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Research Article

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Pathophysiological discussions and statistical results between diabetes caused by obesity versus cancers caused by obesity via SD-VMT areas comparison using data collected from 2010 to 2023 and based on space-domain viscoplastic energy model of GH-Method: Math-Physical Medicine (No. 893, VGT #293)

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Abstract

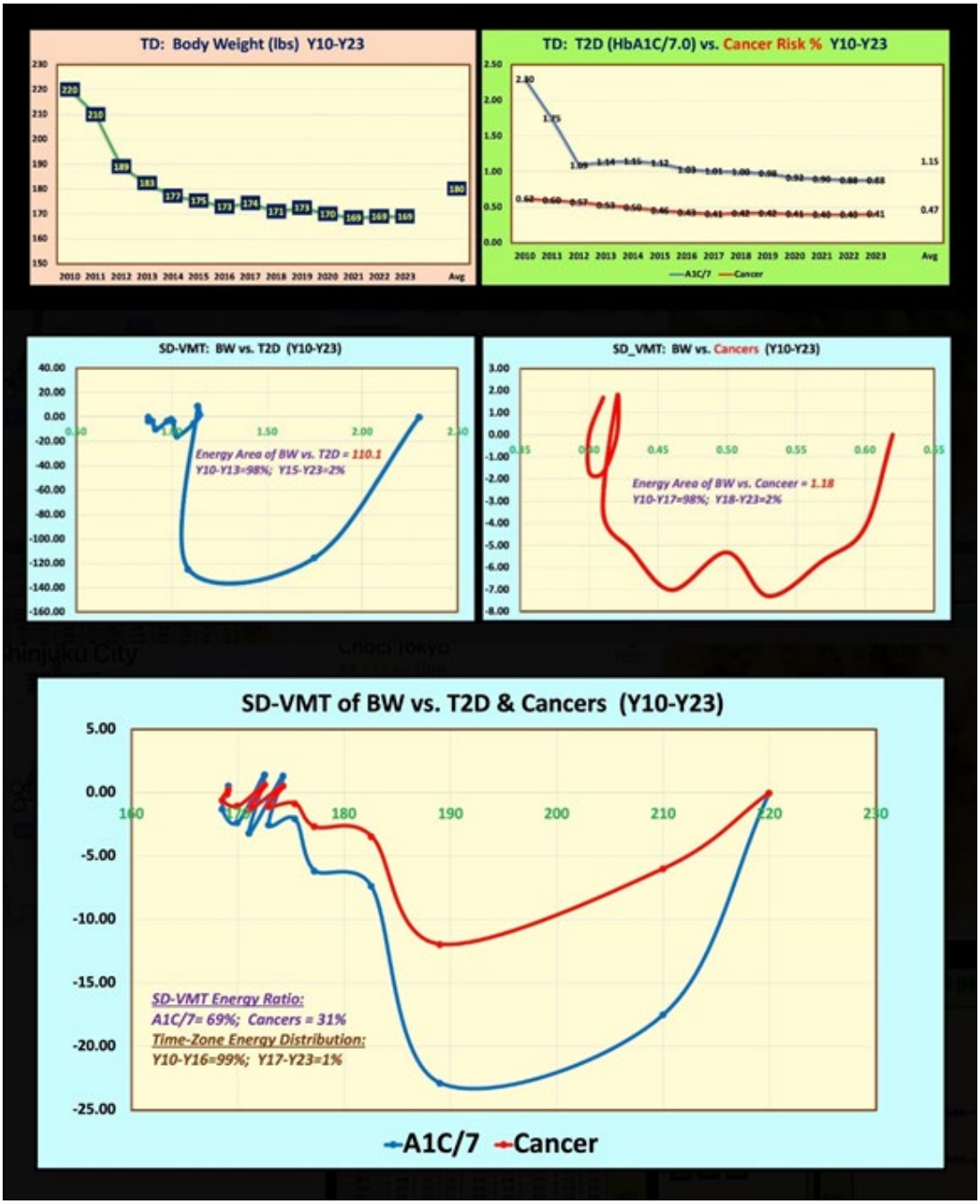
The objective of this methodology article is to assess and compare the risks of developing type 2 diabetes (T2D) and cancers caused by obesity as a single input factor. *The study spans a timeframe of approximately 14 years, from 2010 to 2023. The author personally collected and analyzed their input data, although there were limitations in obtaining sufficient lab-tested data between 2010 and 2012. From 2013 to 2018, the data collection methods involved more lab testing and personal data obtained through finger-piercing glucose testing. A more complete and comprehensive dataset was obtained from 2018 to 2023, utilizing laboratory tests, automated glucose sensors, and wearable health devices. Despite these limitations, the annual dataset used in this study is considered adequate for drawing useful and reasonably accurate conclusions.*

The author has been diagnosed with T2D since 1996 but has not received any cancer diagnoses. The assessment of cancer risk in this study is conducted using a sophisticated metabolism index (MI) model developed by the author in 2014. This model takes into account factors such as lifetime bad habits, environmental factors, genetic concerns, four metabolic disorders, and six lifestyle details.

To analyze and compare the contribution of T2D (measured through HbA1C levels) and cancer risks based on the single input cause of obesity (measured through body weight, m1), this study utilizes the space-domain viscoplastic energy model from GH-Method: Math-Physical Medicine.

In summary*, this research presents two key findings:*
Firstly, ***the author conducts two separate analyses to evaluate the impact of obesity on his existing T2D condition and his future risks of developing cancer.*** *The SD-VMT analysis shows that* ***the energy level for T2D and obesity is 110.1, while the energy level for cancers and obesity is only 1.18, representing just 1% of the diabetes energy level. This finding indicates that despite being a T2D veteran of 26 years, the author has not shown any signs of developing cancer.*** *Furthermore, his SD time-zone analysis reveals that 98% of the total energy is concentrated in the Y10-Y13 for T2D and Y10-Y17 for cancers, while only 2% falls in the Y14-Y23 for T2D and Y18-Y23 for cancers.*

Secondly, ***as an exploratory medical research methodology, he considers obesity as the output symptom, with diabetes and cancers being two separate input causes.*** *The SD-VMT energy ratios demonstrate that* ***diabetes accounts for 69% of the total energy, while cancers account for 31%.*** *Although diabetes has twice as much energy as cancers, the disparity is not as substantial as the 99-fold difference observed in the first case. This finding emphasizes that the author's existing diabetes condition is significantly more severe than his risks of developing cancer.*



Pathophysiological discussions and statistical results between diabetes caused by obesity versus cancers caused by obesity via SD-VMT areas comparison using data collected from 2010 to 2023 and based on space-domain viscoplastic energy model of GH-Method: Math-Physical Medicine (No. 893)

1. Introduction

The aim of this research is to compare the risks of type 2 diabetes (T2D) caused by obesity with the risks of cancer occurrence caused by obesity. The study spanned a period of approximately 14 years, from 2010 to 2023. The author collected all the analyzed data personally, although there were some limitations in the availability of lab-tested data between 2010 and 2012. Subsequently, data collection methods included both lab testing and personal data collection, such as finger-piercing glucose data, from 2013 to 2018, and more complete data was obtained through the use of labs, automated sensors for glucose data, and certain wearable devices from 2018 to 2023. Despite these limitations, the annual data utilized in this study is deemed sufficient to draw some useful and reasonably accurate conclusions.

It is worth noting that the author has been diagnosed with type 2 diabetes (T2D) since 1996 but has not been diagnosed with any cancers. The assessment of cancer risk is calculated using a sophisticated metabolism index (MI) model developed by the author in 2014, which takes into account lifetime bad habits, environmental factors, genetic concerns, 4 metabolic disorders and 6 lifestyle details.

To analyze and compare the total energies (i.e. degrees of influence or contribution) between T2D (measured by HbA1C levels) and obesity (measured by body weight, m1) versus cancer risks and obesity, this study employs the space-domain viscoplastic energy model from GH-Method: Math-Physical Medicine.

2. Background Information

2.1 Pathophysiological description and statistical data regarding type 2 diabetes caused by obesity:

2.1.1 Pathophysiological Description

Obesity is considered a major risk factor for the development of type 2 diabetes (T2D) mellitus. The mechanism underlying this association involves a complex interaction of various physiological factors. Here is a brief pathophysiological description:

1. Insulin Resistance: Obesity is often accompanied by insulin resistance, a condition in which cells become less responsive to the effects of insulin. Adipose tissue, particularly visceral fat, releases inflammatory substances that interfere with insulin signaling. As a result, glucose uptake by peripheral tissues is impaired, leading to raised blood glucose levels.

2. Beta-Cell Dysfunction: In response to insulin resistance, the pancreatic beta cells increase insulin production to compensate for reduced effectiveness. However, over time, excessive demand on the beta cells leads to dysfunction and reduced insulin secretion.

3. Inflammatory Response: Obesity triggers a chronic low-grade inflammatory state due to the release of adipokines and proinflammatory cytokines from adipose tissue. Inflammation further promotes insulin resistance and contributes to the development of diabetes.

4. Lipotoxicity: Excess fatty acids released from adipose tissue are deposited in non-adipose tissues like the liver and muscle. This accumulation of lipids in these tissues disrupts normal metabolic processes, further impairing insulin signaling and worsening insulin resistance.

2.1.2 Statistical Data:

Several studies have highlighted the strong association between obesity and diabetes. Here are some key statistics:

1. Global Trends: According to the World Health Organization, ***around 90% of people with type 2 diabetes are overweight or obese.***

2. Increased Risk: Obesity increases the risk of developing type 2 diabetes by approximately 6 to 7 times compared to individuals with a healthy weight.

3. Prevalence: In the United States, the National Health and Nutrition Examination Survey (NHANES) reported that ***around 87% of adults with diabetes are either overweight or obese.***

4. Body Mass Index (BMI): BMI is a common measure of obesity. Research shows a clear positive correlation between increasing BMI and the risk of developing diabetes. It has been proven that the ***waistline to hipline ratio (WHR) may be a more effective measurement biomarker.***

5. Ethnic Disparities: Certain ethnic groups, such as South Asians, Hispanics, and non-Hispanic Blacks, have a higher prevalence of both obesity and diabetes.

It's important to note that while obesity is a significant risk factor for diabetes, it does not guarantee its development. Other factors like genetics, lifestyle, and overall health also play crucial roles. Additionally, diabetes can occur in individuals who are not obese, emphasizing the multifactorial nature of the disease.

2.2 Pathophysiological description and statistical data regarding various cancers caused by obesity

2.2.1 Pathophysiological Description

Obesity is recognized as a significant risk factor for various types of cancer. The association between obesity and cancer involves complex pathophysiological mechanisms. Here is a general explanation:

1. Chronic Inflammation: Obesity is associated with a state of chronic low-grade inflammation. Adipose tissue releases pro-inflammatory cytokines and adipokines, which can lead to inflammation throughout the body. ***Chronic inflammation promotes tumor development and progression.***

2. Hormonal Imbalance: Adipose tissue produces hormones, such

as estrogen, insulin, and leptin, which are often dysregulated in obesity. ***Elevated levels of circulating hormones, particularly estrogen, can increase the risk of hormone-related cancers like breast and endometrial cancer.***

3. Insulin Resistance: Obesity is frequently linked to insulin resistance, a condition where cells become less responsive to insulin. ***Insulin resistance can lead to increased insulin and insulin-like growth factor (IGF-1) levels, promoting cell proliferation and growth, which can contribute to cancer development.***

4. Alterations in Adipokines: Obese individuals often have disrupted levels of adipokines, including adiponectin and leptin. These alterations can impact cell growth, angiogenesis, and immune functioning, potentially facilitating cancer formation and progression.

5. Dyslipidemia: Obesity is commonly associated with dyslipidemia, characterized by elevated levels of cholesterol and triglycerides. ***High levels of circulating lipids can increase oxidative stress, promote inflammation, and contribute to cancer development.***

2.2.2 Statistical Data

The association between obesity and cancer has been extensively studied. Here are some key statistics:

1. Relative Risk: Epidemiological studies have shown that obesity is associated with an increased risk of several types of cancer, including colorectal, kidney, pancreatic, breast (postmenopausal), endometrial, ovarian, liver, and esophageal cancer.

2. Global Trends: The International Agency for Research on Cancer estimates that ***approximately 20% of all cancer cases worldwide are attributed to excess body weight and physical inactivity.***

3. Impact on Cancer Mortality: Obesity is linked to a higher risk of cancer-related mortality. According to some studies, ***obesity-related cancers account for about 40% of cancer deaths in the United States.***

4. Body Mass Index (BMI): Research consistently demonstrates a positive relationship between BMI and the risk of several cancers. Higher BMI categories are associated with a progressively increased risk.

5. Ethnic Disparities: The association between obesity and cancer can vary among different ethnic groups. For example, ***obesity has a particularly strong link to endometrial cancer among postmenopausal women of non-Hispanic white ethnicity.***

It is important to note that while ***obesity is a recognized risk factor for cancer, it is not the sole determinant. Other factors, such as genetics, lifestyle, and environmental exposures, also influence cancer development. Adopting a healthy lifestyle, including maintaining a balanced weight, engaging in regular physical activity, and following a nutritious diet, can contribute to reducing the risk of obesity-related cancers.***

3. Methods

3.1 MPM Background

To learn more about his developed GH-Method: math-physical medicine (MPM) methodology, readers can read the following three papers selected from his published 760+ papers.

The first paper, No. 386 (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.

3.2 The author's diabetes history

The author was a severe T2D patient since 1995. He weighed 220 lb. (100 kg) at that time. By 2010, he still weighed 198 lb. with an average daily glucose of 250 mg/dL (HbA1C at 10%). During that year, his triglycerides reached 1161 (high risk for CVD and stroke) and his albumin-creatinine ratio (ACR) at 116 (high risk for chronic kidney disease). He also suffered from five cardiac episodes within a decade. In 2010, three independent physicians warned him regarding the need for kidney dialysis treatment and the future high risk of dying from his severe diabetic complications.

In 2010, he decided to self-study endocrinology with an emphasis on diabetes and food nutrition. He spent the entire year of 2014 to develop a metabolism index (MI) mathematical model. During 2015 and 2016, he developed four mathematical prediction models related to diabetes conditions: weight, PPG, fasting plasma glucose (FPG), and HbA1C (A1C). Through using his developed mathematical metabolism index (MI) model and the other four glucose prediction tools, by the end of 2016, his weight was reduced from 220 lbs. (100 kg) to 176 lbs. (89 kg), waistline from 44 inches (112 cm) to 33 inches (84 cm), average finger-piercing glucose from 250 mg/dL to 120 mg/dL, and A1C from 10% to ~6.5%. One of his major accomplishments is that he no longer takes any diabetes-related medications since 12/8/2015.

In 2017, he achieved excellent results on all fronts, especially his glucose control. However, during the pre-COVID period, including both 2018 and 2019, he traveled to ~50 international cities to attend 65+ medical conferences and made ~120 oral presentations. This hectic schedule inflicted damage to his diabetes control caused by stress, dining out frequently, post-meal exercise disruption, and jet lag, along with the overall negative metabolic impact from the irregular life patterns; therefore, his glucose control was somewhat affected during the two-year traveling period of 2018-2019.
He started his COVID-19 self-quarantined life on 1/19/2020.

By 10/16/2022, his weight was further reduced to ~164 lbs. (BMI 24.22) and his A1C was at 6.0% without any medication intervention or insulin injection. In fact, with the special COVID-19 quarantine lifestyle since early 2020, not only has he written and published ~500 new research articles in various medical and engineering journals, but he has also achieved his best health conditions for the past 27 years. These achievements have resulted from his non-traveling, low-stress, and regular daily life routines. Of course, his in-depth knowledge of chronic diseases, sufficient practical lifestyle management experiences, and his own developed high-tech tools have also contributed to his excellent health improvements.

On 5/5/2018, he applied a continuous glucose monitoring (CGM) sensor device on his upper arm and checks his glucose measurements every 5 minutes for a total of 288 times each day. Furthermore, he extracted the 5-minute intervals from every 15-minute interval for a total of 96 glucose data each day stored in his computer software.

Through the author's medical research work over 40,000 hours and read over 4,000 published medical papers online in the past 13 years, he discovered and became convinced that good life habits of not smoking, moderate or no alcohol intake, avoiding illicit drugs; along with eating the right food with well-balanced nutrition, persistent exercise, having a sufficient and good quality of sleep, reducing all kinds of unnecessary stress, maintaining a regular daily life routine contribute to the risk reduction of having many diseases, including CVD, stroke, kidney problems, micro blood vessels issues, peripheral nervous system problems, and even cancers and dementia. In addition, a long-term healthy lifestyle can even "repair" some damaged internal organs, with different required time-length depending on the particular organ's cell lifespan. For example, he has "self-repaired" about 35% of his damaged pancreatic beta cells during the past 10 years.

3.3 Energy theory

The human body and organs have around 37 trillion live cells which are composed of different organic cells that require energy infusion from glucose carried by red blood cells; and energy consumption from labor-work or exercise. When the residual energy (resulting from the plastic glucose scenario) is stored inside our bodies, it will cause different degrees of damage or influence to many of our internal organs.

According to physics, energies associated with the glucose waves are proportional to the square of the glucose amplitude. The residual energies from elevated glucoses are circulating inside the body via blood vessels which then impact all of the internal organs to cause different degrees of damage or influence, e.g. diabetic complications. Elevated glucose (hyperglycemia) causes damage to the structural integrity of blood vessels. When it combines with both hypertension (rupture of arteries) and hyperlipidemia (blockage of arteries), CVD or Stroke happens. Similarly, many other deadly diseases could result from these excessive energies which would finally shorten our lifespan. For an example, the combination of hyperglycemia and hypertension would cause micro-blood vessel's leakage in kidney systems which is one of the major cause of CKD.

The author then applied Fast Fourier Transform (FFT) operations to convert the input wave from a time domain into a frequency domain. The y-axis amplitude values in the frequency domain indicate the proportional energy levels associated with each different frequency component of input occurrence. ***Both output symptom value (i.e. strain amplitude in the time domain) and output symptom fluctuation rate (i.e. the strain rate and strain frequency) are influencing the energy level (i.e. the Y-amplitude in the frequency domain).***

Currently, many people live a sedentary lifestyle and lack sufficient exercise to burn off the energy influx which causes them to become overweight or obese. Being overweight and having obesity leads to a variety of chronic diseases, particularly diabetes. In addition, many types of processed food add unnecessary ingredients and harmful chemicals that are toxic to the bodies, which lead to the development of many other deadly diseases, such as cancers. For example, ~85% of worldwide diabetes patients are overweight, and ~75% of patients with cardiac illnesses or surgeries have diabetes conditions.

In engineering analysis, when the load is applied to the structure, it bends or twists, i.e. deform; however, when the load is removed, it will either be restored to its original shape (i.e, elastic case) or remain in a deformed shape (i.e. plastic case). In a biomedical system, the glucose level will increase after eating carbohydrates or sugar from food; therefore, the carbohydrates and sugar function as the energy supply. After having labor work or exercise, the glucose level will decrease. As a result, the exercise burns off the energy, which is similar to load removal in the engineering case. In the biomedical case, both processes of energy influx and energy dissipation take some time which is not as simple and quick as the structural load removal in the engineering case. Therefore, the age difference and 3 input behaviors are "dynamic" in nature, i.e. time-dependent. *This time-dependent nature leads to a "viscoelastic or viscoplastic" situation.* ***For the author's case, it is "viscoplastic" since most of his biomarkers are continuously improved during the past 13-year time window.***

*3.4 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).***

A1C vs. BW	100%	96%	/ 1	Strain Rate	Strain	Stress 1	Height 1	Area 1	
7/26/23	A1C/7	BW	N. 1	A1C/7 Rate	A1C/7	BW	BW	BW	Time-Zone
2010	2.30	220	220	0.00	2.30	0.00	0.00	0.00	Y10-Y13
2011	1.75	210	210	-0.55	1.75	-115.50	-57.75	31.76	108.2
2012	1.09	189	189	-0.66	1.09	-124.76	-120.13	79.29	98%
2013	1.14	183	183	0.05	1.14	9.13	-57.82	-2.89	
2014	1.15	177	177	0.01	1.15	1.77	5.45	0.05	Y14-Y23
2015	1.12	175	175	-0.03	1.12	-5.26	-1.74	0.05	1.9
2016	1.03	173	173	-0.09	1.03	-15.56	-10.41	0.94	2%
2017	1.01	174	174	-0.02	1.01	-3.49	-9.53	0.19	
2018	1.00	171	171	-0.01	1.00	-1.71	-2.60	0.03	
2019	0.98	173	173	-0.02	0.98	-3.45	-2.58	0.05	
2020	0.92	170	170	-0.06	0.92	-10.20	-6.83	0.41	
2021	0.90	169	169	-0.02	0.90	-3.37	-6.79	0.14	
2022	0.88	169	169	-0.02	0.88	-3.38	-3.38	0.07	
2023	0.88	169	169	0.00	0.88	0.00	-1.69	0.00	
Avg	1.15	180	180	-0.10	1.15	-19.70	-19.70	110.08	
							SD-E:	110.1	

Cancer vs. BW	1.00	0.93	/ 1	Strain Rate	Strain	Stress 1	Height 1	Area 1	
7/26/23	Cancer	BW	BW	Cancer Rate	Cancer	BW	BW	BW	
2010	0.62	220	220	0.00	0.62	0.00	0.00	0.00	Y10-Y17
2011	0.60	210	210	-0.02	0.60	-4.20	-2.10	0.04	1.16
2012	0.57	189	189	-0.03	0.57	-5.67	-4.94	0.15	98%
2013	0.53	183	183	-0.04	0.53	-7.30	-6.49	0.26	
2014	0.50	177	177	-0.03	0.50	-5.32	-6.31	0.19	
2015	0.46	175	175	-0.04	0.46	-7.02	-6.17	0.25	
2016	0.43	173	173	-0.03	0.43	-5.19	-6.10	0.18	
2017	0.41	174	174	-0.02	0.41	-3.49	-4.34	0.09	Y18-Y23
2018	0.42	171	171	0.01	0.42	1.71	-0.89	-0.01	0.03
2019	0.42	173	173	0.00	0.42	0.00	0.86	0.00	3%
2020	0.41	170	170	-0.01	0.41	-1.70	-0.85	0.01	
2021	0.40	169	169	-0.01	0.40	-1.69	-1.69	0.02	
2022	0.40	169	169	0.00	0.40	0.00	-0.84	0.00	
2023	0.41	169	169	0.01	0.41	1.69	0.85	0.01	
Avg	0.47	180	180	-0.02	0.47	-2.73	-2.79	1.18	
							SD-E:	1.18	

Figure 1: Data Tables of Case 1

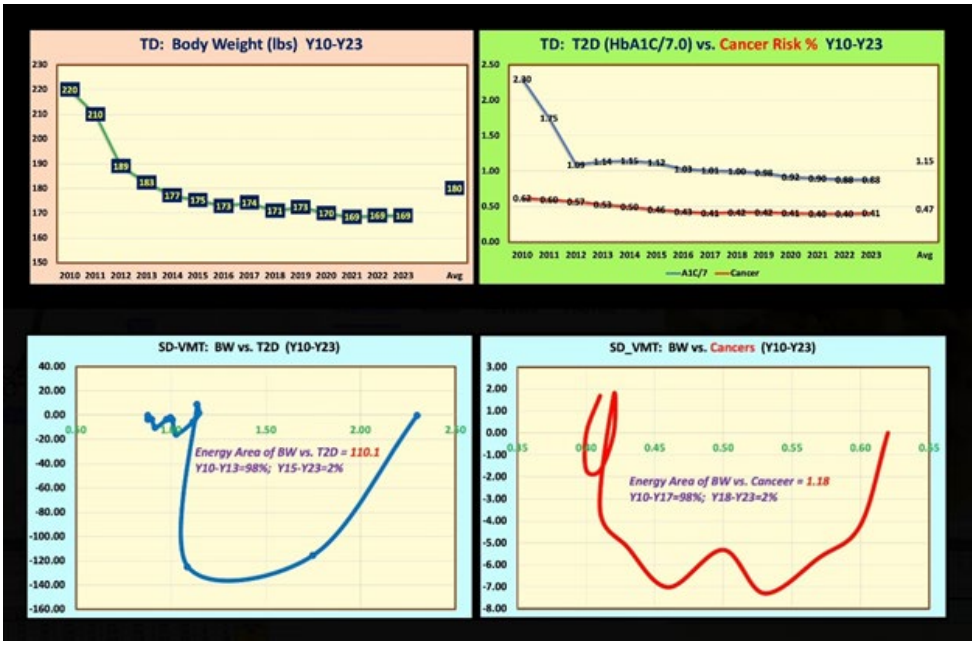
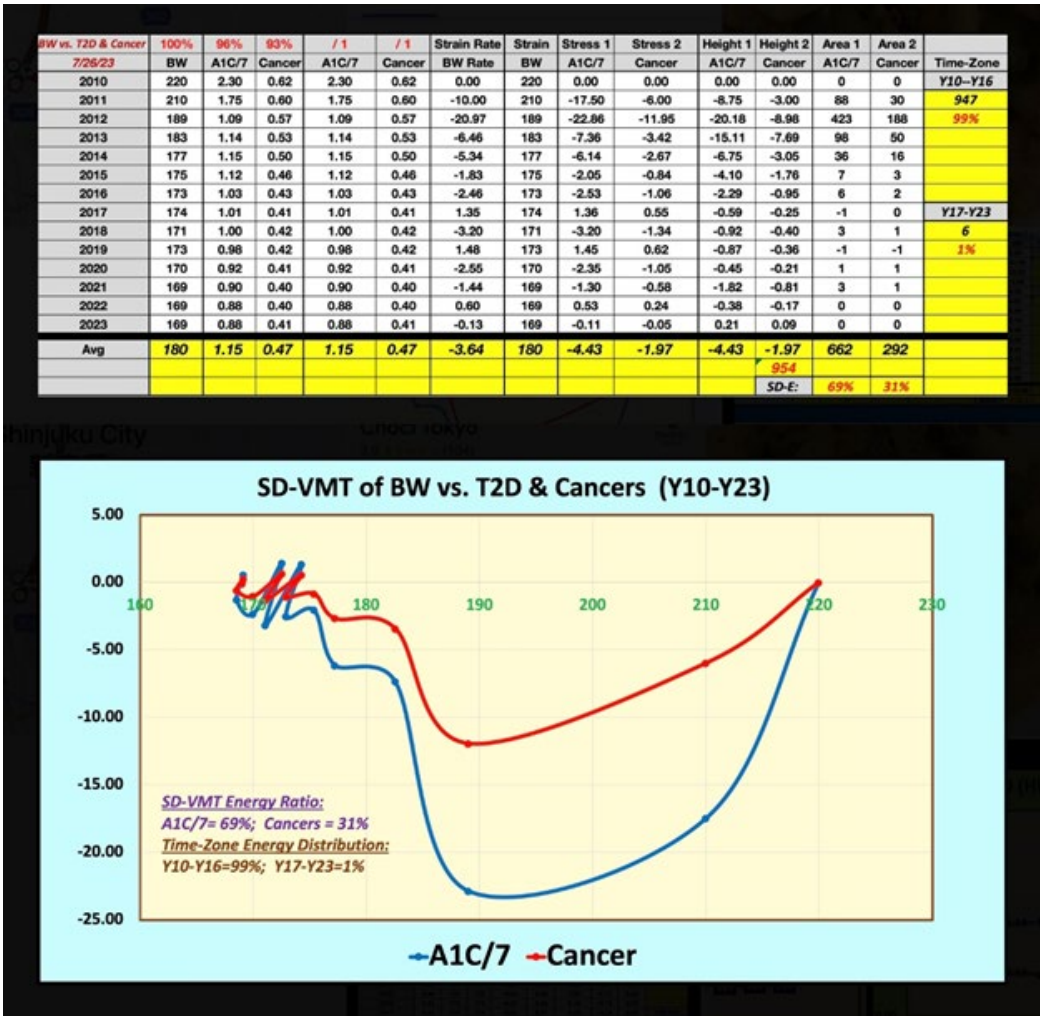


Figure 2: Analysis Results of Case 1



BW vs. T2D & Cancer	100%	96%	93%	/ 1	/ 1	Strain Rate	Strain	Stress 1	Stress 2	Height 1	Height 2	Area 1	Area 2	
7/26/23	BW	A1C/7	Cancer	A1C/7	Cancer	BW Rate	BW	A1C/7	Cancer	A1C/7	Cancer	A1C/7	Cancer	Time-Zone
2010	220	2.30	0.62	2.30	0.62	0.00	220	0.00	0.00	0.00	0.00	0	0	Y10--Y16
2011	210	1.75	0.60	1.75	0.60	-10.00	210	-17.50	-6.00	-8.75	-3.00	88	30	947
2012	189	1.09	0.57	1.09	0.57	-20.97	189	-22.86	-11.95	-20.18	-8.98	423	188	99%
2013	183	1.14	0.53	1.14	0.53	-6.46	183	-7.36	-3.42	-15.11	-7.69	98	50	
2014	177	1.15	0.50	1.15	0.50	-5.34	177	-6.14	-2.67	-6.75	-3.05	36	16	
2015	175	1.12	0.46	1.12	0.46	-1.83	175	-2.05	-0.84	-4.10	-1.76	7	3	
2016	173	1.03	0.43	1.03	0.43	-2.46	173	-2.53	-1.06	-2.29	-0.95	6	2	
2017	174	1.01	0.41	1.01	0.41	1.35	174	1.36	0.55	-0.59	-0.25	-1	0	Y17-Y23
2018	171	1.00	0.42	1.00	0.42	-3.20	171	-3.20	-1.34	-0.92	-0.40	3	1	6
2019	173	0.98	0.42	0.98	0.42	1.48	173	1.45	0.62	-0.87	-0.36	-1	-1	1%
2020	170	0.92	0.41	0.92	0.41	-2.55	170	-2.35	-1.05	-0.45	-0.21	1	1	
2021	169	0.90	0.40	0.90	0.40	-1.44	169	-1.30	-0.58	-1.82	-0.81	3	1	
2022	169	0.88	0.40	0.88	0.40	0.60	169	0.53	0.24	-0.38	-0.17	0	0	
2023	169	0.88	0.41	0.88	0.41	-0.13	169	-0.11	-0.05	0.21	0.09	0	0	
Avg	180	1.15	0.47	1.15	0.47	-3.64	180	-4.43	-1.97	-4.43	-1.97	662	292	
											954			
											SD-E:	69%	31%	

Figure 3: Data and Result of Case 2

5. Conclusions

In summary, this research yields two key observations:

Firstly, from a pathophysiological perspective, obesity is a significant contributing factor to both diabetes and cancers. It leads to issues such as pancreatic beta cell disorders, insulin resistance, chronic inflammation, lipotoxicity, alterations in adipokines, hormonal imbalance, and dyslipidemia, which can contribute to the development of both diabetes and certain cancers. In light of this, the author conducts two separate analyses to assess the influence of obesity on his own type 2 diabetes (T2D) condition and his future risks of having cancers. The SD-VMT analysis reveals that the energy level for T2D and obesity is 110.1, whereas the energy level for cancer risks and obesity is only 1.18, which amounts to just 1% of the diabetes energy level. This finding demonstrates that the author, as a 26-years veteran of T2D, has shown no signs of developing any cancer. Additionally, the author's SD time-zone analysis results indicate that 98% of the total energy is concentrated in the Y10-Y13 period, while Y14-Y23 period accounts for only 2%.

Secondly, as an exploratory medical research methodology, the author reverses the roles by considering obesity as the output symptom, with both diabetes and cancers as two separate input causes. Of course, he acknowledges that this does not align with biomedical understanding, but he still is curious to examine the energy ratios between diabetes and cancers which are associated with obesity in this exploratory scenario. The SD-VMT energy ratios reveal that diabetes accounts for 69% of the total energy, while cancers account for 31%. Although diabetes energy is twice as high as the energy for cancers, it is not as drastically higher as the initial energy ratio of 99 times. From a biomedical perspective, this second research case holds no practical meaning. However, it still highlights that his existing diabetes condition is much more severe than his risks of developing cancers.

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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