model could be the presentation of a gray box model (i.e., presenting a model that considers not only the input/output data but also other information about the system). The integrity and robustness of the model will be more reliable if more characteristics of the model are considered. It is obvious that we cannot introduce white box model for biological systems because of the complex system structure.

Because of the vast range of the recordable Parkinson's disease movement disorders, this disease has attracted scientists and has excited them for mathematical modeling.[1,2] Unfortunately, despite the presence of physiological information about Parkinson's disease, most of the models have studied the disease as a black box. Therefore, we are to present a model that simulates the symptoms based on physiological findings.

Analyzing the clinical recordings such as tremor, posture, and stride-time intervals has elucidated the chaotic nature of this signal. Therefore, a proper model should be able to simulate the chaotic behavior.[3,4] Modeling chaotic systems is difficult because of their high sensitivity. Owing to the variable nature of chaotic signals, it is not suitable to use signal time series for modeling. It is wise to get different signals in different recordings and then assess global features of these chaotic signals (such as Lyapunove exponent, fractal dimension, attractor properties, and poincare section).

Most available models of Parkinson's disease are limited to I/O data (black box). Now, we propose to consider physiological findings in order to have a gray box model. The main cause of the disease is the loss of dopaminergic neurons in basal ganglia. Hence, the healthy persons and patients differ only in basal ganglia structure. Some information is available about basal ganglia segments and the connections between them. We employ Chaotic Neural Network for each block to have the most similarity to neural structure of basal ganglia blocks. We propose using available information about basal ganglia to train Chaotic Neural Network weights.

It is believed that the basal ganglia plays its role in movement control by reinforcement learning. The dopamine neurons' activity encodes the reward prediction error.[5] In fact, healthy persons learn new patterns of movement based on trial and error (reinforcement learning). Therefore, we train our model using reinforcement learning methods. This training network is biologically plausible and helps us to understand the interaction between blocks. In Parkinson's disease, the reward prediction error would be distorted and the reinforcement learning is not done. In addition, the stochastic properties of neurotransmitter behavior are increased because of uptake, up-regulation, and distortion of neurotransmitter diffusion. This stochastic behavior is the main cause of the disease and can simulate disease symptoms. Using the proposed model of Parkinson's disease, early diagnosis and treatment of the disease may be possible.

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Brain-Derived Neurotrophic Factor Polymorphism: More Than a Prognostic Factor During Depression?

To the Editor: Brain-derived neurotrophic factor (BDNF) is a member of the nerve growth factor family, which is widely expressed in the adult mammalian brain. Besides its critical role in the development and maintenance of neuronal systems, it is also involved in neuronal survival, proliferation, plasticity, and neurogenesis.[1]

Studies indicating its role in adaptation to social stress and its association with chronic-stress-related atrophy of the limbic structures and major depressive disorder are rapidly replicating. However, considering that major depressive disorder is a heritable disorder, studies focusing on the reflection of BDNF gene functional polymorphism into the development and course of major depression are rapidly increasing.[2,3]

Can the BDNF gene be considered as one of the multiple susceptibility genes which may play a critical role in the development and/or course of depression?

Although there are inconsistent results indicating the importance of

met allele of the BDNF gene (BDNF polymorphism) on the vulnerability of depression, there is strong evidence regarding the effect of the BDNF polymorphism on cognitive performance, hippocampal volume, and the prefrontal cortex which have been implicated as important structures in mood regulation and memory.[4,5]

In our previous study evaluating the association of the BDNF gene val66met polymorphism with the serum BDNF levels in drug-free depressed patients, we showed that depressed patients had significantly lower mean values of serum BDNF relative to the comparison group ($p<0.05$). Beyond suggesting a significant negative correlation between the Hamilton Depression Rating Scale (HAM-D)-17 scores and serum BDNF levels in the study group, we also showed that depressed patients with met allele had significantly lower serum BDNF levels correlated with more severe depression scores ($p<0.05$).

Although we failed to support the BDNF polymorphism as a susceptibility factor in major depression, our results indicated a strong link between the BDNF polymorphism, the depression severity, and the serum BDNF levels in depressed patients, which suggests its role as a prognostic factor during depression. However, the dilemma of whether the BDNF polymorphism predisposes an individual to an earlier age of developing depression or only increases the risk in the context of other genetic risk factors for depression or other hippocampal impairment should be evaluated with further neuroimaging/genetic studies, including antidepressant treatment outcomes.

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Antipsychotic Induced Catatonia: A Case of Probable Dementia With Lewy Bodies

To the Editor: This report describes the case of a patient who presented with catatonia after rapid escalation of antipsychotic dose. The patient was eventually diagnosed with probable dementia with Lewy bodies (DLB).

Case Report

Mr. A was a 51-year-old man who presented with distressing visual hallucinations and paranoia. After outpatient psychiatric evaluation, risperidone, 0.5 mg twice daily, was changed to paliperidone, 6 mg once daily. After a single dose, he became confused and ataxic and was taken to the emergency room for evaluation. No medical causes for his symptoms were found, and he was admitted to an inpatient psychiatric unit.

His examination was notable for mutism, constricted affect, and lead-pipe rigidity of the upper extremities. His responses were limited to intermittent staring at examiners. When assisted to a standing position, he remained standing but would not move. Catatonia was diagnosed, and lorazepam, 1 mg twice a day, and aripiprazole, 5 mg once daily, were started. Paliperidone was discontinued.

The following day, his mutism persisted and he would not respond to multiple interview attempts. Although he made scant eye contact, he continued to be noninteractive. He required staff assistance with transferring from bed and continued to decline food intake. Vital signs were well within normal limits. Laboratory studies were normal. CT of the head revealed no parenchyma changes or other acute abnormalities. Aripiprazole was discontinued after a single dose due to concern for neuroleptic malignant syndrome (NMS), while lorazepam was increased to 2 mg three times a day. The patient became less responsive after a total of 8 mg of lorazepam, and he was transferred to the neurology service. Laboratory studies and CSF analysis were unremarkable. EEG showed diffuse slowing with no epileptiform discharges. Lorazepam was discontinued.

Over the subsequent 48 hours, the patient's level of consciousness improved to where he started to