

The GH-Method

Viscoelastic Medicine Theory (VMT #307): An Integrated Health Analysis and Medical Research Work Utilizing Qualitative Pathophysiology and Quantitative Math-Physical Modeling (No. 907)

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Abstract

This research article presents a comprehensive health analysis framework that combines qualitative pathophysiological explanations with math-physical quantitative analyses. The work is based on 12 years of data collection (2012-2023) and employs the space-domain viscoplastic energy model from GH-Method: Math-Physical Medicine. The author's investigation into his health and medical conditions over the past 14 years has yielded some significant insights. The examination of over 300 analyses utilizing the viscoplastic medicine technology (VMT) modeling, conducted since December 2021, has provided a deeper understanding of his physical well-being. This study aims to construct a comprehensive health roadmap based on various findings from his VMT research journey of recent 2.5 years. Through 6 interconnected studies, the author has derived the following insights: Obesity results from a combination of overeating, low-quality food, and insufficient exercise. The obesity subsequently leads to three metabolic disorders (MD): diabetes, hypertension, and dyslipidemia. The author further groups these four medical conditions (MC) along with six lifestyle details (LD) into a mathematical model of the metabolism index (MI). Furthermore, he examines the association between five mortality diseases—type 2 diabetes (T2D), cardiovascular diseases (CVD), chronic kidney diseases (CKD), dementia from ADPD (Alzheimer's and Parkinson's diseases), and various cancers—with MC, LD, and MI. The author evaluates his potential lifespan by considering age difference and the impact from these mortality diseases. In a summarized

approach, he compares the energy disparity between the initial years (2013-2014) and recent years (2022-2023) to understand and demonstrate the extent of his health conditions improvement, using the numerical difference in total viscoplastic energies between these two periods. In summary, there are two key findings: 1. The author uncovers energy ratios representing degrees of influence, contribution, or association from six distinct but interconnected SD-VMT studies. - Energy ratios involving body weight: Diet holds a 62% influence (food portion contributes 39%, food quality contributes 23%), compared to exercise at 38%. The diet-to-exercise ratio is 1.63. - Energy ratios for obesity and three metabolic disorders (MD): Diabetes at 45%, hypertension at 35%, and dyslipidemia at 21%. - Energy ratios for medical conditions (MC) with 4 mortality diseases: CKD (35%), CVD (26%), ADPD (20%), and Cancer (19%). - Energy ratios for lifestyle details (LD) with 4 mortality diseases: CKD (34%), CVD (27%), ADPD (20%), and Cancer (19%). - Energy ratios linking longevity perspective with 4 mortality diseases: CKD (34%), CVD (26%), ADPD (20%), Cancer (20%). - Associations revealing MI and five mortality diseases: T2D (34%), CKD (23%), CVD (17%), ADPD (13%), Cancer (13%). 2. Averaged total energy calculations indicate a comparison between two initial years and two recent years: 57% for Y2013-Y2014 versus 3.2% for Y2022-Y2023. The 16-fold higher energy associated with the initial period signifies significant improvement in his health conditions, with no regression to the initial unhealthy state. These observations are reflected in those viscoplastic curves.

Keywords: Viscoelastic; Viscoplastic; Metabolic disorders; Lifestyle; Glucose; Diabetes

Abbreviations: BMI: body mass index; MD: metabolic disorders; LD: lifestyle details; MC: medical conditions; MI: metabolism index; CVD: cardiovascular disease; CKD: chronic kidney disease; T2D: type 2 diabetes; PPG: postprandial plasma glucose; FPG: fasting plasma glucose

1. INTRODUCTION

This research article presents a comprehensive health analysis framework that combines qualitative pathophysiological explanations with math-physical quantitative analyses. The work is based on 12 years of data collection (2012-2023) and employs the space-domain viscoplastic energy model from GH-Method: Math-Physical Medicine.

The author's investigation into his health and medical conditions over the past 14 years has yielded some significant insights. The examination of over 300 analyses utilizing the viscoplastic medicine technology (VMT) modeling, conducted since December 2021, has provided a deeper understanding of his physical well-being.

This study aims to construct a comprehensive health roadmap based on various findings from his VMT research journey of recent 2.5 years. Through 6 interconnected studies, the author has derived the following insights:

Obesity results from a combination of overeating, low-quality food, and insufficient exercise. The obesity subsequently leads to three metabolic disorders (MD): diabetes, hypertension, and dyslipidemia. The author further groups these four medical conditions (MC) along with six lifestyle details (LD) into a mathematical model of the metabolism index (MI).

Furthermore, he examines the association between five mortality diseases—type 2 diabetes (T2D), cardiovascular diseases (CVD), chronic kidney diseases (CKD), dementia from ADPD (Alzheimer's and Parkinson's diseases), and various cancers—with MC, LD, and MI. The author evaluates his potential lifespan by considering age difference and the impact from these mortality diseases.

In a summarized approach, he compares the energy disparity between the initial years (2013-2014) and recent years (2022-2023) to understand and demonstrate the extent of his health conditions improvement, using the numerical difference in total viscoplastic energies between these two periods.

1.1 Biomedical information

The following sections contain condensed information sourced from various published medical articles that the author has reviewed. It is important to acknowledge that these sections are not the original work or creation of the author of this specific article. They have been included for the purpose of later review by the author and to provide useful information to other readers interested in this topic.

Pathophysiological explanations of body weight vs. meal portion, food quality, and exercises:

The relationship between body weight and meals portion, food quality, and exercise can be explained through various pathophysiological mechanisms. Here are some explanations:

1. Meals portion

Consuming larger portion sizes can lead to body weight gain and obesity. When individuals consistently consume more calories than their body requires, the excess energy is stored as fat. Over time, this can lead to an increase in body weight. Additionally, larger portion sizes can contribute to a higher intake of unhealthy nutrients such as sugar, unhealthy fats, and refined carbohydrates, which can also lead to weight gain and related health issues.

2. Food quality

The quality of the food we consume has a significant impact on our body weight. Diets high in processed foods, added sugars, unhealthy fats, and low in essential nutrients can contribute to weight gain and obesity. These types of foods are often energy-dense but nutrient-poor, meaning they provide a high number of calories without offering many essential vitamins, minerals, and fiber. Consuming nutrient-poor foods can lead to overeating as the body seeks out essential nutrients, resulting in weight gain. On the other hand, a diet rich in whole, unprocessed foods such as fruits, vegetables, whole grains, lean proteins, and healthy fats provides essential nutrients while being lower in calories. These types of foods promote satiety and can help maintain a healthy body weight.

3. Exercise

Regular physical activity and exercise play a crucial role in maintaining a healthy body weight. Exercise increases energy expenditure, which helps burn calories and contributes to weight loss or weight maintenance. It also helps to build and maintain lean muscle mass, which increases metabolic rate and supports a healthy weight. Additionally, exercise can improve insulin sensitivity, enhance cardiovascular health, and provide various other health benefits that contribute to weight management. Exercise can also have indirect effects on body weight through its impact on appetite regulation. Physical activity can help regulate hunger hormones and reduce cravings for unhealthy, calorie-dense foods, which can help control calorie intake and support weight management.

In summary, consuming appropriate portion sizes, prioritizing nutritious food choices, and engaging in regular exercise all play significant roles in maintaining a healthy body weight. By understanding these pathophysiological mechanisms behind these bio-factors, individuals can make informed choices and adopt lifestyle habits that support optimal weight management and overall health.

Pathophysiological explanations of body weight vs. glucose levels, blood pressures, blood lipids:

The relationship between body weight and glucose levels, blood pressure, and blood lipids can be explained through different pathophysiological mechanisms. Here are some explanations:

1. Glucose levels

Excess body weight, particularly in the form of excess adipose tissue (fat), can increase the risk of developing insulin resistance and type 2 diabetes. Adipose tissue produces hormones and inflammatory substances that disrupt the normal functioning of insulin, a hormone responsible for regulating blood glucose levels. In individuals with excess body weight, insulin resistance occurs, meaning the cells become less responsive to the effects of insulin. As a result, glucose cannot effectively enter the cells, leading to elevated blood glucose levels and the development of diabetes.

2. Blood pressure

Excess body weight, particularly abdominal or central obesity, is linked to the development of high blood pressure or hypertension. Adipose tissue releases various hormones and substances that increase inflammation and damage blood vessels. This can lead to a decrease in the elasticity of blood vessels, increased resistance to blood flow, and elevated blood pressure. Additionally, obesity leads to an increase in blood volume and cardiac output, further contributing to hypertension.

3. Blood lipids

Excess body weight and obesity are associated with dyslipidemia, characterized by elevated levels of triglycerides and LDL (low-density lipoprotein) cholesterol, and decreased levels of HDL (high-density lipoprotein) cholesterol. Obesity can lead to an imbalance in lipid metabolism, as adipose tissue releases fatty acids into the bloodstream and affects lipid transport and clearance. It also increases the production of triglycerides in the liver. Elevated triglyceride levels and LDL cholesterol contribute to the development of atherosclerosis, the buildup of plaque in the arteries, increasing the risk of cardiovascular disease.

Furthermore, obesity can disrupt the balance of adipokines, hormones produced by adipose tissue. Adipokines like adiponectin, which has anti-inflammatory and cardiovascular protective effects, may be reduced in obesity, further contributing to dyslipidemia and increased cardiovascular risk.

Overall, excess body weight, particularly excess fat deposition, can have detrimental effects on glucose metabolism, blood pressure regulation, and lipid metabolism, increasing the risk of metabolic disorders such as diabetes, hypertension, and dyslipidemia. Managing body weight through healthy lifestyle choices, including a balanced diet and regular exercise, is crucial for maintaining optimal glucose levels, blood pressure, and blood lipid profile.

Pathophysiological explanations of 4 metabolic disorders (obesity, diabetes, hypertension and dyslipidemia) versus 4 diseases (CVD, CKD, dementia and cancers):

1. Obesity

Pathophysiology: Obesity is a chronic condition characterized by excessive accumulation of body fat. It results from an imbalance between caloric intake and energy expenditure. Adipose tissue (fat cells) releases a variety of chemical substances called adipokines, including leptin, resistin, and adiponectin, which contribute to the development of insulin resistance, chronic inflammation, and metabolic dysfunction. These factors disrupt the regulation of appetite, glucose metabolism, and lipid metabolism, leading to the development of obesity-related complications.

Associated conditions:

-Cardiovascular disease (CVD): Obesity increases the risk of developing CVD, such as coronary artery disease, heart failure, and stroke. The chronic inflammation and metabolic abnormalities associated with obesity contribute to the development of atherosclerosis and cardiovascular complications.

-Chronic kidney disease (CKD): Obesity is a risk factor for the development and progression of CKD. It can lead to kidney damage through various mechanisms, including increased blood pressure, inflammation, oxidative stress, and abnormal lipid metabolism.

-Dementia: Obesity in midlife has been linked to an increased risk of dementia in later life. The chronic inflammation, insulin resistance, and impaired vascular function associated with obesity may contribute to cognitive decline and the development of neurodegenerative diseases.

-Cancers: Obesity is associated with an increased risk of various cancers, including breast, colorectal, endometrial, kidney, and pancreatic cancers. Chronic inflammation, insulin resistance, hormonal imbalances, and altered immune function are some of the mechanisms underlying the link between obesity and cancer.

2. Diabetes

Pathophysiology: Diabetes mellitus is a metabolic disorder characterized by elevated blood glucose levels (hyperglycemia) due to insufficient insulin production or impaired

insulin action. In type 1 diabetes, the body fails to produce insulin, whereas in type 2 diabetes, there is resistance to the effects of insulin. Both types involve complex interactions of genetic, environmental, and lifestyle factors. Insulin deficiency or resistance leads to abnormal glucose uptake and utilization in various tissues, resulting in hyperglycemia and metabolic dysfunction.

Associated conditions:

-Cardiovascular disease (CVD): Diabetes is a major risk factor for the development of CVD, including heart disease, heart failure, and stroke. Chronic hyperglycemia, insulin resistance, dyslipidemia, hypertension, and inflammation contribute to endothelial dysfunction, atherosclerosis, and cardiac complications.

-Chronic kidney disease (CKD): Diabetes is the leading cause of CKD. Prolonged exposure to high blood glucose levels damages the small blood vessels and filtration units of the kidneys, impairing their function over time.

-Dementia: Diabetes has been associated with an increased risk of cognitive decline and dementia. Chronic hyperglycemia, insulin resistance, oxidative stress, vascular damage, and inflammation may contribute to neurodegenerative processes.

-Cancers: Diabetes is associated with an increased risk of certain cancers, including liver, pancreatic, colorectal, and bladder cancers. The underlying mechanisms are not fully understood, but factors such as chronic hyperinsulinemia, insulin resistance, chronic inflammation, and altered hormone levels may play a role.

3. Hypertension

Pathophysiology: Hypertension, or high blood pressure, involves the sustained elevation of systemic arterial blood pressure. It can be classified as primary (essential) or secondary. Primary hypertension is the most common form and is multifactorial, typically resulting from a combination of genetic, environmental, and lifestyle factors. Secondary hypertension is caused by an underlying medical condition or medication. Various mechanisms contribute to the development of hypertension, including increased peripheral vascular resistance,

abnormal kidney function, and disrupted regulation of fluid and electrolyte balance.

Associated conditions:

-Cardiovascular disease (CVD): Hypertension is a major risk factor for CVD, including coronary artery disease, heart failure, and stroke. Elevated blood pressure leads to structural and functional changes in the blood vessels, promoting atherosclerosis, arterial stiffness, and cardiac complications.

-Chronic kidney disease (CKD): Hypertension is both a cause and consequence of CKD. Elevated blood pressure damages the blood vessels in the kidneys, impairing their function. Conversely, impaired kidney function can contribute to the development of hypertension.

-Dementia: Hypertension in midlife has been associated with an increased risk of dementia later in life. Chronic high blood pressure can lead to damage and narrowing of the blood vessels supplying the brain, compromising cerebral blood flow and increasing the risk of cognitive decline and dementia.

-Cancers: Chronic hypertension has been associated with an increased risk of certain cancers, such as kidney, colorectal, and breast cancers. The underlying mechanisms explaining this association are not fully understood, but factors like chronic inflammation and altered angiogenesis may contribute.

4. Dyslipidemia

Pathophysiology: Dyslipidemia refers to abnormal levels of lipids (cholesterol and triglycerides) in the bloodstream. It can involve elevated total cholesterol, LDL cholesterol, triglycerides, or low levels of HDL cholesterol. Dyslipidemia often occurs due to a combination of genetic predisposition, dietary factors, physical inactivity, and underlying medical conditions. It can result from an imbalance in lipid synthesis, transportation, or metabolism.

Associated conditions:

-Cardiovascular disease (CVD): Dyslipidemia is a major modifiable risk factor for CVD. Elevated levels of LDL cholesterol and triglycerides, along with low levels of HDL cholesterol, contribute to the development of

atherosclerosis, arterial plaque formation, and increased risk of heart disease, heart attack, and stroke.

-Chronic kidney disease (CKD): Dyslipidemia commonly occurs in CKD, and it contributes to the progression of kidney damage. Abnormal lipid metabolism and impaired clearance of lipid particles can lead to lipid deposition within the kidneys, promoting inflammation and fibrosis.

-Dementia: Dyslipidemia, especially elevated cholesterol levels, has been associated with an increased risk of cognitive decline and Alzheimer's disease. Dyslipidemia may contribute to the development of neuronal damage, inflammation, and impaired cerebral blood flow, all of which are implicated in dementia.

-Cancers: The relationship between dyslipidemia and cancer is complex and not fully understood. Some studies have suggested a link between dyslipidemia, particularly high cholesterol levels, and an increased risk of certain cancers, including breast, colorectal, and prostate cancers. However, the underlying mechanisms are still being investigated.

Pathophysiological explanations of 6 lifestyle details (diet, water drinking, exercise, sleep, stress, life routines) versus CVD, CKD, dementia, and cancers:

1) Diet

-Cardiovascular disease (CVD): High intake of unhealthy fats, cholesterol, and sodium can lead to the development of plaque in the arteries, contributing to atherosclerosis and increasing the risk of CVD.

-Chronic kidney disease (CKD): High intake of protein, especially animal protein, can increase the workload of the kidneys, potentially leading to kidney damage and CKD.

-Dementia: A diet high in saturated fats and sugar has been associated with cognitive decline and an increased risk of developing dementia.

-Cancers: Eating a diet high in processed foods, red meat, and sugary beverages can

increase the risk of various cancers, including colorectal, pancreatic, and breast cancer.

2) Water drinking

-CVD: Dehydration can increase blood viscosity, which in turn increases the risk of blood clot formation and cardiovascular events such as heart attacks and strokes.

-CKD: Chronic dehydration can contribute to the development and progression of CKD, as it puts additional strain on the kidneys.

-Dementia: Dehydration can lead to cognitive impairment and increase the risk of developing dementia.

-Cancers: Staying properly hydrated helps the body eliminate toxins and waste products, reducing the risk of cancer development.

3) Exercise

-CVD: Sedentary behavior and lack of physical activity contribute to weight gain, obesity, high blood pressure, and insulin resistance, all of which are risk factors for CVD.

-CKD: Regular exercise improves cardiovascular health, blood pressure regulation, and can help prevent or delay the progression of CKD.

-Dementia: Physical activity promotes brain health, enhances cognitive function, and reduces the risk of developing dementia.

-Cancers: Regular exercise reduces the risk of various cancers, including colon, breast, and lung cancer, by promoting a healthy weight, supporting immune function, and reducing chronic inflammation.

4) Sleep

-CVD: Poor sleep quality and sleep disorders, such as obstructive sleep apnea, can contribute to high blood pressure, inflammation, obesity, and insulin resistance, increasing the risk of CVD.

-CKD: Sleep disturbances have been associated with increased inflammation and decreased kidney function, potentially

contributing to the development or progression of CKD.

-Dementia: Chronic sleep disturbances and sleep-related breathing disorders have been linked to an increased risk of cognitive decline and dementia.

-Cancers: Disrupted sleep patterns and insufficient sleep have been associated with an increased risk of developing certain cancers, such as breast and colorectal cancer.

5) Stress

-CVD: Chronic stress can lead to increased blood pressure, inflammation, and the dysregulation of various physiological processes, contributing to the development of CVD.

-CKD: Psychological stress can affect renal function through various mechanisms, including increased sympathetic nervous system activity and hormonal changes, potentially accelerating the progression of CKD.

-Dementia: Chronic stress is associated with cognitive decline, accelerated brain aging, and an increased risk of developing dementia.

-Cancers: Prolonged exposure to stress hormones can weaken the immune system, impair DNA repair mechanisms, and promote tumor growth, increasing the risk of cancer development.

6) Life routines

-CVD: Unhealthy lifestyle routines, such as smoking, excessive alcohol consumption, and lack of regular health check-ups, contribute to the development of CVD.

-CKD: Certain routines that involve exposure to toxins, such as occupational hazards or chronic use of nonsteroidal anti-inflammatory drugs, can damage the kidneys and lead to CKD.

-Dementia: Engaging in mentally stimulating activities, maintaining social connections, and having a consistent routine can help reduce the risk of dementia and cognitive decline.

-Cancers: Occupational exposures, such as asbestos, benzene, and certain heavy metals, as well as unhealthy habits like smoking and excessive alcohol consumption, can increase the risk of developing various cancers.

Pathophysiological explanations of longevity versus CVD, CKD, dementia, cancers and T2D:

1) Longevity

-Cardiovascular disease (CVD): Longevity is associated with a lower risk of CVD as individuals who live longer generally have better cardiovascular health, including lower blood pressure, improved blood lipid profiles, and healthier blood vessels.

-Chronic kidney disease (CKD): Longevity is generally associated with a lower risk of CKD, as individuals who live longer tend to have better kidney function and fewer risk factors for CKD, such as diabetes and high blood pressure.

-Dementia: Longevity does not necessarily protect against dementia, as age is still the primary risk factor for dementia. However, certain lifestyle factors associated with longevity, such as physical activity and cognitive engagement, can help reduce the risk of cognitive decline and dementia.

-Cancers: Longevity is associated with an increased risk of developing certain types of cancers, as cells accumulate genetic mutations over time, increasing the likelihood of cancerous growth. However, maintaining a healthy lifestyle, including regular screening and risk reduction strategies, can help mitigate the risk.

-Type 2 diabetes (T2D): Longevity is associated with a higher risk of developing T2D due to the cumulative effects of insulin resistance, age-related changes in metabolism, and other factors. However, lifestyle modifications, such as maintaining a healthy weight and engaging in regular physical activity, can help prevent or manage T2D even in older adults.

It is important to note that longevity alone does not guarantee protection against these diseases, as genetic predispositions, environmental factors, and individual variations play a significant role. However,

adopting a healthy lifestyle that includes proper nutrition, regular exercise, good sleep habits, stress management, and avoiding harmful habits can contribute to overall health and potentially reduce the risk of these diseases, promoting longevity and well-being.

2. METHODS

2.1 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 describes his MPM methodology in a general conceptual format. The second paper, No. 387 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 depicts a general flow diagram containing ~10 key MPM research methods and different tools.

2.2 The author's diabetes history

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

In 2010, he decided to self-study endocrinology with an emphasis on diabetes and food nutrition. He spent the entire year of 2014 to develop a metabolism index (MI) mathematical model. During 2015 and 2016, he developed four mathematical prediction models related to diabetes conditions: weight, PPG, fasting plasma glucose (FPG), and HbA1C (A1C). Through using his developed mathematical metabolism index (MI) model and the other four glucose prediction tools, by the end of 2016, his weight was reduced from

220 lbs. (100 kg) to 176 lbs. (89 kg), waistline from 44 inches (112 cm) to 33 inches (84 cm), average 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 takes any diabetes-related medications since 12/8/2015.

In 2017, he achieved excellent results on all fronts, especially his glucose control. However, during the 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 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 over 40,000 hours and read over 4,000 published medical papers online in the past 13 years, he discovered and became convinced that good life habits of not

smoking, moderate or no alcohol intake, avoiding illicit drugs; along with eating the right food with 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.

2.3 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 an example, the combination of hyperglycemia and hypertension would cause micro-blood vessel's leakage in kidney systems which is one of the major cause of CKD.

The author then applied Fast Fourier Transform (FFT) operations to convert the input wave from a time domain into a frequency domain. The y-axis amplitude

values in the frequency domain indicate the proportional energy levels associated with each different frequency component of input occurrence. Both output symptom value (i.e. strain amplitude in the time domain) and output symptom fluctuation rate (i.e. the strain rate and strain frequency) are influencing the energy level (i.e. the Y-amplitude in the frequency domain).

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

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

2.4 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 reciprocal of Young’s modulus E.

However, in viscoelasticity or viscoplasticity theory, the stress is expressed as:

Stress
= viscosity factor (η : eta) * strain rate ($d\epsilon/dt$)

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 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 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 the data table.

The table contains multiple sections of data, including energy ratios for different categories like BW vs. Food Quality, Medical Conditions vs. 4 Diseases, Glucose/BP/Lipids vs. Lifestyle Details, MI vs. 5 Diseases, and Longevity vs. 4 Diseases. Each section includes numerical values for various parameters and their corresponding energy ratios.

Figure 1: SD-VMT data table.

Figure 2 shows summary results.

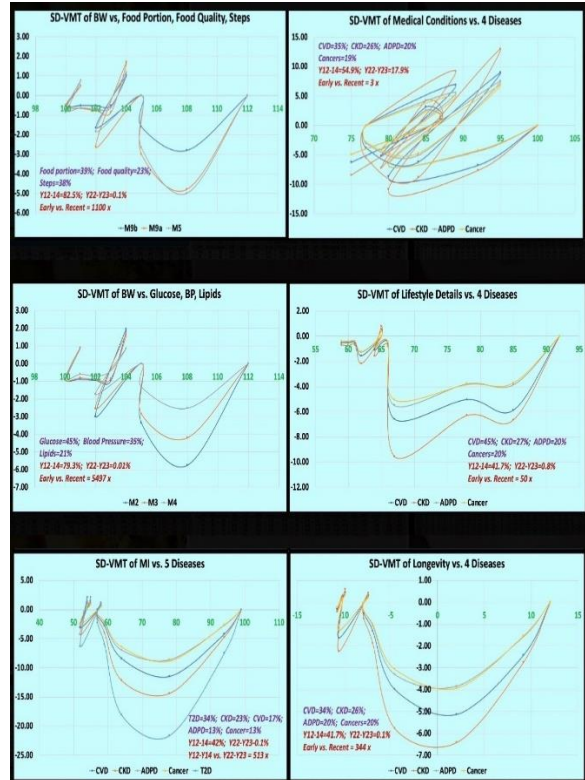


Figure 2: Summary results.

4. CONCLUSION

In summary, there are two key findings:

1. The author uncovers energy ratios representing degrees of influence, contribution, or association from six distinct but interconnected SD-VMT studies.

-Energy ratios involving body weight: Diet holds a 62% influence (food portion contributes 39%, food quality contributes 23%), compared to exercise at 38%. The diet-to-exercise ratio is 1.63.

-Energy ratios for obesity and three metabolic disorders (MD): Diabetes at 45%, hypertension at 35%, and dyslipidemia at 21%.

-Energy ratios for obesity and three metabolic conditions (MC) with 4 mortality diseases: CKD (35%), CVD (26%), ADPD (20%), and Cancer (19%).

-Energy ratios for lifestyle details (LD) with 4 mortality diseases: CKD (34%), CVD (27%), ADPD (20%), and Cancer (19%).

-Energy ratios linking longevity perspective with 4 mortality diseases: CKD (34%), CVD (26%), ADPD (20%), and Cancer (20%).

-Associations revealing MI and five mortality diseases: T2D (34%), CKD (23%), CVD (17%), ADPD (13%), Cancer (13%).

2. Averaged total energy calculations indicate a comparison between two initial years and two recent years: 57% for Y2013-Y2014 versus 3.2% for Y2022-Y2023. The 16-fold higher energy associated with the initial period signifies significant improvement in his health conditions, with no regression to the initial unhealthy state. These observations are reflected in those viscoplastic curves.

5. REFERENCES

For editing purposes, majority of the references in this paper, which are self-references, have been removed for this article. Only references from other authors' published sources remain. The bibliography

of the author's original self-references can be viewed at www.eclaircmd.com.

Readers may use this article as long as the work is properly cited, and their use is educational and not for profit, and the author's original work is not altered.

For reading more of the author's published VGT or FD analysis results on medical applications, please locate them through 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).

Viscoelastic and Viscoplastic Glucose Theory Application in Medicine

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