

188N: 2639-0108

Research Article

Relationships of Diabetic Neuropathy Risks and Three Glycemic Intensities of Type 2 Diabetes Using Viscoplastic Energy Model of GH-Method: Math Physical Medicine (no. 988, vmt #387, 12/4/2023)

Gerald C Hsu*

EclaireMD Foundation, USA

*Corresponding Author

Gerald C Hsu, EclaireMD Foundation, USA

Advances in Theoretical & Computational Physics

Submitted: 2023, Dec 04; Accepted: 2024, Jan 08; Published: 2024, Jan 24

Citation: Hsu, G. C. (2024). Relationships of Diabetic Neuropathy Risks and Three Glycemic Intensities of Type 2 Diabetes Using Viscoplastic Energy Model of GH-Method: Math Physical Medicine (no. 988, vmt #387, 12/4/2023). *Adv Theo Comp Phy*, 7(1), 01-06.

Abstract

Based on PubMed research, it is estimated that approximately 50% of individuals with type 1 or type 2 diabetes experience diabetic neuropathy, while another article reports a prevalence of 40.8% for this condition in diabetes patients. From a pathophysiological standpoint, diabetic neuropathy is strongly connected to metabolic disorders, particularly type 2 diabetes.

The American Diabetes Association classifies glucose levels as follows:

- Hyperglycemia (glucose above 180 mg/dL)
- Hypoglycemia (glucose below 70 mg/dL)
- Normal Glycemic (glucose between 70 and 180 mg/dL)

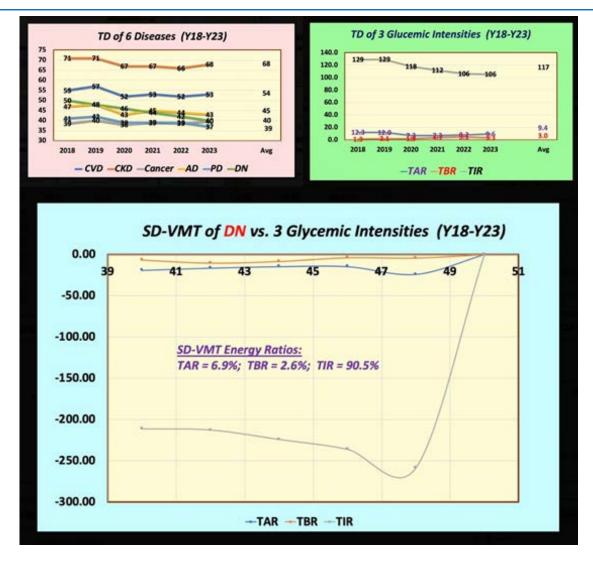
This study delves into the author's risk of developing diabetic neuropathy (DN) in relation to their type 2 diabetes. The author introduces a novel concept known as "glycemic intensities (GI)" as new biomarkers to examine various diabetic complications. These GI values are calculated as the product of average glucose levels and their frequency of occurrence, potentially shedding light on the impact of diabetes control and glucose management on the development of various health issues, including DN. In this research, the author assesses hid DN risk using three T2D-GI measures: TAR-GI (TAR), TBR-GI (TBR), and TIR-GI (TIR), utilizing his personal data collected from August 1, 2018, to December 2, 2023.

In summary, the author utilizes the space-domain viscoplastic energy (SD-VMT) method to explore the underlying connections and dynamics (i.e. energies) between three diabetic glycemic intensity (GI) inputs and the annual diabetic neuropathy (DN) risk output:

- Energy from Time Above Range (TAR): 6.9%
- Energy from Time Below Range (TBR): 2.6%
- Energy from Time in Range (TIR): 90.5%

Key Message

It is expected that the predominant contribution of TIR energy to his Parkinson's risk is significant. Notably, the finding indicating that the TAR-GI contribution (6.9%) to various cancers risk is nearly 2.7 times higher than that of TBR-GI (2.6%) is of particular importance. The intensity of hyperglycemia can negatively impact internal organs, while the intensity of hypoglycemia can potentially lead to sudden death due to insulin shock.



1. Introduction

Based on PubMed research, it is estimated that approximately 50% of individuals with type 1 or type 2 diabetes experience diabetic neuropathy, while another article reports a prevalence of 40.8% for this condition in diabetes patients.

From a pathophysiological standpoint, diabetic neuropathy is strongly connected to metabolic disorders, particularly type 2 diabetes.

The American Diabetes Association classifies glucose levels as follows:

- Hyperglycemia (glucose above 180 mg/dL)
- Hypoglycemia (glucose below 70 mg/dL)
- Normal Glycemic (glucose between 70 and 180 mg/dL)

This study delves into the author's risk of developing diabetic neuropathy (DN) in relation to their type 2 diabetes. The author introduces a novel concept known as "glycemic intensities (GI)" as new biomarkers to examine various diabetic complications. These GI values are calculated as the product of average glucose levels and their frequency of occurrence, potentially shedding light on the impact of diabetes control and glucose management on the development of various health issues, including DN.

In this research, the author assesses hid DN risk using three T2D-GI measures: TAR-GI (TAR), TBR-GI (TBR), and TIR-GI (TIR), utilizing his personal data collected from August 1, 2018, to December 2, 2023.

2. Biomedical Information

The following sections contain excerpts and concise information drawn from multiple medical articles, which have been meticulously reviewed by the author of this paper. The author has adopted this approach as an alternative to including a conventional reference list at the end of this document, with the intention of optimizing his valuable research time. It is essential to clarify that these sections do not constitute part of the author's original contribution but have been included to aid the author in his future reviews and offer valuable insights to other readers with an interest in these subjects.

2.1. Pathophysiological Explanations of Relationships Between Diabetic Neuropathy and Three Diabetes Biomarkers, tar, tbt, tir:

Diabetic neuropathy is a common complication of diabetes, and its relationship with diabetes biomarkers can be complex. Here is a brief overview:

• TAR (Time Above Range): Elevated TAR levels (above 180 mg/dL) in diabetes may contribute to nerve damage seen in

diabetic neuropathy. Higher blood glucose levels can lead to oxidative stress and inflammation, further damaging nerves over time.

- TBR (Time Below Range): Prolonged periods of blood glucose levels below the target range (below 70 mg/dL) can also be very harmful. *Hypoglycemia, especially severe episodes, can damage nerves and exacerbate neuropathy symptoms.*
- TIR (Time in Range): Maintaining blood glucose levels within the recommended range (between 70 and 180 mg/ dL) is crucial in preventing diabetic neuropathy. Consistently high TIR helps reduce the risk of nerve damage by minimizing exposure to high glucose levels.

In summary, diabetic neuropathy is closely linked to glucose control. Elevated TAR and prolonged TBR can contribute to nerve damage, while optimizing TIR is essential to reduce the risk and progression of neuropathy in diabetes. Proper glycemic management through medication, diet, exercise, and lifestyle changes is key in preventing or managing this complication.

3. MPM Background

To learn more about his developed GH-Method: math-physical medicine (MPM) methodology, readers can read the following three papers selected from his published 760+ papers. The first paper, No. 386 (Reference 1) describes his MPM methodology in a general conceptual format. The second paper, No. 387 (Reference 2) outlines the history of his personalized diabetes research, various application tools, and the differences between biochemical medicine (BCM) approach versus the MPM approach. The third paper, No.

397 (Reference 3) depicts a general flow diagram containing \sim 10 key MPM research methods and different tools.

4. The Author's Diabetes History

The author was a severe T2D patient since 1995. He weighed 220 lb. (100 kg) at that time. By 2010, he still weighed 198 lb. with an average daily glucose of 250 mg/dL (HbA1C at 10%). During that year, his triglycerides reached 1161 (high risk for CVD and stroke) and his albumin-creatinine ratio (ACR) at 116 (high risk for chronic kidney disease). He also suffered from five cardiac episodes within a decade. In 2010, three independent physicians warned him regarding the need for kidney dialysis treatment and the future high risk of dying from his severe diabetic complications.

In 2010, he decided to self-study endocrinology with an emphasis on diabetes and food nutrition. He spent the entire year of 2014 to develop a metabolism index (MI) mathematical model. During 2015 and 2016, he developed four mathematical prediction models related to diabetes conditions: weight, PPG, fasting plasma glucose (FPG), and HbA1C (A1C). Through using his developed mathematical metabolism index (MI) model and the other four glucose prediction tools, by the end of 2016, his weight was reduced from 220 lbs. (100 kg) to 176 lbs. (89 kg), waistline from 44 inches (112 cm) to 33 inches (84 cm), average fingerpiercing glucose from 250 mg/dL to 120 mg/dL, and A1C from 10% to ~6.5%. One of his major accomplishments is that he no longer takes any diabetes-related medications since 12/8/2015.

In 2017, he achieved excellent results on all fronts, especially his glucose control. However, during the preCOVID period, including both 2018 and 2019, he traveled to ~50 international cities to attend 65+ medical conferences and made ~120 oral presentations. This hectic schedule inflicted damage to his diabetes control caused by stress, dining out frequently, postmeal exercise disruption, and jet lag, along with the overall negative metabolic impact from the irregular life patterns; therefore, his glucose control was somewhat affected during the two-year traveling period of 2018-2019.

He started his COVID-19 selfquarantined life on 1/19/2020. By 10/16/2022, his weight was further reduced to ~164 lbs. (BMI 24.22) and his A1C was at 6.0% without any medication intervention or insulin injection. In fact, with the special COVID-19 quarantine lifestyle since early 2020, not only has he written and published ~500 new research articles in various medical and engineering journals, but he has also achieved his best health conditions for the past 27 years. These achievements have resulted from his non-traveling, low-stress, and regular daily life routines. Of course, his indepth knowledge of chronic diseases, sufficient practical lifestyle management experiences, and his own developed high-tech tools have also contributed to his excellent health improvements.

On 5/5/2018, he applied a continuous glucose monitoring (CGM) sensor device on his upper arm and checks his glucose measurements every 5 minutes for a total of 288 times each day. Furthermore, he extracted the 5minute intervals from every 15minute interval for a total of 96 glucose data each day stored in his computer software.

Through the author's medical research work over 40,000 hours and read over 4,000 published medical papers online in the past 13 years, he discovered and became convinced that good life habits of not smoking, moderate or no alcohol intake, avoiding illicit drugs; along with eating the right food with wellbalanced nutrition, persistent exercise, having a sufficient and good quality of sleep, reducing all kinds of unnecessary stress, maintaining a regular daily life routine contribute to the risk reduction of having many diseases, including CVD, stroke, kidney problems, micro blood vessels issues, peripheral nervous system problems, and even cancers and dementia. In addition, a long-term healthy lifestyle can even "repair" some damaged internal organs, with different required time-length depending on

the particular organ's cell lifespan. For example, he has "self-repaired" about 35% of his damaged pancreatic beta cells during the past 10 years.

5. Energy theory:

The human body and organs have around 37 trillion live cells which are composed of different organic cells that require energy infusion from glucose carried by red blood cells; and energy consumption from laborwork or exercise. When the residual energy (resulting from the plastic glucose scenario) is stored inside our bodies, it will cause different degrees of damage or influence to many of our internal organs.

According to physics, energies associated with the glucose waves are proportional to the square of the glucose amplitude. The residual energies from elevated glucoses are circulating inside the body via blood vessels which then impact all of the internal organs to cause different degrees of damage or influence, e.g. diabetic complications. Elevated glucose (hyperglycemia) causes damage to the structural integrity of blood vessels. When it combines with both hypertension (rupture of arteries) and hyperlipidemia (blockage of arteries), CVD or Stroke happens. Similarly, many other deadly diseases could result from these excessive energies which would finally shorten our lifespan. For an example, the combination of hyperglycemia and hypertension would cause micro-blood vessel's leakage in kidney systems which is one of the major cause of CKD.

The author then applied Fast Fourier Transform (FFT) operations to convert the input wave from a time domain into a frequency domain. The y-axis amplitude values in the frequency domain indicate the proportional energy levels associated with each different frequency component of input occurrence. Both output symptom value (i.e. strain amplitude in the time domain) and output symptom fluctuation rate (i.e. the strain rate and strain frequency) are influencing the energy level (i.e. the Y-amplitude in the frequency domain).

Currently, many people live a sedentary lifestyle and lack sufficient exercise to burn off the energy influx which causes them to become overweight or obese. Being overweight and having obesity leads to a variety of chronic diseases, particularly diabetes. In addition, many types of processed food add unnecessary ingredients and harmful chemicals that are toxic to the bodies, which lead to the development of many other deadly diseases, such as cancers. For example, ~85% of worldwide diabetes patients are overweight, and ~75% of patients with cardiac illnesses or surgeries have diabetes conditions.

In engineering analysis, when the load is applied to the structure, it bends or twists, i.e. deform; however, when the load is removed, it will either be restored to its original shape (i.e. elastic case) or remain in a deformed shape (i.e. plastic case). In a biomedical system, the glucose level will increase after eating carbohydrates or sugar from food; therefore, the carbohydrates and sugar function as the energy supply. After having labor work or exercise, the glucose level will decrease. As a result, the exercise burns off the energy, which is similar to load removal in the engineering case. In the biomedical case, both processes of energy influx and energy dissipation take some time which is not as simple and quick as the structural load removal in the engineering case. Therefore, the age difference and 3 input behaviors are

"dynamic" in nature, i.e. timedependent. This time-dependent nature leads to a "viscoelastic or viscoplastic" situation.

For the author's case, it is "viscoplastic" since most of his biomarkers are continuously improved during the past 13-year time window.

Time-Dependent Output Strain and Stress of (Viscous Input*Output Rate)

Hooke's law of linear elasticity is expressed as:

Strain (ε : epsilon) = Stress (σ : sigma) / Young's modulus (E)

For biomedical glucose application, his developed linear elastic glucose theory (LEGT) is expressed as:

PPG (strain) = carbs/sugar (stress) * GH.p-Modulus (a positive number) + post-meal walking ksteps * GH.w-Modulus (a negative number)

Where GH.p-Modulus is reciprocal of Young's modulus E. However, in viscoelasticity or viscoplasticity theory, the stress is expressed as:

Stress = viscosity factor (η : eta) * strain rate ($d\varepsilon/dt$) Where strain is expressed as Greek epsilon or ε .

In this article, in order to construct an "ellipse-like" diagram in a stressstrain space domain (e.g. "hysteresis loop") covering both the positive side and negative side of space, he has modified the definition of strain as follows:

Strain = (body weight at certain specific time instant)

He also calculates his strain rate using the following formula: Strain rate = (body weight at next time instant) - (body weight at present time instant)

The risk probability % of developing into CVD, CKD, Cancer is calculated based on his developed metabolism index model (MI) in 2014. His MI value is calculated using inputs of 4 chronic conditions, i.e. weight, glucose, blood pressure, and lipids; and 6 lifestyle details, i.e. diet, drinking water, exercise, sleep, stress, and daily routines. These 10 metabolism categories further contain ~500 elements with millions of input data collected and processed since 2010. For individual deadly disease risk probability %, his mathematical model contains certain specific weighting factors for simulating certain risk percentages associated with different deadly diseases, such as metabolic disorderinduced CVD, stroke, kidney failure, cancers, dementia; artery damage in heart and brain, micro-vessel damage in kidney, and immunity-related infectious diseases, such as COVID death.

Some of explored deadly diseases and longevity characteristics using the *viscoplastic medicine theory (VMT)* include stress relaxation, creep, hysteresis loop, and material stiffness, damping effect *based on time-dependent stress and strain* which are different from his previous research findings using *linear elastic glucose theory (LEGT)* and *nonlinear plastic glucose theory (NPGT)*.

7. Results

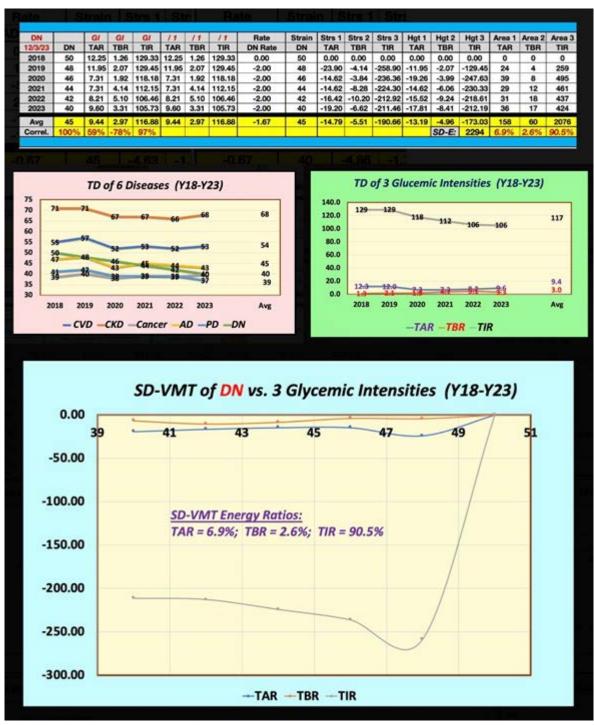


Figure 1: Data table, Time-domain curves and SD-VMT energies

8. Conclusions:

In summary, the author utilizes the space-domain viscoplastic energy (SD-VMT) method to explore the underlying connections and dynamics (i.e. energies) between three diabetic glycemic intensity (GI) inputs and the annual diabetic neuropathy (DN) risk output:

- Energy from Time Above Range (TAR): 6.9%
- Energy from Time Below Range (TBR): 2.6%
- Energy from Time in Range (TIR): 90.5%

Key message:

It is expected that the predominant contribution of TIR energy to his Parkinson's risk is significant. Notably, the finding indicating that the TAR-GI contribution (6.9%) to various cancers risk is nearly 2.7 times higher than that of TBR-GI (2.6%) is of particular importance.

The intensity of hyperglycemia can negatively impact internal organs, while the intensity of hypoglycemia can potentially lead to sudden death due to insulin shock.

References:

For editing purposes, majority of the references in this paper, which are self-references, have been removed for this article. Only references from other authors' published sources remain. The bibliography of the author's original self-references can be viewed at www.eclairemd.com. Readers may use this

article as long as the work is properly cited, and their use is educational and not for profit, and the author's original work is not altered. For reading more of the author's published VGT or FD analysis results on medical applications, please locate them through platforms for scientific research publications, such as ResearchGate, Google Scholar, etc.

Copyright: ©2024 Gerald C Hsu. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.