

A Neurocomputational Approach to Delusions

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Neuronal networks process information in parallel. The cortex can be viewed as a computational surface that creates and maintains dynamic maps of representations of important sensorimotor and higher-level aspects of the environment and the organism. Its functions can be modeled by a particular type of neural network, the self-organizing feature map. Most importantly, representations of information in the cortex and in these maps have been demonstrated to change dynamically according to the salience and frequency of the input. This feature is referred to as neuroplasticity.

HOW DO NEURONS produce and maintain delusions? Although it seems natural to ask this question—after all, we know that the neurons of the brain somehow must produce delusions—at the same time, it appears that neuronal activity and delusional judgment are too far apart to allow an answer. Delusions, according to common wisdom, are the product of “somehow dysfunctional” higher-order cognitive processes that are generated by the interaction of millions (or billions) of neurons, and hence are beyond the reach of neurophysiological explanations.

However, during the last decade we have seen enormous breakthroughs in our understanding of how assemblies of neurons produce cognitive functions. Not only can we watch single neurons and neuronal populations learn and perform cognitive tasks,^{1,2} but we also have the conceptual tools to understand neuronal activity on an unprecedentedly complex level. Neuroscientists have become “computational” in that they use mathematical tools to understand the flow of information in networks of neurons.³⁻⁷ The implications of these scientific accomplishments for the medical specialty that deals with disorders of the mind and brain, psychiatry, can hardly be overestimated.

The focus of this report are the neurocomputational mechanisms that produce and maintain

The fact that general operational characteristics of computational maps in the cortex can be fine-tuned according to specific processing needs is referred to as neuromodulation. Within this framework of cortical maps and their computational models, acute and chronic delusions are discussed in terms of neuromodulation and neuroplasticity. This neurocomputational approach provides new insights into the phenomena in question, is detailed enough to allow empirical testing, and has therapeutic implications.

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delusions. It will be argued that although many details are yet unresolved, the conceptual tools of cognitive neuroscience can be used to gain a deeper understanding of delusions, which is theoretically plausible and may already have practical implications. To do so, the principles of parallel distributed processing in neuronal networks are introduced, and a particular type of network, the so-called self-organizing feature map, is discussed in some detail. This provides a framework for the discussion of acute and chronic delusions in terms of neuromodulation and neuroplasticity. The neurocomputational approach provides new insights into the phenomena in question, is detailed enough to allow empirical testing, and has therapeutic implications.

COMPUTERS AND NEURONS

Neurons can be understood as switching elements that process information. Compared with silicon computer chips capable of performing

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0010-440X/95/3602-0008\$03.00/0*

millions of switchings per second, neurons are quite slow. Even fast ion-channel-coupled neuronal switching takes about 1 to 2 milliseconds. Thus, it is approximately 10,000 times slower than the switching taking place in the small personal computer on which this report is written. In addition to being slow, neurons are prone to malfunctioning.⁸ It has been estimated that the difference between neurons and computer chips in terms of proneness to malfunction is approximately 1 to more than 10^9 . (To function, an ordinary personal computer must not make a single mistake in several billion switchings.) In other words, with respect to accuracy, neurons fare even worse when compared with a silicon chip than with respect to speed.

Keeping this in mind, it is clear that the brain has to function very differently from a computer. We know that we can recognize a familiar face or distinguish a word from a meaningless string of characters within a few hundred milliseconds. A serial computer can be programmed to perform such tasks, but only by breaking the task down and iteratively calculating thousands of single steps. If the same algorithms were used by the brain, we could never do these tasks as quickly and flawlessly as we actually can with ease.

Within artificial intelligence, this became known as the “100-step program” constraint⁹: The algorithm that the brain uses has to be such that even complex tasks must not take more than roughly a hundred steps to be accomplished, i.e., they must involve a series of no more than approximately 100 switchings. The solution to this problem was long known in general terms: it has to be the sheer quantity of neurons that somehow makes up for their slowness and faultiness of operation. However, only during the last decade have the mathematical and biological principles of neuronal mass action been discovered. By now, we know—in principle—how thousands of neurons can act together and thereby perform tasks in only a few computational steps, although the same tasks would involve, if performed by a serial computer, thousands of steps. We can simulate networks of neurons in the computer and not only prove the existence of solutions to compu-

tational problems of biological systems,^{10,11} but also deduce hypotheses, test them, and thereby make genuine discoveries. Moreover, in this report the view is held that the theory of neuronal networks provides a new framework for the understanding of normal and pathological mental phenomena, which will have not only theoretical but also practical clinical consequences.

NEURONS AS COMPUTING DEVICES

Neurons receive up to 10,000 input signals and produce one output signal. The formal aspect of this process is depicted in Fig 1, in which for the sake of simplicity only two axonal inputs are displayed. The input signal can be represented by a number, which for a given moment in time is either 0 or 1, i.e., the respective axon is either firing or not. When a period of time is considered, the number of action potentials per amount of time can also be represented by any number. Since there are many input fibers, it is convenient to represent them mathematically by an indexed variable (“ x ” in Fig 1) such that input 1 is x_1 , input 2 is x_2 , etc.

We know that action potentials are transferred from one neuron to the next via synapses, i.e., gaps between neurons where the incoming action potential triggers the release of substances (neurotransmitters) that lead—in the case of fast ion-channel-coupled synapses—to the depolarization of the membrane of the postsynaptic neuron. However, depending on the amount of transmitter released and the number and sensitivity of postsynaptic receptors (as well as other mechanisms), the incoming signal can be transmitted strongly or weakly. The signal is modified by the “strength” or “weight” of the synapse, and this weight again can be mathematically formalized as a number. Like the numbers representing the inputs through all the incoming connecting fibers, the weights of all the respective synapses can be represented by an indexed variable. So we have input x_1 being transmitted through a synapse with weight w_1 , input x_2 being transmitted through a synapse with weight w_2 , etc. Then, the weighted input that a neuron receives through a single fiber—for example, fiber 9—can easily be

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