Dysmodularity: A Neurocognitive Model for Schizophrenia

by Anthony S. David

Abstract

Hoffman and McGlashan's introduction of the term neurodynamics prompts a neurocognitive account of schizophrenia along the same lines, called "dysmodularity." In cognitive terms dysmodularity describes an impairment in the function of specialized processors due to a breakdown in one of their prime attributes: informational encapsulation. In neural terms dysmodularity implies increased structural and functional connectivity, reduced anatomical specialization such as lateral asymmetries, and increased white matter in relation to gray. Reduced cortical pruning would be one mechanism for dysmodularity. This model is opposite to the excessive pruning model proposed by Hoffman and McGlashan, but we believe it is more firmly supported by the literature, including some articles in the same issue of Schizophrenia Bulletin (Vol. 19, No. 1, 1993).


The term neurodynamics as introduced by Hoffman and McGlashan (1993a) encompasses many different and novel ways of studying causes and mechanisms of schizophrenia, as reviewed in the Schizophrenia Bulletin (Vol. 19, No. 1, 1993). The most novel of these, and arguably the most exciting, is the use of neural network modeling. Hoffman and McGlashan (1993b) discuss data from a parallel distributed processing simulation of the acoustic analysis of speech (Hoffman and Dobscha 1989). When increasing numbers of neural connections were removed, the network exhibited a number of changes. Its output became less related to input, and subpopulations of neurons began to fire autonomously, sometimes in stereotyped ways. This phenomenon was described as a "parasitic focus," a term that seemed analogous to auditory verbal hallucinations and other characteristic symptoms of schizophrenia. The authors suggest that excessive cortical pruning or at least impaired corticocortical communication is the pathophysiology underlying schizophrenic disorders.

This article takes issue with the latter stages of Hoffman and McGlashan's argument and proposes that the weight of neuroanatomical, physiological, but most importantly, psychological evidence points toward hyperconnectivity as a more plausible general underlying dysfunction. The article draws on the concept of modularity (Fodor 1983a, 1983b) as an overall framework for understanding human cognitive architecture, the pathology of which may be called "dysmodularity." However, it must be stated at the outset that the network model will stand or fall on its own merits. The fact that the network produces hallucinatory analogs is impressive but does not prove that this is how hallucinations are produced in the brainmind of the person with schizophrenia. Indeed, as stated elsewhere in the same volume (Cohen and Servan-Schreiber 1993), the "interesting behaviors exhibited by..."
The concept of modularity owes its foundation to faculty psychology and the phrenologists of the 19th century. Philosopher of psychology Jerry Fodor (1983a, 1983b) laid out the defining properties of a mental module. These properties included domain specificity, informational encapsulation, mandatory processing, and others. Examples of Fodorean modules include speech perception and object recognition. Fodor contrasted these functions with nonmodular or central functions, which have different properties such as attention and "thought." Some cognitive psychologists have criticized this scheme, with most disapproval leveled at the characterization of nonmodular functions (Marshall 1984). Fodor conceived of these functions as using information from a variety of sources and modalities (isotropy), as in complex problem solving.

For our purposes a simplified idea of modularity will be used. The activity of the mind can be divided into separate processing systems, each of which is specialized to carry out its own tasks. In general, it is assumed that modules correspond to localized regions of the cortex, but this may not necessarily be the case. Unlike Fodor, who viewed modules as low-level processors, the network model places modular functions at an intermediate level, effectively splitting central nonmodular functions. Modules thus lie between unconscious, automatic, but generally lower level functions such as attention (see Sternberg 1983)—submodular processes—and effortful, conscious, and relatively time-consuming functions such as problem solving at a higher level—supramodular processes. The latter processes have not been explained completely, but they probably represent the coordinated activity of many modules (Gardner 1983) or even a separate "executive" module (Shallice 1984).

Predictions From Dysmodularity

General Intelligence. The guiding principle behind modularity is that it is an efficient way for the mind to operate. Any complex organization needs specialists with unique skills who are best left alone to get on with their job. Without specialists, the organization will be inefficient and cannot progress, but it need not grind to a halt. The basic workers will continue regardless, and those with higher training can always perform lower level functions. This metaphor is compatible with what we know about premorbid IQ in schizophrenia. Even highly intelligent people who develop the disorder were probably operating at lower general efficiency (i.e., intelligence) than they might have, say, in comparison to their sibs (Aylloward et al. 1984). However, there is nothing that marks them as different, nor is there any particular pattern of deficits. Isolated abilities such as those found in the idiot savant do not occur in schizophrenia (cf. autism), and there is no convincing evidence of enhanced creativity (cf. manic depression).

Cognitive Deficits. Put simply, the free flow of information sustains nonmodular cognition but is the enemy of modularity. Modules are by definition informationally encapsulated and carry out their functions regardless of other demands. For example, the Müller-Lyer optical illusion persists despite knowledge of the lengths of the two lines. Also, one cannot process spoken words for their meaning. Therefore, dysmodularity in the form of excessive connectivity and excessive flow of information will hamper modular functions but leave low level nonmodular ones undisturbed. In general, schizophrenia subjects are less susceptible to standard optical illusions (Letourneau 1974) and can understand speech, but according to Hoffman and McGlashan, subjects make mistakes if the input is degraded. Supramodular functions may fail if they depend on "subordinate modules" or if large amounts of information have to be consciously processed, thus leading to vulnerability to overload or an increase in signal-to-noise ratio. Another consequence of failure of supramodular functions would be misconnections, such as the production of spurious associations in thought (i.e., thought disorder and disturbances in executive functions such as planning) leading to bizarre or impoverished behavior.

This is precisely the pattern seen in schizophrenia. Automatic processes such as short-term memory, recognition, simple stimulus response tasks, and so on are gener-
should be enhanced, and intermodal processes will be derailed by input from other modalities, resulting in Stroop-like interference (Liddle and Morris 1991) and, at worst, synesthesia or crosstalk, first suggested by Randall (1983). Perception will be excessively biased by contextual information, including affects and prior expectation, thus leading to errors in belief formation, for example, illusions and delusions (Fleminger 1992). Linguistic analysis will "overflow" into perceptual systems, resulting in thought insertion and auditory hallucinations (Frith 1979; Nasrallah 1985). Higher level, nonautomatic, nonmodular tasks are also found wanting in schizophrenia (Weinberger et al. 1986; Shallice et al. 1991; Frith 1992). But the pattern of errors in certain tasks, such as those focusing on semantic memory or cognitive flexibility, also points to a lack of informational encapsulation and hence categorization and perseverative errors (Shallice et al. 1991; Frith 1992). Dysmodularity could be further probed by exploring priming (Kwapil et al. 1990) with cross-modal designs, which should be enhanced, and interference, which should be increased. Complex auditory–visual illusions such as the McGurk effect should be preserved in the face of other deficits (Myslobodsky et al. 1992).

Finally, subjective accounts have long stressed the difficulty of dealing with incoming information, a problem that is usually conceived of as a defect in perceptual filtering (McGhie and Chapman 1961). Despite the attractiveness of Hoffman and colleagues' excessive cortical pruning model, the notion of decreased pruning seems a more plausible model. A dysmodularity trait may be exposed by heavy incoming stimuli in more than one modality, for example, that seen in disturbed family interaction and other stressful environments.

Pausing at this point, we may ask whether dysmodularity offers any real advantages over such concepts as impaired internal–external (McGhie and Chapman 1961) and conscious–unconscious (Frith 1979) filtering. Do we need more jargon? The answer to this reasonable question lies in the facile mapping from neurology to cognition implied by the concept of dysmodularity.

**Neurocognitive Development.**

Modularity is the difference between a general purpose processor and a clever computer. We must assume that the neural hardware supports this development of clever skills. We cannot see modularity under the microscope or on a brain scan. However, let us pursue philosopher Gilbert Ryle's (1949) famous example of the university's not being located in a place: Where there is a large collection of books, desks, and young people reading at them, for those of us who are literal-minded, this is in a sense where the university is likely to be if it is anywhere. Similarly, where there is a discrete mass of gray matter—a collection of cell assemblies perhaps—with its own cytoarchitecture (e.g., different from adjacent gray matter and bigger or different from that found in infants or lower mammals), the suspicion must be that there lurks a module. On the other hand, where there are large swathes of connections (white matter, for the sake of simplicity) and "undifferentiated" regions or simply reductions in the amount of gray matter, modularity is compromised. The few university students get distracted by what's going on in the world around them instead of learning the second law of thermodynamics. Of course, modules must talk to each other at some level (the best students are not clausiles), but the balance between sufficient input and overload or crosstalk is a delicate one.

The most obviously differentiated thing about the human brain, visible even to the naked eye, is its asymmetry. This characteristic underlies the first mental module to be discovered: that responsible for speech production. Dysmodularity predicts the following effects: (1) reduced cerebral asymmetry (Crow 1990; Bilder et al. 1993); (2) reduced behavioral asymmetry (Colbourn and Lishman 1979; Flor-Henry and Gruzelier 1983; Gur et al. 1985; Green et al. 1989; David and Cutting 1990); (3) lack of gray matter relative to white (Jernigan et al. 1991; Ron et al. 1992; Zipursky et al. 1992); and (4) structural abnormalities in the most differentiated brain regions underlying the most sophisticated modules, for example, the language areas of the left hemisphere (Suddath et al. 1990; Waddington 1993).

It should be noted that almost all of the work on reduced behavioral asymmetry has looked at a specialized ability such as syllable perception in dichotic listening or dot localization with tachistoscopically divided visual field tasks. The results typically show a
loss of the expected right ear (left hemisphere) advantage and left visual field (right hemisphere) advantage. Rather than arguing whether the right or left hemisphere bears the brunt of the pathology, dysmodularity predicts some impairment on both tasks but a loss of hemisphere advantage in either direction amounting to equilibration. This is consistent with Crow's work and theorizing but is much more general (Crow et al. 1990). While Crow proposes that the "gene" for language lateralization is the gene for schizophrenia, dysmodularity proposes that the gene or genes or other mechanisms that lead to the aspect of cortical development that supports the expression of specialized modules (including those for language) also lead to schizophrenia. This proposition has the advantage of accounting for a wide range of findings while remaining refutable.

A hemisphere, or a whole brain for that matter, that is less modular is less affected by focal lesions. The novel prediction arising from this is that schizophrenia subjects should be relatively resistant, from a cognitive deficit point of view, to acquired focal brain lesions. I know of no data that addresses this question, although the lack of efficacy or indeed the effect of frontal leucotomy may be a case in point (Stuss et al. 1981).

The proposed dysmodularity may be present at birth but only becomes evident when modularity becomes most evident—late in cognitive development. A failure of cortical pruning has been suggested by many to be directly relevant and is a mechanism for demodularizing the brain. Evidence for this is reviewed by Feinberg (1982), Goodman (1989), Jones and Murray (1991), and Stevens (1992). As a theory, defective pruning is flourishing, but the weight of evidence points to a defect in a direction opposite to that proposed by Hoffman and McGlashan.

Just as dysmodularity through hyperconnection should be evident when looking at gross examples of differentiation such as asymmetry, the largest corticocortical tract in the brain, the corpus callosum (CC), cannot escape scrutiny. The CC may indeed be increased in size in some cases. These findings are not universal but suggestive (Günther et al. 1991; David, in press). Even if no absolute size increase is found, the possibility of increased neuronal connections in schizophrenia relative to the reduced volume cortex that it connects remains a plausible and refutable hypothesis (Rosenthal and Bigelow 1972; Raine et al. 1990). However, the crude equation between size and connectivity must not be overemphasized. Functional connectivity is more important (Zaidel et al. 1990). Functional connectivity is difficult to assess, but one study (David 1993) has attempted to do so by measuring Stroop interference (and facilitation) across the CC. David found that functional connectivity was increased only in schizophrenic patients and not in psychiatric controls and that it was correlated with anterior callosal size. Other neuropsychological studies that have found similar effects include those by Green et al. (1986), who showed impairments with a binaural versus monaural comprehension task, and Oepen et al. (1987), who showed increased reactivity in one visual field to emotional material presented to the opposite field.

**Neurophysiology and Neurochemistry.** The large body of research in this area will not be reviewed (see e.g., Kerwin and Murray 1992). Neurophysiology studies using event-related potentials and electroencephalographic measures of coherence indicate increased interhemispheric connectivity (Gulmann et al. 1982; Merrin et al. 1989). One explanation for the increased left medial temporal activity inferred from regional cerebral blood flow studies and found to be a unifying factor in all behavioral subsyndromes of schizophrenia (Friston et al. 1992) is "dysinhibition" between this and related but functionally discrete regions (i.e., dysmodularity). Recent data from studies using magnetic resonance spectroscopy, reviewed by Pettegrew et al. (1993) show that the increased phosphodiester found in schizophrenia correlates positively with CC area that is presumed to reflect in turn reduced frontal lobe gray matter. Deakin and colleagues (1989) found increased glutamate receptor density in the frontal regions supplied by the anterior CC.

**Conclusion**

The idea of neurodynamics as introduced by Hoffman and McGlashan is a good one. Working backward from symptoms to neurons is worthwhile, and modeling computer networks is one way to do this. However, experiments that look at increased gain (Cohen and Servan-Schreiber 1993), as well as signal-to-noise ratio and cross-talk, are promising and concur with a range of data that appear to point to an abnormality in corticocortical connections. This abnormality can account for stable impairments and the pattern of symptoms seen in schizophrenia.
The proposed mechanism of dysmodularity is an example of a neurodynamic but mostly neurocognitive account of schizophrenia. It may be criticized as being too loyal to its phrenological roots in assuming that increased connections equal increased connectivity. I agree with Hoffman and McGlashan (1993b) when they acknowledge that loss of some kinds of neurons corresponds to disinhibition and vice versa. Where this encouraging Anglo-American harmony breaks down is in our views of the nature of the neural connectivity problem. They say too little, and I say too much.

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Announcement

Longtime readers of the Schizophrenia Bulletin may have noticed a
change in the masthead—Dr. Samuel J. Keith, Editor-in-Chief of the
Schizophrenia Bulletin since 1980, has become Editor Emeritus at the
end of 1993. Dr. Keith has ac-
cepted the positions of Professor of Psychiatry and Psychology and
Chairman of the Department of Psychiatry at the University of
New Mexico. During his 20-year
tenure at the National Institute of Mental Health, Dr. Keith's hall-
mark has been creativity and sci-
entific excellence in pursuing a better understanding of the causes,
treatment, prevention, and impact of schizophrenia on patients and
their families. His leadership style is perhaps best captured in his
crafting of the “National Plan for Schizophrenia Research.” In bringing
together over 150 of the world's leading scientists, he led a process that both synthesized the
current findings of the field of schizophrenia research and generated a “blueprint” for the future. The scientific accomplishments of the late eighties and early nineties were made possible through the interest and resources this plan attracted. Furthermore, throughout this complex 18-month process, he insisted that everyone involved remain mindful of the needs and impact of the ongoing deliberations on those most afflicted by schizophrenia—patients and their families. His enduring interest and efforts to maintain the highest sci-
entific quality of the Bulletin also deserve special recognition. The Bulletin staff and its readership are deeply appreciative to Dr. Keith for his commitment to maintaining the breadth of the coverage of the Bulletin without compromising its scientific excellence. We wish him well in his work at the University of New Mexico. We will miss him.