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Manuscript Title: Genetic effect of Type 2 Diabetes to the progression of Neurological diseases

Md Habibur Rahman[1,2,3], Silong Peng[1,2], Chen Chen[1], Pietro Lio'[4], Mohammad Ali Moni[5]

[1]Institute of Automation, Chinese Academy of Sciences, Beijing 100190, China.
[2]University of Chinese Academy of Sciences, Beijing 100190, China.
[3]Dept. of Computer Science and Engineering, Islamic University, Kushtia-7003, Bangladesh.
[4]Computer Laboratory, The University of Cambridge, UK.
[5]Faculty of Medicine and Health, The University of Sydney, Australia.

Corresponding Author:
Mohammad Ali Moni
Faculty of Medicine and Health, The University of Sydney, Australia.
Email address: mohammad.moni@sydney.edu.au

Abstract

Neurological diseases (NDs) are progressive disorder often advances with age and comorbidities of Type 2 diabetes (T2D). Epidemiological, clinical and neuropathological evidence advocate that patients with T2D are at an increased risk of getting NDs. However, it is very little known how T2D affects the risk and severity of NDs.

To tackle these problems, we employed a transcriptional analysis of affected tissues using agnostic approaches to identify overlapping cellular functions. In this study, we examined gene expression microarray human datasets along with control and disease-affected individuals. Differentially expressed genes (DEG) were identified for both T2D and NDs that includes Alzheimer Disease (AD), Parkinson Disease (PD), Amyotrophic Lateral Sclerosis (ALS), Epilepsy Disease (ED), Huntington Disease (HD), Cerebral Palsy (CP) and Multiple Sclerosis Disease (MSD).

We have developed genetic association and diseasome network of T2D and NDs based on the neighborhood-based benchmarking and multilayer network topology approaches. Overlapping DEG sets go through protein-protein interaction and gene enrichment using pathway analysis and gene ontology methods, identifying numerous candidate common genes and pathways.

Gene expression analysis platforms have been extensively used to investigate altered pathways and to identify potential biomarkers and drug targets. Finally, we validated our

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identified biomarkers using the gold benchmark datasets which identified corresponding relations of T2D and NDs. Therapeutic targets aimed at attenuating identified altered pathway could ameliorate neurological dysfunction in a T2D patient.