

Relationships of Alzheimer's disease Risks and Five Inputs of Bmi, Whr, Eag, Diet and Exercise Using Viscoplastic Energy Model of Gh-Method: Math Physical Medicine

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Abstract

Diabetes is estimated to impact up to 81% of individuals with Alzheimer's. Furthermore, 50% to 80% (averaging 65%) of Parkinson's disease patients may also have diabetes. In a study involving 10,095 participants (67.3% men; aged 35 to 55 years in 1985 to 1988), 1,710 cases have diabetes (17%) and 639 cases have dementia (6%) were recorded over 31.7 years.

From a pathophysiological standpoint, Alzheimer's disease is intricately linked to metabolic disorders, particularly obesity and type 2 diabetes. Body weight, assessed through BMI and WHR, along with blood glucose levels, directly correlates with both diet and exercise.

Hence, the author explores his estimated Alzheimer's disease (AD) risks associated with five inputs: BMI, WHR, eAG, diet (food portion and meal quality), and walking steps, based on personal data collected between 1/1/2013 and 11/30/2023.

The author, having experienced complications from metabolic disorders, has been motivated to conduct numerous studies exploring the connection between mortality diseases and their key inputs. Notably, the waist-to-hip ratio (WHR), reflective of visceral fat, is introduced as a new variable in this analysis—an aspect seldom considered in the author's prior studies.

In summary, a statistical correlation analysis unveils robust correlations (90% to 93%) between the author's AD risk and four inputs, with a lower 67% correlation with walking steps. This lower correlation is attributed to variations in his walking steps, ranging from 7.5k in 2013, increasing to 18.5k in 2018, later reducing to 12.7k in 2023 due to agerelated discomfort from excessive walking.

Applying the space-domain viscoplastic energy (SD-VMT) method, the author reveals hidden relationships and dynamics (i.e. energies) between these 5 inputs and annual AD risk output:

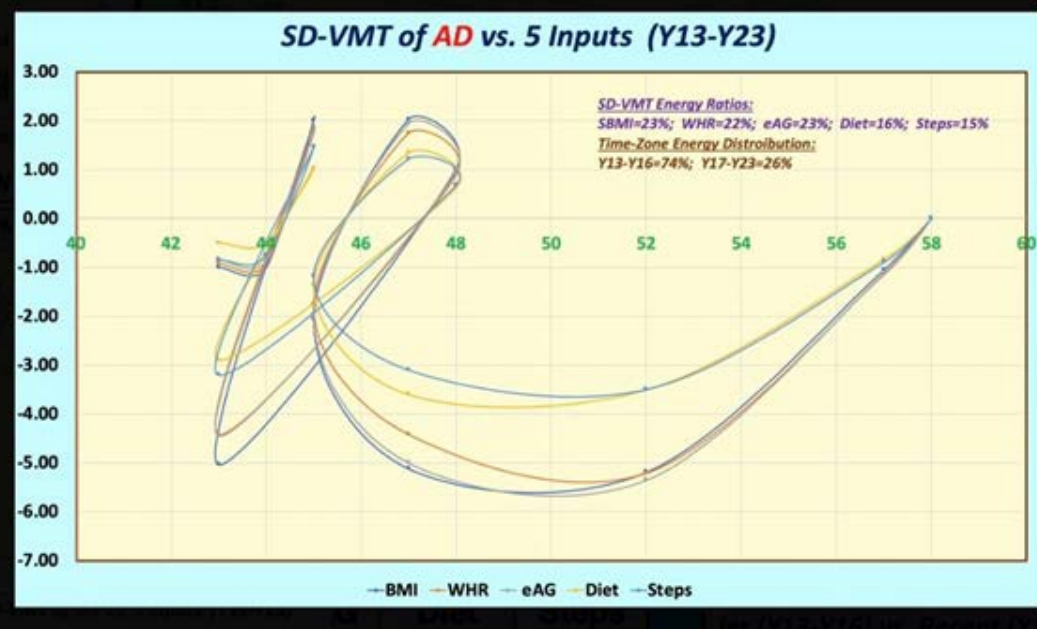
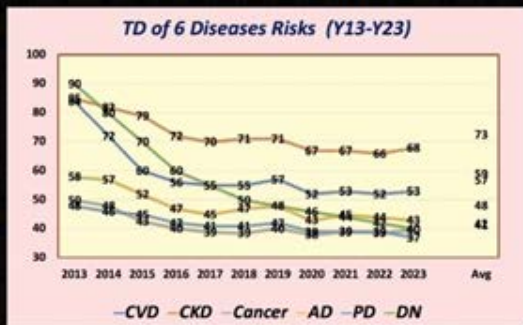
- **BMI Energy: 23%**
- **WHR Energy: 22%**
- **eAG Energy: 23%**
- **Diet Energy: 16% - Steps Energy: 15%**

Key Message

The author's three metabolic biomarkers, BMI, WHR, and eAG, contribute 22% to 23% each (totaling 68%), while lifestyle details, diet and exercise, contribute 15% to 16% each (totaling 31%). This 68% to 31% split aligns with the author's defined Da-Vinci code of medicine, reflecting a 70 to 30 ratio. Lifestyles significantly impact metabolic disorders, thereby influencing mortality diseases like AD. It is important to note that the author hasn't been diagnosed with any signs of Alzheimer's diseases. Despite lacking real data observations for direct comparison, this research, from a preventive medicine perspective, offers valuable insights into the author's health maintenance program.

Viscoelastic Medicine Theory (VMT #377) Relationships of Alzheimer's disease risks and five inputs of BMI, WHR, eAG, Diet and Exercise using viscoplastic energy model of GH-Method: mathphysical medicine (No. 978)

AD	BMI	WHR	eAG	Diet	Steps	N.1	N.2	N.3	N.4	N.5	Rate	Strain	Strs 1	Strs 2	Strs 3	Strs 4	Strs 5	Hgt 1	Hgt 2	Hgt 3	Hgt 4	Hgt 5	Area 1	Area 2	Area 3	Area 4	Area 5	Time Zone		
2013	27.02	1.10	1.30	1.00	1.25	1.08	1.18	1.30	1.00	1.25	0.00	98	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	Y13-Y18
2014	27.23	1.08	1.12	0.83	0.80	1.05	1.14	1.12	0.83	0.80	-1.00	57	-1.05	-1.14	-1.12	-0.83	-0.80	-0.52	-0.57	-0.56	-0.41	-0.45	0.5	0.8	0.8	0.4	0.5	182	74%	
2015	25.86	0.96	1.07	0.70	0.70	1.04	1.04	1.07	0.70	0.70	-5.00	52	-5.19	-5.21	-5.20	-3.90	-3.90	-3.12	-3.17	-3.24	-3.17	-3.19	15.8	15.9	16.2	10.8	11.0	174	74%	
2016	25.59	0.84	1.00	0.72	0.62	1.02	0.88	1.00	0.72	0.62	-5.00	47	-5.12	-4.42	-5.00	-3.00	-3.10	-5.16	-4.82	-5.10	-3.55	-3.30	25.8	24.1	25.9	17.8	16.5	182	74%	
2017	25.79	0.84	0.86	0.68	0.59	1.03	0.88	0.96	0.68	0.59	-2.00	45	-2.08	-1.77	-1.86	-1.36	-1.18	-3.59	-3.08	-3.48	-2.48	-2.14	7.2	6.2	7.0	5.0	4.3	177	Y17-Y23	
2018	25.32	0.83	0.97	0.67	0.61	1.01	0.87	0.97	0.67	0.61	2.00	47	2.03	1.79	1.94	1.34	1.22	-0.02	-0.01	-0.01	-0.01	0.02	0.0	0.0	0.0	0.0	0.0	95	95	
2019	25.54	0.85	0.95	0.63	0.69	1.02	0.89	0.95	0.63	0.69	1.00	48	1.02	0.89	0.95	0.63	0.69	1.02	1.30	1.44	0.99	0.95	1.0	1.3	1.4	1.0	1.0	20%	20%	
2020	25.16	0.84	0.89	0.58	0.64	1.01	0.88	0.89	0.58	0.64	-5.00	43	-5.03	-4.42	-4.45	-2.90	-3.20	-2.01	-1.76	-1.75	-1.14	-1.26	10.5	8.8	9.8	8.7	6.3	182	74%	
2021	24.95	0.87	0.87	0.51	0.73	1.00	0.92	0.87	0.51	0.73	2.00	45	2.00	1.83	1.74	1.62	1.46	-1.52	-1.29	-1.35	-0.94	-0.87	-3.0	-3.6	-2.7	-1.9	-1.7	182	74%	
2022	25.04	0.88	0.86	0.50	0.76	1.00	0.93	0.86	0.50	0.76	-1.00	44	-1.00	-0.83	-0.80	-0.90	-0.76	0.90	0.45	0.44	0.26	0.35	-0.5	-0.5	-0.4	-0.3	-0.4	182	74%	
2023	24.80	0.87	0.84	0.50	0.86	1.00	0.92	0.84	0.50	0.86	-1.00	43	-1.00	-0.82	-0.84	-0.90	-0.86	-1.00	-0.92	-0.88	-0.50	-0.81	1.0	0.8	0.9	0.5	0.8	182	74%	
Avg	25.50	0.91	0.90	0.67	0.76	1.02	0.96	0.99	0.67	0.76	-1.36	48	-1.40	-1.30	-1.38	-0.93	-0.92	-1.36	-1.26	-1.32	-0.90	-0.88	56.1	54.7	57.5	39.0	36.2	182	74%	
Correl	800%	21%	22%	23%	20%	67%																								



1. Introduction

Diabetes is estimated to impact up to 81% of individuals with Alzheimer's.

Furthermore, 50% to 80% (averaging 65%) of Parkinson's disease patients may also have diabetes. In a study involving 10,095 participants (67.3% men; aged 35 to 55 years in 1985 to 1988), 1,710 cases have diabetes (17%) and 639 cases have dementia (6%) were recorded over 31.7 years.

From a pathophysiological standpoint, Alzheimer's disease is intricately linked to metabolic disorders, particularly obesity and type 2 diabetes. Body weight, assessed through BMI and WHR, along with blood glucose levels, directly correlates with both diet and exercise.

Hence, the author explores his estimated Alzheimer's disease (AD) risks associated with five inputs:

BMI, WHR, eAG, diet (food portion and meal quality), and

walking steps, based on personal data collected between 1/1/2013 and 11/30/2023.

The author, having experienced complications from metabolic disorders, has been motivated to conduct numerous studies exploring the connection between mortality diseases and their key inputs. Notably, the waist-to-hip ratio (WHR), reflective of visceral fat, is introduced as a new variable in this analysis—an aspect seldom considered in the author's prior studies.

1.1. Biomedical Information

The following sections contain excerpts and concise information drawn from multiple medical articles, which have been meticulously reviewed by the author of this paper. The author has adopted this approach as an alternative to including a conventional reference list at the end of this document, with the intention of optimizing his valuable research time. It is essential to clarify that these sections do not constitute part of the author's

original contribution but have been included to aid the author in his future reviews and offer valuable insights to other readers with an interest in these subjects.

1.2. Pathophysiological Explanations of Relationships Between Alzheimer's Diseases and Obesity / Diabetes

The relationship between Alzheimer's disease and obesity/diabetes involves complex pathophysiological mechanisms:

1.2.1. Insulin Resistance and Brain Function: In type 2 diabetes and obesity, insulin resistance can extend to the brain. Insulin plays a crucial role in brain function, and resistance may contribute to cognitive decline and the development of Alzheimer's disease.

1.2.2. Inflammation: Obesity and diabetes are associated with chronic inflammation, and this systemic inflammation can impact the brain. Inflammation in the brain has been linked to the progression of Alzheimer's disease.

1.2.3. Vascular Effects: Diabetes and obesity can lead to vascular changes, including atherosclerosis and impaired blood flow. Vascular issues are known to contribute to cognitive decline and increase the risk of vascular dementia, which often coexists with Alzheimer's disease.

1.2.4. Amyloid and Tau Proteins: In Alzheimer's disease, abnormal accumulations of proteins, such as beta-amyloid plaques and tau tangles, are found in the brain. Some studies suggest that insulin resistance and metabolic dysfunction may contribute to the buildup of these proteins.

1.2.5. Hormonal Imbalances: Obesity and diabetes can disrupt hormonal balance, including insulin and other hormones that influence brain health. These imbalances may contribute to neurodegenerative processes.

1.2.6. Impaired Mitochondrial Function: Both obesity and diabetes can affect mitochondrial function, crucial for energy production in cells. Impaired mitochondrial function in the brain cells may contribute to Alzheimer's disease progression.

Understanding the interplay between metabolic disorders like obesity and diabetes with Alzheimer's disease is an active area of research. Lifestyle modifications, including a healthy diet, regular exercise, and managing metabolic health, are considered important in reducing the risk or slowing the progression of Alzheimer's disease.

1.3. What is Mitochondrial Function?

Mitochondrial function refers to the health and efficiency of mitochondria, which are cellular organelles responsible for producing energy in the form of adenosine triphosphate (ATP). Proper mitochondrial function is essential for various cellular processes and overall cellular health.

In the context of health and disease, disturbances in mitochondrial function can have significant implications. Impaired mitochondrial function is associated with various

conditions, including neurodegenerative diseases, metabolic disorders, and aging. Mitochondrial dysfunction may lead to a decrease in energy production, increased production of reactive oxygen species (ROS), and disruption of cellular processes.

Maintaining healthy mitochondrial function is crucial for overall cellular and organ health. Lifestyle factors such as regular exercise, a balanced diet, and stress management can positively influence mitochondrial function. Researchers are actively exploring the link between mitochondrial health and various diseases to develop strategies for preventing or treating conditions associated with mitochondrial dysfunction.

1.4. Which Foods Contain Antioxidants and Anti-Inflammatory Compounds?

Foods rich in antioxidants and antiinflammatory compounds can help combat oxidative stress and inflammation. Here are some examples:

1.4.1. Fruits

- Berries (blueberries, strawberries, raspberries)
- Citrus fruits (oranges, grapefruits)
- Cherries
- Apples
- Grapes

1.4.2. Vegetables

- Leafy greens (spinach, kale, Swiss chard)
- Broccoli
- Brussels sprouts
- Bell peppers
- Tomatoes

1.4.3. Nuts and Seeds

- Almonds
- Walnuts
- Chia seeds
- Flaxseeds

1.4.4. Herbs and Spices

- Turmeric
- Ginger
- Garlic
- Cinnamon
- Rosemary

1.4.5. Fatty Fish

- Salmon
- Mackerel
- Sardines
- Trout

1.4.6. Green Tea

- Contains polyphenols with antioxidant and anti-inflammatory properties.

1.4.7. Dark Chocolate

- In moderation, dark chocolate with a high cocoa content provides antioxidants.

1.4.8. Olive Oil

- Extra virgin olive oil contains polyphenols and has antiinflammatory effects.

1.4.9. Whole Grains

- Quinoa
- Brown rice - Oats

1.4.10. Legumes

- Lentils
- Chickpeas
- Black beans

Incorporating a variety of these foods into your diet can contribute to a well-rounded intake of antioxidants and anti-inflammatory compounds. It's essential to focus on a balanced and diverse diet to provide your body with a range of nutrients.

1.5. MPM Background

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

The first paper, No. 386 (Reference 1) describes his MPM methodology in a general conceptual format. The second paper, No. 387 (Reference 2) outlines the history of his personalized diabetes research, various application tools, and the differences between biochemical medicine (BCM) approach versus the MPM approach. The third paper, No. 397 (Reference 3) depicts a general flow diagram containing ~10 key MPM research methods and different tools.

1.6. The Author's Diabetes History

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

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

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

He started his COVID-19 selfquarantined life on 1/19/2020. By 10/16/2022, his weight was further reduced to ~164 lbs. (BMI 24.22) and his A1C was at 6.0% without any medication intervention or insulin injection. In fact, with the special COVID-19 quarantine lifestyle since early 2020, not only has he written and published ~500 new research articles in various medical and engineering journals, but he has also achieved his best health conditions for the past 27 years. These achievements have resulted from his non-traveling, low-stress, and regular daily life routines. Of course, his indepth knowledge of chronic diseases, sufficient practical lifestyle management experiences, and his own developed high-tech tools have also contributed to his excellent health improvements.

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

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

1.7. Energy Theory

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

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

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

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

In engineering analysis, when the load is applied to the structure, it bends or twists, i.e. deform; however, when the load is removed, it will either be restored to its original shape (i.e. elastic case) or remain in a deformed shape (i.e. plastic case). In a biomedical system, the glucose level will increase after eating carbohydrates or sugar from food; therefore, the carbohydrates and sugar function as the energy supply. 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.

1.8. Time-Dependent Output Strain and Stress of (Viscous Input*Output Rate)

Hooke’s law of linear elasticity is expressed as:

$$\text{Strain } (\epsilon: \text{epsilon}) = \text{Stress } (\sigma: \text{sigma}) / \text{Young's modulus } (E)$$

For biomedical glucose application, his developed linear elastic glucose theory (LEGT) is expressed as:

$$\text{PPG (strain)} = \text{carbs/sugar (stress)} * \text{GH.p-Modulus (a positive number)} + \text{post-meal walking ksteps} * \text{GH.w-Modulus (a negative number)}$$

Where GH.p-Modulus is reciprocal of Young’s modulus E.

However, in viscoelasticity or viscoplasticity theory, the stress is expressed as:

$$\text{Stress} = \text{viscosity factor } (\eta: \text{eta}) * \text{strain rate } (d\epsilon/dt)$$

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:

$$\text{Strain} = (\text{body weight at certain specific time instant})$$

He also calculates his strain rate using the following formula:

$$\text{Strain rate} = (\text{body weight at next time instant}) - (\text{body weight at present time instant})$$

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)*.

2. Results

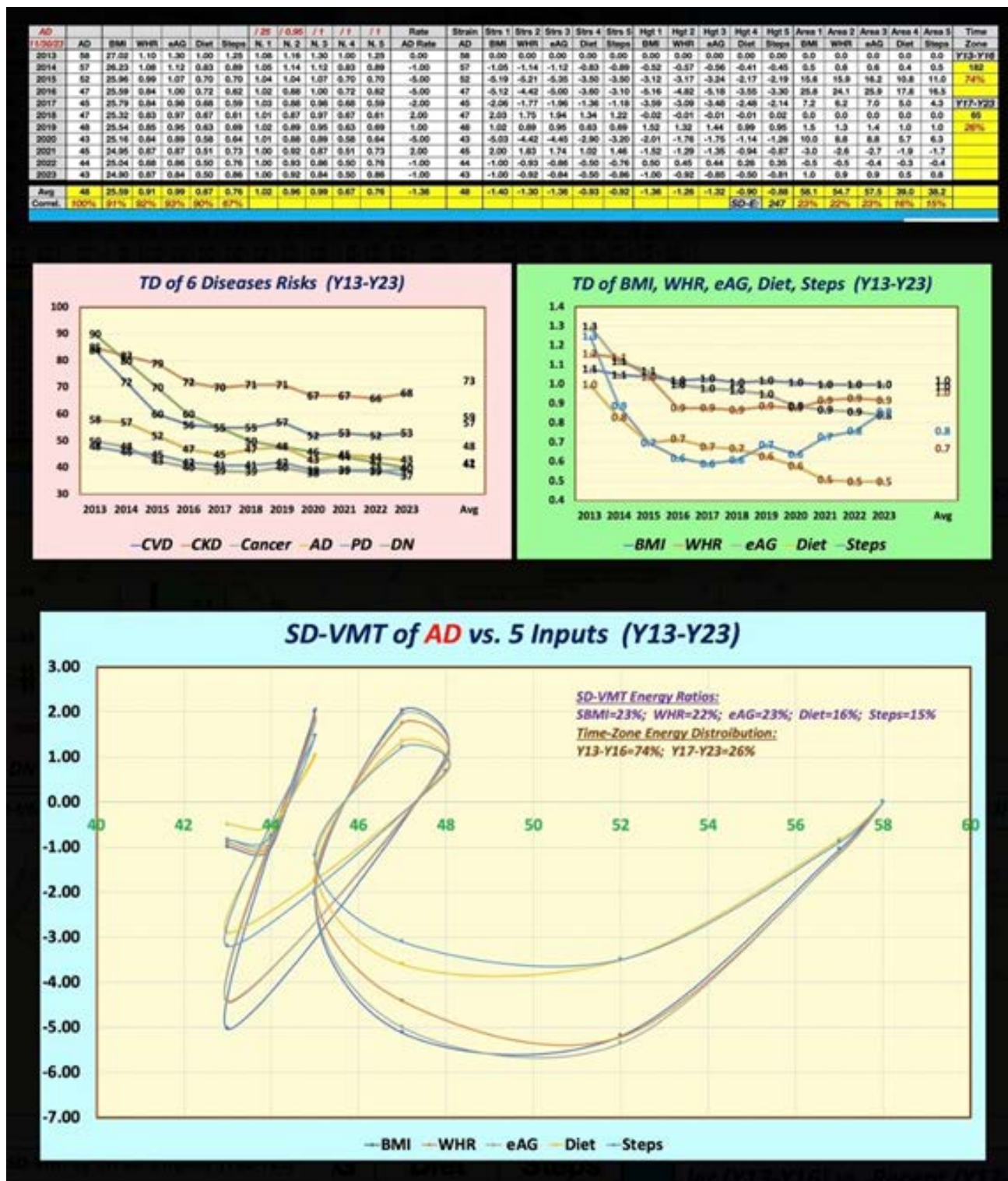


Figure 1: Data Table, Time-Domain Curves and SD-VMT Energies

3. Conclusions

3.1. In Summary

A statistical correlation analysis unveils robust correlations (90% to 93%) between the author's AD risk and four inputs, with a lower 67% correlation with walking steps.

This lower correlation is attributed to variations in his walking steps, ranging from 7.5k in 2013, increasing to 18.5k in 2018,

later reducing to 12.7k in 2023 due to agerelated discomfort from excessive walking.

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The author's three metabolic biomarkers, BMI, WHR, and eAG, contribute 22% to 23% each (totaling 68%), while lifestyle details, diet and exercise, contribute 15% to 16% each (totaling 31%). This 68% to 31% split aligns with the author's defined Da-Vinci code of medicine, reflecting a 70 to 30 ratio. Lifestyles significantly impact metabolic disorders, thereby influencing mortality diseases like AD. It is important to note that the author hasn't been diagnosed with any signs of Alzheimer's diseases. Despite lacking real data observations for direct comparison, this research, from a preventive medicine perspective, offers valuable insights into the author's health maintenance program.

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.

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