

# The GH-Method

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## Viscoelastic Medicine Theory (VMT #331): Variations in Cardiovascular Diseases Risk Analysis Using Measured and Predicted Body Weight and Glucose Inputs from 2015 to 2023 Applying the Viscoplastic Energy Model of GH-Method: Math-Physical Medicine (No. 931)

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### Abstract

Since 2015, the author has focused on developing prediction equations for certain important biomarkers. Specifically, he has created the following 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 H2O 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 sumSY is the summation of the squared X or squared Y; Calculate correlation R between X and Y;  $sdX = \sqrt{\text{sum}X / \text{number of } X}$ ,  $sdY = \sqrt{\text{sum}Y / \text{number of } Y}$ ,  $b = R * sdY / sdX$ ,  $a = \text{avg}Y - b * \text{avg}X$ . Predicted glucose  $Y = a + b * \text{weight } X$ . (3) Predicted PPG using linear elastic glucose theory (LEGT): Predicted LEGT PPG =  $\text{FPG} * 0.9 + (\text{carbs/sugar grams}) * 3.4 - (\text{post-meal walking steps} / 1000) * 4$ . This study focuses on using three significant input factors, body weight, fasting glucose (FPG), and post-meal glucose (PPG), to estimate his cardiovascular diseases (CVD) risks from January 1, 2015, to September 20, 2023. He

performs two quantitative analyses using the viscoplastic energy model (VMT) with two different input datasets, both measured and predicted. He also utilized his VMT-based prediction model to calculate another CVD risk for comparative analysis. The purpose is to assess the differences between using these two datasets. If the disparities of results are minimal, indicating a close alignment, it then demonstrates the high accuracy and practical applicability of the author's predicted biomarker equations in real-life patient scenarios. In summary, this analysis reveals two observations: 1. Space-domain viscoplastic energy (SD-VMT) analysis: The energy ratios for both measured and predicted body weight, FPG, and PPG are almost identical, with body weight accounting for 32.8%, FPG accounting for 35.7%-35.9%, and PPG accounting for 31.3%-31.5%. Similarly, the distribution of energy in the time zones is identical, with Y15-Y19 accounting for 74% and Y20-Y23 accounting for 26%. 2. VMT-based CVD risk curves: The CVD risk curves generated using VMT for both measured and predicted data closely match each other. These two curves exhibit correlation coefficients of 79% (measured) and 78% (predicted) when compared to the MI-based CVD risk.

**Keywords:** Viscoelastic; Viscoplastic; Chronic kidney diseases; 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

## 1. INTRODUCTION

Since 2015, the author has focused on developing prediction equations for certain important biomarkers. Specifically, he has created the following 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 H2O 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 sumSY is the summation of the squared X or squared Y;  
Calculate correlation R between X and Y;  
 $sdX = \sqrt{\text{sumX} / \text{number of X}}$   
 $sdY = \sqrt{\text{sumY} / \text{number of Y}}$   
 $b = R * sdY / sdX$   
 $a = \text{avgY} - b * \text{avgX}$   
Predicted glucose Y  
 $= a + b * \text{weight X}$

(3) Predicted PPG using linear elastic glucose theory (LEGT):

Predicted LEGT PPG  
 $= \text{FPG} * 0.9 + (\text{carbs/sugar grams}) * 3.4 - (\text{post-meal walking steps} / 1000) * 4$

This study focuses on using three significant input factors, body weight, fasting glucose (FPG), and post-meal glucose (PPG), to estimate his cardiovascular diseases (CVD) risks from January 1, 2015, to September 20, 2023. He performs two quantitative analyses using the viscoplastic energy model (VMT) with two different input datasets, both measured and predicted. He also utilized his VMT-based prediction model to calculate another CVD risk for comparative analysis.

The purpose is to assess the differences between using these two datasets. If the disparities of results are minimal, indicating a close alignment, it then demonstrates the high accuracy and practical applicability of the author's predicted biomarker equations in real-life patient scenarios.

### 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 cardiovascular diseases 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<sup>2</sup>) 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.

## 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 ( $\epsilon$ : epsilon)  
= Stress ( $\sigma$ : 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:



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2. VMT-based CVD risk curves: The CVD risk curves generated using VMT for both measured and predicted data closely match each other. These two curves exhibit correlation coefficients of 79% (measured) and 78% (predicted) when compared to the MI-based CVD risk.

## **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.eclairermd.com](http://www.eclairermd.com).

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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.

# Viscoelastic and Viscoplastic Glucose Theory Application in Medicine

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