Special Issue 5
2022

The GH-Method

Viscoelastic Medicine Theory (VMT #332): Differences of VMT-Based Total Energies Associated with Chronic Kidney Diseases, Cardiovascular Diseases, and Cancers Using Three Measured Inputs of Body Weight, FPG, PPG from 2015 to 2023 Based on GH-Method: Math-Physical Medicine (No. 932)

Gerald C. Hsu*

eclaireMD Foundation, USA

Abstract

Since 2015, the author has dedicated on developing accurate prediction equations for some biomarkers. Specifically, he has formulated three prediction equations for body weight, fasting glucose (FPG), and post-meal glucose (PPG). (1) Body weight prediction: Predicted BW in the early morning = Yesterday's BW in early morning + Yesterday's food quantity (mIa) + Yesterday's H20 drinking (m6) - Yesterday's bowel movement / 5 - Last night's sleeping hours / 10. (2) Statistical glucose prediction: Calculate standard deviations of X (body weight) and Y (FPG or PPG); Calculate sumSX or sunSY is the summation of the squared X or squared Y; Calculate correlation R between X and Y; sdX = sqrt (sumX / number of X), sdY = sqrt (sumY / number of Y), b = R * sdY / sdX, a = avgY - b * avgX. Predicted glucose Y = a + b * weight X. (3) Predicted PPG using linear elastic glucose theory (LEGT): Predicted LEGT PPG = FPG*0.9 + (carbs/sugar grams) * 3.4 - (post-meal walking steps / 1000) * 4. This study examines the interactions between three identical inputs as shown above, and the risks of three diseases, namely chronic kidney diseases (CKD), cardiovascular diseases (CVD), and cancers over a period from 1/1/2015 to 9/22/2023. The main objective of this study is to assess the variations in total energy associated with three diseases (CKD, CVD, Cancers) in relation to three identical inputs (body weight, FPG, PPG). The total energy is calculated through the size of enclosed area of the output-input curve in the SD-VMT diagram. These disparities of total energy can provide some useful insights into the relative risks of developing these three mortality diseases. The author also provides his personal medical history to interpret these findings. In summary, this analysis yields four observations: 1. The VMT analysis reveals the following three total energy ratios: CKD has 133 energy (4.0 times higher than cancers), CVD has 86 energy (2.6 times higher than cancers), and cancer has 33 energy (1.0 times of its own energy). These quantitative observations matches well with the author's medical history of CKD between 2009 and 2011, 5 CVD episodes between 1994 and 2004, and the absence of any symptoms or diagnosis of cancers. 2. The VMT analysis exhibits almost identical energy contribution ranges from these three measured inputs: body weight is 32%-33%, FPG is 36%, and PPG is 31%. 3. The VMT-based predicted disease risks exhibit a 100% prediction accuracy with strong correlations compared against the metabolism-index (MI) based disease risks: CKD is 83%-85%, CVD is 78%-79%, and Cancer is 81%-85%. 4. Three disease-input curves have quite similar shapes of waveform resulted from using the same three measured inputs, but they have quite different magnitudes of curves due to different rates of risk change.

Keywords: Viscoelastic; Viscoplastic; Cardiovascular diseases; Chronic kidney diseases; Cancers; Body weight; Diabetes; Exercise

Abbreviations: MI: metabolism index; CVD: cardiovascular diseases; CKD: chronic kidney diseases; T2D: type 2 diabetes; PPG: postprandial plasma glucose; FPG: fasting plasma glucose

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*Corresponding author: Gerald C. Hsu, eclaireMD Foundation, USA

1. INTRODUCTION

Since 2015, the author has dedicated on developing accurate prediction equations for some biomarkers. Specifically, he has formulated three prediction equations for body weight, fasting glucose (FPG), and post-meal glucose (PPG).

(1) Body weight prediction:
Predicted BW in the early morning
= Yesterday's BW in early morning
+ Yesterday's food quantity (mIa)
+ Yesterday's H20 drinking (m6)
- Yesterday's bowel movement / 5
- Last night's sleeping hours / 10

(2) Statistical glucose prediction:
Calculate standard deviations of X (body weight) and Y (FPG or PPG);
Calculate sumSX or sunSY is the summation of the squared X or squared Y;
Calculate correlation R between X and Y;
sdX = sqrt (sumX / number of X)
sdY = sqrt (sumY / number of Y)
b = R * sdY / sdX
a = avgY - b * avgX
Predicted glucose Y
= a + b * weight X

(3) Predicted PPG using linear elastic glucose theory (LEGT):
Predicted LEGT PPG
= FPG*0.9 + (carbs/sugar grams) * 3.4 - (post-meal walking steps / 1000) * 4

This study examines the interactions between three identical inputs as shown above, and the risks of three diseases, namely chronic kidney diseases (CKD), cardiovascular diseases (CVD), and cancers over a period from 1/1/2015 to 9/22/2023.

The main objective of this study is to assess the variations in total energy associated with three diseases (CKD, CVD, Cancers) in relation to three identical inputs (body weight, FPG, PPG). The total energy is calculated through the size of enclosed area of the output-input curve in the SD-VMT diagram. These disparities of total energy can provide some useful insights into the relative risks of developing these three mortality diseases. The author also provides his personal medical history to interpret these findings.

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.

Notes from the author of this paper:

Upon reviewing the upcoming excerpts from other published articles, it becomes evident that these findings are predominantly conveyed using qualitative statements. On occasion, these statements include a limited number of numerical values, typically sourced from statistical data within epidemiological studies. However, a recurring deficiency among them is the lack of robust quantitative findings to underpin their qualitative conclusions. Consequently, the author of this paper has deliberately opted to leverage his familiar methodologies from mathematics, physics, and engineering fields in his medical research pursuits. This strategic choice is intended to yield substantial conclusions supported by sound proofs via quantitative data, effectively bridging the current gap in the realm of biomedical research.

Pathophysiological explanations and statistical data regarding relationships between chronic kidney diseases (CKD) versus both body weight and glucoses:

Chronic kidney diseases (CKD) have been extensively studied in relation to body weight and glucose levels, with pathophysiological explanations and statistical data shedding light on their complex relationships.

Pathophysiological explanations

1. Body weight and CKD

Excess body weight, particularly obesity, is often associated with an increased risk of

CKD development and progression. Several mechanisms contribute to this relationship, such as:

-Hemodynamic changes: Obesity can lead to elevated blood pressure and increased renal blood flow, resulting in glomerular hyperfiltration and subsequent kidney damage.

-Inflammatory processes: Adipose tissue secretes pro-inflammatory cytokines, which promote inflammation and oxidative stress, creating a pathological environment that can contribute to renal damage.

-Insulin resistance: Obesity often coincides with insulin resistance, which may impair renal function through various pathways, including the activation of the renin-angiotensin-aldosterone system.

2. Glucose levels and CKD

Elevated glucose levels, as seen in diabetes, play a crucial role in the development and progression of CKD. Key mechanisms involve:

-Glomerular hemodynamic changes: Hyperglycemia affects the delicate balance of vasoactive substances within the kidney, leading to glomerular hyperfiltration and subsequent damage to the renal structures.

Accumulation of advanced glycation end products (AGEs): High glucose levels contribute to the formation of AGEs, which can promote inflammation, fibrosis, and oxidative stress in the kidneys.

-Activation of inflammatory pathways: Hyperglycemia triggers the release of cytokines and chemokines, promoting low-grade inflammation within the renal tissue.

Statistical data

Statistical studies have provided robust evidence supporting the relationships between CKD, body weight, and glucose levels. Here are a few noteworthy findings:

1. Body weight and CKD:

-Population-based studies have consistently demonstrated an increased risk of CKD among individuals with higher body mass index (BMI) values. Higher BMI is associated with a higher prevalence of albuminuria and declining glomerular filtration rate (GFR).

-Longitudinal studies have shown that weight loss interventions, such as lifestyle modifications or bariatric surgery, can improve renal outcomes and slow the progression of CKD.

2. Glucose levels and CKD

-Diabetes, characterized by elevated glucose levels, is a well-established risk factor for CKD. Diabetic nephropathy is one of the leading causes of end-stage renal disease (ESRD).

-Glycemic control, as reflected by hemoglobin A1c (HbA1c) levels, has a significant impact on the development and progression of CKD in individuals with diabetes. Tight glycemic control has been associated with reduced risk of CKD complications.

These pathophysiological explanations and statistical findings underscore the importance of addressing body weight and glucose levels in the prevention and management of CKD. They provide valuable insights for developing targeted interventions and strategies aimed at mitigating the burden of CKD on affected individuals.

Please note that for specific and detailed statistical data, referring to recent research articles and studies in this field will provide the most accurate and up-to-date information.

Pathophysiological explanations and statistical data regarding relationships between cardiovascular diseases (CVD) versus both body weight and glucoses:

Pathophysiological explanations

The relationships between cardiovascular diseases (CVD) and body weight as well as glucose levels are complex and multifactorial. However, there are several pathophysiological explanations that help elucidate these relationships:

1. Body weight and CVD

Obesity is a well-established risk factor for the development of CVD. Excess body weight, particularly abdominal obesity, is associated with a higher prevalence of metabolic syndrome, which includes multiple cardiovascular risk factors such as hypertension, dyslipidemia, and insulin resistance.

Adipose tissue produces and releases various bioactive substances called adipokines, which can directly contribute to endothelial dysfunction, atherosclerosis, and the development of CVD.

Inflammatory processes and oxidative stress associated with obesity can promote the formation of plaques in blood vessels, leading to atherosclerosis and subsequent cardiovascular events.

2. Glucose levels and CVD

Elevated glucose levels, as seen in diabetes, significantly increase the risk of CVD. Several mechanisms contribute to this relationship:

Hyperglycemia can damage the endothelial lining of blood vessels, impairing vascular function and promoting atherosclerosis.

Diabetes is often associated with dyslipidemia, characterized by elevated triglycerides, lower high-density lipoprotein (HDL) cholesterol levels, and higher levels of small, dense low-density lipoprotein (LDL) particles, all of which contribute to the development of atherosclerosis.

Insulin resistance, commonly observed in diabetes, can lead to abnormalities in lipid metabolism and blood pressure regulation, further increasing the risk of CVD.

Statistical data

Extensive research has examined the relationships between CVD, body weight, and glucose levels. Here are a few key findings from statistical studies:

1. Body weight and CVD

Population-based studies consistently show a strong association between obesity and increased risk of CVD, including coronary artery disease, stroke, and heart failure.

The risk of CVD increases progressively with increasing body mass index (BMI), with individuals classified as obese (BMI>30 kg/m^2) having a substantially higher risk compared to those within the normal weight range.

Weight loss interventions, including lifestyle modifications and bariatric surgery, have been shown to reduce CVD risk factors and improve cardiovascular outcomes.

2. Glucose levels and CVD

Individuals with diabetes have a significantly higher risk of CVD compared to those without diabetes. The risk is further elevated in individuals with poorly controlled glucose levels.

Long-term epidemiological studies, such as the Framingham Heart Study, have demonstrated a strong association between diabetes and the incidence of CVD, including coronary artery disease, heart failure, and stroke.

Glycemic control, measured by hemoglobin A1c (HbA1c) levels, has been found to have a significant impact on reducing the risk of CVD complications in individuals with diabetes.

These pathophysiological explanations and statistical findings underline the importance of managing body weight and glucose levels in the prevention and management of CVD. They provide critical insights for developing targeted interventions and strategies aimed at reducing the burden of CVD on affected individuals.

Please note that for specific and detailed statistical data, referring to recent research articles and studies in this field will provide the most accurate and up-to-date information.

Pathophysiological explanations and statistical data regarding relationships between cancers versus both body weight and glucoses:

Pathophysiological mechanisms and statistical data provide valuable insights into the relationships between cancers and body weight, as well as glucose levels such as

fasting plasma glucose (FPG) and postprandial plasma glucose (PPG).

Various studies have shown that obesity is strongly associated with an increased risk of several types of cancers. Excess body weight can lead to chronic inflammation, insulin resistance, and hormonal imbalances, creating an environment that promotes cancer development and progression. The adipose tissue itself releases pro-inflammatory molecules and hormones that can contribute to cancer initiation and growth. Additionally, obesity is linked to changes in adipokines, which are involved in regulating cell growth and differentiation.

Regarding glucose levels, elevated FPG and PPG levels have also been associated with an increased risk of cancer. Prolonged exposure to high glucose levels can lead to oxidative stress and the production of advanced glycation end-products (AGEs), which can damage cells and DNA. This, in turn, can promote cancer formation and growth.

Statistical data supports these pathophysiological explanations. Research studies have consistently shown a positive correlation between obesity and the incidence of various cancers, including breast, colorectal, endometrial, kidney, liver, pancreatic, and ovarian cancer, among others. Similarly, studies have found that individuals with higher FPG and PPG levels are more likely to develop certain types of cancer, such as colorectal, pancreatic, and liver cancer.

Taken together, pathophysiological mechanisms and statistical data emphasize the importance of maintaining a healthy body weight and managing glucose levels to reduce the risk of cancer development and progression. These findings highlight the significance of lifestyle modifications, including regular exercise, balanced diet, and glucose control, in cancer prevention and management.

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 (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 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 6 input data tables.

Figure 2 shows 2 output charts.

Figure 3 shows 3 SD-VMT diagrams.

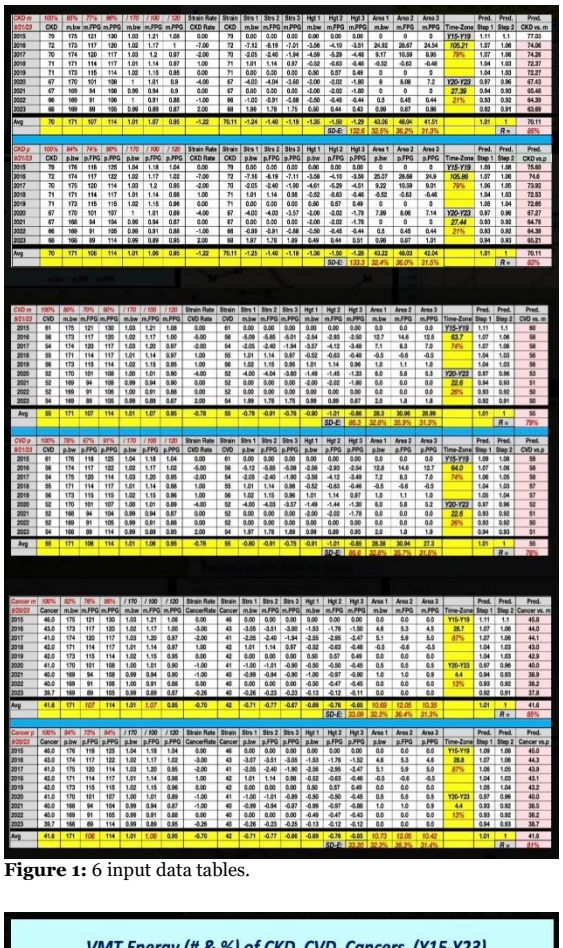
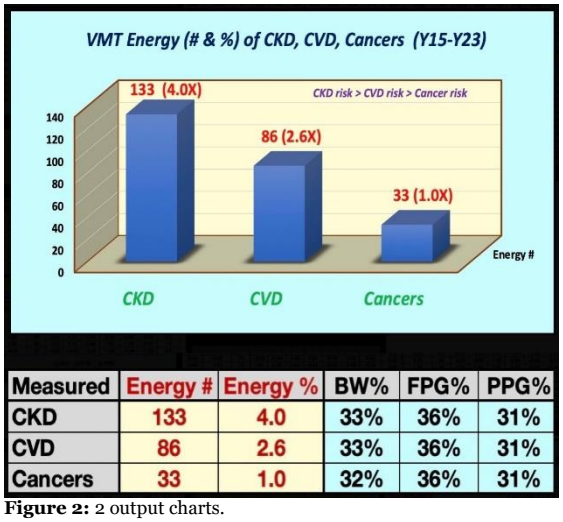


Figure 1: 6 input data tables.



Measured	Energy #	Energy %	BW%	FPG%	PPG%
CKD	133	4.0	33%	36%	31%
CVD	86	2.6	33%	36%	31%
Cancers	33	1.0	32%	36%	31%

Figure 2: 2 output charts.

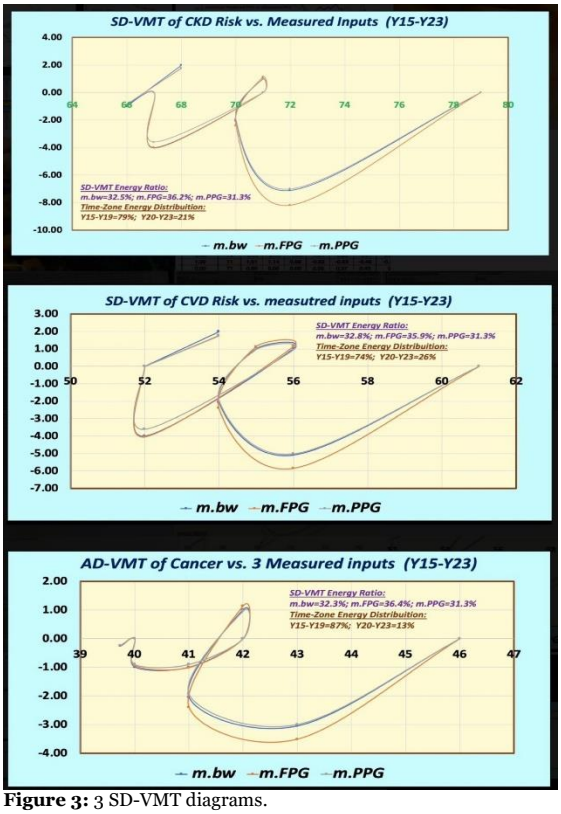


Figure 3: 3 SD-VMT diagrams.

4. CONCLUSION

In summary, this analysis yields four observations:

1. The VMT analysis reveals the following three total energy ratios: CKD has 133 energy (4.0 times higher than cancers), CVD has 86 energy (2.6 times higher than cancers), and cancer has 33 energy (1.0 times of its own energy). These quantitative observations matches well with the author's medical history of CKD between 2009 and 2011, 5 CVD episodes between 1994 and 2004, and the absence of any symptoms or diagnosis of cancers.

2. The VMT analysis exhibits almost identical energy contribution ranges from these three measured inputs: body weight is 32%-33%, FPG is 36%, and PPG is 31%.

3. The VMT-based predicted disease risks exhibit a 100% prediction accuracy with strong correlations compared against the metabolism-index (MI) based disease risks: CKD is 83%-85%, CVD is 78%-79%, and Cancer is 81%-85%.

4. Three disease-input curves have quite similar shapes of waveform resulted from

using the same three measured inputs, but they have quite different magnitudes of curves due to different rates of risk change.

5. REFERENCES

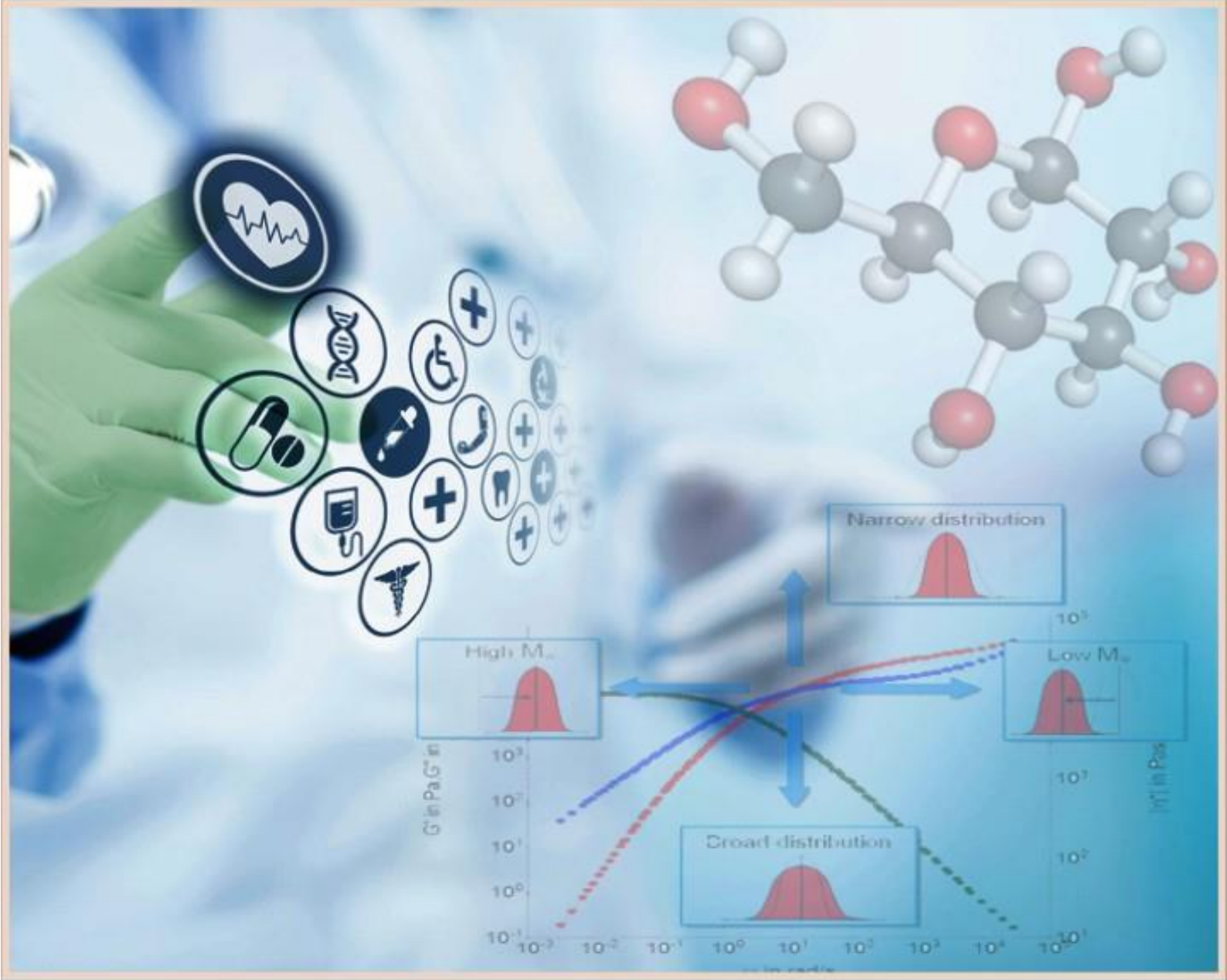
For editing purposes, majority of the references in this paper, which are self-references, have been removed for this article. Only references from other authors' published sources remain. The bibliography of the author's original self-references can be viewed at www.eclairemd.com.

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

Gerald C. Hsu



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