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The GH-Method

Viscoelastic Medicine Theory (VMT #271): Pathophysiological Interpretations of Dementia versus Hyperglycemia Intensity, Hypoglycemia Intensity, Glucose Fluctuations, and Exercise Using Collected Data between 5/1/2018 to 6/17/2023 and a Quantitative Analysis Applying SD-VMT Quantitative Energy Model of GH-Method: Math-Physical Medicine (No. 870)

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Abstract

Glucose is the primary fuel source for the brain, and studies have shown that both high glucoses, low glucoses, and glucose fluctuations can have harmful effects on the brain. Hyperglycemia intensity (Hyper I), or high glucoses, has been shown to increase inflammation in the brain and contribute to the formation of plaques in the brain which are a hallmark of Alzheimer's disease. Hypoglycemia intensity (Hypo I), or low glucoses, can result in reduced energy to the brain, leading to impaired cognitive function and an increased risk of dementia. The fluctuations of glucoses (GF) within a day has also brought burdens and damages on brain cells. Regular exercise can reduce the risk of cognitive impairment and dementia. The author has chosen the daily walking (Ksteps) as his main exercise option. Overall, there are strong evidences to suggest that maintaining healthy glucose levels and engaging in regular exercise may reduce the risk of cognitive impairment and dementia diseases. First, he has included some pathophysiological concluding remarks from a few medical publication and articles regarding this subject of dementia. He has cited some of those summarized qualitative statements which have indicated that glucose control and regular exercise have directly impact on reduction of risk of developing dementia. He then applies the engineering and physics space-domain viscoplastic medicine theory (SD-VMT) energy model to quantitatively determine the degree of influences from hyperglycemia intensity, hypoglycemia intensity, glucose fluctuations, and walking exercise on his own risk investigation of dementia. He uses his collected data between 5/1/2018 and 6/17/2023 as the base sources. In summary, there are 4 findings from this particular dementia study: A) In time domain, his averaged hyper I value is 193 mg/dL and occurrence frequency is 5.5%, while his averaged hypo I value is 66 mg/dL and occurrence frequency is 7.2%. His averaged GF (max of 288 mg/dL minus min of 48 mg/dL) is 91 mg/dL and his averaged daily walking exercise is 15,380 steps. B) His SD-VMT energies are: Hyper I = 11%; Hypo I = 6%, GF = 35%; and Ksteps = 48%. C) Due to the lower occurrence frequencies of hyper I (5.5%) and hypo I (7.2%), these two influential factors contributions are much lower than other two influential factors with 100% daily occurrences (GF and Ksteps). Therefore, he decides to compare them separately. In terms of contribution %, Hyper I > Hypo I; and Ksteps > GF. D) Time-zone analysis results are: Pre-COVID of Y17-Y19 = 36%; COVID of Y20-Y23 = 64%. COVID period has higher contribution on his dementia risk. The author must continuously lower his Hyperglycemia intensity, hypoglycemia intensity, and GF. His walking exercise amount is sufficient regarding his dementia concerns.

Keywords: Viscoelastic; Viscoplastic; Dementia; Hyperglycemia; Hypoglycemia; Glucose; Exercise

Abbreviations: PPG: postprandial plasma glucose; FPG: fasting plasma glucose; SD: space-domain

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1. INTRODUCTION

1.1 Pathophysiological interpretation

Pathophysiological explanation of Dementia versus hyperglycemia, hypoglycemia and glucose fluctuations:

The pathophysiological explanation of the relationship between dementia vs. hyperglycemia, hypoglycemia, glucose fluctuation, and exercise is complex and not fully understood. However, there is evidence to suggest that these factors may play a role in the development and progression of dementia.

Glucose is the primary fuel source for the brain, and studies have shown that both high and low glucose levels can have harmful effects on the brain. For example, hyperglycemia, or high blood glucose levels, can result in damage to blood vessels in the brain, leading to reduced blood flow and increased risk of stroke. Hyperglycemia has also been shown to increase inflammation in the brain and contribute to the formation of beta-amyloid plaques, which are a hallmark of Alzheimer's disease.

On the other hand, hypoglycemia, or low blood glucose levels, can result in reduced energy to the brain, leading to impaired cognitive function and an increased risk of dementia. Repeated episodes of hypoglycemia have been associated with increased risk of cognitive decline and dementia.

Exercise has been shown to have many benefits for brain health, including improving glucose regulation and reducing inflammation. In fact, studies have demonstrated that regular exercise can reduce the risk of cognitive impairment and dementia.

There are many quantitative studies that have investigated the relationship between glucose regulation, exercise, and dementia. For example, a 2017 study published in the Journal of Alzheimer's Disease found that individuals with better glucose regulation had a lower risk of developing dementia. Another study published in the Journal of Alzheimer's Disease in 2018 found that moderate-intensity exercise was associated with increased brain volume and improved cognitive function in older adults.

Overall, while the exact pathophysiological explanation of the relationship between dementia, glucose regulation, and exercise is not fully understood, there is evidence to suggest that maintaining healthy glucose levels and engaging in regular exercise may reduce the risk of cognitive impairment and dementia.

This particular article:

This article discusses relationships between dementia risk versus four input variables, hyperglycemia intensity (Hyper I), hypoglycemia intensity (Hypo I), glucose fluctuation (GF). and daily elealkibg steos (Ksteps). The author has used his collected data from 5/1/2018 through 6/17/2023.

His selected single output variable is his dementia risk percentages from Y2017 to Y 2023. The year of Y2017 has dementia risk data but without glucose data due to his Libre sensor data collection started on 5/1/2018. Therefore, Y2017 serves as the "initial condition" with zero stress and height for his SD-VMT calculations.

In the body of this article, at first, the author describes the relationship between dementia versus hyper I and hypo I as well as GF and Ksteps using pathophysiological interpretations (a qualitative approach). Then, he applies time-domain analysis and the energy models in space-domain (SD) to provide a quantitative picture of their respective contribution levels (or influence levels) from each of these 4 input component on his dementia risk.

Furthermore, he calculated his 2 time-zone energies in his SD-VMT analysis results regarding his dementia risk.

2. METHODS

2.1 Alzheimer's dementia (AD)

The following paragraphs in this sub-section are excerpts from different published papers about dementia.

"What is the relationship between diabetes and dementia?"
by Andrew E. Budson, MD on Harvard Health Publishing of Harvard Medical School on July 12, 2021:

"It has been known for many years that type 2 diabetes increases your risk for strokes and heart disease. More recent studies have shown that diabetes also increases your risk of dementia. What has not previously been investigated, however, is whether the age of onset of diabetes makes a difference in your risk of developing dementia.

New research about age at diabetes onset and the risk of developing dementia

A newly published study examined the association between age of onset of diabetes and the development of dementia using a large, ongoing cohort study. The cohort was established in 1985–88 among 10,308 employees aged 35 to 55 years (33% women, 88% white) in London-based government departments. Data on diabetes exposure, including fasting glucose and the Finnish Diabetes Risk Score, were obtained at ages 55, 60, 65, and 70. (The Finnish Diabetes Risk Score includes age, family history of diabetes, personal history of elevated blood glucose, fruit and vegetable consumption, blood pressure medication, physical activity, body mass index, and measured waist circumference.) (The author's note: these factors have been included in his metabolism model for dementia.)

Dementia due to any cause was the primary outcome measure. In addition to diabetes, they also examined the effects of age, sex, race, smoking, alcohol consumption, physical activity, fruit and vegetable consumption, high blood pressure, body mass index, coronary heart disease, heart failure, stroke, medications, and the Alzheimer's risk factor gene, apolipoprotein E.

The long-term effects of diabetes on dementia

From 1985 to 2019, 1,710 cases of diabetes and 639 cases of dementia were recorded. For every 1,000 people, examined yearly, the rates of dementia were 8.9 in those without diabetes at age 70. Comparable rates of dementia for those with diabetes were 10.0 for those with onset up to five years earlier, 13.0 for six to 10 years earlier, and 18.3 for more than 10 years earlier. These striking results clearly show that the earlier you develop diabetes, the greater (about two times higher) your risk is for developing dementia.

How diabetes can lead to dementia?

There are multiple reasons why years of type 2 diabetes may lead to dementia. One reason is related to the effects that diabetes has on the heart, as heart health is related to brain health. Heart disease and elevated blood pressure are both associated with strokes that, in turn, can lead to dementia. However, strokes do not appear to be the complete answer, as some studies found that diabetes led to an increased risk of dementia even when strokes were controlled for.

Another factor relates to the episodes of hypoglycemia that commonly occur in diabetes. Although tight control of blood sugars has been proven to reduce the long-term risks of heart disease and strokes, tight control can also lead to hypoglycemia, memory loss, and dementia. Here, the reason is likely because low blood sugars are known to damage the hippocampus — the memory center of the brain.

One of the more intriguing hypotheses is that diabetes directly causes Alzheimer's disease. Indeed, Alzheimer's disease has even been called "type 3 diabetes" because of shared molecular and cellular features among diabetes and Alzheimer's. For example, insulin plays a critical role in the formation of amyloid plaques, and insulin is also involved in the phosphorylation of tau, which leads to neurofibrillary tangles. In other words, whereas insulin resistance in the body can lead to type 2 diabetes, insulin resistance in the brain can lead to the plaques and tangles of Alzheimer's disease.

Reduce your risk of diabetes and dementia

The good news is that you can reduce your risk of type 2 diabetes — and your risk of dementia. Speak with your doctor today about whether the following lifestyle modifications would be right for you. Note that these life changes are helpful even if you have a diagnosis of diabetes or prediabetes.

- Engage in aerobic exercise at least 30 minutes each day, five days each week.
- Eat a Mediterranean-style menu of foods.
- Maintain a healthy body weight.
- Treat high blood pressure.
- Treat high cholesterol.
- Don't smoke.

Lastly, social activities, a positive attitude, learning new things, and music can all help your brain work at its best and reduce your risk of dementia. (The author's note: these lifestyle factors have also been considered in his metabolism model for dementia.)"

2.2 Overall picture of dementia

"Alzheimer's dementia is the most common form of dementia, and probably the best studied. Alzheimer disease is on the rise in the United States, and the facts are daunting. According to the Alzheimer Association's 2020 Alzheimer's Disease Facts and Figures report, approximately 5.8 million Americans age 65 and older currently have the Alzheimer dementia (AD) disease, with this number expected to triple over the next 30 years, and nearly two-thirds of those are women. It's the sixth leading cause of death in the United States—a ranking that's expected to skyrocket as the US population ages (reference 2).

And it's not just an American problem. The WHO estimates there are about 50 million people across the globe living with dementia, with nearly 10 million cases being added each year (Reference 3). According to a review published in Therapeutic Advances in Chronic Disease, this number is projected to increase to 75 million by 2030, and to 135 million by 2050.

The cost of dementia on the worldwide healthcare system is currently more than $800 billion and is expected to mushroom to $2 trillion by the year 2030, and that does not account for the costs associated with informal caregiving. As a matter of fact, over 16 million Americans care for patients with Alzheimer or other dementias, without pay."

2.3 Dementia and lifestyle

"The following news is based on an analysis of data collected over 8 years from almost 200,000 adults aged 60 and over in the UK (Reference 4). This study was conducted by researchers from the UK (University of Exeter Medical School, University of Oxford, University College London, The Alan Turing Institute), the US (University of Michigan, Veterans Affairs Center for Clinical Management Research in Michigan), Australia (University of South Australia), and Germany (University of Hamburg, Hamburg Center for Health Economics). The study was published in the peer-reviewed Journal of American Medical Association (JAMA).

There are measures we can take individually to help combat the problem. According to the authors of the aforementioned review, almost half of all dementia cases can be attributed to a small number of modifiable lifestyle risk factors, including smoking, obesity, and physical inactivity.

The research project used volunteers completed questionnaires at the start of the study about their lifestyles, and researchers looked at their DNA to see who carried genetic variations that have been associated with increased risk of Alzheimer's – the most common type of dementia.

The researchers found that among participants who had a higher genetic risk of getting dementia, only about 11 in every 1,000 with healthier lifestyles developed the condition during follow-up, compared with about 18 in every 1,000 with unhealthy lifestyles.

Overall, however, the findings are good news. We cannot change our genetics, but this study suggests that regardless of this, changing our lifestyles could help everyone reduce their dementia risk. This large UK cohort study has suggested that a healthy lifestyle can reduce overall dementia risk, even in people with genetic risk factors for Alzheimer's disease (Reference 5). The Daily Telegraph reported that "Bad dementia genes can be overcome through healthy living, study finds". The newspaper reports that regular exercise, not smoking, drinking sensibly, and eating a healthy diet have been found to reduce risk of getting dementia even if a person has a higher genetic risk of developing the condition."

2.4 Glycemic fluctuations & dementia

The following is an excerpt from an article by Kaneto, Kinoshita, Shimoda, and Kaku,
"The Presence of Dementia as one of Diabetic Complications: Hyperglycemia, Hypoglycemia and Glycemic Fluctuation are Associated with the Development of Dementia".

"Mainly, there are two types of dementia; one type is Alzheimer's disease and another type is vascular dementia. Chronic hyperglycemia and repeated hypoglycemia are closely associated with the onset and/or development of dementia. It is well known that the presence of diabetes mellitus leads to various complications such as microangiopathy (nephropathy, retinopathy, neuropathy) and macroangiopathy (ischemic heart disease, stroke). Dementia is often complicated in elderly subjects with diabetes and thereby thought to be one of diabetic complications. Indeed, it was reported that the incidence of Alzheimer's disease and vascular dementia was significantly higher in subjects with diabetes compared to non-diabetic subjects. It has been suggested that in the diabetic state chronic hyperglycemia leads to the formation and accumulation of advanced glycation end products in the brain which could lead to the development of dementia [8] (Figure 1). In addition, while it is known that hyperglycemia and subsequent oxidative stress reduce insulin signaling in various insulin target tissues [9,10], insulin receptors are highly expressed in the brain as well [11]. It was reported that such reduction of insulin signaling in the brain induced hyper-phosphorylation of Tau protein and accumulation of beta amyloid protein both of which are well known as main characteristics in Alzheimer's disease [8,12-14] (Figure 1). In subjects with diabetes, there are several macro- and micro-angiopathy. Diabetic macroangiopathy (e.g. atherosclerosis) leads to the onset of cerebrovascular disease such as stroke which is closely associated with the development of vascular dementia. In addition, diabetic microangiopathy brings out brain ischemia which is also closely associated with the onset and progression of vascular dementia (Figure 1).

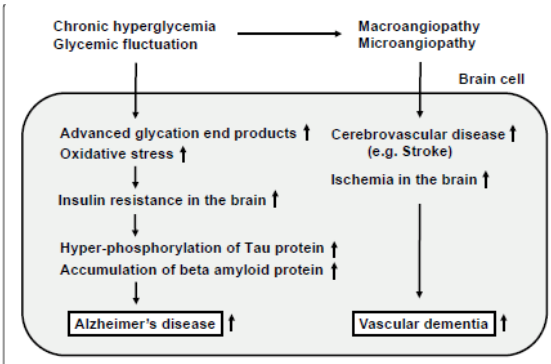

Figure 1: Association between diabetes and dementia.

There was also a significant linear association between an increased risk of dementia and an increased number of hypoglycemia. It is known that recurrent hypoglycemia causes brain damage especially in the cerebral cortex and hippocampus. There is a kind of vicious cycle between the frequency of hypoglycemia and the development of dementia including both Alzheimer's disease and vascular dementia (Figure 2).

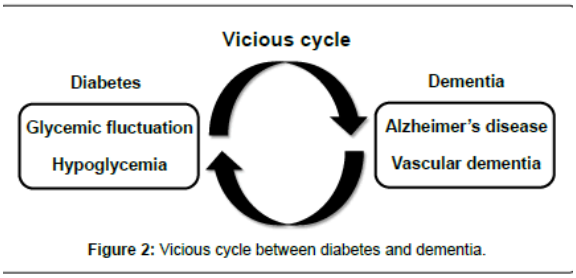

Figure 2: Vicious cycle between diabetes and dementia.

It was reported that the risk of Alzheimer's disease and vascular dementia was significantly associated with postprandial glucose levels but not fasting glucose levels [7]. These data support the hypothesis that the fluctuation of blood glucose levels is likely associated with the development of various types of dementia. It has been reported that glycoalbumin (GA)/hemoglobin A1c (HbA1c) ratio is a good marker for the fluctuation of blood glucose levels regardless of glycemic control situations [20].

The fluctuation of blood glucose levels, but not the incidence of hypoglycemia, was significantly associated with the decrease of cognitive impairment in this study. In order to avoid the onset and/or development of dementia, we should be careful for reducing

the fluctuation of blood glucose levels in addition to avoiding hypoglycemia. Taken together, glycemic fluctuation is closely associated with the onset of and/or progression of dementia in elderly subjects with type 2 diabetes. We should avoid glycemic fluctuation especially in elderly subjects with type 2 diabetes in order to avoid the development of dementia.

Dementia is often complicated in elderly subjects with diabetes mellitus and thereby thought to be one of diabetic complications. Not only chronic hyperglycemia and repeated hypoglycemia but also the fluctuation of blood glucose levels is associated with the onset and/or progression of dementia in elderly subjects with diabetes mellitus."

2.5 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 the 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.

2.6 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 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 lb. (100 kg) to 176 lb. (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 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 self-quarantined life on 1/19/2020. By 10/16/2022, his weight was further reduced to ~164 lb. (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 in-depth knowledge of chronic diseases, sufficient practical lifestyle management experiences, and his 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 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-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.

2.7 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 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 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) are influencing the energy level (i.e. the Y-amplitude in the frequency domain).

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 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 energy influx and dissipation processes 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. 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 are continuously improved during the past 13-year time window.

2.8 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, and 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 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).

Note: For a more detailed description, please refer to the "consolidated method" section which is given at the beginning of the special issue.

3. RESULTS

Figure 1 shows data tables.

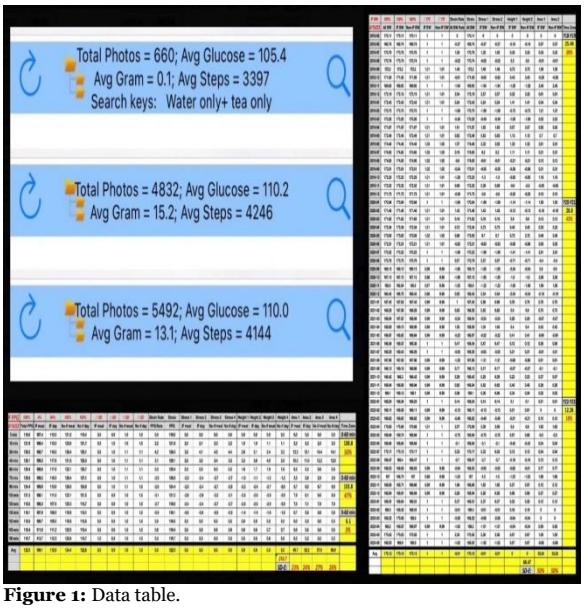

Figure 1: Data table.

Figure 2 shows TD and SD-VMT analysis results for both PPG and BW.

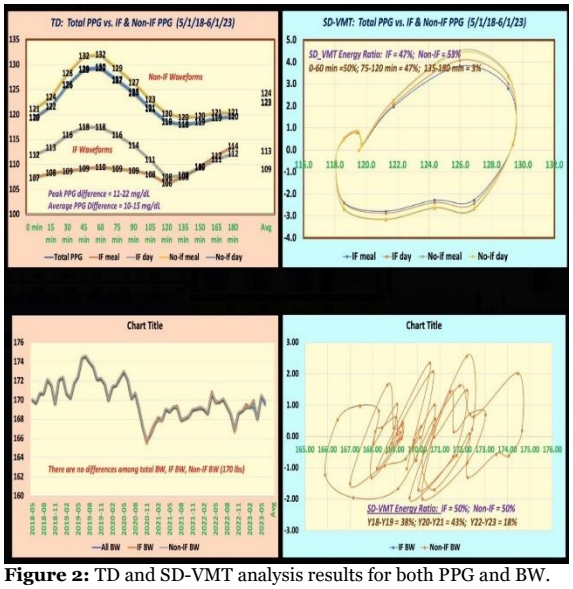

Figure 2: TD and SD-VMT analysis results for both PPG and BW.

4. CONCLUSION

In summary, there are 6 findings from this particular IF study:

A. In time domain, his peak total and Non-IF PPG is 11-22 mg/dL higher than IF PPG with his averaged Non-IF PPG is 10-15 mg/dL higher than IF PPG.

B. His space-domain viscoplastic SD-VMT energy contributions are: IF PPG = 47% and Non-IF PPG = 53%.

C. In time domain, his total BW, IF BW and Non-IF BW are identical at 170 lbs.

D. His space-domain viscoplastic SD-VMT energy contributions are: IF = 50% and Non-IF = 50%.

E. In conclusion, IF brings 6% on his PPG reduction while IF brings no benefit on his weight control efforts.

F. Time-zone analysis results in SD are: For PPG: 0-60 minutes = 50%; 75-120 minutes = 47%; 135-180 = 47%. For BW: Y18-Y19 = 38%; Y20-Y21 = 43%; Y22-Y23 = 18%.

Behind the conclusion of IF has no impact on his body weight control, the author has guessed the following 3 possible reasons:

A. His body weight are daily weight data, not meal associated weight.

B. After having one IF meal, he may compensate his food consumption by eating slightly more on other two meals of the IF day.

C. Although he has collected 660 IF data, it could still need more IF data in order to observe the pathophysiology's qualitative conclusion.

5. REFERENCES

For editing purposes, the majority of the references in this paper, which are self-references, have been removed. 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, 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 three published special editions from the following three specific journals:

(1) Special Issue. The GH-Method. (https://www.theghmethod.com).

(2) Journal of Applied Material Science & Engineering Research (contact: Catherine).

(3) Advances in Bioengineering and Biomedical Science Research (contact: Sony Hazi).

Special Issue | Issue No. 5 The GH-Method

Viscoelastic and Viscoplastic Glucose Theory Application in Medicine

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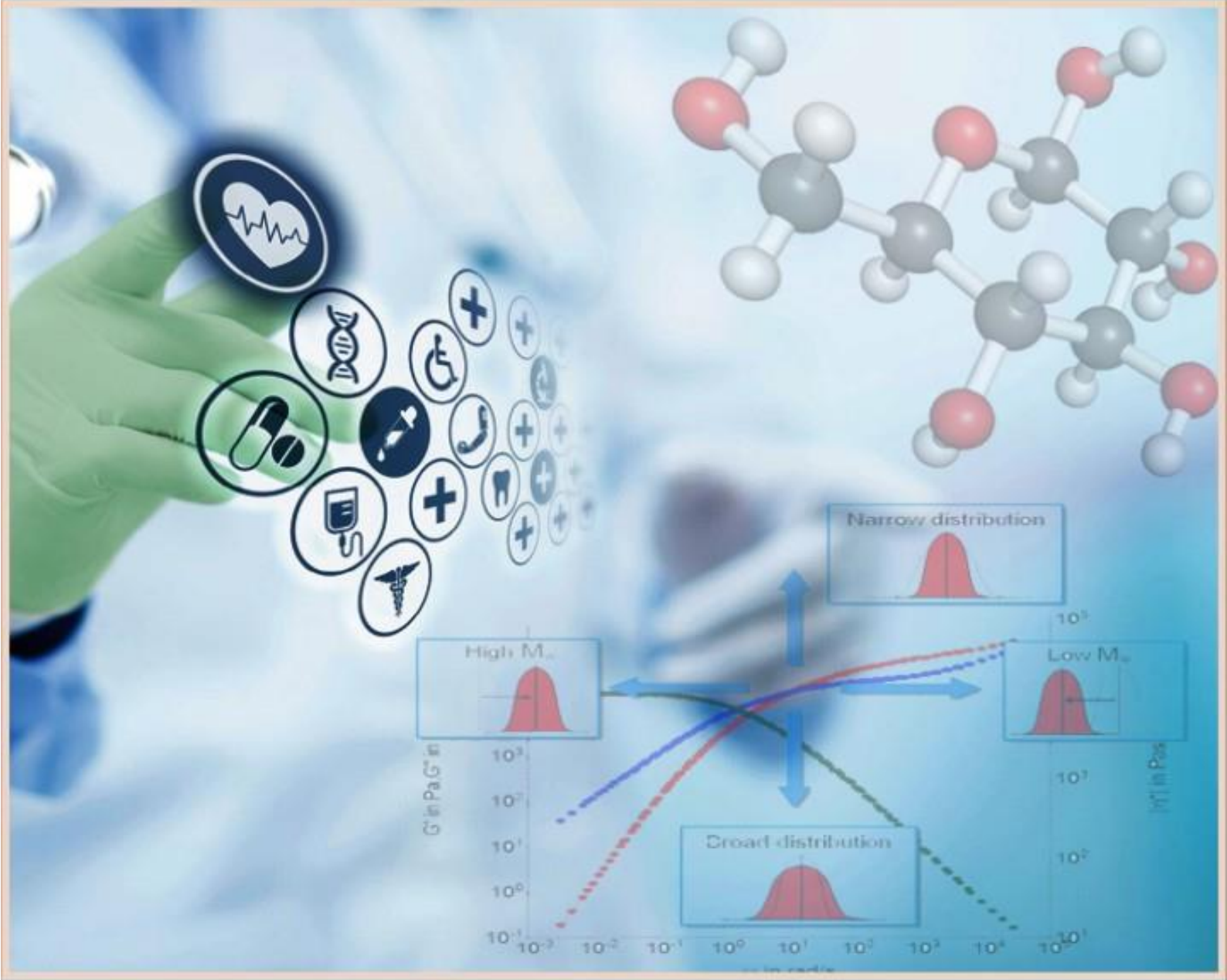

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