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Research Article

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Relationships of Parkinson's Disease Risks and Three Glycemic Intensities of Type 2 Diabetes Using Viscoplastic Energy Model of Ghmethod: Math-Physical Medicine

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Abstract

Diabetes is believed to affect a significant portion, approximately 81%, of individuals with Alzheimer's disease (AD Additionally, a range of 50% to 80% (with an average of 65%) of Parkinson's disease (PD) patients may also have diabetes. In a comprehensive meta-analysis comprising seven observational cohort studies involving over 1,761,000 individuals, it was observed that individuals with diabetes had a 38% higher risk of developing PD compared to those without diabetes. This risk was even more pronounced, with a 50% increase in females and a 40% increase in males.

From a pathophysiological perspective, there exists a intricate connection between Parkinson's disease and 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)

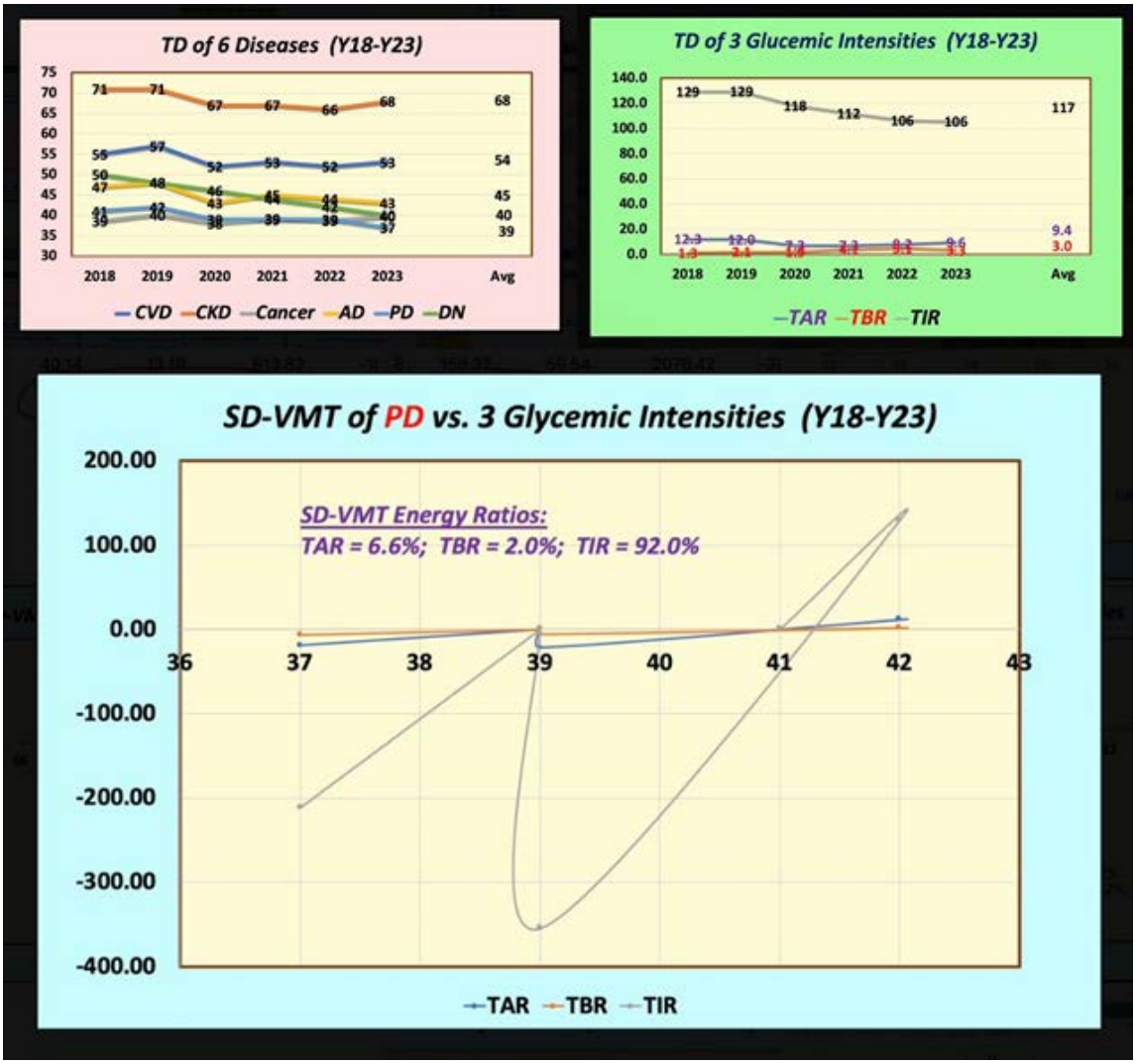
This paper delves into the author's risk probability of developing Parkinson's disease (PD) in relation to his type 2 diabetes condition. He introduces a novel concept called "glycemic intensities (GI)" as new biomarkers to study various diabetic complications. ***Those GI values are calculated as the 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 mortality conditions, such as PD.*
This study assesses the author's PD risk using three T2D-GI measures: TAR-GI (TAR), TBR-GI (TBR), and TIR-GI (TIR), utilizing 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* ***Parkinson's diseases (PD) risk output:***
Energy from Time Above Range (TAR): 6.0%
Energy from Time Below Range (TBR): 2.0%
Energy from Time in Range (TIR):92.0%

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 (6.0%) to various cancers risk is 3 times higher than that of TBR-GI (2.0%) 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.

Viscoelastic Medicine Theory (VMT #386)
Relationships of Parkinson's disease risks and three glycemic intensities of type 2 diabetes using viscoplastic energy model of GHMethod: math-physical medicine (No. 987)



1. Introduction

Diabetes is believed to affect a significant portion, approximately 81%, of individuals with Alzheimer's disease (AD). Additionally, a range of 50% to 80% (with an average of 65%) of Parkinson's disease (PD) patients may also have diabetes. In a comprehensive meta-analysis comprising seven observational cohort studies involving over 1,761,000 individuals, it was observed that individuals with diabetes had a 38% higher risk of developing PD compared to those without diabetes. This risk was even more pronounced, with a 50% increase in females and a 40% increase in males.

Hence, from a pathophysiological perspective, there exists a intricate connection between Parkinson's disease and metabolic disorders, particularly type 2 diabetes.

The American Diabetes Association classifies glucose levels as follows:

1.1. Hyperglycemia (glucose above 180 mg/dL)

1.2. Hypoglycemia (glucose below 70 mg/dL)

1.3. Normal Glycemic (glucose between 70 and 180 mg/dL):

This paper delves into the author's risk probability of developing Parkinson's disease (PD) in relation to his type 2 diabetes condition. He introduces a novel concept called ***"glycemic intensities (GI)"*** as new biomarkers to study various diabetic complications. ***Those 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 mortality conditions, such as PD.

This study assesses the author's PD risk using three T2D-GI measures:
TAR-GI (TAR), TBR-GI (TBR), and TIR-GI (TIR), utilizing personal data collected from August 1, 2018, to December 2, 2023.

1.4. 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.

1.5. Pathophysiological Explanations of Relationships Between Parkinson's Diseases and Diabetes Biomarkers, such as TAR, TBT, TIR

The relationship between Parkinson's disease and diabetes biomarkers, such as TAR, TBR, and TIR, involves various pathophysiological mechanisms:

1.5.1. Insulin Dysfunction: Both Parkinson's disease and diabetes are associated with insulin dysfunction. Insulin plays a critical role in the brain's neurotransmitter function and abnormal insulin signaling may contribute to the development of Parkinson's disease. Dysregulation of insulin in diabetes is associated with altered glucose levels, which could indirectly affect the progression of Parkinson's disease.

1.5.2. Oxidative Stress: Diabetes and its associated biomarkers, including TAR, TBR, and TIR, contribute to oxidative stress in the body.

Oxidative stress has been implicated in the pathogenesis of Parkinson's disease, as it can lead to cellular damage and neurodegeneration. The interplay between diabetes-related oxidative stress and the pathophysiology of Parkinson's disease is an area of ongoing research.

1.5.3. Inflammation: Both diabetes and Parkinson's disease involve inflammatory processes. Diabetesrelated biomarkers TAR, TBR, and TIR can contribute to chronic inflammation, which may exacerbate the neuroinflammatory processes seen in Parkinson's disease.
Inflammation can impact the central nervous system and contribute to neuronal damage in Parkinson's.

1.5.4. Vascular Effects: Diabetes can lead to vascular changes and endothelial dysfunction, which contribute to microvascular and macrovascular complications. These vascular changes may also affect brain health, potentially contributing to the pathophysiology of Parkinson's disease.

1.5.5. Glucose Dysregulation: Fluctuations in blood glucose levels represented by TIRT, TBR, and TAR can impact brain function.

Dysglycemia may disrupt neuronal function and affect neurotransmitter balance, which could affect the progression of Parkinson's disease.

It is important to note that the relationship between Parkinson's disease and diabetes biomarkers is complex, and the precise mechanisms linking the two conditions are not yet fully understood. Further research into the interactions between diabetes biomarkers and Parkinson's disease pathophysiology is necessary to provide a more comprehensive understanding of the relationship and potential targets for therapeutic intervention.

1.6. Dopaminergic System

The dopaminergic system is a neural network in the brain that relies on the neurotransmitter dopamine. Dopamine plays a crucial role in various physiological functions, including mood regulation, motor control, and reward-driven learning. The dopaminergic system is particularly significant in conditions like Parkinson's disease and is involved in complex processes such as:

1.6.1. Motor Control: Dopamineproducing neurons in specific brain regions, including the substantia nigra, are essential for coordinating smooth and controlled movements. *Dysfunction of the dopaminergic system, as seen in Parkinson's disease, can result in motor impairments like tremors, stiffness, and bradykinesia (slowed movements).*

1.6.2. Reward and Pleasure: Dopamine is involved in the brain's reward system, influencing motivation, reinforcement learning, and the experience of pleasure.
Disruptions in the dopaminergic pathways can contribute to conditions like addiction or mood disorders.

1.6.3. Cognition: Dopamine is implicated in cognitive functions such as memory, attention, and problem-solving. Changes in the dopaminergic system are associated with cognitive decline in conditions like Parkinson's disease and some forms of dementia.

1.6.4. Emotional Regulation: Dopamine plays a role in regulating emotions, and imbalances in the dopaminergic system are linked to mood disorders such as depression and bipolar disorder.

In Parkinson's disease, the degeneration of dopaminergic neurons in the substantia nigra leads to a shortage of dopamine, causing motor symptoms. Understanding the dopaminergic system is crucial for developing treatments that aim to modulate dopamine levels and improve the symptoms associated with disorders like Parkinson's.

1.7. Mitochondrial Function

Mitochondrial function refers to the health and efficiency of mitochondria, which are cellular organelles responsible for producing energy in the form of adenosine triphosphate (ATP). Proper mitochondrial function is essential for various cellular processes and overall cellular health.

In the context of health and disease, disturbances in mitochondrial function can have significant implications. Impaired mitochondrial function is associated with various conditions, including neurodegenerative diseases, metabolic disorders, and aging. Mitochondrial dysfunction may lead to a decrease in energy production, increased production of reactive oxygen species (ROS), and disruption of cellular processes.

Maintaining healthy mitochondrial function is crucial for overall cellular and organ health. Lifestyle factors such as regular exercise, a balanced diet, and stress management can positively influence mitochondrial function. Researchers are actively exploring the link between mitochondrial health and various diseases to develop strategies for preventing or treating conditions associated with mitochondrial dysfunction.

1.8. Neurotrophic Factors

Neurotrophic factors are a family of proteins that support the growth, survival, and differentiation of neurons (nerve cells) in the nervous system. These factors play a crucial role in the development, maintenance, and repair of the nervous system. Here are key points about neurotrophic factors:

1.8.1. Promoting Neuronal Growth: Neurotrophic factors stimulate the growth of nerve fibers (axons and dendrites) and promote the formation of new synapses, which are essential for communication between neurons.

1.8.2. Preventing Cell Death: They help prevent apoptosis, a process of programmed cell death, in neurons. This anti-apoptotic effect is vital for the survival and maintenance of neurons throughout the lifespan.

1.8.3. Facilitating Synaptic Plasticity: Neurotrophic factors contribute to synaptic plasticity, which is the ability of synapses to strengthen or weaken over time. This is crucial for learning and memory processes.

1.8.4. Supporting Nerve Regeneration: In cases of injury or damage to the nervous system, neurotrophic factors play a role in supporting nerve regeneration and repair.

1.8.5. Diverse Functions: Different types of neurotrophic factors exist, including nerve growth factor (NGF), brain-derived neurotrophic factor (BDNF), and neurotrophin-3 (NT-3), among others. Each has specific functions and is active in different regions of the nervous system.

1.8.6. Implications in Neurological Disorders: Dysregulation of neurotrophic factors has been implicated in various neurological disorders, including neurodegenerative diseases such as Alzheimer's and Parkinson's. Therapeutic strategies targeting these factors are being explored for potential neuroprotective effects.

Understanding the role of neurotrophic factors is crucial for advancing research and developing treatments that aim to support and protect neurons in both health and disease.

1.9. 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 ~10 key MPM research methods and different tools.

1.10. 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, post-meal 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.

1.11. 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.***

*1.12. 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 (dε/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)

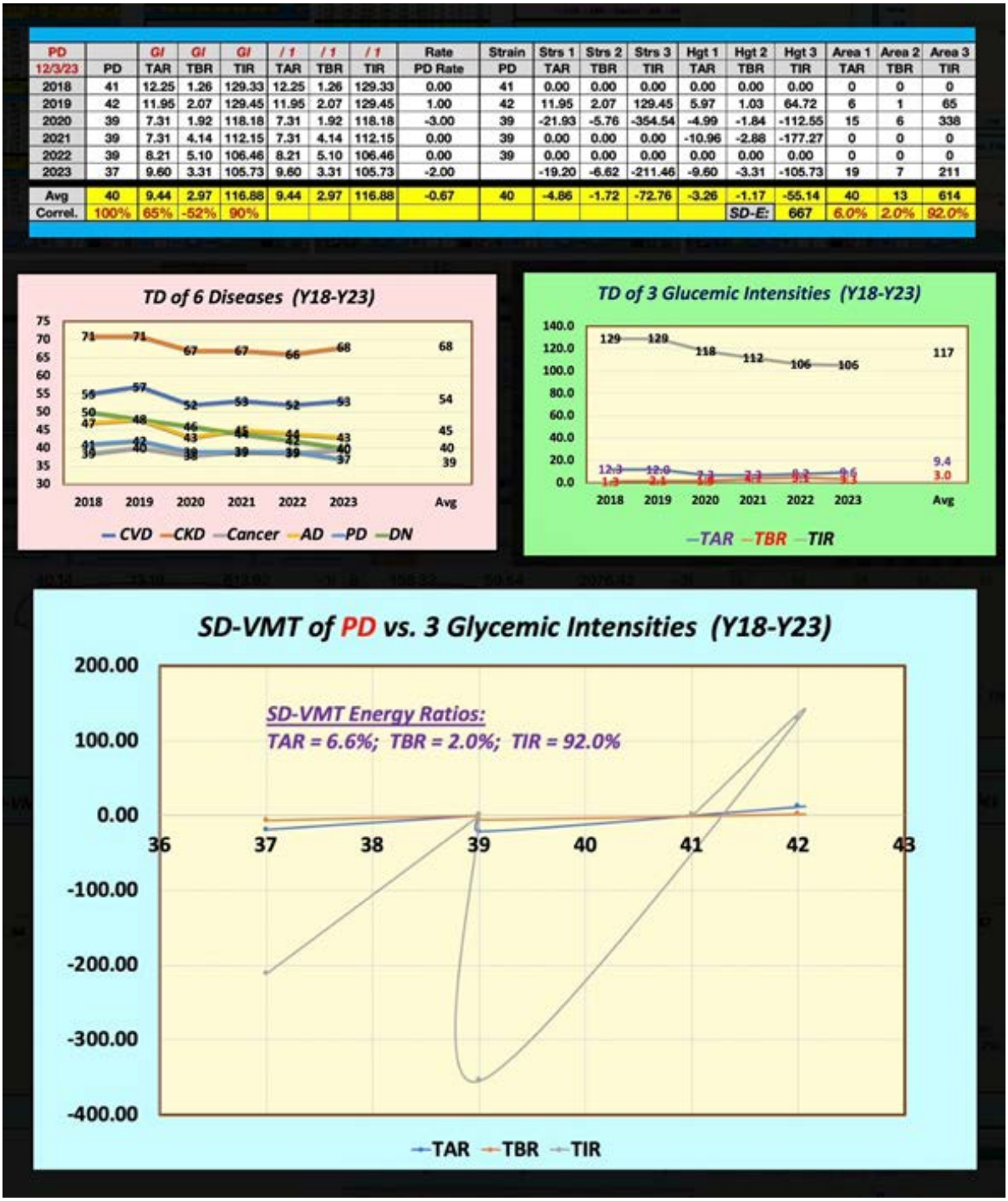
He also calculates his strain rate using the following formula:
Strain rate = (body weight at next time instant) - (body weight at present time instant)

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).***

2. Results



PD		GI	GI	GI	/ 1	/ 1	/ 1	Rate	Strain	Strs 1	Strs 2	Strs 3	Hgt 1	Hgt 2	Hgt 3	Area 1	Area 2	Area 3
12/3/23	PD	TAR	TBR	TIR	TAR	TBR	TIR	PD Rate	PD	TAR	TBR	TIR	TAR	TBR	TIR	TAR	TBR	TIR
2018	41	12.25	1.26	129.33	12.25	1.26	129.33	0.00	41	0.00	0.00	0.00	0.00	0.00	0.00	0	0	0
2019	42	11.95	2.07	129.45	11.95	2.07	129.45	1.00	42	11.95	2.07	129.45	5.97	1.03	64.72	6	1	65
2020	39	7.31	1.92	118.18	7.31	1.92	118.18	-3.00	39	-21.93	-5.76	-354.54	-4.99	-1.84	-112.55	15	6	338
2021	39	7.31	4.14	112.15	7.31	4.14	112.15	0.00	39	0.00	0.00	0.00	-10.96	-2.88	-177.27	0	0	0
2022	39	8.21	5.10	106.46	8.21	5.10	106.46	0.00	39	0.00	0.00	0.00	0.00	0.00	0.00	0	0	0
2023	37	9.60	3.31	105.73	9.60	3.31	105.73	-2.00		-19.20	-6.62	-211.46	-9.60	-3.31	-105.73	19	7	211
Avg	40	9.44	2.97	116.88	9.44	2.97	116.88	-0.67	40	-4.86	-1.72	-72.76	-3.26	-1.17	-55.14	40	13	614
Correl.	100%	65%	-52%	90%										SD-E:	667	6.0%	2.0%	92.0%

Figure 1: Data Table, Time-Domain Curves and SD-VMT Energies

3. Conclusions

3.1. 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 Parkinson's diseases (PD) risk output:

Energy from Time Above Range (TAR): 6.0%
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3.2. 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 (6.0%) to various cancers risk is 3 times higher than that of TBR-GI (2.0%) 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.

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