

Editorial

Electroconvulsive Therapy and Heat Shock Gene Inactivation in Neurodegenerative Diseases

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EDITORIAL

The global diabetes epidemic [1] is associated with accelerated aging and relevant to neurodegenerative diseases such as multiple sclerosis, Parkinson's disease and Alzheimer's disease. The major mental illnesses in diabetics are schizophrenia, bipolar disorders and depression. These mental illnesses lead to changes in mood, memory, cognition and behaviour in these individuals with potential screening plasma tests for early detection of these conditions. These mental disorders may be related to sleep disorders with relevance to alterations in the sleep/wake cycle [2,3]. In diabetes and neurodegenerative diseases the major defect in the brain is related to the suprachiasmatic nucleus (SCN) and its dysregulation is connected to core body temperature regulation, non alcoholic fatty liver disease and metabolic disease [4,5]. Electroconvulsive therapy (ECT) is performed in patients with diabetes mellitus and psychiatric disorders such as severe depression and relevant to the use of anti-psychotic/antidepressant drugs [6,7]. The use of electroconvulsive therapy (Figure 1) is an established means to improve function in a variety of psychiatric and neurologic conditions such as multiple sclerosis, Parkinson's disease and Alzheimer's disease [8-11]. The therapeutic benefit of ECT has been indicated in various studies and in Alzheimer's disease its use in particular is involved in the prevention uncontrolled agitation and aggression [10,11]. ECT studies indicate brain temperature increases [12] with relevance to electroconvulsive therapy stimulation [13,14]. Concerns over heat stress relevant to ECT associated memory loss/brain damage [15,16] may be relevant to irreversible inactivation of the genes and brain heat shock gene Sirtuin 1 (Sirt 1) expression [17-19]. Sirt 1 is a NAD(+) dependent class III histone deacetylase (HDAC) protein that targets transcription factors to adapt gene expression to metabolic activity, insulin resistance and inflammation [4,5]. Sirt 1 is involved in neurogenesis, gluconeogenesis in the liver, cholesterol metabolism, mitochondrial biogenesis and adipocyte senescence/energy metabolism. Sirt 1 is a heat shock gene [20,21] that is responsible for the circadian regulation of heat shock proteins (HSP) with HSP associated with amyloid beta

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aggregation critical to the survival of neurons and peripheral cells [21,22]. Sirt 1 is important to prevent mitochondrial apoptosis and epilepsy induced stroke [23].

Sirt 1 is sensitive to temperature changes with elevated brain temperature associated with core body temperature disturbances [5]. Sirt 1 and the SCN [24] are closely connected with physiological temperatures relevant to central circadian control in the SCN [25] and related to mental illness in diabetes and neurodegenerative diseases [4]. SCN can synchronize circadian rhythms in other brain regions and peripheral tissues but ECT stimulation (dose/frequency) should be carefully controlled [26,27] to avoid heat stress and complete SCN disruption (Figure 1) associated with heat therapy [28] in various neurologic conditions such as diabetes, multiple sclerosis, Parkinson's disease and Alzheimer's disease. Food quality can inactivate Sirt 1 with nutrigenomic diets essential for the maintenance and treatment of diabetes and neurologic conditions [29]. In the developing world diets that contain bacterial lipopolysaccharides (LPS), mycotoxin and xenobiotics repress Sirt 1. In the developed world unhealthy diets (without Sirt 1 activators) that contain excess calories inactivate Sirt 1 and the SCN with relevance to mental illnesses in diabetes and neurodegenerative diseases. The therapeutic benefit of ECT will depend on patients that are from the developing or developed world with relevance to diet, core body temperature disorders and synaptic plasticity defects [30,31]. ECT treatments in diabetic and psychiatric patients that disturb Sirt 1 regulation of the SCN (Figure 1) should involve blood tests that include plasma Sirt 1 regulated proteins and hormones such as HSP 70, melatonin, brain derived neurotrophic factor, neuropeptide Y, fibroblast growth factor and adiponectin [5,32-40].

CONCLUSION

ECT as an established technique and technology in a variety of psychiatric and neurologic conditions may require reassessment with relevance to memory loss and brain damage. The therapeutic benefit of ECT will depend on heat stress induction and inactivation of the heat shock gene Sirt 1 with

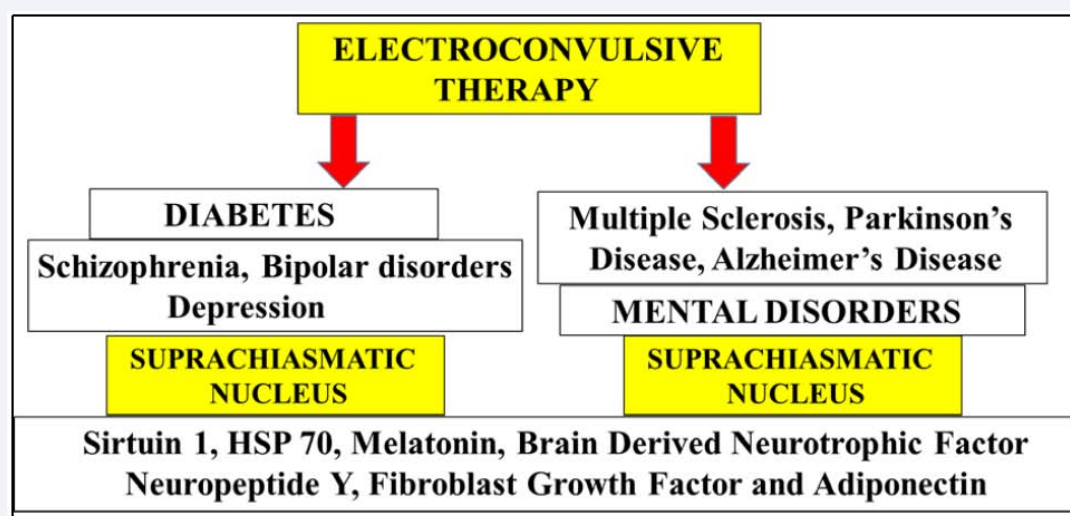


Figure 1 Electroconvulsive therapy stimulation (dose/frequency) should avoid heat stress and complete inactivation of the heat shock gene Sirtuin 1 and the suprachiasmatic nucleus relevant to mental disorders in diabetes and neurodegenerative diseases. Electroconvulsive therapy require assessment for SCN function with relevance to neuron death (mitochondrial apoptosis). ECT therapeutic technology should involve screening tests for Sirtuin 1 regulated proteins and hormones such as HSP 70, melatonin, brain derived neurotrophic factor, neuropeptide Y, fibroblast growth factor and adiponectin.

relevance to mitochondrial apoptosis and neuron death. ECT and neurostimulation in diabetes and neurodegenerative diseases should allow intact SCN function and activation to avoid sleep/wake disturbances, heat shock response dysregulation and induction of circadian abnormalities. ECT technology should be used with caution (dose/frequency) in psychiatric individuals with synaptic plasticity defects with relevance to unhealthy diets and core body temperature dysregulation. ECT application may now involve plasma tests for various Sirt 1 regulated protein hormones with relevance to SCN function and circadian signals.

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