ISSN: 2689-1204

Research Article

Journal of Applied Material Science & Engineering Research

A Preventive Medicine and Public Health Study of Close Relationship Between Chronic Kidney Diseases Versus Diet, Obesity, and Diabetes Based on One T2d Patient's Collected Data Using The Viscoplastic Energy Model Of GH-Method: Math-Physical Medicine (No. 1038, Viscoelastic Medicine Theory #436)

Gerald C. Hsu

EclaireMD Foundation, USA

***Corresponding Author**
Gerald C. Hsu, EclaireMD Foundation, USA

Submitted: 2024, Feb 26; **Accepted:** 2024, Mar 21; **Published:** 2024, Apr 02

Citation: Hsu, G. C. (2024). A Preventive Medicine and Public Health Study of Close Relationship Between Chronic Kidney Diseases Versus Diet, Obesity, and Diabetes Based on One T2d Patient's Collected Data Using The Viscoplastic Energy Model Of GH-Method: Math-Physical Medicine (No. 1038, Viscoelastic Medicine Theory #436). *J App Mat Sci & Engg Res, 8*(1), 01-07.

Category: Nephrology & Diabetes

Abstract

The author reviewed a paper published in 2021 by Jonathan Pearson-Stuttard and colleagues, named "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." Referred to as the "UK study," it focused on health complications related to diabetes. Among the findings, the UK study noted a decrease in renal complications rate per 1,000 cases from 0.4 in 2001 to 0.2 in 2018 (averaged 0.3) among diabetic patients and from 0.4 in 2001 to 0.1 in 2018 among non-diabetics (averaged 0.25). ***Therefore, the UK study highlighted a CKD ratio of 1.2 (0.3 divided by 0.25) between diabetes and non-diabetes****.*

Inspired by these findings, the author conducted an in-depth analysis of dietary habits, body weight, fasting glucoses, carbohydrate/sugar intake, fasting and postprandial glucoses, and hemoglobin A1c values, and their impact on his CKD or renal complications. This exploration was based on a personal dataset of 3 million data gathered over 15 years (2010-2024). The comparison of biomarker ratios from two distinct periods—2010-2011 when the author battled obesity and diabetes, and 2023-2024 when a healthier state was achieved—revealed compelling evidence on how lifestyle and health improvements can lower the risk of developing renal complications.

In summary, *this research provides certain practical guidelines for patients aiming to minimize their CKD and renal complications risk, integrating 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.*
- ***CKD risk reduced from 112% to 65%, with a ratio of 1.72.***
- *All of correlations between any set of two variables are within the range of 88% to 97%.*

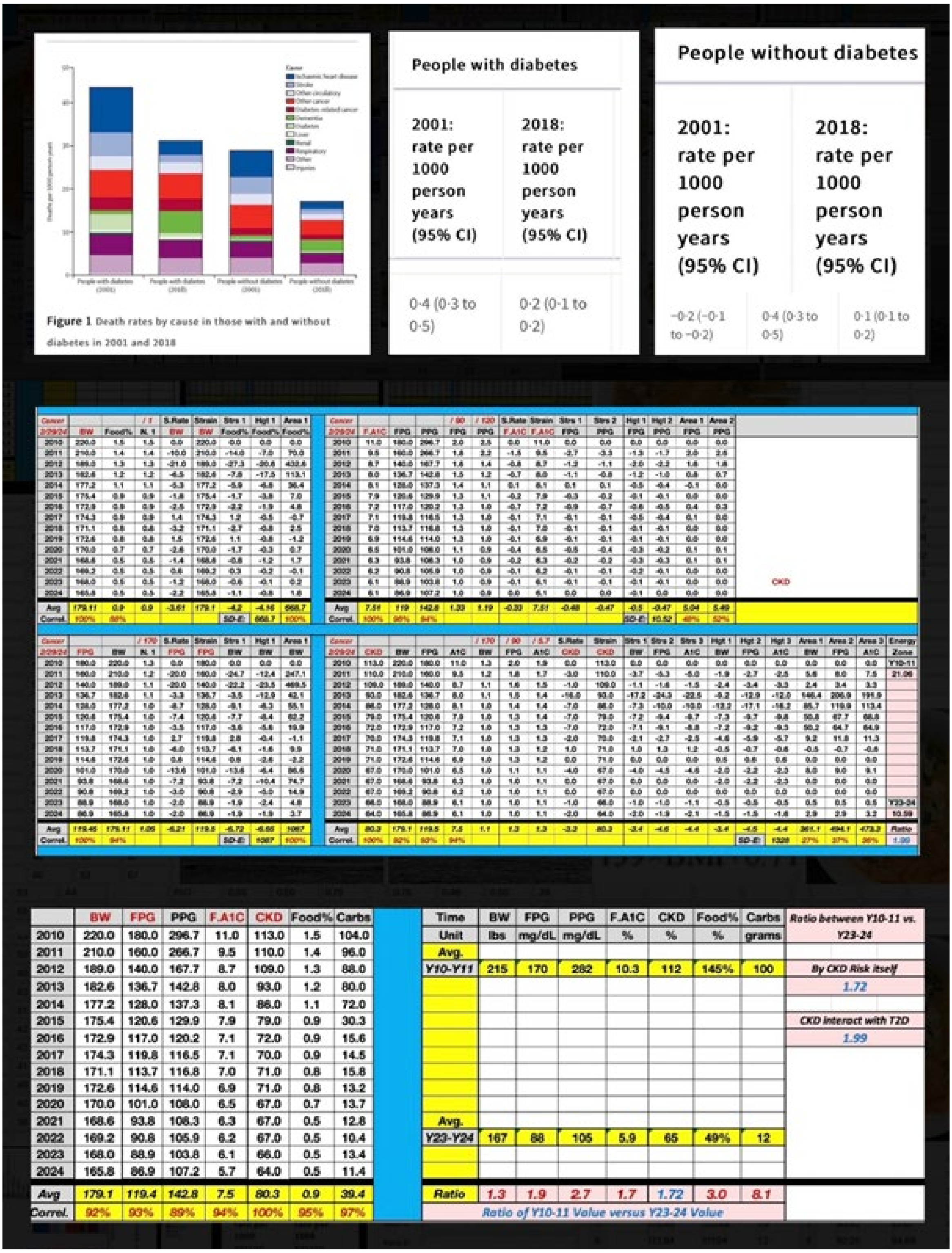
His calculated CKD risk ratio of 1.71 (from 112% to 65%) aligns with the UK study's CKD ratio of 1.20 *which was based on traditional statistical methods and extensive data from many patients.*

Furthermore, using the viscoplastic energy method (VMT), the author calculated the energy ratio from the interaction between his CKD risk and three biomarkers: obesity, FPG, and A1C. ***The VMT energy ratio was found to be 1.99 when comparing data from the initial period to the present period.***

Unlike his low CVD energy ratio of 1.04, this demonstrated that his CKD was at its peak condition in 2010 and then continuously decreased in the following years. It should be noted that data on his lifestyle and chronic conditions before 2010 were not gathered.

Key Message

Managing portion sizes of meals can lead to body weight reduction, which lowers FPG level and benefits pancreatic insulin function. Then, reducing intake of carbohydrates and sugars can decrease PPG levels, subsequently reducing A1C levels—a critical biomarker of type 2 diabetes. ***Effective diabetes management can indeed lower the risk of developing CKD or renal complications.***



	BW	FPG	PPG	F.A1C	CKD	Food%	Carbs
2010	220.0	180.0	296.7	11.0	113.0	1.5	104.0
2011	210.0	160.0	266.7	9.5	110.0	1.4	96.0
2012	189.0	140.0	167.7	8.7	109.0	1.3	88.0
2013	182.6	136.7	142.8	8.0	93.0	1.2	80.0
2014	177.2	128.0	137.3	8.1	86.0	1.1	72.0
2015	175.4	120.6	129.9	7.9	79.0	0.9	30.3
2016	172.9	117.0	120.2	7.1	72.0	0.9	15.6
2017	174.3	119.8	116.5	7.1	70.0	0.9	14.5
2018	171.1	113.7	116.8	7.0	71.0	0.8	15.8
2019	172.6	114.6	114.0	6.9	71.0	0.8	13.2
2020	170.0	101.0	108.0	6.5	67.0	0.7	13.7
2021	168.6	93.8	108.3	6.3	67.0	0.5	12.8
2022	169.2	90.8	105.9	6.2	67.0	0.5	10.4
2023	168.0	88.9	103.8	6.1	66.0	0.5	13.4
2024	165.8	86.9	107.2	5.7	64.0	0.5	11.4
Avg	179.1	119.4	142.8	7.5	80.3	0.9	39.4
Correl.	92%	93%	89%	94%	100%	95%	97%

Time	BW	FPG	PPG	F.A1C	CKD	Food%	Carbs	Ratio between Y10-11 vs. Y23-24
Unit	lbs	mg/dL	mg/dL	%	%	%	grams	
Avg.								
Y10-Y11	215	170	282	10.3	112	145%	100	By CKD Risk itself
								1.72
								CKD interact with T2D
								1.99
Avg.								
Y23-Y24	167	88	105	5.9	65	49%	12	
Ratio	1.3	1.9	2.7	1.7	1.72	3.0	8.1	
Ratio of Y10-11 Value versus Y23-24 Value								

1. Introduction

The author reviewed a paper published in 2021 by Jonathan Pearson-Stuttard and colleagues, named "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." Referred to as the "UK study," it focused on health complications related to diabetes. Among the findings, the UK study noted a decrease in renal complications rate per 1,000 cases from 0.4 in 2001 to 0.2 in 2018 (averaged 0.3) among diabetic patients and from 0.4 in 2001 to 0.1 in 2018 among non-diabetics (averaged 0.25). ***Therefore, the UK study highlighted a CKD ratio of 1.2 (0.3 divided by 0.25) between diabetes and non-diabetes.***

Inspired by these findings, the author conducted an in-depth analysis of dietary habits, body weight, fasting glucoses, carbohydrate/sugar intake, fasting and postprandial glucoses, and hemoglobin A1c values, and their impact on his CKD or renal complications. This exploration was based on a personal dataset of 3 million data gathered over 15 years (2010-2024). The comparison of biomarker ratios from two distinct periods—2010-2011 when the author battled obesity and diabetes, and 2023-2024 when a healthier state was achieved—revealed compelling evidence on how lifestyle and health improvements can lower the risk of developing renal complications.

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 Chronic Kidney Diseases or Renal Complications

The relationship between diabetes and chronic kidney disease (CKD) or renal complications is complex and involves multiple pathophysiological mechanisms. Here are the key explanations:

- **Hyperglycemia:** Prolonged high blood glucose levels in diabetes damage the kidneys' filtering units, the nephrons. This damage impairs the nephrons' ability to filter waste from the blood efficiently, leading to the accumulation of waste products and the development of CKD.
- **Advanced Glycation End-products (AGEs):** High blood sugar levels increase the formation of AGEs, which can alter the structure and function of various proteins in the kidneys. AGEs can induce inflammation and fibrosis in kidney tissues, contributing to the progression of kidney disease.
- **Glomerular Hyperfiltration:** Diabetes can lead to glomerular hyperfiltration, where the kidneys filter blood at an abnormally high rate. Over time, this increased workload can damage the glomeruli, leading to proteinuria (excess protein in the urine) and a decline in kidney function.
- **Hemodynamic Changes:** Diabetes can cause changes in the blood vessels within the kidneys, affecting their ability to regulate blood pressure and filtration. *High blood pressure (hypertension) is both a cause and a consequence of kidney damage.* The increased pressure in the glomeruli can lead to further damage, exacerbating kidney disease.
- **Inflammation and Oxidative Stress:** Diabetes is associated with increased levels of inflammation and oxidative stress, which can damage kidney tissues. Inflammatory cytokines and oxidative stress markers can promote fibrosis and scarring in the kidneys, further impairing their function.
- **Insulin Resistance:** In type 2 diabetes, insulin resistance can contribute to kidney disease by promoting hyperfiltration and increasing the kidneys' exposure to toxic levels of glucose. Insulin resistance is also linked to other risk factors for CKD, such as hypertension and dyslipidemia (abnormal levels of lipids in the blood).
- **Renal Tubular Damage:** Diabetes can lead to damage of the renal tubules, which are responsible for reabsorbing substances the body needs and excreting waste. High blood sugar can directly damage these tubules and impair their function, leading to the loss of essential nutrients and the accumulation of waste products.

The interplay of these mechanisms contributes to the progression of kidney disease in individuals with diabetes. Early detection and management of diabetes and its complications are critical for slowing the progression of CKD and preventing end-stage renal disease (ESRD).

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

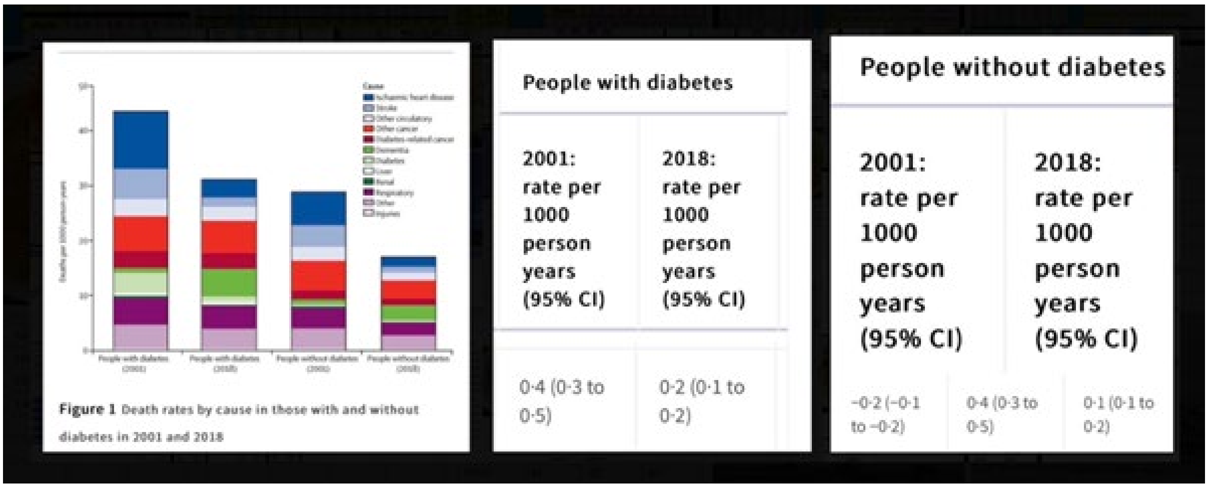
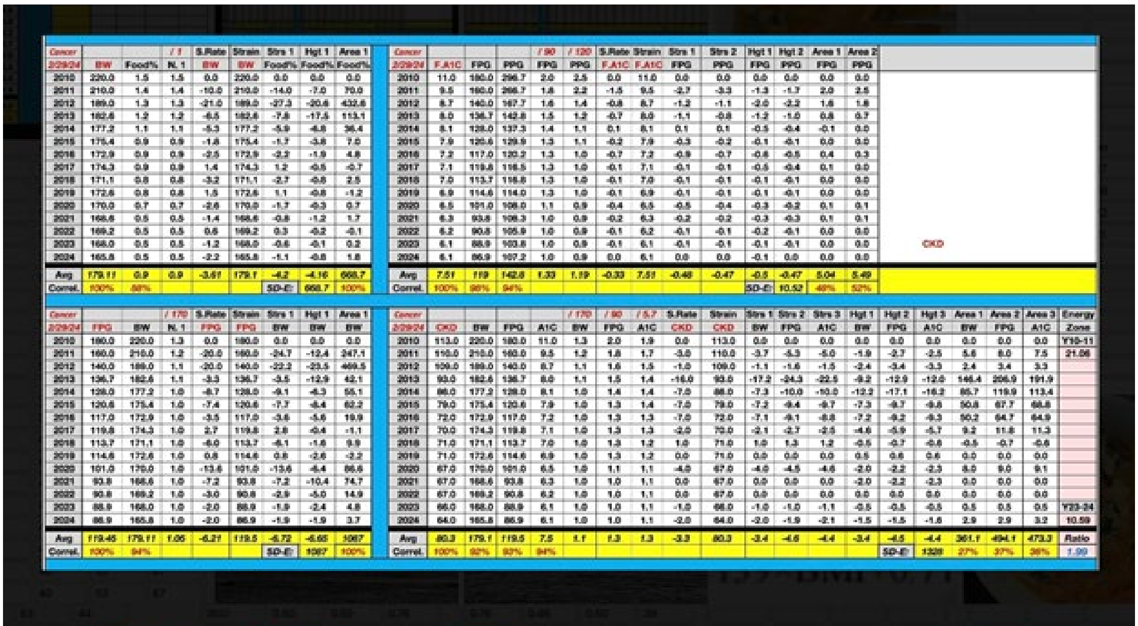


Figure 1: The UK report data of CKD Risk



	BW	FPG	PPG	F.A1C	CKD	Food%	Carbs
2010	220.0	180.0	296.7	11.0	113.0	1.5	104.0
2011	210.0	160.0	266.7	9.5	110.0	1.4	96.0
2012	189.0	140.0	167.7	8.7	109.0	1.3	88.0
2013	182.6	136.7	142.8	8.0	93.0	1.2	80.0
2014	177.2	128.0	137.3	8.1	86.0	1.1	72.0
2015	175.4	120.6	129.9	7.9	79.0	0.9	30.3
2016	172.9	117.0	120.2	7.1	72.0	0.9	15.6
2017	174.3	119.8	116.5	7.1	70.0	0.9	14.5
2018	171.1	113.7	116.8	7.0	71.0	0.8	15.8
2019	172.6	114.6	114.0	6.9	71.0	0.8	13.2
2020	170.0	101.0	108.0	6.5	67.0	0.7	13.7
2021	168.6	93.8	108.3	6.3	67.0	0.5	12.8
2022	169.2	90.8	105.9	6.2	67.0	0.5	10.4
2023	168.0	88.9	103.8	6.1	66.0	0.5	13.4
2024	165.8	86.9	107.2	5.7	64.0	0.5	11.4
Avg	179.1	119.4	142.8	7.5	80.3	0.9	39.4
Correl.	92%	93%	89%	94%	100%	95%	97%

Time	BW	FPG	PPG	F.A1C	CKD	Food%	Carbs	Ratio between Y10-11 vs. Y23-24
Unit	lbs	mg/dL	mg/dL	%	%	%	grams	
Avg.								
Y10-Y11	215	170	282	10.3	112	145%	100	By CKD Risk itself
								1.72
								CKD interact with T2D
								1.99
Avg.								
Y23-Y24	167	88	105	5.9	65	49%	12	
Ratio	1.3	1.9	2.7	1.7	1.72	3.0	8.1	
Ratio of Y10-11 Value versus Y23-24 Value								

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

7. Conclusions

In summary, this research provides certain practical guidelines for patients aiming to minimize their CKD and renal complications risk, integrating 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.
- ***CKD risk reduced from 112% to 65%, with a ratio of 1.72.***
- All of correlations between any set of two variables are within the range of 88% to 97%.

His calculated CKD risk ratio of 1.71 (from 112% to 65%) aligns with the UK study's CKD ratio of 1.20 *which was* based on traditional statistical methods and extensive data from many patients.

Furthermore, using the viscoplastic energy method (VMT), the author calculated the energy ratio from the interaction between his CKD risk and three biomarkers: obesity, FPG, and A1C. ***The VMT energy ratio was found to be 1.99 when comparing data from the initial period to the present period***.

Unlike his low CVD energy ratio of 1.04, this demonstrated that his CKD was at its peak condition in 2010 and then continuously decreased in the following years. It should be noted that data on his lifestyle and chronic conditions before 2010 were not gathered.

Key Message

Managing portion sizes of meals can lead to body weight reduction, which lowers FPG level and benefits pancreatic insulin function. Then, reducing intake of carbohydrates and sugars can decrease PPG levels, subsequently reducing A1C levels—a critical biomarker of type 2 diabetes. ***Effective diabetes management can indeed lower the risk of developing CKD or renal complications***.

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.

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

For reading more of the author’s published VGT or FD analysis results on medical applications, please locate them through platforms for scientific research publications, such as ResearchGate, Google Scholar, etc.