Available online at www.sciencedirect.com
SCIENCE DIRECT®
Schizophrenia Research 61 (2003) 255–263
SCHIZOPHRENIA RESEARCH
www.elsevier.com/locate/schres

Associative memory in chronic schizophrenia: a computational model

S. Duke Han [a], Paul G. Nestor [a,b], Martha E. Shenton [b], Margaret Niznikiewicz [b,c], Gordon Hannah [a], Robert W. McCarley [b,*]

[a] *University of Massachusetts, Boston, MA, USA*
[b] *Psychiatry 116A, Brockton VA Medical Center, Harvard Medical School, 940 Belmont Street, Brockton, MA 02301, USA*
[c] *Massachusetts Mental Hospital, USA*

Received 16 November 2001; revised 20 March 2002; accepted 25 March 2002

Abstract

We developed a computer model to simulate associative memory recall of patients with chronic schizophrenia. Model inputs consisted of words derived from normative data that differed in terms of connectivity and network size, with the former quantitatively represented by parametric weights and the latter by the specific number of word associates that formed a particular network. Previous behavioral studies of normal subjects indicated better recall for words of high connectivity-small network (HCSN), followed by low connectivity-small network (LCSN), high connectivity-large network (HCLN), and low connectivity-large network (LCLN). This pattern of recall differed from that observed in behavioral studies of schizophrenic patients, which showed better recall for high connectivity words, regardless of network size. Holding constant network size while manipulating connection weights effectively simulated this schizophrenic pattern of recall. That is, manipulation of parametric weights coupled with a slight increase in noise significantly and reliably elicited the response pattern of abnormal connectivity demonstrated in the prior behavioral study of patients with chronic schizophrenia. An increase in noise was a necessary, but insufficient step in modeling the response pattern of abnormal connectivity. These findings provide support for the use of computational models to investigate dynamics of associative word recall in patients with chronic schizophrenia.

Keywords: Schizophrenia; Computer modeling; Word association; Associative memory; Connectivity

1. Introduction

A hallmark of the neuropsychology of schizophrenic thought disturbance is a breakdown in the associative threads that serve to interweave words, thoughts, and ideas into coherent discourse (Nestor et al., 1998). Bleuler (1911/1950) first observed the intrusion of dominant but contextually inappropriate associations in schizophrenic thought as illustrated by his now classic example of his patient describing her family members as "father, son, and Holy Ghost". More direct experimental evidence comes from the rigorous work of Chapman and Chapman (1973), who demonstrated a pronounced schizophrenic bias for dominant meanings of homonyms (e.g., "pen" as a writing instrument) even when preceding sentential

* Corresponding author. Tel.: +1-508-583-4500x2479; fax: +1-508-586-0894.

0920-9964/02/$ - see front matter © 2002 Elsevier Science B.V. All rights reserved.
doi:10.1016/S0920-9964(02)00289-X

context called for secondary meanings (e.g., ''pen'' as an enclosed fence). The locus and mechanisms that underlie schizophrenic associative disturbance have, however, remained elusive. It is unclear, for example, whether the schizophrenic associative disturbance reflects aberrant spread of activation within a presumed lexicon that is widely distributed across frontal and temporal lobes (Nestor et al., 1997, 1998; Caramazza, 1996). Nor has it been established as to whether the disease-related associative disturbance reflects neuronal loss or faulty modulation of preexisting synapses or connections (Nestor and O'Donnell, 1998).

Connectionist modeling provides a conceptual and empirical vehicle to examine and simulate the dynamics of schizophrenic associative memory, and by extension, associative disturbance. A special, intrinsically parallel architecture lends conceptual definition and neurological plausibility to these models. In their generic and simplest forms, these massively parallel models consist of a number of interconnected units or nodes, each characterized by an activation value. The connections between the units are weighted quantitatively and serve to govern the flow of activation from one unit to the other. These idealized neural elements help to form very ''broad abstractions of brain models'' of a relatively simple scale that are intended to simulate a very small set of functional characteristics and properties of biological networks (Protopapas, 1999). In reality, however, these generic connectionist or network models are best viewed not even as abstractions of brain models, but as mathematical descriptions of selected sets of dynamic cognitive processes with no clearly established link to underlying neural events and properties.

Such generic models can hypothetically simulate the spread of activation within associative memory, typically using nodes or sets of nodes to represent words and parametric connection weights to represent associative links among nodes. Words and their corresponding nodes are assumed to be organized into networks, which may differ in size, as in number of associates, and in the strength of connections linking these associates. Activation spreads as a function of both size and associative strength of a network, with maximum activation and, hence, better recall for words of small, highly connected networks (Nelson et al., 1993). Connectionist models thus allow for the novel examination of how size and connectivity of word networks govern activation spread in associative memory of schizophrenia. Moreover, these models can address the extent to which schizophrenic recall might be simulated by altering connection weights or by ''lesioning'' units. By the same token, these models all make provisions for the effect of ''noise'', typically defined by adding random error to the implementation program (Hoffman, 1987; Cohen and Servan-Schreiber, 1992; Cohen et al., 1990).

We now present a neural network simulation of a word recall experiment that compared patients with chronic schizophrenia and normal control subjects. In this experiment, subjects studied a list of 32 words, each rated on the dimensions of connectivity (associative strength) and network size (number of associates). The list contained equal proportions of four types of words: high connectivity-small network (HCSN); low connectivity-small network (LCSN); high connectivity-large network (HCLN); and low connectivity-large network (LCLN). Consistent with prior studies, healthy subjects showed a pattern of cued recall of HCSN>LCSN>HCLN>LCLN (Nelson et al., 1993). However, patients with schizophrenia showed a different pattern of recall of HCSN>HCLN>LCSN>LCLN (Nestor et al., 1998). The schizophrenic pattern suggested an abnormal connectivity effect as reflected by the significant statistical interaction of diagnosis and word connectivity, but not word network size. This pattern of word recall occurred against a backdrop of overall reduced recall in patients with schizophrenia as reflected by a statistically significant group effect.

These behavioral data therefore underscored two aspects of performance of associative memory in patients with schizophrenia. First is the overall reduction in word recall, which is hardly surprising and analogous to the near ubiquitous, disease-related generalized impairment seen across a variety of behavioral tasks. To address this robust but general feature of schizophrenic performance, the proposed model incorporates random noise to account for the overall reduced rate of word recall, regardless of connectivity and network size, demonstrated in our prior behavioral study of patients with schizophrenia. In other words, random noise represents a general feature of the proposed schizophrenic model of associative memory, designed to capture the robust empirical finding that