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DO NEURONS HAVE MOOD AND HOW CAN IT BE STABILISED?

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Polarity of affective tone has taken the role of the core defining feature of bipolar affective disorder (BAD) eclipsing Falret's and Kraepelin's emphasis on cyclicity and recurrence. Yet in many clinical cases polarity is lost in the course of time while cyclic recurrence persists for decades, completely disrupting patients' lives. That's why true “mood stabilisation” aims not as much at reducing episode severity as at reducing episode number to a minimum.

On the neuronal level polarity could be envisaged as interplay between arousal and inhibition due to trans-membrane ion currents, either brief and adaptive or pathologically sustained. Cyclicity and recurrence could arise from permanent deficits in neuronal plasticity and resilience which are to a large extent under genetic control. The “mood stabilising” properties of different pharmacological agents are due to multi-level actions on the intracellular signal transduction pathways affecting the function of Gi and Go proteins, of some intracellular messengers (mioinositol, Bcl-2, ER stress proteins, MARKS, the complex calmodulin-synapsin1-synaptotagmin), and of some crucially important enzymes: adenilyl cyclase, various protein kinases (ά, ε, C, MAP, Akt), mitochondrial respiratory chain enzyme complexes, histone deacetylase and acetyltransferase, GSK-3. Thus, in the long run "mood-stabilising agents" exert blockade of excitotoxic/apoptotic pathways, phosphorilation of microtubular proteins, and regulation of the expression of genes implicated in processes involved in neuroplasticity, neuroprotection, even neurogenesis, and hamper the long-term neuronal (and by extension, mental) deterioration. Lithium displays the broadest spectrum of such actions and provides, by far, the best control over the chronic protean symptoms of BAD.

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