Special Issue 5
2022

The GH-Method

Viscoelastic Medicine theory (VMT #413): A partial view of the gut-brain axis study from lifestyle details through metabolic disorders to dementia using viscoplastic energy model of GH-Method: Math-Physical Medicine (No. 1015)

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Abstract

The gut-brain axis is essential for regulating metabolism, which can impact the development of type 2 diabetes (T2D) and obesity, both of which are linked to dementia, particularly in the context of neurodegenerative conditions such as Alzheimer's and Parkinson's. In short, the gut-brain axis influences various brain functions, including cognition and motor control.

As a long-term T2D patient without access to medical facilities, instruments, or the means to collect and analyze specific gut-brain axis biomarkers like gut hormones (ghrelin, leptin, cholecystokinin, peptide YY), gut motility, gut microbiota information, and pathological brain proteins such as beta-amyloid and tau, the author relied on available and collectible data as a patient. For example, both obesity and diabetes represent not only metabolic disorders associated with insulin resistance but also demonstrate situations of low-grade inflammation. Additionally, fasting plasma glucose in the early morning (FPG) was used as an indicator of the level of insulin resistance. The author also chose to incorporate the TyG index (Triglyceride-glucose index) as a biomarker for both insulin resistance (TyG value greater than 4.49) and non-alcoholic fatty liver disease (NAFLD, TyG value greater than 8.5).

The TyG index is calculated based on fasting levels of triglycerides (TG) and fasting glucose, using the formula: TyG = ln [fasting triglycerides (mg/dL) × fasting glucose (mg/dL)/2].

This paper details a two-stage analysis centered around a partial view of the gut-brain axis. The initial stage delves into the correlation between lifestyle factors and two metabolic disorders. For obesity, influential factors such as food portions (Food P), daily exercise (Exercise), and sleep quality (Sleep) are considered, while for type 2 diabetes, impactful factors include fasting plasma glucose (FPG), carbohydrate and sugar intake (Carbs), and post-meal walking steps (Steps).

The second stage of the analysis investigates the link between five selected metabolic disorders and two specific neurodegenerative diseases: Alzheimer's (AD) and Parkinson's (PD). Both diseases are examined in relation to five common metabolic factors, namely body weight (BW), daily glucose levels (eAG), insulin resistance (via FPG), a normalized TyG index for insulin resistance (IR via TyG with a normalization factor of 4.49), and non-alcoholic fatty liver disease (NAFLD via TyG with a normalization factor of 8.5).

In summary, the first stage of two SD-VMT energy analysis results for metabolic disorders versus lifestyle are:

Obesity of BW versus: food portion = 45%; sleep = 29%; daily exercise = 26% (Food portion is the predominant factor)

T2D of eAG versus: FPG = 46%; Carbs = 33%; post-meal steps = 21% (Insulin resistance of FPG is the predominant factor; ratio of carbs versus exercise is 1.6)

The second stage of two SD-VMT energy analysis results for dementia versus five common inputs are:

Alzheimer's risk versus: Obesity BW = 19%; T2D eAG = 19%; IR via FPG = 31%; IR via TyG = 20%; NAFLD via TyG = 11% (Total IR contributed 51%, Obesity and T2D contributed 38%, and NAFLD contributed only 11%).

Parkinson's risk versus: Obesity BW = 20%; T2D eAG = 19%; IR via FPG = 30%; IR via TyG = 20%; NAFLD via TyG = 11% (Total IR contributed 50%, Obesity and T2D contributed 39%, and NAFLD contributed only 11%).

In all four analysis cases, the time-zone energy distribution ratios exhibit a consistent pattern:

Available online: 4 April 2024

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Y2014-Y2023 contributed higher percentages (63% to 85%), while Y2019-Y2023 showed lower contribution percentages (15% to 37%).

Key message:

Based on a partial view of the gut-brain axis study, insulin resistance emerges as the primary influencer for Alzheimer's and Parkinson's diseases, mirroring the situation observed in T2D's eAG case. As anticipated, fatty liver (NAFLD) demonstrates the least influence. In the context of obesity and weight management, portion size of food consumption emerges as the most critical influencer.

Keywords: Viscoelastic; Viscoplastic; Diabetes; Glucose; Biomarkers; Insulin; Hyperglycemia; Dementia

Abbreviations: CGM: continuous glucose monitoring; eAG: estimated average glucose; T2D: type 2 diabetes; PPG: postprandial plasma glucose; FPG: fasting plasma glucose; SD: space-domain; VMT: viscoelastic medicine theory; FFT: Fast Fourier Transform

1. INTRODUCTION

The gut-brain axis is essential for regulating metabolism, which can impact the development of type 2 diabetes (T2D) and obesity, both of which are linked to dementia, particularly in the context of neurodegenerative conditions such as Alzheimer's and Parkinson's. In short, the gut-brain axis influences various brain functions, including cognition and motor control.

As a long-term T2D patient without access to medical facilities, instruments, or the means to collect and analyze specific gut-brain axis biomarkers like gut hormones (ghrelin, leptin, cholecystokinin, peptide YY), gut motility, gut microbiota information, and pathological brain proteins such as beta-amyloid and tau, the author relied on available and collectible data as a patient. For example, both obesity and diabetes represent not only metabolic disorders associated with insulin resistance but also demonstrate situations of low-grade inflammation. Additionally, fasting plasma glucose in the early morning (FPG) was used as an indicator of the level of insulin resistance. The author also chose to incorporate the TyG index (Triglyceride-glucose index) as a biomarker for both insulin resistance (TyG value greater than 4.49) and non-alcoholic fatty liver disease (NAFLD, TyG value greater than 8.5).

The TyG index is calculated based on fasting levels of triglycerides (TG) and fasting glucose, using the formula:

TyG = ln[fasting triglycerides (mg/dL) × fasting glucose (mg/dL)/2]

Condition	Cut-off value	Values below cut-off	Values above cut-off
Insulin resistance	4.49	Insulin resistance unlikely	Suggestive of insulin resistance
Nonalcoholic fatty liver disease	8.5	NAFLD diagnosis is unlikely	High likelihood of NAFLD

This paper details a two-stage analysis centered around a partial view of the gut-brain axis. The initial stage delves into the correlation between lifestyle factors and two metabolic disorders. For obesity, influential factors such as food portions (Food P), daily exercise (Exercise), and sleep quality (Sleep) are considered, while for type 2 diabetes, impactful factors include fasting plasma glucose (FPG), carbohydrate and sugar intake (Carbs), and post-meal walking steps (Steps).

The second stage of the analysis investigates the link between five selected metabolic disorders and two specific neurodegenerative diseases: Alzheimer's (AD) and Parkinson's (PD). Both diseases are examined in relation to five common metabolic factors, namely body weight (BW), daily glucose levels (eAG), insulin resistance (via FPG), a normalized TyG index for insulin resistance (IR via TyG with a normalization factor of 4.49), and non-alcoholic fatty liver disease (NAFLD via TyG with a normalization factor of 8.5).

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, 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.

What are the food-sugar-gut-axis connecting brain activities, such as Alzheimer's cognition brain cells and Parkinson's motion brain cells?

The connection between the brain and the stomach through glucose involves the release of hormones and neurotransmitters. When glucose enters the bloodstream after a meal, it triggers the release of insulin, which helps to regulate blood sugar levels. Additionally, the brain responds to the presence of glucose by releasing neurotransmitters such as serotonin and dopamine, which can influence feelings of hunger, satisfaction, and overall mood. This intricate interaction between glucose, insulin, and neurotransmitters helps to regulate appetite, digestion, and overall energy balance in the body.

The connection between brain activities, such as cognition in Alzheimer's disease and motion in Parkinson's disease, and the conversion of food digestion and conversion into glucose via gut functions involves a complex interplay of various physiological processes. Here are some key components:

Glucose Metabolism:
Food digestion leads to the breakdown of carbohydrates into glucose, which is absorbed into the bloodstream. The levels of glucose in the blood are carefully regulated to ensure a steady supply of energy to the brain and other organs.

Hormonal Regulation:
Hormones such as insulin, released from the pancreas, play a crucial role in regulating glucose levels. Insulin facilitates the uptake of glucose by various cells in the body, including neurons in the brain.

Brain Function:
Neurons in the brain, including those involved in cognition and movement, rely on glucose as their primary energy source. Disruptions in glucose metabolism can impact the function of these neurons, potentially contributing to neurodegenerative conditions like Alzheimer's and Parkinson's.

Neurotransmitters:
Glucose metabolism in the brain is involved in the production of neurotransmitters, which are essential for cell-to-cell communication. Imbalances in neurotransmitter levels can influence cognitive function and motor control.

Gut-Brain Axis:
Emerging research suggests that the gut microbiota and the central nervous system communicate bidirectionally through neural, endocrine, and immune pathways. This gut-brain axis may influence various brain functions, including cognition and motor control.

Overall, the convergence of glucose metabolism, hormonal regulation, brain function, neurotransmitter activity, and the gut-brain axis highlights the intricate connections between food digestion, glucose utilization, and brain activities, especially in the context of neurodegenerative diseases such as Alzheimer's and Parkinson's.

Pathophysiological explanations of the gut-brain axis in terms of neural, endocrine, and immune pathways:

The gut-brain axis is a bidirectional communication system that involves complex interactions between the central nervous system (CNS), the enteric nervous system (ENS) of the gut, the endocrine system, and the immune system. Dysregulation in any of these pathways can lead to various pathophysiological conditions. Here are explanations of the gut-brain axis in terms of neural, endocrine, and immune pathways:

Neural Pathway:
The neural pathway involves bidirectional communication between the CNS and the ENS. The ENS is often referred to as the "second brain" due to its extensive network of neurons in the brain that control gastrointestinal functions independently of the CNS. This pathway is essential for regulating gastrointestinal motility, secretion, and blood flow. Disruptions in neural signaling within the gut-brain axis can lead to functional gastrointestinal disorders such as irritable bowel syndrome (IBS) or contribute to neurodegenerative diseases such as Parkinson's disease, both of which are characterized by altered gut motility and sensory processing.

Endocrine Pathway:
The gut-brain axis also involves the endocrine system, primarily through the secretion of gut hormones such as ghrelin, leptin, peptide YY, and cholecystokinin. These hormones play a vital role in regulating appetite, satiety, and metabolism, and they communicate with the brain to convey information about the nutritional status of the body. Dysregulation of these hormones and their signaling pathways can contribute to conditions such as obesity, eating disorders, and metabolic syndrome.

Immune Pathway:
The immune system also plays a crucial role in the gut-brain axis, as the gut is home to a large proportion of the body's immune cells. Local immune responses in the gut can impact systemic immune function and neuroinflammation. Disruption of the immune pathway in the gut-brain axis has been implicated in the pathophysiology of various neurological and psychiatric disorders, including depression, anxiety, autism spectrum disorders, and neurodegenerative diseases. Additionally, inflammatory bowel diseases (IBD) such as Crohn's disease and ulcerative colitis involve complex interactions between the immune system and the nervous system, leading to both gastrointestinal and neurological symptoms.

Overall, the gut-brain axis is a multidimensional and intricate system that involves neural, endocrine, and immune pathways. Dysregulation of these pathways can contribute to a wide range of pathophysiological conditions, making the understanding and modulation of the gut-brain axis vital in the management of various disorders.

What is the enteric nervous system ENS?

The enteric nervous system (ENS) is a complex network of neurons that is often described as the "second brain" due to its extensive capabilities and its ability to function largely independently of the central nervous system (CNS). The ENS is embedded within the lining of the gastrointestinal tract, extending from the esophagus to the rectum, and it plays a critical role in regulating various gastrointestinal functions, including motility, secretion, and blood flow.

The ENS contains a vast number of neurons, estimated to be in the hundreds of millions, as well as glial cells, which support and modulate the activity of the neurons. This intricate network allows the ENS to coordinate and control the movement of food through the digestive system, the secretion of digestive enzymes and hormones, and the sensation of visceral pain. The ENS can also regulate local blood flow and immune responses within the gut.

In addition to its autonomous functions, the ENS is in constant communication with the central nervous system through various pathways, including the sympathetic and parasympathetic nervous systems. This bidirectional communication allows for the integration of gut functions with higher brain functions to maintain overall homeostasis.

Disruptions in the enteric nervous system have been implicated in various gastrointestinal disorders, such as irritable bowel syndrome (IBS), inflammatory bowel diseases (IBD), and functional dyspepsia. Furthermore, emerging research suggests that the ENS may also play a role in conditions that extend beyond the gut, including certain neurological disorders, mental health conditions, and even metabolic diseases.

In summary, the enteric nervous system is a highly specialized and autonomous network that governs many aspects of gastrointestinal function. Its complex interplay with the central nervous system and its role in various disorders make it an area of great interest in both neurology and gastroenterology.

How many different kinds of dementia?

Dementia is an umbrella term that encompasses various diseases and conditions that impair cognitive function, memory, and the ability to perform daily activities. Alzheimer's disease and Parkinson's disease are two well-known causes of dementia, but there are several other diseases that can also lead to dementia, including:
1. Vascular dementia
2. Lewy body dementia
3. Frontotemporal dementia
4. Creutzfeldt-Jakob disease
5. Huntington's disease
6. Wernicke-Korsakoff syndrome
7. Normal pressure hydrocephalus

These are just a few examples, and there are other less common causes of dementia as well. Each of these conditions has its distinct characteristics and underlying causes, leading to different patterns of cognitive impairment and functional decline.

Pathophysiological explanations of relationships between gut-brain axis and Alzheimer's and Parkinson's diseases:

The gut-brain axis has been implicated in the pathophysiology of Alzheimer's disease and Parkinson's disease through various mechanisms involving neural, endocrine, and immune pathways. Here are the pathophysiological explanations of the relationships between the gut-brain axis and these two neurodegenerative diseases, namely dementia:

Alzheimer's Disease:

1. Neural Pathway:
Dysfunction in the gut-brain axis may contribute to the development and progression of Alzheimer's disease. The enteric nervous system (ENS) is known to play a role in regulating gastrointestinal motility and secretion. Studies have suggested that alterations in gut motility and gut microbiota composition could lead to dysregulation in the gut-brain axis, possibly contributing to the spread of pathological proteins, such as beta-amyloid and tau, through neural connections between the gut and the brain.

2. Endocrine Pathway:
Hormones and signaling molecules produced in the gastrointestinal tract can also influence synaptic plasticity and neuroinflammation, which are key aspects of Alzheimer's disease pathology. Disruptions in gut hormone signaling, such as ghrelin and leptin, may impact cognitive function and contribute to the pathogenesis of Alzheimer's disease.

3. Immune Pathway:
The gut houses a large portion of the body's immune cells, and dysregulation of the gut microbiota can lead to increased intestinal permeability and systemic inflammation. This systemic inflammation can directly impact the progression of neuroinflammation in Alzheimer's disease, potentially exacerbating cognitive decline and neuronal damage.

Parkinson's Disease:

1. Neural Pathway:
The gut-brain axis has been implicated in the pathophysiology of Parkinson's disease through the concept of the "gut-first" hypothesis. This hypothesis suggests that the pathophysiological process of Parkinson's disease may initiate in the gastrointestinal tract and then spread to the brain via the vagus nerve. This could be facilitated by alpha-synuclein aggregation and spread, influencing both gut motility and brain function.

2. Endocrine Pathway:
Hormonal and metabolic changes in the gut-brain axis have been linked to the development of Parkinson's disease. For example, alterations in gut hormone signaling and metabolism may influence the accumulation of alpha-synuclein aggregates and impact neurodegenerative processes.

3. Immune Pathway:
Inflammation and immune dysregulation within the gut have been associated with Parkinson's disease. The gut microbiota may play a role in modulating systemic immune responses and neuroinflammation, affecting the progression of Parkinson's disease pathology.

Overall, these pathophysiological explanations highlight the intricate interplay between the gut-brain axis and the development and progression of neurodegenerative diseases such as Alzheimer's and Parkinson's diseases. Understanding these relationships may offer insights into potential therapeutic targets for these conditions.

Is TyG a biomarker for gut-brain axis study?

The triglyceride-glucose index (TyG) is a marker used to assess insulin resistance and non-alcoholic fatty liver disease (NAFLD) which is calculated by the following formula: ln [fasting triglycerides (mg/dL) x fasting glucose (mg/dL)/2].

It has been suggested that TyG may be associated with various health conditions, including metabolic syndrome, cardiovascular disease, and non-alcoholic fatty liver disease (NAFLD).

While the gut-brain axis primarily focuses on the bidirectional communication between the

gut and the brain, including the influence of the gut microbiota on brain function, mood, and behavior, as well as the role of the central nervous system in regulating gut function, there is no direct evidence indicating that TyG could serve as a biomarker for the study of the gut-brain axis.

However, given the association between metabolic disturbances, insulin resistance, and conditions such as NAFLD, there may be indirect links between TyG, gut health, and neurological function. For example, alterations in the gut microbiota and subsequent inflammation associated with insulin resistance and metabolic dysfunction could impact the gut-brain axis.

As research in the field of the gut-brain axis continues to evolve, it is essential to consider the potential systemic and metabolic factors that could impact the interplay between the gut and the brain, including markers such as TyG. However, at present, the role of TyG as a specific biomarker for the direct study of the gut-brain axis remains uncertain and requires further investigation.

Is NAFLD a part of the gut-brain axis?

Non-alcoholic fatty liver disease (NAFLD) is not traditionally considered a direct part of the gut-brain axis. The gut-brain axis primarily refers to the bidirectional communication between the central nervous system (CNS) and the enteric nervous system, linking cognitive and emotional centers in the brain with peripheral intestinal functions.

However, emerging research has suggested potential links between NAFLD and the gut microbiota, leading to indirect associations with the gut-brain axis. The gut microbiota plays a crucial role in metabolic processes, immune function, and the regulation of inflammation, and dysbiosis (an imbalance in the gut microbial community) has been implicated in the development and progression of NAFLD.

Furthermore, studies have proposed that alterations in the gut microbiota composition can influence systemic inflammation, insulin resistance, and metabolic dysfunction, which are all factors contributing to NAFLD development. These changes in the gut microbiota and subsequent metabolic disturbances may indirectly impact the gut-brain axis by influencing neuroinflammation, cognitive function, and mental health.

While the gut-brain-liver axis is an evolving area of research that investigates the complex interplay between the gut, brain, and liver, it is important to acknowledge that the direct involvement of NAFLD in the traditional gut-brain axis remains an area of ongoing study and debate.

(Note: The author included TyG in this analysis due to its potential or indirect involvement in the gut-brain axis and attempted to provide some quantitative evidence of its role.).

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 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.

The author's diabetes history:

The author has been 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 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 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 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 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).

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, 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. 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, in order 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 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 tables.

Figure 2 shows inputs and SD-VMT energy output diagram.

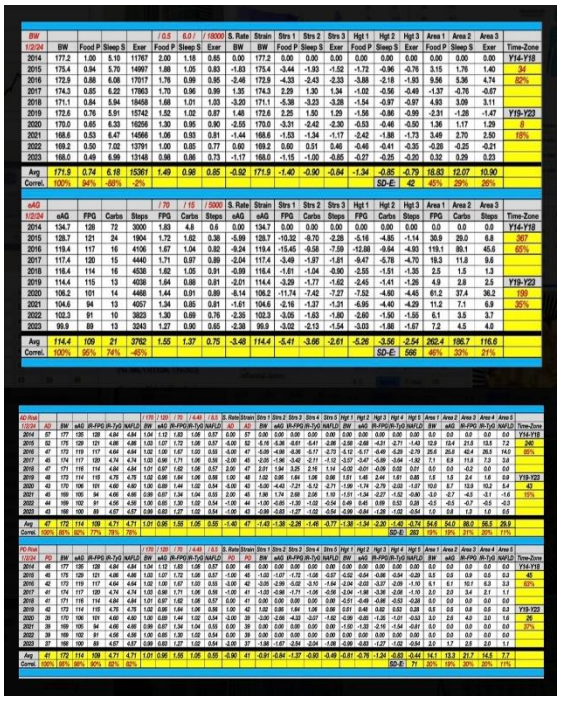

Figure 1: Data tables

3. CONCLUSION

In summary, the first stage of two SD-VMT energy analysis results for metabolic disorders versus lifestyle are:

Obesity of BW versus:

food portion = 45%; sleep = 29%; daily exercise = 26%

(Food portion is the predominant factor)

T2D of eAG versus:

FPG = 46%; Carbs = 33%; post-meal steps = 21%

(Insulin resistance of FPG is the predominant factor; ratio of carbs versus exercise is 1.6)

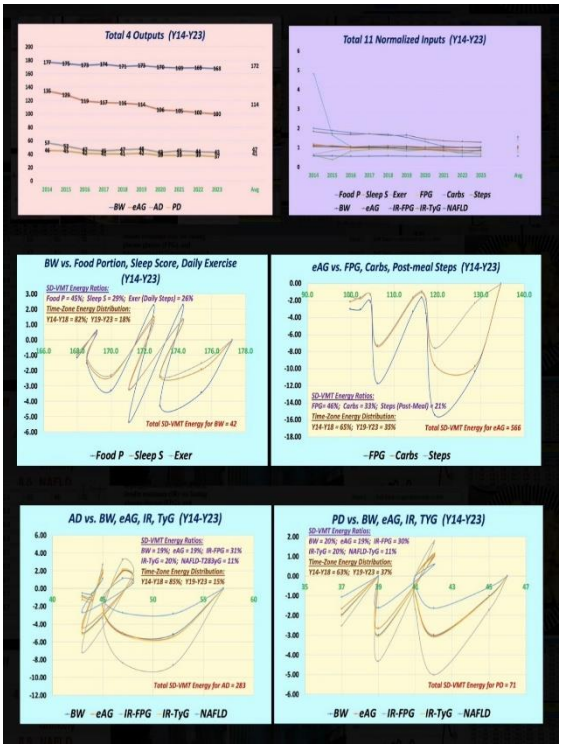

Figure 2: Inputs and SD-VMT energy output diagram

The second stage of two SD-VMT energy analysis results for dementia versus five common inputs are:

Alzheimer's risk versus:

Obesity BW = 19%; T2D eAG = 19%; IR via FPG = 31%; IR via TyG = 20%; NAFLD via TyG = 11%.

(Total IR contributed 51%, Obesity and T2D contributed 38%, and NAFLD contributed only 11%).

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(Total IR contributed 50%, Obesity and T2D contributed 39%, and NAFLD contributed only 11%).

In all four analysis cases, the time-zone energy distribution ratios exhibit a consistent pattern: Y2014-Y2023 contributed higher percentages (63% to 85%), while Y2019-Y2023 showed lower contribution percentages (15% to 37%).

Key message:

Based on a partial view of the gut-brain axis study, insulin resistance emerges as the primary influencer for Alzheimer's and Parkinson's diseases, mirroring the situation observed in T2D's eAG case. As anticipated, fatty liver (NAFLD) demonstrates the least influence. In the context of obesity and weight management, portion size of food consumption emerges as the most critical influencer.

4. REFERENCES

For editing purposes, most 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.eclairemd.com.

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.

Special Issue | Issue No. 5 The GH-Method

Viscoelastic and Viscoplastic Glucose Theory Application in Medicine

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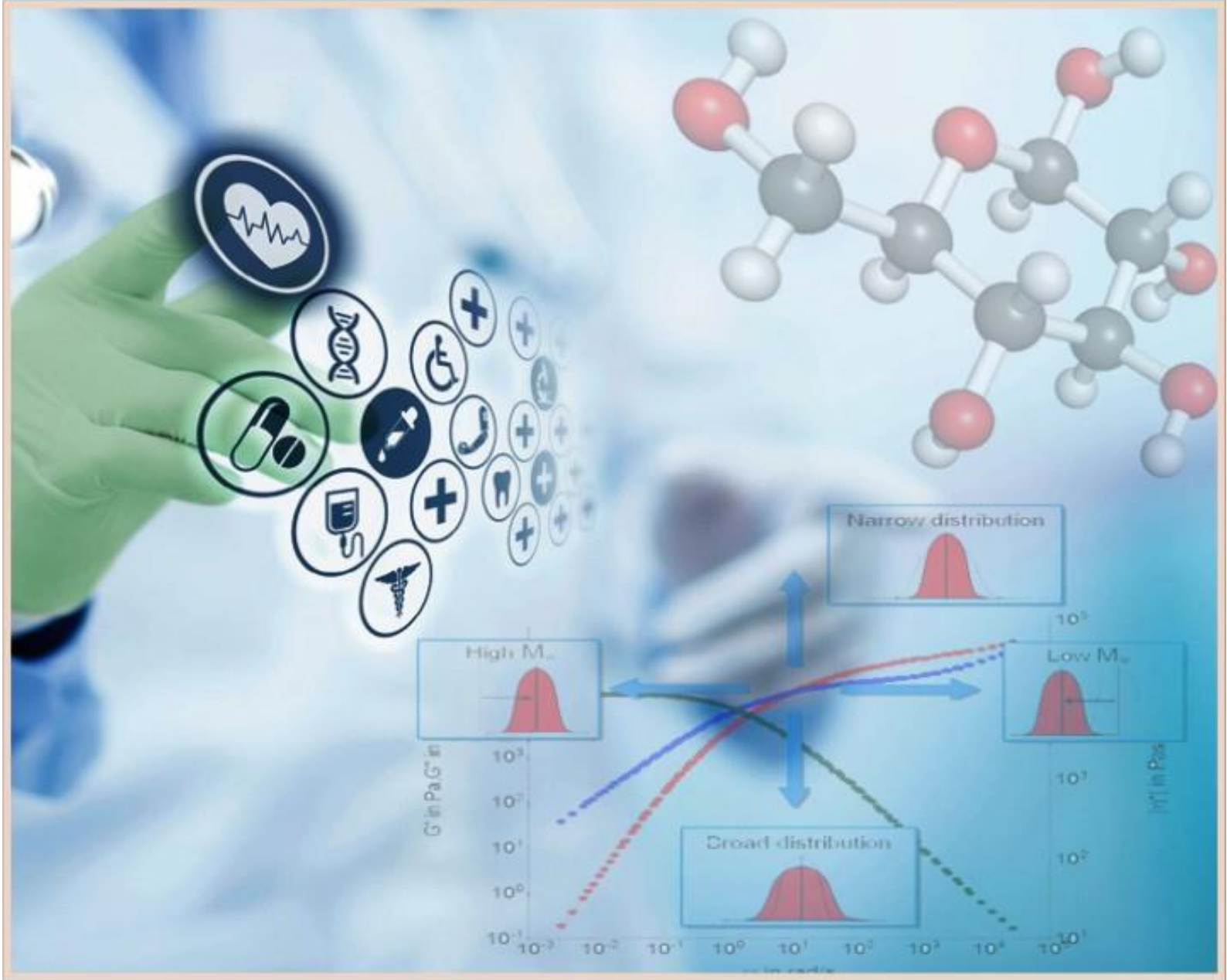

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2022