

Depression 2019: E-BABE: The hidden relation, clues of Autism, ADHD and Depression which reveals the effective cause and cure - Pharmacist, Vietnam

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Introduction: Antipsychotic drugs are prevalent for the treatment of psychological disorder like schizophrenia. Schizophrenia is a chronic, severe, and hampered brain disorder that has affected people throughout history. It affects how a person thinks, feels and acts and may have difficulty distinguishing between what is real and what is imaginary. The cause of schizophrenia still remains unclear though some researches stated that this disease may be triggered by genetical problem running in families and biological factors like the imbalance in the brain's chemistry, viral infections and immune disorders. Is there any relation among autism, ADHD, poor learning, stress, depression & mental health? Some by genes, some are not by genes but they are comorbid! What if we can have the diagnostic since the kids 2,3,4,5,6,7, or 8 months old? What if we can have the diagnostic long before they can officially be diagnosed? then what these kids will be if we can give them effective intervention since that time 24/7 for kids with love, compassion, and patience? What if autism is just viewed as having substantially poor social skills? From the definition, will kids recover from autism if they have full communication skills, emotional skills, and social skills? The big question for autism is: which cause make kids poor at these skills? This cause is also the cause of autism. And in the changing world, all are changing with different speed, genes, and brain change also. The changing of the brain described by NEUROPLASTICITY.

Objectives: To assess clinically relevant symptom improvement in patients with major depressive disorder (MDD) receiving vilazodone by using the Montgomery-Aberg Depression Rating Scale (MADRS), a clinician-rated scale used to measure MDD symptom severity and improvement. Method: Pooled data from 2 positive, phase 3, 8-week, double-blind, randomized, placebo-controlled trials in patients with MDD were analyzed. Patients received vilazodone 40 mg/d or placebo; post hoc analyses were conducted on study completers. Depression symptom improvement was evaluated by analyzing the proportions of patients who shifted from the baseline MADRS single-item symptom severity category. The neuroanatomy of panic disorder largely overlays with that of most anxiety disorders. Neuropsychological, neurosurgical, and neuroimaging studies associate the insula, amygdala, hippocampus, anterior cingulate cortex (ACC), lateral prefrontal cortex, and periaqueductal grey. During serious panic attacks, viewing expressively exciting words, and rest, most studies find elevated blood flow or metabolism. However, the remark of amygdala hyperactivity is not totally consistent, especially in studies that evoke panic attacks chemically. Hippocampus hyperactivity has been observed during rest and watching emotionally charged pictures, which has been conjectured to be related to recall retrieval bias near anxious recollections. If the defect in genes and brain is by nature, scientists can test and detect it to predict autism since the time of pregnancy, infant, ...but the facts is no, they cannot see any warning until the behaviors, poor skills are obvious! This makes me think problems in genes and brain are just the symptoms in the rainbow of autism. The primary objective of

this article is to provide a concise review of the clinical relevance of sleep and vigilance in major depressive disorder. Data Sources: PubMed was reviewed and English-language articles were identified using the key words sleep and depression and sleep and antidepressants. Secondary searches included articles cited in sources identified by the primary search. Study Selection: The narrative review provides brief descriptions of the normal physiology of sleep and changes associated with depression, as well as the impact of various treatments on these processes. Data Synthesis: Although it has long been known that sleep disturbances are an important characteristic of depression, relatively few studies have been conducted with the newer generation antidepressants. Neither of the most widely used classes of antidepressants, the selective serotonin reuptake inhibitors and the serotonin-norepinephrine reuptake inhibitors, have particularly beneficial effects on sleep and, among the medications that reliably improve sleep efficiency, including mirtazapine and the tricyclic antidepressants, problems with daytime sedation can offset therapeutic benefit. Despite relatively widespread use, trazodone has not been demonstrated to be an effective and safe hypnotic in patients taking other antidepressants. For many patients, ongoing concomitant treatment with benzodiazepines and related drugs is the preferred option, again without convincing empirical support of longer-term efficacy. Among newer and investigational antidepressants, agomelatine shows promise with respect to both overall safety and effects on insomnia, although possible negative effects on liver function warrant further study. Conclusions: Sleep disturbances are a significant aspect of depressive syndromes, and relief of insomnia remains an important unmet need in antidepressant therapeutics. Development of a well-tolerated antidepressant medication that rapidly.

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