Research Article

Assessment of Six Diseases Resulted From Four Metabolic Biomarkers Using both Statistical Correlation and Viscoplastic Energy Model of Gh-Method: Math-Physical Medicine (No. 955, VMT #354, 11/15-17/2023)

Gerald C Hsu*

EclaireMD Foundation, USA

*Corresponding Author
Gerald C Hsu, EclaireMD Foundation, USA

Submitted: 2023, Dec 04; **Accepted:** 2024, Jan 08; **Published:** 2024, Jan 24

Abstract

After completing three studies on fundamental aspects of metabolic disorders, specifically body weight (BW), fasting glucose in the early morning (FPG), and post-meal glucose (PPG), the author has scientifically demonstrated that his food portion is the primary factor influencing his BW. And his BW is intricately linked with his FPG, indicating the health status of pancreatic beta cells' insulin quality and emission. Furthermore, his FPG serves as the baseline for his PPG, contributing 50% or more, with diet and exercise as two most important secondary influential factors. The specific ratio between his carbs/sugar intake and post-meal walking steps exhibits a notable ratio of 1.6 (31% dividing by 19%), mirroring the ratio between two pathophysiological pathways of diet (8) versus exercise (5).

Building on these foundations, the author extended his research to investigate six deadly diseases versus four basic biomarkers, including body weight (m1), glucose levels (m2), blood pressures (m3), and blood lipids or cholesterols (m4). This article explores hidden relationships and dynamics (i.e. energies) between these 6 diseases and 4 metabolic biomarkers. His data collection started in 2012 (with incomplete data), focusing on an 11year period from 1/1/2013 to 11/14/2023. Correlations between the six deadly diseases and both body weight (m1) and glucose levels (m2) are higher than those with blood pressure (m3) and cholesterol (m4). These statistical findings resonate closely with the author's observation of his personal health journey, having experienced obesity (220 lbs with BMI 32.5) and type 2 diabetes (averaged daily glucose around 200 mg/dL and postprandial glucose hyperglycemia around 300 mg/dL). Notably, his blood pressure (m3) was elevated during 2013-2014 only, while his cholesterol (m4) exhibited fluctuations below a normalized 100% level (safe ranges).

In summary, *the annual averaged risks of six deadly diseases exhibit a downward trend resembling different skiing slopes from 2013 to 2023. Correspondingly, the annual averaged biomarker values, including body weight and glucose, are also decreasing, while blood pressure and blood lipids fluctuate within safe ranges (below 100%).*

*Applying traditional statistical calculations reveals **the "averaged" correlations between these six deadly diseases and four distinct biomarkers:***
- ***6 diseases vs. weight: 90%***
- ***6 diseases vs. glucose: 90%***
- ***6 diseases vs. blood pressure: 73% - 6 diseases vs. lipids: -26%***

*Utilizing space-domain viscoplastic energy analysis, his **study identifies four energy contribution** margins for these six deadly diseases:*
- ***Energy from body weight: 29%***
- ***Energy from glucose: 29%***
- ***Energy from blood pressure: 25% - Energy from blood lipids: 16% Key Message:***
- ***The majority of Type 2 diabetes patients face overweight or obesity challenges. The interplay between obesity and diabetes escalates the risk of six diseases: heart attacks & strokes, kidney problems, cancers, Alzheimer's disease, Parkinson's disease, and diabetic neuropathy (e.g., foot ulcers leading to amputations), all linked to high mortality rates.***

Correlation	M1	M2	M3	M4
CVD	92%	80%	81%	-22%
CKD	91%	94%	73%	-32%
Cancer	86%	81%	84%	-18%
AD	87%	92%	74%	-25%
PD	92%	96%	62%	-28%
DN	94%	95%	65%	-33%
Average	90%	90%	73%	-26%

VMT Energy	M1	M2	M3	M4
CVD	29%	30%	26%	15%
CKD	29%	29%	25%	17%
Cancer	29%	29%	25%	17%
AD	29%	29%	25%	17%
PD	30%	29%	25%	16%
DN	29%	30%	26%	16%
Average	29%	29%	25%	16%

1. Introduction

After completing three studies on fundamental aspects of metabolic disorders, specifically body weight (BW), fasting glucose in the early morning (FPG), and post-meal glucose (PPG), the author has scientifically demonstrated that his food portion is the primary factor influencing his BW. And his BW is intricately linked with his FPG, indicating the health status of pancreatic beta cells' insulin quality and emission. Furthermore, his FPG serves as the baseline for his PPG, contributing 50% or more, with diet and exercise as two most important secondary influential factors. The specific ratio between his carbs/sugar intake and post-meal walking steps exhibits a notable ratio of 1.6 (31% dividing by 19%), mirroring the ratio between two pathophysiological pathways of diet (8) versus exercise (5). Building on these foundations, the author extended his research to investigate six deadly diseases versus four basic biomarkers, including body weight (m1), glucose levels (m2), blood pressures (m3), and blood lipids or cholesterols (m4).

This article explores hidden relationships and dynamics (i.e. energies) between these 6 diseases and 4 metabolic biomarkers. His data collection started in 2012 (with incomplete data), focusing on an 11year period from 1/1/2013 to 11/14/2023. Correlations between the six deadly diseases and both body weight (m1) and glucose levels (m2) are higher than those with blood pressure (m3) and cholesterol (m4). These statistical findings resonate closely with the author's observation of his personal health journey, having experienced obesity (220 lbs with BMI 32.5) and type 2 diabetes (averaged daily glucose around 200 mg/dL and postprandial glucose hyperglycemia around 300 mg/dL). Notably, his blood pressure (m3) was elevated during 2013-2014 only, while his cholesterol (m4) exhibited fluctuations below a normalized 100% level (safe ranges).

2. 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.

3. Pathophysiological Explanations and Statistics Data Regarding 6 Diseases, Specifically CVD, CKD, Cancers, Alzheimer's Diseases, Parkinson's disease, and Diabetic Neuropathy

There is extensive scientific research and literature on the pathophysiological explanations and statistics concerning various diseases. Here's a brief overview:

3.1 Cardiovascular Disease (CVD)

• CVD encompasses a range of conditions affecting the heart and blood vessels, including coronary artery disease, heart failure, and stroke. Common pathophysiological causes include atherosclerosis, hypertension, and chronic inflammation.

• Statistics: According to the World Health Organization (WHO), CVD is the leading cause of death globally, accounting for approximately 32% of all deaths. Around 17.9 million people die from CVDs each year, and this number is expected to rise.

3.2 Chronic Kidney Disease (CKD):
• CKD involves the gradual loss of kidney function over time and is often associated with conditions like diabetes and hypertension. Pathophysiological mechanisms include glomerular damage, tubulointerstitial fibrosis, and impaired renal function.
• Statistics: The National Kidney Foundation reports that over 37 million American adults have CKD, and millions more are at increased risk. CKD is a major risk factor for cardiovascular events and is associated with a higher risk of mortality.

3.3 Cancer
• Cancer is a complex group of diseases characterized by the uncontrolled division and spread of abnormal cells. Pathophysiological mechanisms vary depending on the type of cancer and can include genetic mutations, environmental factors, and immune system dysfunction.
• Statistics: According to WHO, cancer is the second leading cause of death globally, responsible for an estimated 9.6 million deaths in 2018. The number of new cancer cases is expected to rise to 29.5 million by 2040.

3.4 Alzheimer's Disease
• Alzheimer's is a progressive neurodegenerative disorder characterized by cognitive decline and memory loss. Pathophysiologically, it involves the accumulation of beta-amyloid plaques and tau protein tangles in the brain and neuronal degeneration.
• Statistics: Alzheimer's is the most common cause of dementia, and an estimated 50 million people worldwide are living with dementia. This number is projected to triple by 2050.

3.5 Parkinson's Disease
• Parkinson's is a neurodegenerative disorder characterized by motor symptoms, such as tremors and bradykinesia. Pathophysiologically, it involves the loss of dopamineproducing neurons in the brain's substantia nigra.
• Statistics: According to the Parkinson's Foundation, about 60,000 Americans are diagnosed with Parkinson's disease each year, and an estimated 1 million people in the United States are living with the disease.

3.6 Diabetic Neuropathy
• Diabetic neuropathy is a type of nerve damage that can occur in people with diabetes. It is caused by chronically high blood sugar levels and can affect nerves throughout the body.
• Statistics: Diabetic neuropathy is a common complication of diabetes, occurring in up to 50% of people with diabetes. It can lead to significant morbidity, including foot ulcers, infections, and lower limb amputations.
These statistics and pathophysiological explanations underscore the significant public health impact of these diseases and the need for ongoing research, preventive strategies, and effective treatments to address them.

4. Pathophysiological Explanations of Aforementioned 6 Deadly Diseases with Inputs of obesity, Diabetes, Hypertension and Dyslipidemia
Here are the pathophysiological explanations of the six diseases in relation to obesity, diabetes, hypertension, and dyslipidemia:

4.1 Cardiovascular Disease (CVD)
• Obesity: Excessive body fat can lead to dyslipidemia (abnormal levels of cholesterol and triglycerides) and insulin resistance, contributing to the development of atherosclerosis and subsequently increasing the risk of CVD.
• Diabetes: High blood sugar levels can damage blood vessels and lead to atherosclerosis, leading to an increased risk of heart attacks, stroke, and peripheral arterial disease.
• Hypertension: High blood pressure puts strain on the arterial walls, leading to potential damage and atherosclerosis, increasing the risk of CVD.
• Dyslipidemia: Abnormal levels of cholesterol and triglycerides can lead to plaque formation in the arteries, leading to atherosclerosis and increasing the risk of CVD.

4.2 Chronic Kidney Disease (CKD)
• Obesity: Excess adipose tissue can lead to inflammation and oxidative stress, contributing to kidney damage and the development of CKD.
• Diabetes: High blood sugar levels can damage the small blood vessels in the kidneys, leading to diabetic nephropathy and CKD.
• Hypertension: Chronic high blood pressure can damage the kidneys over time, leading to CKD.
• Dyslipidemia: Abnormal lipid levels can contribute to the development and progression of CKD through mechanisms involving inflammation and endothelial dysfunction.

4.3 Cancer
• Obesity: Adipose tissue can produce hormones and growth factors that may promote the growth of cancer cells. Additionally, chronic low-grade inflammation associated with obesity can promote the development of some cancers.
• Diabetes: *High insulin levels and insulin resistance, common in diabetes, may promote tumor growth.*
• Hypertension: Chronic inflammation and oxidative stress associated with hypertension may promote cancer development.
• Dyslipidemia: Dyslipidemia may influence cancer development through various mechanisms such as promoting inflammation, altering cell membrane function, and impacting cell signaling pathways.

4.4 Alzheimer's Disease
• Obesity: Adipose tissue-derived hormones and inflammatory cytokines may contribute to neuroinflammation and oxidative stress, which are implicated in the development of Alzheimer's disease.
• Diabetes: High blood sugar levels, insulin resistance, and chronic inflammation in diabetes may contribute to the development and progression of Alzheimer's disease.
• Hypertension: Chronic high blood pressure can lead to vascular changes in the brain, which may increase the risk of Alzheimer's disease.
• Dyslipidemia: Abnormal lipid metabolism and cholesterol levels may impact the formation of amyloid plaques, which are

characteristic of Alzheimer's disease.

4.5 Parkinson's Disease
• Obesity: Adipose tissue-derived hormones and inflammatory molecules may contribute to oxidative stress and neuroinflammation, potentially impacting the development of Parkinson's disease.
• Diabetes: Insulin resistance and dysregulated glucose metabolism may contribute to neurodegeneration and the development of Parkinson's disease.
• Hypertension: Chronic high blood pressure and associated vascular changes may impact neuronal function and increase the risk of Parkinson's disease.
• Dyslipidemia: Dyslipidemia and cholesterol metabolism may influence neurodegenerative processes associated with Parkinson's disease.

4.6 Diabetic Neuropathy
• Obesity: Chronic low-grade inflammation associated with obesity may contribute to nerve damage and neuropathy.
• Diabetes: Prolonged high blood sugar levels can damage nerves throughout the body, leading to diabetic neuropathy.
• Hypertension: High blood pressure and vascular changes associated with hypertension may contribute to nerve damage and diabetic neuropathy.
• Dyslipidemia: Abnormal lipid levels and impaired lipid metabolism may contribute to nerve damage and the development of diabetic neuropathy.
These pathophysiological explanations highlight the intricate connections between obesity, diabetes, hypertension, dyslipidemia, and the development and progression of these complex conditions. Effective management and prevention strategies for these interconnected diseases should take into account their shared underlying pathophysiological mechanisms.

5. MPM Background
To learn more about his developed
GH-Method: math-physical medicine (MPM) methodology, readers can read the following three papers selected from his published 760+ papers.
The first paper, No. 386 (Reference 1) describes his MPM methodology in a general conceptual format. The second paper, No. 387 (Reference 2)
outlines the history of his personalized diabetes research, various application tools, and the differences between 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.

6. The Author's Diabetes History
The author was a severe T2D patient since 1995. He weighed 220 lb. (100 kg) at that time. By 2010, he still weighed 198 lb. with 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 fingerpiercing 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 preCOVID 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 selfquarantined 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 indepth 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 5minute intervals from every 15minute 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 wellbalanced 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.

7. Energy Theory

The human body and organs have around 37 trillion live cells which are composed of different organic cells that require energy infusion from glucose carried by red blood cells; and energy consumption from laborwork 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. N 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. timedependent. *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.**

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 ksteps * 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 stressstrain 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 disorderinduced 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).
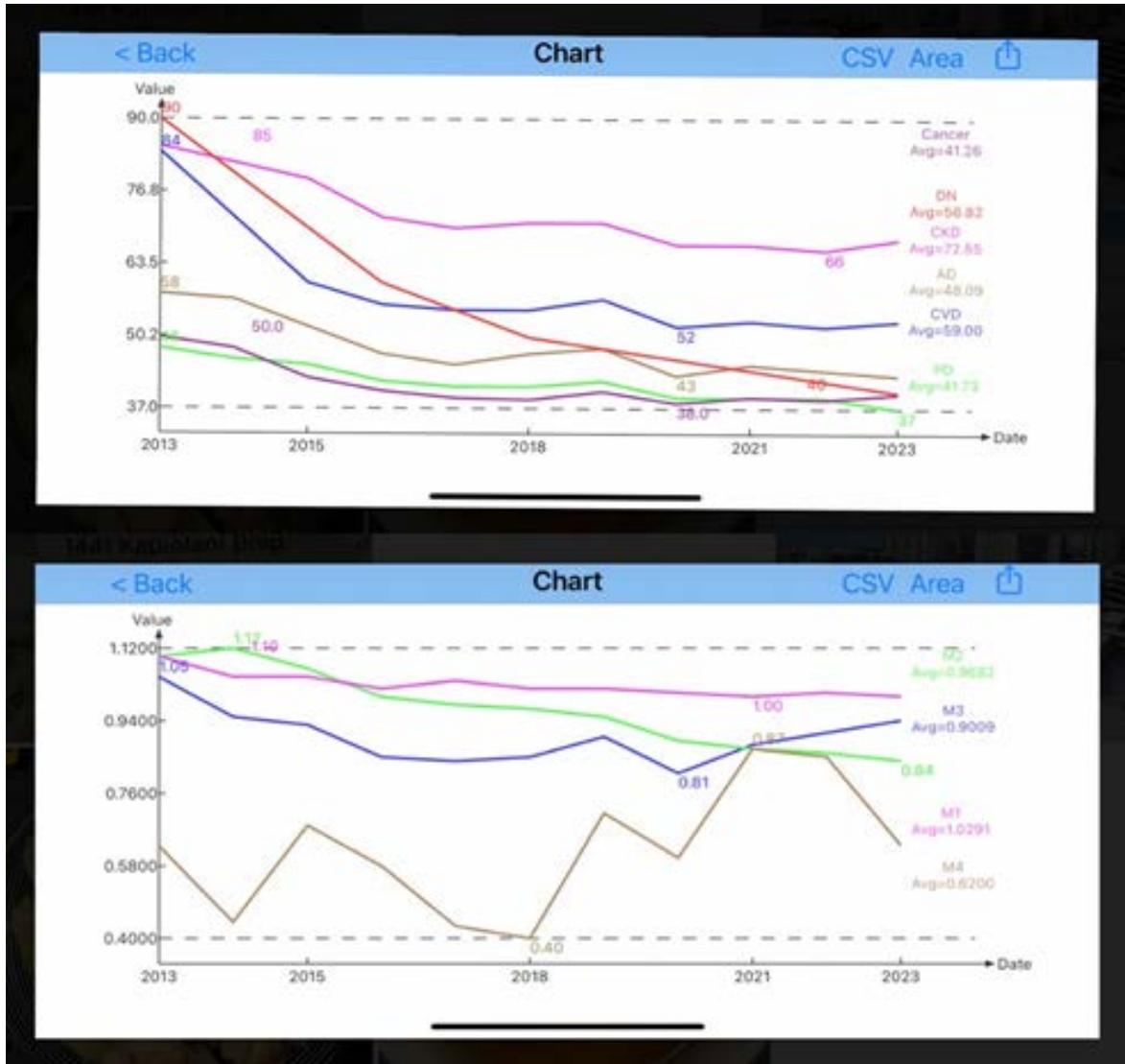
8. Results

Figure 1: time-domain curves of 6 diseases and 4 biomarkers.

Figure 2: 6 data tables of deadly diseases.

Correlation	M1	M2	M3	M4
CVD	92%	80%	81%	-22%
CKD	91%	94%	73%	-32%
Cancer	86%	81%	84%	-18%
AD	87%	92%	74%	-25%
PD	92%	96%	62%	-28%
DN	94%	95%	65%	-33%
Average	90%	90%	73%	-26%

VMT Energy	M1	M2	M3	M4
CVD	29%	30%	26%	15%
CKD	29%	29%	25%	17%
Cancer	29%	29%	25%	17%
AD	29%	29%	25%	17%
PD	30%	29%	25%	16%
DN	29%	30%	26%	16%
Average	29%	29%	25%	16%

Figure 3: Summarized Conclusions

9. Conclusions

In summary, the annual averaged risks of six deadly diseases exhibit a downward trend resembling different skiing slopes from 2013 to 2023. Correspondingly, the annual averaged biomarker values, including body weight and glucose, are also decreasing, while blood pressure and blood lipids fluctuate within safe ranges (below 100%).

Applying traditional statistical calculations reveals the *"averaged" correlations between these six deadly diseases and four distinct biomarkers:*
•   *6 diseases vs. weight: 90%*
•   *6 diseases vs. glucose: 90%*
•   *6 diseases vs. blood pressure: 73% - 6 diseases vs. lipids: -26%*

Utilizing space-domain viscoplastic energy analysis, his study identifies four energy contribution margins for these six deadly diseases:
•   *Energy from body weight: 29%*
•   *Energy from glucose: 29%*
•   *Energy from blood pressure: 25% - Energy from blood lipids: 16%*

Why these 6 diseases risks have negative correlations with cholesterols?

Disease risks having negative correlations with cholesterol levels may be due to several factors. First, it's important to note that the relationship between cholesterol and disease risks is complex and not fully understood. However, some potential reasons for the negative correlation include:

1. Low cholesterol levels may be associated with malnutrition or other underlying health issues, which in turn can increase disease risk.

1. Some diseases, such as cancer and liver disease, can lead to decreased cholesterol levels due to the body's response to the illness.

2. Genetic factors and metabolism can play a role, as individuals with naturally low cholesterol levels may have an increased risk for certain diseases.

It's important to keep in mind that the relationship between cholesterol levels and disease risks is not fully linear, and many other factors, such as diet, lifestyle, and overall health, also contribute to disease risk.

Why these 6 Disease risks have high positive correlations with obesity, diabetes and hypertension?

Disease risks have high positive correlations with obesity, diabetes, and hypertension due to the following reasons:

1. Obesity: Excess body fat, particularly around the abdomen, can lead to a variety of health issues such as heart disease, stroke, type 2 diabetes, and certain types of cancer. Obesity is associated with chronic low-grade inflammation and an increased risk of developing insulin resistance, both of which contribute to the development of these diseases.

2. Diabetes: Diabetes, especially type 2 diabetes, is characterized by high blood sugar levels resulting from the body's inability to effectively use insulin. Uncontrolled diabetes can lead to serious complications including cardiovascular disease, kidney disease, and nerve damage, all of which increase disease risks.

3. Hypertension: High blood pressure, or hypertension, forces the heart to work harder and can contribute to the development of heart disease, stroke, and other health issues. It puts strain on the arteries and can lead to damage over time, increasing the risk of cardiovascular diseases.

In summary, the conditions of obesity, diabetes, and hypertension are linked to a range of physiological and metabolic dysfunctions that significantly raise the risks of developing various diseases, particularly those related to the cardiovascular system and metabolic health.

10. Key Message

The majority of Type 2 diabetes patients face overweight or obesity challenges. The interplay between obesity and diabetes escalates the risk of six diseases: heart attacks & strokes, kidney problems, cancers, Alzheimer's disease, Parkinson's disease, and diabetic neuropathy (e.g., foot ulcers leading to amputations), all linked to high mortality rates.

References