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Research Article

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A Preventive Medicine And Public Health Study Of Close Relationship Between Cardiovascular And Cerebrovascular Diseases Versus Diet, Obesity, and Diabetes Based on one T2d Patient's Collected Data Using the Viscoplastic Energy Model ff GH-Method: Math-Physical Medicine (No. 1037, Viscoelastic Medicine Theory #435)

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Abstract

The author read a paper published on NIH and Lancet in 2021 by Jonathan Pearson-Stuttard and colleagues, titled "Trends in Predominant Causes of Death in Individuals with and without Diabetes in England from 2001 to 2018: An Epidemiological Analysis of Linked Primary Care Records." This investigation, henceforth referred to as the "UK study," scrutinized various health complications in relation to diabetes. This UK study highlighted a decrease in cardiovascular-related mortality rates per 1,000 deaths from 18.5 in 2001 to 7.5 in 2018 among diabetic patients (averaged 13.0), and from 12.2 in 2001 to 4.3 in 2018 among non-diabetics (averaged 8.25). ***The UK Study is showing an increase in the CVD mortality rate ratio of 1.58 (13.0 divided by 8.25) for diabetics compared to non-diabetics****.*

Motivated by these findings, the author delved into an in-depth analysis exploring the interplay among dietary habits, body weight, fasting glucoses, carbohydrates/sugar intake, fasting and postprandial glucoses, and hemoglobin A1c values, and their impact on cardiovascular and cerebrovascular diseases (CVD) risks. This exploration was based on a personal dataset of 3 million data gathered over 15 years, from 2010 to 2024. By comparing average ratios of these biomarkers from two distinct periods, 2010 to 2011 when the author battled his obesity and diabetes, and 2023 to 2024 when he achieved a healthier state. The conclusive findings from this study provide a compelling narrative on how substantial lifestyle and health improvements can lead to a lower risk of having cardiovascular and cerebrovascular diseases.

In summary, *this research not only offers practical guidelines for patients aiming to minimize their CVD and stroke risks but also integrates perspectives from both preventive medicine and public health.*

• Food portion per meal decreased from 145% in the initial period to 49% in the current period, with a ratio of 3.0.
• Body weight was reduced from 215 lbs to 167 lbs through food portion control, resulting in a ratio of 1.3.
• FPG dropped from 170 mg/dL to 88 mg/dL, with a ratio of 1.9.
• Carbohydrate/sugar intake went from 100 grams to 12 grams, with a ratio of 8.3.
• PPG decreased from 282 mg/dL to 105 mg/dL, with a ratio of 2.7.
• HbA1C levels fell from 10.3% to 5.9%, with a ratio of 1.7.
• CVD risk reduced from 88% to 51.5%, with a ratio of 1.71.
• All of correlations between any set of two variables are within the range of 88% to 98%.

His calculated cardiovascular disease (CVD) risk ratio of 1.71 closely matches the findings from the UK study, which reported CVD risk ratios of 1.58. *The UK study utilized traditional statistical methods and involved extensive data from hundreds of thousands of patients.*

Additionally, by employing the viscoplastic energy method or VMT, the author calculated the energy ratio

stemming from the interplay between his CVD risk and three biomarkers: obesity, FPG, and A1C. ***This VMT energy ratio was found to be 1.04, comparing data from the initial period to the present period.*** *The explanation for this low ratio lies in the fact that his five cardiovascular episodes occurred much earlier, between 1994 and 2004, whereas his VMT energy model spans from 2010 to 2024. Moreover, he did not gather data on his lifestyle and chronic conditions before 2010.*

Key Message

Controlling meal's portion size can lead to a body weight reduction, which benefits pancreatic insulin function through lower FPG levels. Reducing intake of carbohydrates and sugars can decrease PPG levels, which, in turn, reduces A1C levels, a critical biomarker of type 2 diabetes. ***Effective diabetes management can indeed lower the risk of developing cardiovascular and cerebrovascular diseases.***

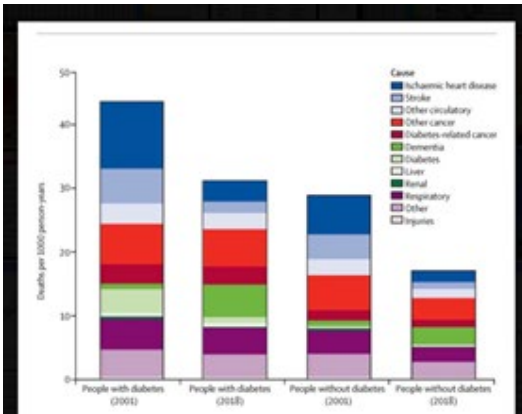

Figure 1 Death rates by cause in those with and without diabetes in 2001 and 2018

		People with diabetes	People without diabetes
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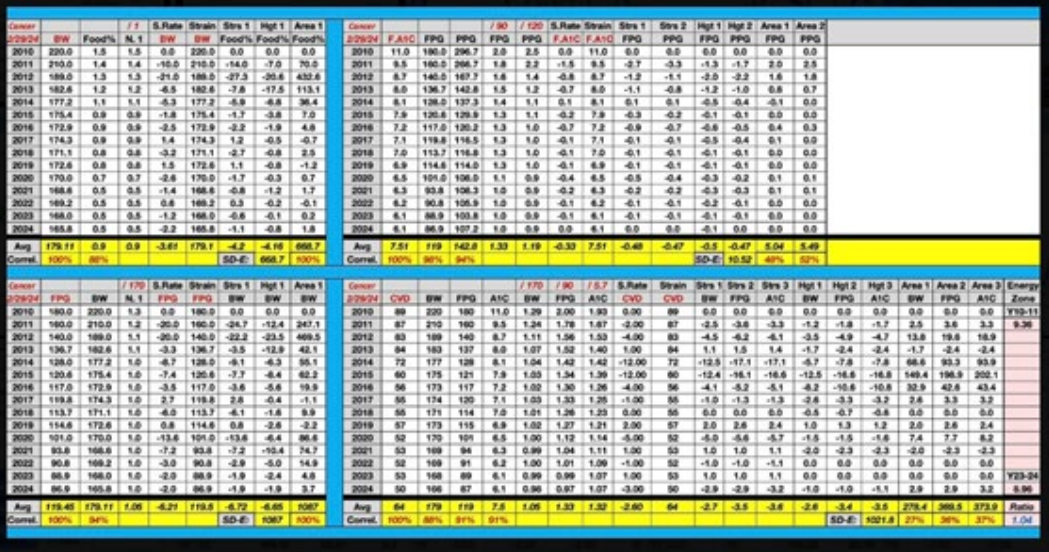
Men					
All-cause		40·7 (40·0 to 41·5)	27·8 (27·3 to 28·3)	28·4 (27·7 to 29·0)	16·7 (16·3 to 17·0)
Vascular causes		18·5 (18·0 to 19·1)	7·5 (7·3 to 7·7)	12·2 (11·7 to 12·7)	4·3 (4·2 to 4·5)
	Ischaemic heart disease	11·2 (10·7 to 11·6)	4·0 (3·8 to 4·1)	6·2 (6·0 to 7·0)	2·2 (2·1 to 2·3)
	Stroke	4·7 (4·4 to 5·0)	1·5 (1·4 to 1·6)	3·3 (3·0 to 3·5·3)	0·9 (0·8 to 1·0)



	BW	FPG	PPG	F.A1C	CVD	Food%	Carbs
2010	220.0	180.0	296.7	11.0	89.0	1.5	104.0
2011	210.0	160.0	266.7	9.5	87.0	1.4	96.0
2012	189.0	140.0	167.7	8.7	83.0	1.3	88.0
2013	182.6	136.7	142.8	8.0	84.0	1.2	80.0
2014	177.2	128.0	137.3	8.1	72.0	1.1	72.0
2015	175.4	120.6	129.9	7.9	60.0	0.9	30.3
2016	172.9	117.0	120.2	7.1	56.0	0.9	15.6
2017	174.3	119.8	116.5	7.1	55.0	0.9	14.5
2018	171.1	113.7	116.8	7.0	55.0	0.8	15.8
2019	172.6	114.6	114.0	6.9	57.0	0.8	13.2
2020	170.0	101.0	108.0	6.5	52.0	0.7	13.7
2021	168.6	93.8	108.3	6.3	53.0	0.5	12.8
2022	169.2	90.8	105.9	6.2	52.0	0.5	10.4
2023	168.0	88.9	103.8	6.1	53.0	0.5	13.4
2024	165.8	86.9	107.2	5.7	50.0	0.5	11.4
Avg	*179.1*	*119.4*	*142.8*	*7.5*	*63.9*	*0.9*	*39.4*
Correl.	*88%*	*91%*	*84%*	*91%*	*100%*	*94%*	*99%*

Time	BW	FPG	PPG	F.A1C	CVD	Food%	Carbs	*Ratio between Y10-11 vs. Y23-24*
Unit	lbs	mg/dL	mg/dL	%	%	%	grams	
Avg.								
Y10-Y11	215	170	282	10.3	88	145%	100	*By CVD Risk itself*
								1.71
								CVD interact with T2D
								1.04
Avg.								
Y23-Y24	167	88	105	5.9	52	49%	12	
Ratio	*1.3*	*1.9*	*2.7*	*1.7*	*1.71*	*3.0*	*8.1*	

Ratio of Y10-11 Value versus Y23-24 Value

1. Introduction

The author read a paper published on NIH and Lancet in 2021 by Jonathan Pearson-Stuttard and colleagues, titled "Trends in Predominant Causes of Death in Individuals with and without Diabetes in England from 2001 to 2018: An Epidemiological Analysis of Linked Primary Care Records." This investigation, henceforth referred to as the "UK study," scrutinized various health complications in relation to diabetes. This UK study highlighted a decrease in cardiovascular-related mortality rates per 1,000 deaths from 18.5 in 2001 to 7.5 in 2018 among diabetic patients (averaged 13.0), and from 12.2 in 2001 to 4.3 in 2018 among non-diabetics (averaged 8.25). ***The UK Study is showing an increase in the CVD mortality rate ratio of 1.58 (13.0 divided by 8.25) for diabetics compared to non-diabetics***.

Motivated by these findings, the author delved into an in-depth analysis exploring the interplay among dietary habits, body weight, fasting glucoses, carbohydrates/sugar intake, fasting and postprandial glucoses, and hemoglobin A1c values, and their impact on cardiovascular and cerebrovascular diseases (CVD) risks. This exploration was based on a personal dataset of 3 million data gathered over 15 years, from 2010 to 2024. By comparing average ratios of these biomarkers from two distinct periods, 2010 to 2011 when the author battled his obesity and diabetes, and 2023 to 2024 when he achieved a healthier state. The conclusive findings from this study provide a compelling narrative on how substantial lifestyle and health improvements can lead to a lower risk of having cardiovascular and cerebrovascular diseases.

1.1 Biomedical or certain Technical Information

The following sections contain excerpts and concise information on 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.

2. Pathophysiological explanations of relationship between diabetes and cardiovascular and cerebrovascular diseases

The relationship between diabetes and cardiovascular (CVD) and cerebrovascular diseases is complex and multifaceted, involving a range of pathophysiological mechanisms. Here is an overview of the brief explanations for this connection:

2.1 Hyperglycemia and Glycemic Variability

• Atherosclerosis: Chronic high blood sugar levels in diabetes damage the endothelium (inner lining) of blood vessels, leading to atherosclerosis, which is the buildup of plaques in the arteries. This condition is a primary cause of CVD and can also affect the arteries that supply blood to the brain, contributing to cerebrovascular disease.

• Oxidative Stress: Elevated glucose levels increase oxidative stress, which further damages blood vessels and accelerates atherosclerosis.

• Inflammation: Diabetes is associated with chronic inflammation, which exacerbates the development of atherosclerotic plaques and can lead to both CVD and cerebrovascular diseases.

2.2 Insulin Resistance

• Dyslipidemia: Insulin resistance, a hallmark of type 2 diabetes, contributes to dyslipidemia, characterized by high levels of triglycerides, low levels of HDL cholesterol, and often elevated levels of LDL cholesterol. These lipid abnormalities enhance atherosclerotic plaque formation.

• Hypertension: Insulin resistance is also associated with hypertension, a major risk factor for CVD and stroke, due to the effects on kidney function and sodium retention.

2.3 Coagulation Abnormalities

• Hyper-coagulability: Diabetes leads to changes in the blood, making it more prone to clotting. This increased tendency for thrombosis can block arteries, leading to heart attacks or strokes.

2.4 Microvascular Damage

• Micro-angioplasty: Diabetes causes damage to small blood vessels (microangiopathy), impairing blood flow to various organs, including the heart and brain. This can contribute to heart failure and microvascular ischemic disease in the brain.

2.5 Endothelial Dysfunction

• Impaired Vasodilation: Diabetes impairs endothelial function, reducing the ability of arteries to dilate. This endothelial dysfunction is a critical early step in the development of atherosclerosis and contributes to hypertension.

2.6 Autonomic Neuropathy

• Cardiovascular Autonomic Neuropathy (CAN): Diabetes can affect the autonomic nervous system, which controls the heart and blood vessels. CAN may result in abnormal heart rate control and vascular dynamics, increasing the risk of cardiac arrhythmias, sudden death, and ischemic stroke.

2.7 Hyperinsulinemia

• In Type 2 Diabetes: High levels of insulin (hyperinsulinemia) are common in the early stages of type 2 diabetes due to insulin resistance. Insulin has growth-promoting effects on the arterial wall, contributing to atherosclerosis.

In summary, the interplay of these mechanisms leads to an increased risk of developing cardiovascular and cerebrovascular diseases in individuals with diabetes. Management strategies focusing on controlling blood sugar levels, blood pressure, and lipid levels, along with lifestyle modifications, are crucial for reducing this risk.

3. 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.

4. 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.

5. 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.***

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.

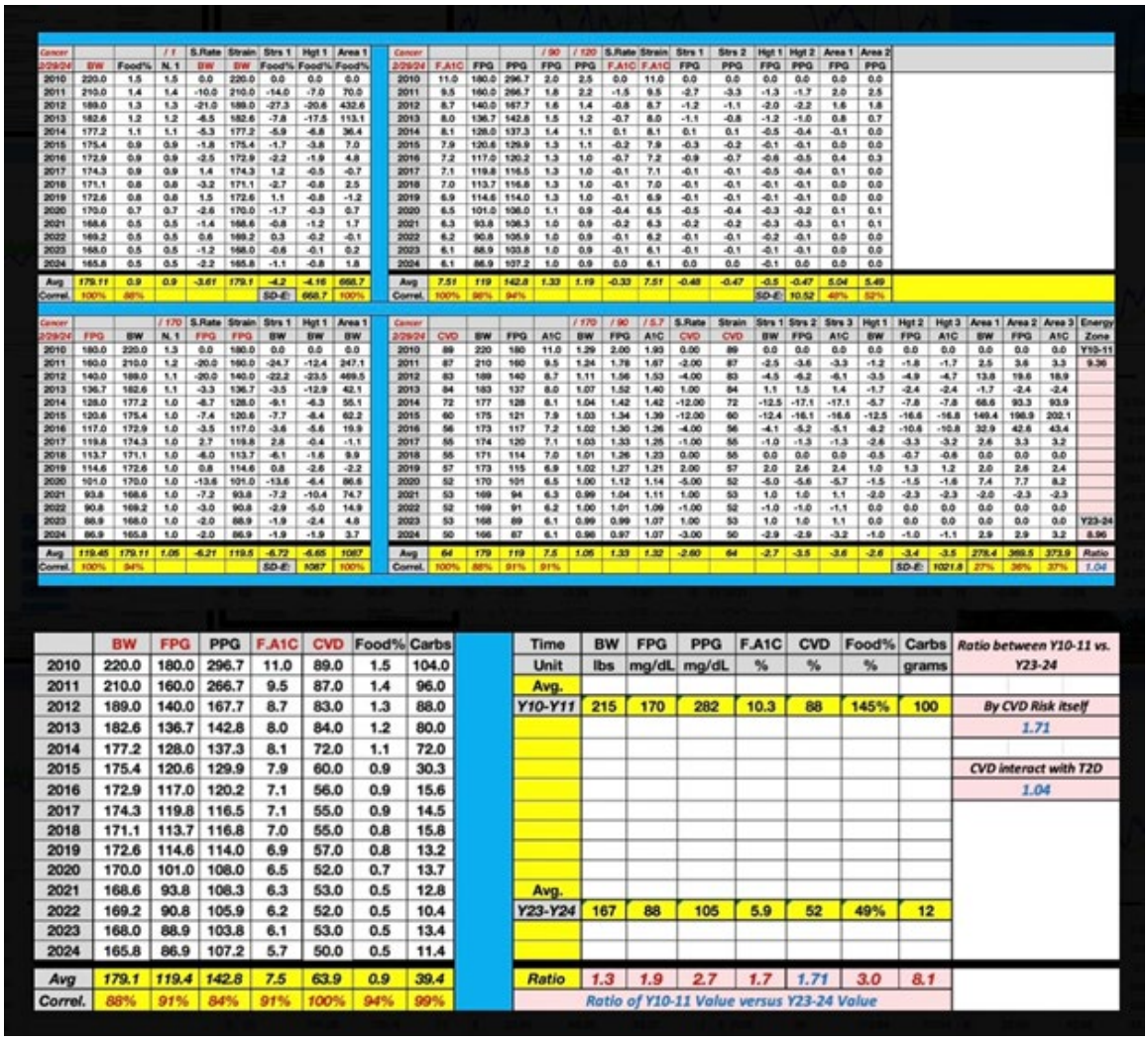
Some of explored deadly diseases and longevity characteristics using the ***viscoplastic medicine theory (VMT)*** include stress relaxation, creep, hysteresis loop, and material stiffness, damping effect ***based on time-dependent stress and strain*** which are different from his previous research findings using ***linear elastic glucose theory (LEGT)*** and nonlinear plastic glucose theory (NPGT).

6. Results

Figure 1 Death rates by cause in those with and without diabetes in 2001 and 2018

		People with diabetes		People without diabetes	
		2001: rate per 1000 person years (95% CI)	2018: rate per 1000 person years (95% CI)	2001: rate per 1000 person years (95% CI)	2018: rate per 1000 person years (95% CI)
Men					
All-cause		40·7 (40·0 to 41·5)	27·8 (27·3 to 28·3)	28·4 (27·7 to 29·0)	16·7 (16·3 to 17·0)
Vascular causes		18·5 (18·0 to 19·1)	7·5 (7·3 to 7·7)	12·2 (11·7 to 12·7)	4·3 (4·2 to 4·5)
	Ischaemic heart disease	11·2 (10·7 to 11·6)	4·0 (3·8 to 4·1)	6·2 (6·0 to 7·0)	2·2 (2·1 to 2·3)
	Stroke	4·7 (4·4 to 5·0)	1·5 (1·4 to 1·6)	3·3 (3·0 to 3·5·3)	0·9 (0·8 to 1·0)

Figure 1: The UK report data of CVD Risk



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Time	BW	FPG	PPG	F.A1C	CVD	Food%	Carbs	*Ratio between Y10-11 vs. Y23-24*
Unit	lbs	mg/dL	mg/dL	%	%	%	grams	
Avg.								
Y10-Y11	215	170	282	10.3	88	145%	100	*By CVD Risk itself*
								1.71
								CVD interact with T2D
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Avg.								
Y23-Y24	167	88	105	5.9	52	49%	12	
Ratio	*1.3*	*1.9*	*2.7*	*1.7*	*1.71*	*3.0*	*8.1*	
Ratio of Y10-11 Value versus Y23-24 Value								

Figure 2: Input Information, TD and SD results of CVD Risk

7. Conclusions

In summary, this research not only offers practical guidelines for patients aiming to minimize their CVD and stroke risks but also integrates perspectives from both preventive medicine and public health.

• Food portion per meal decreased from 145% in the initial period to 49% in the current period, with a ratio of 3.0.
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Key Message

Controlling meal's portion size can lead to a body weight reduction, which benefits pancreatic insulin function through lower FPG levels. Reducing intake of carbohydrates and sugars can decrease PPG levels, which, in turn, reduces A1C levels, a critical biomarker of type 2 diabetes. ***Effective diabetes management can indeed lower the risk of developing cardiovascular and cerebrovascular diseases***.

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.

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