

The GH-Method

Viscoelastic Medicine theory (VMT #362): Relationships of Alzheimer's disease risks and four biomarkers of type 2 diabetes disease, insulin resistance via FPG, glycemic control via HbA1c and eAG, hyperglycemia intensity using viscoplastic energy model of GH-Method: math-physical medicine (No. 963)

Gerald C. Hsu*

eclairMD Foundation, USA

Abstract

The interaction between diet quality and portion control significantly influences body weight, a crucial factor in the progression of type 2 diabetes (T2D). T2D is characterized by four key biomarkers: insulin resistance (measured via morning fasting glucose - FPG), daily glycemic control (averaged glucose - eAG), quarterly glycemic control (HbA1c levels), and hyperglycemia situation control (hyperglycemia intensity - HyGI). HbA1c and eAG are similar biomarkers. However, both lack representation of insulin resistance influences and damage caused by hyperglycemia. HyGI is calculated as averaged glucose above 180 mg/dL multiplied by the occurrence frequency of glucose above 180 mg/dL

This study explores the author's Alzheimer's disease (AD) risks associated with these four T2D biomarkers, drawing from personal data collected over the past six years (5/1/2018 to 11/20/2023).

Traditional statistical analysis reveals strong correlations (77% to 86%) between the author's AD risk and the four T2D biomarkers. Additionally, the author employs the space-domain viscoplastic energy (SD-VMT) method to unveil hidden relationships and dynamics (i.e., energies) between these four T2D biomarkers and his annual AD risk output.

Keywords: Viscoelastic; Viscoplastic; Diabetes; Glucose; Dementia; Biomarkers; Insulin; Hyperglycemia; Alzheimer's Disease; Dementia

Abbreviations: CGM: continuous glucose monitoring; T2D: type 2 diabetes; PPG: postprandial plasma glucose; FPG: fasting plasma glucose; SD: space-domain; VMT: viscoelastic medicine theory; CVD: cardiovascular disease; CKD: chronic kidney disease

In summary, traditional statistical correlations uncovered significant associations between the author's CVD risks and his four T2D biomarkers: - AD vs. HbA1c: 85%; - AD vs. FPG: 85%; - AD vs. eAG: 86%; - AD vs. HyGI: 77%.

These differ from low positive correlations (14% to 41%) between his cancer risks and the same four T2D biomarkers, reflecting distinct characteristics in the risk waveforms of these two diseases.

Using SD-VMT energy results, four energy contribution margins on AD risks from T2D biomarkers were identified:

- Energy from HbA1c: 24%; - Energy from FPG: 28%; - Energy from eAG: 24%; - Energy from HyGI: 24%.

Insulin resistance via FPG is the strongest influential factor for his AD risks.

Key message: The author's T2D conditions are indeed linked to his risks of developing AD. Insulin resistance, indicated by FPG contributes the highest energy to CVD risks (28%), followed by 24% each from HbA1c, eAG, and HyGI. Despite representing only 2% occurrence of the total glucose dataset, hyperglycemia intensity contributes 27% of the total impact or energy on his overall AD risks.

1. INTRODUCTION

The interaction between diet quality and portion control significantly influences body weight, a crucial factor in the progression of type 2 diabetes (T2D). T2D is characterized by four key biomarkers: insulin resistance (measured via morning fasting glucose - FPG), daily glycemic control (averaged glucose - eAG), quarterly glycemic control (HbA1c levels), and hyperglycemia situation control (hyperglycemia intensity - HyGI). HbA1c and eAG are similar biomarkers. But, both of them lack representation of insulin resistance influences and damage caused by hyperglycemia. HyGI is calculated as averaged glucose above 180 mg/dL multiplied by the occurrence frequency of glucose above 180 mg/dL.

This study explores the author's Alzheimer's diseases (AD) risks associated with these four T2D biomarkers, drawing from personal data collected over the past six years (5/1/2018 to 11/20/2023).

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1.1 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, intending to optimize 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.

A growing body of research links type 2 diabetes with risk for Alzheimer's

March 16, 2022; (Keck school of medicine, USC):

Type 2 diabetes, also known as adult onset diabetes, is a chronic disease impacting an estimated 34 million Americans. If left untreated, it can lead to a host of complications including blindness, heart disease and stroke. Researchers are also now finding possible connections between diabetes and increased risk for developing Alzheimer's.

A recently completed British study of over 10,000 men and women, tracked the development of diabetes and dementia within the group from 1985-2019. What they learned was that the earlier the onset of diabetes, the greater the risk of developing dementia later. Even with controlling for other risk factors for dementia, such as race, heart conditions, and physical activity, researchers found a correlation between diabetes and dementia.

The researchers observed the age at which diabetes was initially diagnosed correlated with the risk of developing dementia. For example, a 70-year-old diagnosed with type 2 diabetes had an 11 percent increased risk for later developing dementia. Comparatively, those who developed diabetes younger had a much greater risk for dementia later in life: a 53 percent chance risk of later developing dementia for those diagnosed with diabetes at age 65 and a 77 percent risk of at age 60.

Up to 81 percent of people living with Alzheimer's are estimated to have type 2 diabetes. While scientists are still investigating how diabetes is linked to dementia, it is well known that the brain uses enormous amounts of glucose. A hallmark of type 2 diabetes is associated with hyperglycemia, high blood sugar, due to insulin resistance. With insulin resistance, cells no longer respond to insulin, the hormone that regulates cellular energy and metabolism. A recent study conducted by researchers at UNLV showed that chronic hyperglycemia impairs and alters working memory networks. The study revealed the hippocampus and the anterior cingulate cortex—parts of the brain central to forming and retrieving memories—were over-communicating and, as a result causing mistakes.

Insulin resistance in the brain is another growing area of investigation. In fact, Alzheimer's Disease is sometimes referred to as Type 3 Diabetes. By studying brain tissue

from deceased Alzheimer's patients, a team of BYU researchers discovered the genes used to break down glucose in the brain had been compromised. Insulin resistance makes it difficult for the brain to break down the glucose it needs for energy. But, the brain could potentially draw from another source of energy known as ketones, molecules the body makes when it's burning relatively high amounts of fat and when insulin levels are low. However, the average person consuming a Western diet high in carbohydrates or insulin spiking foods, has fewer ketones available as alternative fuel for the brain. What's promising is this line of investigation points to lifestyle and diet as a possible origin for Alzheimer's.

Nearly 1 in 7 Americans have diabetes, a disease that's on its own can have devastating consequences. But Type 2 diabetes can be managed and its complications reduced by monitoring blood sugar and following a personalized program of exercise, diet and medication. A growing body of evidence now supports diabetes prevention and management can also reduce risk of developing Alzheimer's down the road. Pathophysiological explanations of Parkinson's disease and certain biomarkers of type 2 diabetes, such as insulin resistance via FPG, diabetic control via HbA1C and eAG, and hyperglycemia intensity:

Pathophysiological explanations of Alzheimer's disease and four biomarkers of type 2 diabetes, such as insulin resistance via FPG, diabetic control via HbA1C and eAG, and hyperglycemia intensity:

Alzheimer's disease involves the progressive degeneration of brain cells, leading to memory loss and cognitive decline. Pathophysiological mechanisms include the accumulation of beta-amyloid plaques and tau tangles, disrupting communication between neurons and causing neuronal death.

Regarding type 2 diabetes biomarkers:

Insulin Resistance (Fasting Plasma Glucose - FPG):

Elevated FPG levels indicate insulin resistance, a factor linking type 2 diabetes to Alzheimer's. Insulin resistance may affect brain function, contributing to cognitive impairment.

Diabetic Control (HbA1C and eAG):

Effective diabetic control, reflected in stable HbA1C and estimated Average Glucose (eAG) levels, is crucial. Chronic hyperglycemia in diabetes may increase the risk of Alzheimer's by promoting inflammation and oxidative stress in the brain.

Hyperglycemia Intensity:

Monitoring the intensity of hyperglycemia, particularly postprandial glucose levels, is relevant. Elevated glucose levels can adversely impact brain health, potentially influencing Alzheimer's progression.

Advanced Glycation End Products (AGEs):

In diabetes, increased formation of AGEs contributes to vascular and neuronal damage. This process may play a role in the pathogenesis of Alzheimer's, linking diabetes-related vascular changes to cognitive decline.

Understanding these biomarkers helps in recognizing the intricate relationship between type 2 diabetes and Alzheimer's disease. Proper management of diabetes and monitoring these factors may contribute to reducing the risk of cognitive impairment.

1.2 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.

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 developing 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 finger-piercing glucose from 250 mg/dL to 120 mg/dL, and A1C from 10% to ~6.5%. One of his major accomplishments is that he has no longer taken any diabetes-related medications since 12/8/2015.

In 2017, he achieved excellent results on all fronts, especially his glucose control. However, during the pre-COVID period, including both 2018 and 2019, he travelled 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 travelling period of 2018-2019.

He started his COVID-19 self-quarantined 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-travelling, low-stress, and regular daily life routines. Of course, his in-depth 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 checked his glucose measurements every 5 minutes for a total of 288 times each day. Furthermore, he extracted the 5-minute intervals from every 15-minute interval for a total of 96 glucose data each day stored in his computer software.

Through the author's medical research work of over 40,000 hours and reading 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 well-balanced 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 lengths 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.

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 labor-work 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 glucose circulate 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 example, the combination of hyperglycemia and hypertension would cause micro-blood vessel leakage in kidney systems which is one of the major causes 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) influence 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., deforms; 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, carbohydrates and sugar function as the energy supply. After having labour 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 behaviours are “dynamic” in nature, i.e. time-dependent. This time-dependent nature leads to a “viscoelastic or viscoplastic” situation. For the author’s case, it is “viscoplastic” since most of his biomarkers have 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 k-steps * GH.w-Modulus (a negative number)

where GH.p-Modulus is the 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, to construct an “ellipse-like” diagram in a stress-strain 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 a 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 disorder-induced 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 the 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

Figure 1 shows data table, Time-domain curves and SD-VMT energies.

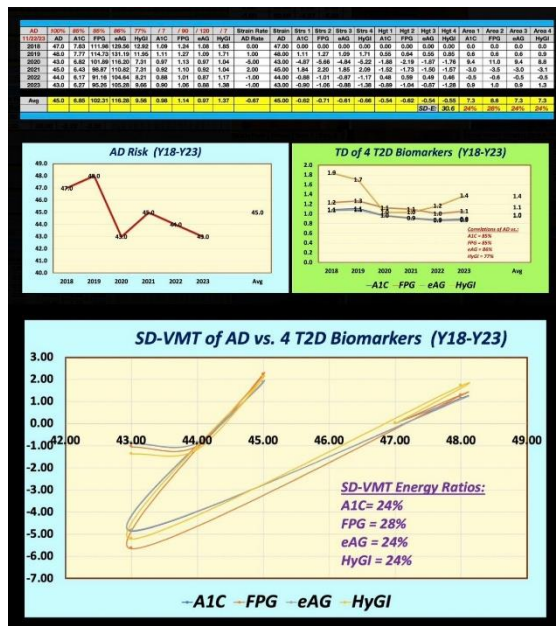


Figure 1: Data table, Time-domain curves and SD-VMT energies.

3. CONCLUSION

In summary, traditional statistical correlations uncovered significant associations between the author's CVD risks and his four T2D biomarkers:

- AD vs. HbA1c: 85%
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4. REFERENCES

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

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Viscoelastic and Viscoplastic Glucose Theory Application in Medicine

Gerald C. Hsu

