

Apelinergic System Defects with Relevance to Mental Disorders in Diabetes

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Editorial

Mental illness in Western communities has increased with the global depression crisis a major disorder in managing psychiatric disturbances. The association between diabetes and depression indicate that psychiatric disturbances such as schizophrenia and bipolar disorders are far greater in diabetic individuals than individuals in the general population. Stress and anxiety are closely associated with mental illness, behaviour and cognition disorders and these psychiatric disorders may be involved in the induction of neurodegeneration, organ disease and diabetes [1].

Interests in diet, lifestyle, stress and sleep factors have been the focus of many communities as factors that influence specific genes involved in the onset of depression and neurodegeneration and may be the primary cause of psychiatric disturbances in diabetic individuals. Epigenetic alterations in diabetes [2-5] induced by the environment [6] have become important to diabetes with post-transcriptional changes linked to various disease processes in diabetes. Diabetics have appetite dysregulation and therole of genes such as the anti-aging gene Sirtuin 1 (Sirt 1) regulated by magnesium [7] and unhealthy diets [8] has escalated that links depression and appetite control to be of major importance in the treatment of various psychiatric disturbances. Sirt 1 is connected to food intake, depression, schizophrenia, cognition, memory and learning [9-15] with research directed to maintain brain Sirt 1 activity such as magnesium therapy [7] to prevent neurodegeneration in diabetes (Figure 1).

Recent interests in Type 3 diabetes [16,17] implicate Sirt 1 as the defective gene with relevance to appetite control and chronic disease. The hypothalamic-pituitary axis implicates the peptide apelinto be involved in the neuroendocrine system with central co-ordination between the brain and peripheral tissues (intestine, liver, kidney, heart). The interference of apelin by stress related disorders generates angiotensin II and inactivates nitric oxide (NO) metabolism with interference of Sirt 1 regulation of Nitric Oxide (NO) balance [18] relevant to appetite control, stroke and dementia [19] (Figure 1). Apelinergic system and appetite control are now important to diabetic treatment with close connection of the defective apelinergic system involved in brain NO production with relevance to NO induced post-transcriptional alterations [20]. Inactivation of NO metabolism (Figure 1) interferes with Sirt 1 role in epigenetic changes [6] associated with the induction of circadian rhythm abnormalities, mental illness, endocrine disease and appetite dysregulation [21-25].

Magnesium is involved in the release of NO [26,27] with magnesium deficiency connected to neurodegeneration, mental illness and cardiovascular disease [7]. NO is an intercellular messenger involved in signal transduction [28] in the brain and is intimately involved in synaptic plasticity with relevance to programmed cell death. Its role as a hormone [29] has become important and is closely associated with the neuroendocrine system [30-32]. The nature of low calorie diets that regulate Sirt 1 indicate its critical role in brain NO homeostasis important to hypothalamic function with relevance to appetite regulation and circadian rhythm disorders [22,23,25] in diabetes and various chronic diseases. Nutrigenomic diets [8] and the prevention of stress maintain Sirt 1 and brain NO balance that activate the neuroendocrine system [18] linked to appetite control and psychiatric conditions in diabetes and associated organ diseases. Drug therapy is essential for the maintenance of NO metabolism [33,34] in mental health disorders and an intact apelinergic system [19] is required to prevent psychiatric disturbances and peripheral organ disease. The use of nutrition and drug therapy is now connected to magnesium with magnesium signaling important in the prevention of NO toxicity and programmed cell death. The effects of magnesium therapy [35,36]

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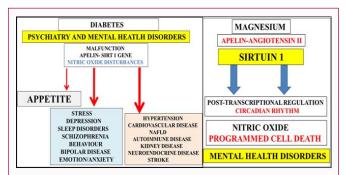


Figure 1: Psychiatric disturbances and neurodegeneration is associated with mental illness, behavioral and cognition disorders in diabetes. The Sirt 1 gene and the peptide apelin is important to nitric oxide homeostasis and post-transcriptional induction of various organ diseases. Angiotensin II generated from apelin interferes with Sirt 1 regulation of epigenetic alterations and NO balance with relevance to mental illness and organ disease. Magnesium interacts with Sirt 1 and its involvement with NO stabilizes psychiatric disturbances that are connected to mental health disorders.

in various communities involve an intact apelingergic pathway that is central to mental health disorders and organ disease. Communities with excessive dietary NO, dietary bacterial lipopolysaccharide and xenobiotic contents may override the beneficial magnesium/NO homeostasis. In these developing communities inactivation of drug therapy for brain NO homeostasis by these lipophilic components may be relevant to competition with lipophilic NO diffusion within/between cells [37] with inactivation of magnesium therapy in neuroendocrine disease, appetite dysregulation and depression in various communities.

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References

- Martins IJ. Diabetes and Organ Dysfunction in the Developing and Developed. GJMR. 2015;15(1):15-21.
- 2. Al-Haddad R, Karnib N, Assaad RA, Bilen Y, Emmanuel N, Ghanem A, et al. Epigenetic changes in diabetes. Neurosci Lett. 2016;625:64-9.
- Villeneuve LM, Reddy MA, Natarajan R. Epigenetics: deciphering its role in diabetes and its chronic complications. Clin Exp Pharmacol Physiol. 2011;38(7):451-9.
- 4. Albert PR. Epigenetics in mental illness: hope or hype? J Psychiatry Neurosci. 2010;35(6):366-8.
- 5. Isles AR. Neural and behavioral epigenetics; what it is, and what is hype. Genes Brain Behav. 2015;14(1):64-72.
- Martins IJ. Increased Risk for Obesity and Diabetes with Neurodegeneration in Developing Countries. J Mol Genet Med. 2013;1-8.
- Martins IJ. Magnesium Therapy Prevents Senescence with the Reversal of Diabetes and Alzheimer's disease. Health. 2016;8:694-710.
- 8. Martins IJ. Unhealthy Nutrigenomic Diets Accelerate NAFLD and Adiposity in Global communities. J Mol Genet Med; 2015; 9(1):1-11.
- 9. Abe-Higuchi N, Uchida S, Yamagata H, Higuchi F, Hobara T, Hara K, et al. Hippocampal Sirtuin 1 Signaling Mediates Depression-like Behavior. Biol Psychiatry. 2016;80(11):815-26.
- Kim HD, Hesterman J, Call T, Magazu S, Keeley E, Armenta K, et al. SIRT1 Mediates Depression-Like Behaviors in the Nucleus Accumbens. J Neurosci. 2016;36(32):8441-52.

- 11. Ng F, Wijaya L, Tang BL. SIRT1 in the brain-connections with aging-associated disorders and lifespan. Front Cell Neurosci. 2015;9:64.
- Michán S, Li Y, Chou MM, Parrella E, Ge H, Long JM, et al. SIRT1 is essential for normal cognitive function and synaptic plasticity. J Neurosci. 2010;30(29):9695-707.
- 13. Drew LJ, Hen R. Food for thought: linking caloric intake to behavior via sirtuin activity. Cell. 2011;147(7):1436-7.
- 14. Kishi T, Fukuo Y, Kitajima T, Okochi T, Yamanouchi Y, Kinoshita Y, et al. SIRT1 gene, schizophrenia and bipolar disorder in the Japanese population: an association study. Genes Brain Behav. 2011;10(3):257-63.
- 15. Wang Y, Huang Y, Peng M, Cong Z, Li X, Lin A, et al. Association between Silent Information Regulator 1 (SIRT1) gene polymorphisms and schizophrenia in a Chinese Han population. Psychiatry Res. 2015;225(3):744-5.
- 16. Martins IJ. Diet and Nutrition reverse Type 3 Diabetes and Accelerated Aging linked to Global chronic diseases. J Diab Res Ther. 2016;2(2):1-6.
- 17. Martins IJ. Type 3 diabetes with links to NAFLD and Other Chronic Diseases in the Western World. Int J Diabetes. 2016;1(1):1-5.
- 18. Martins IJ. Nutritional diets accelerate amyloid beta metabolism and prevent the induction of chronic diseases and Alzheimer's disease. Photon eBooks.
- Martins IJ. The Global Obesity Epidemic is Related to Stroke, Dementia and Alzheimer's disease. ISM Alzheimers Dis Relat Dement. 2014;1(2):1-9.
- 20. Vasudevan D, Bovee RC, Thomas DD. Nitric oxide, the new architect of epigenetic landscapes. Nitric Oxide. 2016;59:54-62.
- 21. Peterson LG, O'Shanick GJ. Psychiatric symptoms in endocrine diseases. Keys to identifying the underlying disorder. Postgrad Med. 1985;77(5):233-9
- Golombek DA, Agostino PV, Plano SA, Ferreyra GA. Signaling in the mammalian circadian clock: the NO/cGMP pathway. Neurochem Int. 2004;45(6):929-36.
- 23. Kojima S, Shingle DL, Green CB. Post-transcriptional control of circadian rhythms. J Cell Sci. 2011;124:311-20.
- Nasyrova RF, Ivashchenko DV, Ivanov MV, Neznanov NG. Role of nitric oxide and related molecules in schizophrenia pathogenesis: biochemical, genetic and clinical aspects. Front Physiol. 2015;6:139.
- Morley JE, Farr SA, Sell RL, Hileman SM, Banks WA. Nitric oxide is a central component in neuropeptide regulation of appetite. Peptides. 2011;32(4):776-80.
- 26. Teragawa H, Kato M, Yamagata T, Matsuura H, Kajiyama G. Magnesium causes nitric oxide independent coronary artery vasodilation in humans. Heart. 2001;86(2):212-6.
- Howard AB, Alexander RW, Taylor WR. Effects of magnesium on nitric oxide synthase activity in endothelial cells. Am J Physiol. 1995;269:C612-8.
- 28. Ignarro LJ. Nitric oxide. A novel signal transduction mechanism for transcellular communication. Hypertension. 1990;16(5):477-83.
- Ghasemi A, Zahediasl S. Is nitric oxide a hormone? Iran Biomed J. 2011;15(3):59-65.
- Peterson LG, O'Shanick GJ. Psychiatric symptoms in endocrine diseases.
 Keys to identifying the underlying disorder. Postgrad Med. 1985;77(5):233-9
- 31. Altemus M. Hormone-specific psychiatric disorders: do they exist? Arch Womens Ment Health. 2010;13(1):25-6.
- Dawson TM, Dawson VL. Nitric oxide synthase: role as a transmitter/ mediator in the brain and endocrine system. Annu Rev Med. 1996;47:219-27

- 33. Mason RP, Cockcroft JR. Targeting nitric oxide with drug therapy. J Clin Hypertens (Greenwich). 2006;8(12):40-52.
- 34. Burgaud JL, Ongini E, Del Soldato P. Nitric oxide-releasing drugs: a novel class of effective and safe therapeutic agents. Ann N Y Acad Sci. 2002;962:360-71.
- 35. Eby GA, Eby KL. Rapid recovery from major depression using magnesium treatment. Med Hypotheses. 2006;67(2):362-70.
- 36. Serefko A, Szopa A, WlaŰ P, Nowak G, RadziwoÅ-Zaleska M, Skalski M, et al. Magnesium in depression. Pharmacol Rep. 2013;65(3):547-54.
- Figueroa XF, Lillo MA, Gaete PS, Riquelme MA, Sáez JC. Diffusion of nitric oxide across cell membranes of the vascular wall requires specific connexin-based channels. Neuropharmacology. 2013;75:471-8.