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Investigation of Close Relationship Between Cancers and Diabetes Based on an UK Study and One T2d Patient's Collected Data Using the Viscoplastic Energy Model of GH-Method: Math-Physical Medicine (No. 1034, Viscoelastic Medicine Theory #432)

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Category: Cancer & Diabetes

Abstract

The author recently reviewed a published report from the NIH and Lancet 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" by Jonathan Pearson-Stuttard et al., published in Lancet Diabetes Endocrinol in 2021 (volume 9, issue 3, pages 165-173). This study, referred to here as the ***"UK study", examined various medical complications versus diabetes but the author of this paper decided to specifically focus on cancers.***

The NIH/Lancet study found that out of every 1,000 individuals, there are 7.7 (range 7.4-8.0) with diabetes and 4.4 (range 4.3-4.6) without diabetes, leading to the UK Study's cancer ratio of 1.75 between those with diabetes and those without diabetes.

Utilizing a self-developed mathematical model called the Metabolic Index (MI) Model, the author calculated his annual cancer risk percentage based on 10 selected influential categories and 500 defined input elements. ***The MI model calculated cancer risk ratio between the first two years (2010 and 2011, marked by severe diabetes with an average A1C of 10.25%) and the most recent two years (2023 and 2024, with well-controlled A1C at 6.1%) was 1.74 (62.5% earlier cancer risk divided by 36.0% recent cancer risk).***

Further, applying the Space-Domain Viscoplastic Energy Model (SD-VMT), the author estimated the energy generated through the interaction between cancer risk percentage (output strain) and three influential stresses (FPG indicating pancreatic beta cells health state, HbA1C indicating the severity of Type 2 Diabetes, and body weight indicating the primary cause of many metabolic disorders, including both T2D and cancers). ***The SD-VMT model showed a cancer energy ratio of 1.88 between the initial two years (2010 and 2011 with severe diabetes) and the most recent two years (2023 and 2024 with controlled diabetes), calculated as earlier energy of 56 divided by recent energy of 30.***

In summary, *this study presents* ***his cancer risks calculated using three distinct methods, yielding closely aligned results within the range of 1.74 to 1.88.***

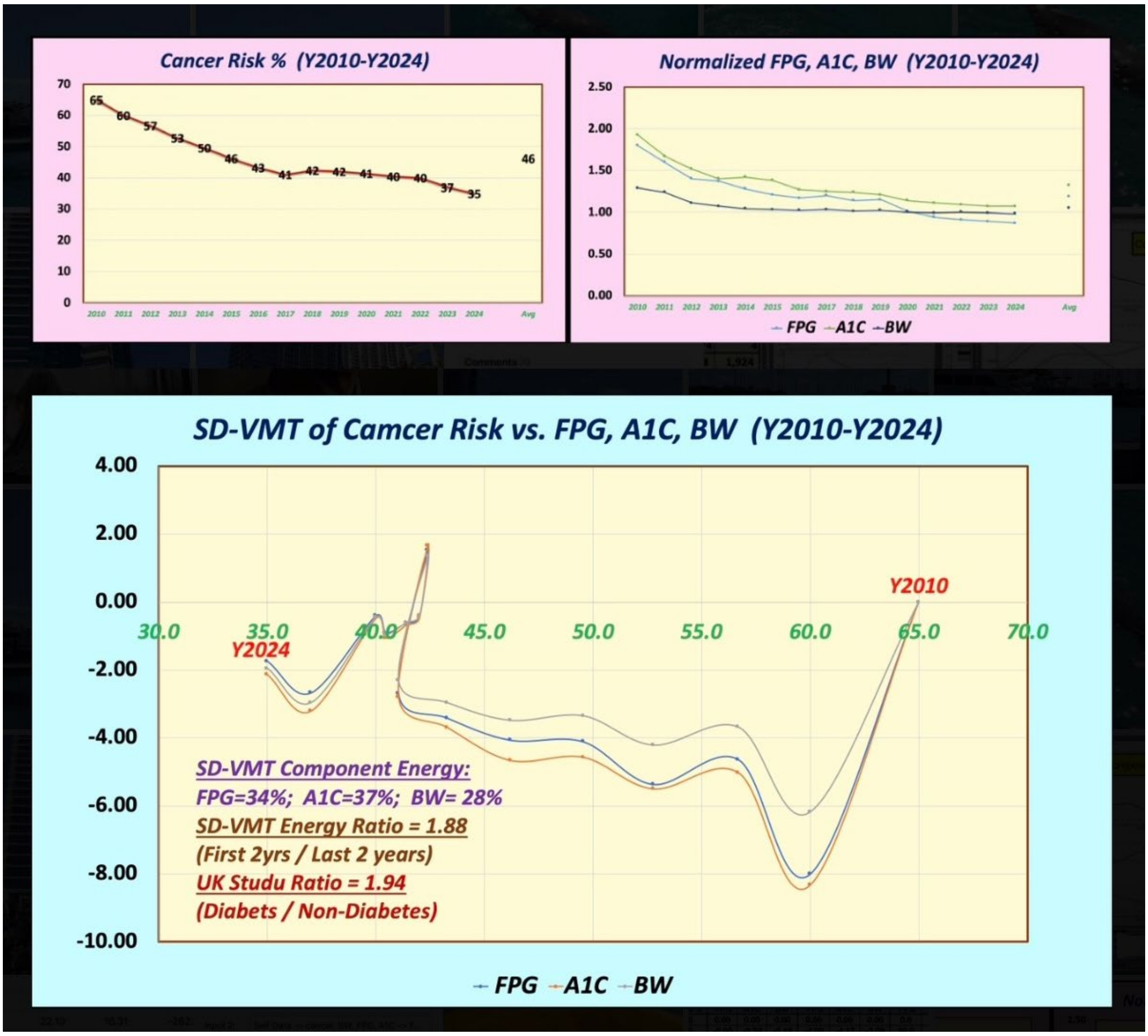
Furthermore, the SD-VMT energy ratios were distributed as follows:

A1C (diabetes) at 37%,
FPG (insulin) at 34%,
Body weight (primary cause) at 28%.

Although the author is an individual patient with Type 2 Diabetes (T2D) who transitioned from a severe condition (A1C at 10.25) in 2010-2011 to well-managed T2D without medication intervention in 2023-2024 (A1C at 6.1), his case simulation reflects the broader comparison between individuals with and without diabetes.

Key Message

Individuals with diabetes face nearly twice the risk (ranging between 1.74 and 1.94) of developing cancers compared to those without diabetes. This underscores the critical importance of managing diabetes to mitigate cancer risks.



1. Introduction

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The NIH/Lancet study found that out of every 1,000 individuals, there are 7.7 (range 7.4-8.0) with diabetes and 4.4 (range 4.3-4.6) without diabetes, leading to the ***UK Study’s cancer ratio of 1.75 between those with diabetes and those without diabetes.***

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Further, applying the Space-Domain Viscoplastic Energy Model (SD-VMT), the author estimated the energy generated through the interaction between cancer risk percentage (output strain) and three influential stresses (FPG indicating pancreatic beta cells health state, HbA1C indicating the severity of Type 2 Diabetes, and body weight indicating the primary cause of many metabolic disorders, including both T2D and cancers). ***The SD-VMT model showed a cancer energy ratio of 1.88 between the initial two years (2010 and 2011 with severe diabetes) and the most recent two years (2023 and 2024 with controlled diabetes), calculated as earlier energy of 56 divided by recent energy of 30.***

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 Cancers

The relationship between diabetes and cancer is a complex and multifaceted one, and the underlying pathophysiological explanations are still being studied. Here are a few key points that help explain the relationship:

2.1 Hyperinsulinemia and Insulin Resistance

Type 2 diabetes is often associated with insulin resistance and hyperinsulinemia, where the body's cells become less responsive to insulin, leading to higher insulin levels in the bloodstream. Elevated insulin levels can promote cell proliferation and growth, which may contribute to the development and progression of cancer.

2.2 Chronic Inflammation

Both diabetes and cancer are associated with chronic inflammation. Inflammatory processes can create a microenvironment that is conducive to cancer development and growth. In diabetes, chronic inflammation is often related to elevated blood sugar levels, which can trigger the release of pro-inflammatory cytokines.

2.3 Hyperglycemia

Diabetes is characterized by elevated blood sugar levels (hyperglycemia). High glucose levels provide a source of energy for cancer cells, potentially promoting tumor growth and progression.

2.4 Shared Risk Factors

Diabetes and cancer share common risk factors such as obesity, physical inactivity, and poor diet. These lifestyle-related risk factors can contribute to the development of both conditions.

2.5 Hormonal Factors

Diabetes can disrupt the balance of various hormones such as insulin, IGF-1 (insulin-like growth factor 1), and sex hormones. These hormonal imbalances can impact cell growth, apoptosis (cell death), and other processes related to cancer development.

2.6 Shared Genetic and Molecular Pathways

Some evidence suggests that diabetes and cancer share common genetic and molecular pathways. For example, alterations in genes involved in insulin signaling may be linked to both conditions.

While these points provide a partial understanding of the relationship between diabetes and cancer, it's important to note that ongoing research is continually shedding light on the complex interplay of genetic, metabolic, and environmental factors that underlie this relationship.

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:

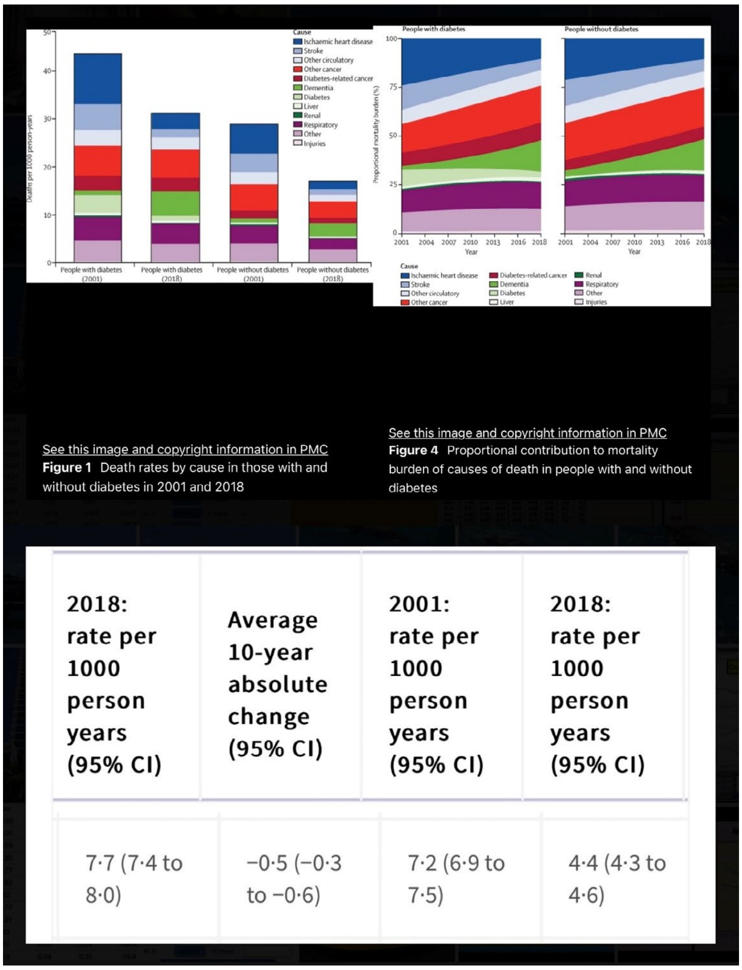
Strain rate
= *(body weight at next time instant) - (body weight at present time instant)*

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).

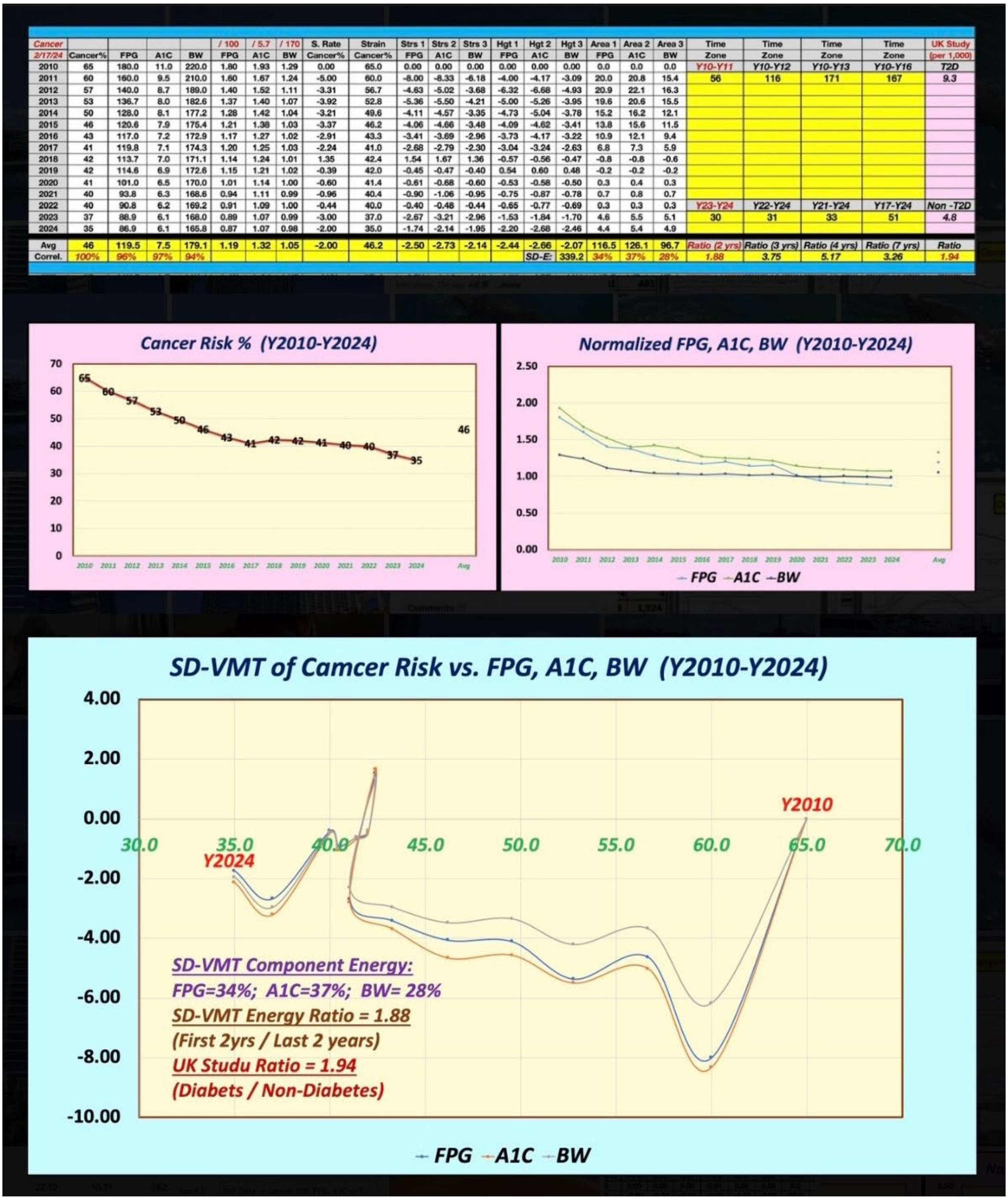
6. Results

Figure 1. Show the UK report data.



2018: rate per 1000 person years (95% CI)	Average 10-year absolute change (95% CI)	2001: rate per 1000 person years (95% CI)	2018: rate per 1000 person years (95% CI)
7·7 (7·4 to 8·0)	−0·5 (−0·3 to −0·6)	7·2 (6·9 to 7·5)	4·4 (4·3 to 4·6)

Figure 1: The UK report data



Cancer 2/17/24	Cancer%	FPG	A1C	BW	/ 100 FPG	/ 5.7 A1C	/ 170 BW	S. Rate Cancer%	Strain Cancer%	Strs 1 FPG	Strs 2 A1C	Strs 3 BW	Hgt 1 FPG	Hgt 2 A1C	Hgt 3 BW
2010	65	180.0	11.0	220.0	1.80	1.93	1.29	0.00	65.0	0.00	0.00	0.00	0.00	0.00	0.00
2011	60	160.0	9.5	210.0	1.60	1.67	1.24	-5.00	60.0	-8.00	-8.33	-6.18	-4.00	-4.17	-3.09
2012	57	140.0	8.7	189.0	1.40	1.52	1.11	-3.31	56.7	-4.63	-5.02	-3.68	-6.32	-6.68	-4.93
2013	53	136.7	8.0	182.6	1.37	1.40	1.07	-3.92	52.8	-5.36	-5.50	-4.21	-5.00	-5.26	-3.95
2014	50	128.0	8.1	177.2	1.28	1.42	1.04	-3.21	49.6	-4.11	-4.57	-3.35	-4.73	-5.04	-3.78
2015	46	120.6	7.9	175.4	1.21	1.38	1.03	-3.37	46.2	-4.06	-4.66	-3.48	-4.09	-4.62	-3.41
2016	43	117.0	7.2	172.9	1.17	1.27	1.02	-2.91	43.3	-3.41	-3.69	-2.96	-3.73	-4.17	-3.22
2017	41	119.8	7.1	174.3	1.20	1.25	1.03	-2.24	41.0	-2.68	-2.79	-2.30	-3.04	-3.24	-2.63
2018	42	113.7	7.0	171.1	1.14	1.24	1.01	1.35	42.4	1.54	1.67	1.36	-0.57	-0.56	-0.47
2019	42	114.6	6.9	172.6	1.15	1.21	1.02	-0.39	42.0	-0.45	-0.47	-0.40	0.54	0.60	0.48
2020	41	101.0	6.5	170.0	1.01	1.14	1.00	-0.60	41.4	-0.61	-0.68	-0.60	-0.53	-0.58	-0.50
2021	40	93.8	6.3	168.6	0.94	1.11	0.99	-0.96	40.4	-0.90	-1.06	-0.95	-0.75	-0.87	-0.78
2022	40	90.8	6.2	169.2	0.91	1.09	1.00	-0.44	40.0	-0.40	-0.48	-0.44	-0.65	-0.77	-0.69
2023	37	88.9	6.1	168.0	0.89	1.07	0.99	-3.00	37.0	-2.67	-3.21	-2.96	-1.53	-1.84	-1.70
2024	35	86.9	6.1	165.8	0.87	1.07	0.98	-2.00	35.0	-1.74	-2.14	-1.95	-2.20	-2.68	-2.46
Avg	46	119.5	7.5	179.1	1.19	1.32	1.05	-2.00	46.2	-2.50	-2.73	-2.14	-2.44	-2.66	-2.07
Correl.	100%	96%	97%	94%										SD-E:	339.2

Cancer 2/17/24	Area 1 FPG	Area 2 A1C	Area 3 BW	Time Zone	Time Zone	Time Zone	Time Zone	UK Study (per 1,000)
2010	0.0	0.0	0.0	Y10-Y11	Y10-Y12	Y10-Y13	Y10-Y16	T2D
2011	20.0	20.8	15.4	56	116	171	167	9.3
2012	20.9	22.1	16.3					
2013	19.6	20.6	15.5					
2014	15.2	16.2	12.1					
2015	13.8	15.6	11.5					
2016	10.9	12.1	9.4					
2017	6.8	7.3	5.9					
2018	-0.8	-0.8	-0.6					
2019	-0.2	-0.2	-0.2					
2020	0.3	0.4	0.3					
2021	0.7	0.8	0.7					
2022	0.3	0.3	0.3	Y23-Y24	Y22-Y24	Y21-Y24	Y17-Y24	Non -T2D
2023	4.6	5.5	5.1	30	31	33	51	4.8
2024	4.4	5.4	4.9					
Avg	116.5	126.1	96.7	Ratio (2 yrs)	Ratio (3 yrs)	Ratio (4 yrs)	Ratio (7 yrs)	Ratio
Correl.	34%	37%	28%	1.88	3.75	5.17	3.26	1.94

Figure 2: Input Information, TD and SD results

7. Conclusions

In summary, this study presents ***his cancer risks calculated using three distinct methods, yielding closely aligned results within the range of 1.74 to 1.88.***

Furthermore, the SD-VMT energy ratios were distributed as follows:

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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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