Abstract
There are several findings on the action of magnesium ions supporting their possible therapeutic potential in affective disorders. Examinations of the sleep-electroencephalogram (EEG) and of endocrine systems point to the involvement of the limbic–hypothalamus–pituitary–adrenocortical axis as magnesium affects all elements of this system. Magnesium has the property to suppress hippocampal kindling, to reduce the release of adrenocorticotropic hormone (ACTH) and to affect adrenocortical sensitivity to ACTH. The role of magnesium in the central nervous system could be mediated via the N-methyl-D-aspartate antagonistic, g-aminobutyric acidA-agonistic or a angiotensin II-antagonistic property of this ion. A direct impact of magnesium on the function of the transport protein p-glycoprotein at the level of the blood–brain barrier has also been demonstrated, possibly influencing the access of corticosteroids to the brain. Furthermore, magnesium dampens the calciumion–proteinkinase C related neurotransmission and stimulates the Na–K-ATPase. All these systems have been reported to be involved in the pathophysiology of depression. Despite the antagonism of lithium to magnesium in some cell-based experimental systems, similarities exist on the functional level, i.e. with respect to kindling, sleep-EEG and endocrine effects. Controlled clinical trials examining the effect of Mg in affective disorder are warranted.