory of mind, and be able to explore our own minds, may depend on at least some modicum of safeness, that allows (affiliative) interactions for learning.

There is increasing evidence that a sizable subgroup of people suffering from this disorder have been traumatised in various ways and suffer from posttraumatic stress disorder (Neria et al. 2002; Resnick et al. 2003). While major traumas, including various forms of abuse, may interact with genetic vulnerabilities, there are a host of other ways that children can grow up in a climate of high threat and low safeness. Birchwood and Chadwick (1997) have shown that voice-hearers usually experience their voices as omnipotent, powerful, and dominating. Moreover, the more subordinate a person feels to others in general in their every day lives, the more subordinate does the person feel to their voice(s) (Birchwood et al. 2000), and of course malevolent voices are also repetitive threat signals (Gilbert et al. 2001).

People may generate hostile voices using similar processing systems for simulating and predicting the social behaviour of others, and for simulating, thinking about, and understanding self-generated thoughts and intentions (Gilbert 2000). Thus, for example, when people feel ashamed, they are self-critical and engage in the same type of self-talk (e.g., I am useless, ugly, a pervert) as would another person who sought to shame them might (e.g., you are useless, ugly, a pervert). They may cue emotional memories of others who have shamed them and where they have internalised these judgements of the self (e.g., "they think I am useless therefore I am useless"; Gilbert 2003).

Many forms of cognitive therapy focus on threat-filled cognitions related to evaluations of other people's intentions, or the source, power, and origins of internal voices (Byrne et al. 2003; Morrison 2002). Helping people with feelings of shame or stigma for this disorder itself, fear of the disorder, and grief, can be major interventions. Any coherent model of psychosis and psychotic phenomena must therefore direct attention not only to various social cognitive processing difficulties but also to the origins and organisation of threat and safeness processing located in old brain systems. Trying to build relationships in which a person can feel safe may be one of the most salient of interventions for people with this distressing disorder.

Schizophrenia: A benign trait

Valerie Gray Hardcastle

Department of Science and Technology in Society, Virginia Tech, Blacksburg, VA 24061-0247. **valerie@vt.edu** **http://mind.phil.vt.edu**

Abstract: While schizophrenia may be genetically determined up to a point, neither it nor its nearest relatives offer any sort of reproductive advantage to its sufferers. Instead, from an evolutionary point of view, schizophrenia is benign – it neither promotes nor inhibits survival to reproduction. Because it is benign, its rate of occurrence should remain fairly constant over time.

Let us assume that everything Burns said about the evolution of the human brain being one of progressive demodularization is correct. Further, let us assume that demodularization coupled with increased neural complexity and connectivity produces an organ more vulnerable to insult, injury, and disease. Finally, let us assume that developmental diseases can have a cascading effect across the human brain, so that one rather minor glitch has the potential to wreak devastation across many regions. All of these are points that Burns skillfully and persuasively argues for in the text. The question I want to examine is whether these claims are enough to support the conclusion that schizophrenia is a heritable trait that has been maintained at a relatively constant level in our population for centuries because it is linked to other genes that offer a selective advantage. I believe that they are not.

I am willing to concede that schizophrenia has a genetic component, though environment certainly plays a large role in its etiology. How are we to explain the fact that approximately 1% of the human population, across cultures and time, is schizophrenic? If schizophrenia were just an overwhelming and ruinous mental disorder, than surely it would have been weeded out over evolutionary history, for many schizophrenics are incapable of caring for themselves, much less reproducing successfully. The conclusion that Burns draws is that there must be something about schizophrenia – or something about what schizophrenia is tied to – that confers an evolutionary advantage.

Burns argues that (1) our social brain is advantageous; (2) genes for schizophrenia predate our evolution into hominids (though they likely weren't expressed as such before our cortex developed such complexity); and (3) genes for schizophrenia are closely tied to the neuroregulatory genes that facilitate our brain development. Therefore, he concludes, genes for schizophrenia piggyback on the neuroregulatory genes such that if both are present, then both are inherited.

Let me suggest that there is a problem with this argument, even if we agree to all of Burns' premises. Consider two obvious facts. First, not everyone has genes for schizophrenia – or even some sort of schizotypal trait. Second, according to Burns, those with schizophrenia are less likely to reproduce than those without. If these premises are true, and Burns seems to believe that they are, then, over time, the carriers of the schizophrenia genes would reproduce less than those who don't carry the gene. Hence, over time, the incidence of schizophrenia should decrease and ultimately disappear altogether.

The only way that rates of schizophrenia could remain constant is if schizophrenia or one of its neighboring traits offered some sort of evolutionary advantage that offsets the reproductive disadvantage of full-blown schizophrenia. In this case, schizophrenia would be something like sickle-cell anemia: carriers of the partial disease would be fitter than normals, which could compensate for the decreased fitness of those with the disease proper.

But, as Burns points out, we have no way of making that argument, for there doesn't seem to be any reproductive advantage given to schizoptypal individuals.

So, then, how are we to explain schizophrenia's constancy across time and cultures? If it does not convey a reproductive advantage, then why didn't natural selection weed it out long ago? Let me suggest an alternative analogy in explanation. Schizophrenia is not

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like sickle-cell anemia. Rather, it is like Huntington's Chorea, another genetic disease; but this one kills its victims after destroying their brains. Huntington's Chorea has managed to survive over evolutionary history because its symptoms don't present themselves until the victim is middle-aged, long after he or she has already reproduced.

I submit that schizophrenia is very similar. Although its symptoms are often visible earlier, the average age of the psychotic break associated with schizophrenia is early twenties. For most of our history, we would have already reproduced by then. Hence, like Huntington's Chorea, schizophrenia strikes after it could make any evolutionary difference at all. From the perspective of evolutionary history, schizophrenia would then be a benign trait, neither enhancing nor curtailing survival to reproduction. It is only from the perspective of recent modern times, in which we reproduce so much later than we ever have before, that schizophrenia decreases fitness. For most of our history, it hasn't. Hence, for most of our history, its rate of occurrence has remained constant.

Auditory hallucinations, network connectivity, and schizophrenia

Ralph E. Hoffman,[a] Michelle Hampson,[b] Maxine Varanko,[a] and Thomas H. McGlashan[a]

[a]*Department of Psychiatry, Yale University School of Medicine, Yale-New Haven Psychiatric Hospital, New Haven, CT 06519;* [b]*Department of Diagnostic Radiology, Yale University School of Medicine, New Haven CT 06520-8043.* **ralph.hoffman@yale.edu michelle.hampson@yale.edu maxine.varnako@yale.edu thomas.mcglashan@yale.edu**

Abstract: Multidisciplinary studies indicate that auditory hallucinations may arise from speech perception neurocircuitry without disrupted theory of mind capacities. Computer simulations of excessive pruning in speech perception neural networks provide a model for these hallucinations and demonstrate that connectivity reductions just below a "psychotogenic threshold" enhance information processing. These data suggest a process whereby vulnerability to schizophrenia is maintained in the human population despite reproductive disadvantages of this illness.

Burns proposes an evolutionary model of schizophrenia based on impaired theory of mind abilities that provides an interesting framework in which to consider this disorder. Below we consider one prominent manifestation of schizophrenia that does not readily fit Burns' explanatory model of schizophrenia: auditory hallucinations, or voices.

Auditory hallucinations reflect the misidentification of self-generated language as spoken speech arising from some nonself agency. Burns argues that if our brains hadn't evolved a capacity to infer intentions of others, there would be no vulnerability for hallucinated voices, because there could be no attribution of alien, nonself willfulness. He therefore posits that disruptions in theory of mind cause these hallucinations. Our research challenges this view.

We are conducting a phenomenological study to determine what factors prompt individual patients to differentiate their voices from their ordinary verbal thoughts. Most commonly, patients rely on three factors: (1) the voice actually sounds like another speaker with distinct, recognizable acoustic characteristics (e.g., low pitched, growling male voice); (2) the verbal content of the hallucination – which is often vulgar, aggressive, or self-critical – is not characteristic of their ordinary verbal thoughts; and (3) voices are experienced as occurring outside of their own willful control. These preliminary data suggest that the otherness attribution of voices arise from inferences about the mind that are actually reasonable. Unintended speech percepts sounding like another speaker saying things that one ordinarily does not say to one's self are common occurrences for all of us – they occur generally when we perceive ordinary utterances produced by actual other speakers. That persons with schizophrenia often attribute language events with these features to other speakers therefore suggests the intactness of at least some experience-based inferences that differentiate self versus nonself agency.

On the other hand, the neurobiological basis for schizophrenia proposed by Burns (namely, disrupted functional connectivity in the brain) is consistent with our research on auditory hallucinations. Computer simulations of parallel-processing neural networks have demonstrated that eliminating connections based on "Darwinian" principles (pruning connections that are functionally the weakest) produces attractor states that intrude into information processing (Hoffman & Dobscha 1989; Hoffman & McGlashan 1997). Complexity and flexibility of neural responses under these conditions are reduced, and spurious outputs are produced in the any absence of sensory inputs, thus simulating hallucinated voices (Hoffman & McGlashan 1997). Such outputs, if generated by actual human speech perception networks, could be cast in the speaking voices of other speakers and experienced as alien and unintended. A model of auditory hallucinations considered as autonomous outputs of speech perception neurocircuitry does not require postulation of specific breakdown of theory of mind capacities.

Consistent with a speech perception neurocircuitry activation model of voices is recent fMRI work by our group. These data demonstrate activation of subregions of the superior temporal gyrus (STG) and Broca's area during auditory hallucination periods that overlaps with activation elicited by listening to external speech in the same patients (Hoffman et al., in preparation; see also Dierks et al. 1999). We also have detected activation unique to hallucination periods in the anterior cingulate and prefrontal cortex that involve neurocircuitry implicated in theory of mind processes, per Burns' discussion. Therefore, our fMRI data do suggest involvement of higher-order neurocognitive processes. However, direction of causality is unclear. Cingulate activation could, for example, arise from attentional/arousal shifts due to intrusion of hallucinated speech percepts.

The aforementioned computer simulation studies demonstrated a second unexpected finding that led us to evolutionary speculations relevant to the model described by Burns. Darwinian pruning of networks to levels just below the "psychotogenic threshold" actually enhanced network performance in detecting linguistic meaning (Hoffman & McGlashan 1997). Burns asserts that the basic mechanisms causing schizophrenia are not in themselves adaptive. Instead, the persistence of this disorder, which is known to produce distinct reproductive disadvantages, is due to linkage between vulnerability loci (presumably generating connectivity loss) and other adaptive loci that enhance function of the social brain. In contrast, our neural network findings highlight information-processing enhancements due to connectivity reductions themselves and suggest that schizophrenia is an extension of this ubiquitous neurodevelopmental process. Consequently, we have hypothesized that there has been and may continue to be an adaptive advantage to robust network pruning which enhances cognitive functioning until it pushes up against a psychotogenic threshold (McGlashan & Hoffman 2000). In this model, higher levels of pruning are selected over generations, with only the small number of individuals at the highest end of the "pruning" curve suffering from frank breakdowns in cortical integration, induction of spurious attractors, and perceptual impairments.

Burns rejects such ideas, because they seem to force the view that schizotypal individuals (who carry genes for schizophrenia but do not suffer from overt illness) should demonstrate some reproductive advantage. We concur with Burns that there is little evidence that individuals with attenuated forms of the schizophrenic phenotype are more fertile. However, there is good statistical evidence to suggest that schizophrenia arises primarily from 2–3 gene epistasis, where multiple distinct susceptibility alleles interact to produce illness risk (Risch 1990). If so, schizotypal individuals in the population could be those who carry "at-risk" combinations of susceptibility alleles but who have not converted to

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