EDITORIAL

Clinical, Experimental and Modeling Studies in AD/HD

This issue of Neural Plasticity covers advances in our understanding of the basic processes that underpin the psycho-patho-physiology of Attention Deficit Hyperactivity Disorder (AD/HD). The papers are divided into three sections: human studies with clinical populations, animal model studies, and mathematical modeling studies.

The clinical studies focus on the issue of neuropsychological and developmental heterogeneity in AD/HD. They attempt to identify some of the potentially multiple patho-physiological pathways to the disorder. The papers by Dalen and colleagues and Stevenson and colleagues provide evidence for the existence of at least two neuropsychological quite distinct routes to the expression of AD/HD symptoms, a motivational pathway associated with intolerance for delay and a cognitive pathway associated with executive dysfunction. The paper by Sonuga-Barke highlights the importance of taking account of the role of this heterogeneity in AD/HD when developing new and potentially more effective non-pharmacological therapeutic options. The paper by Auerbach provides preliminary evidence for the way that developmental trajectories toward AD/HD are likely to be shaped by the interactions of genetic and environmental factors operating in early environments. All these papers challenge the view that AD/HD is a single psycho-patho-physiological entity, understandable in terms of simple deficits. On the other hand, animal studies focus on the dopamine (DA) and norepinephrine (NE) systems, their interactions in the control of executive functions and sustained attention. They pertain to in vivo and in vitro observations, using techniques like behavior, pharmacology, molecular biology, morphometry, microdialysis, electrophysiology, and cell culture. Volpicelli et al. analyze the mechanisms involved in the acquisition of the dopamine phenotype, that is important for the developmental theories of AD/HD. Masuo et al. investigate motor activity, catecholamine content, gene expression alterations in the striatum and midbrain and TH1 immunoreactivity in rats made hyperactive by postnatal 6-OH DA and endocrine disruptors. Carboni et al. review in vivo and in vitro the dopamine system of animal models of AD/HD. Viggiano et al. review the effects of NET blockers and the NE-DA interaction in animal models of ADHD. These studies address the complexity of events that are involved in the expression of the dopamine phenotype, their susceptibility to environmental stressors, plasticity, and interactions in adult animals.

Finally, computational modeling attempts have been made based on the wealth of data emerging from clinical and experimental studies. For instance, Williams and Taylor explore AD/HD in terms of the temporal difference model that accounts for the prediction error by dopamine cell activity. Viggiano et al. review changes in the dopamine systems in hyperactive animals and model them by kinetic analysis. These attempts in modeling a human syndrome are highly promising for future development of this interdisciplinary approach.

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