

The relationship between hypothyroidism and chronic kidney conditions, type 2 diabetes, hyperlipidemia and hypertension using the viscoplastic energy model of GH-Method: math-physical medicine (No. 950, VMT #349, 11/7-11/2023)

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Abstract

In 2010, the author faced significant health challenges reflected in following key biomarkers:

- **Body weight: 220 lbs (100 kg) and BMI: 32 (indicating obesity) - Daily averaged glucose: 280 mg/dL and HbA1C: 11%, (indicating type 2 diabetes)**
- **Thyroid stimulating hormone (TSH) above 5.0 (indicating hypothyroidism)**
- **Albumin-to-creatinine ratio (ACR): around 150 (indicating CKD risk)**
- **Normalized blood pressure above 120% (indicating hypertension) - Normalized blood cholesterols above 140% (indicating hyperlipidemia)**
- **Triglycerides at 1161 (indicating hyperlipidemia)**

Over the past 15 years, the author prioritized a health improvement program, starting with his body weight reduction which impacting his glucose levels, blood pressures, and blood lipids. This lifestyle-focused approach aimed to minimize his risks of having cardiovascular diseases (CVD), strokes, chronic kidney disease (CKD), dementia (Alzheimer's and Parkinson's diseases), and various cancers. His ultimate objective has been longevity with happiness.

*His recent health data in 2023 shows significant improvements: - **Body weight: 162 lbs (73.6 kg) with BMI: 24 years, from 2010 to 2023, regarding these important biomarkers?** Notably, meaningful data was absent for 2010-2011, with the author's useful datasets spanning from 2012 to 2023.*

- **Daily averaged glucose: 1005**
- **TSH: 15**
- **ACR: 145**
- **Normalized blood pressure: 95%.....5**
- **Normalized cholesterols: 63% - Triglycerides: 102 What transpired over the past.....14 (HbA1C: 6.1%)**

A few Japanese scientists published a research paper on July 10, 2023 which connecting thyroid health to kidney conditions, diabetes, and cholesterol, based on 200 hospital patients over a period of 2 years.

Inspired by aforementioned article, the author of this paper decides to conduct a similar analysis based on his own data collected from 2012 to 2023 (over 12 years) using a unique math-physical engineering research method.

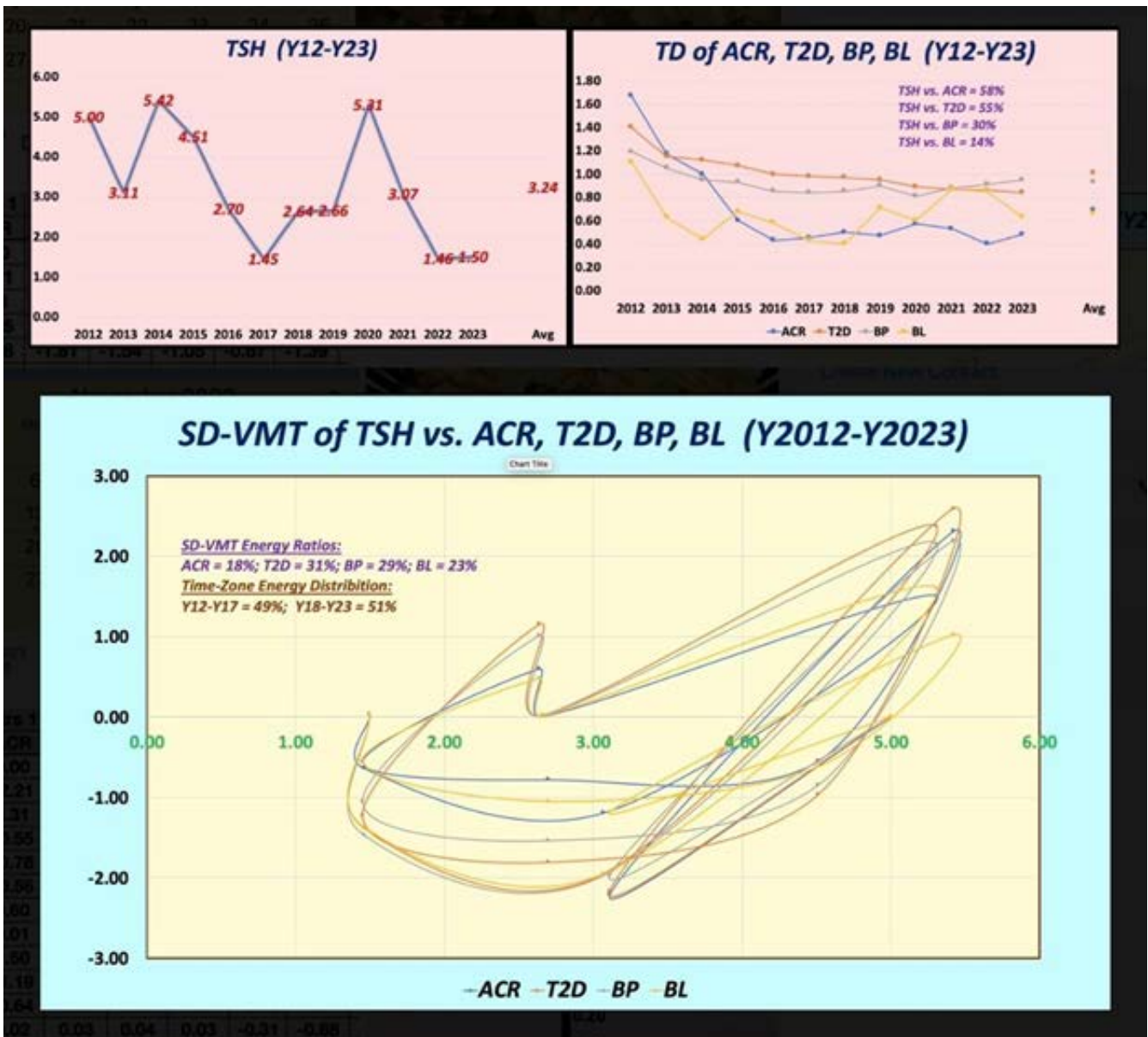
In summary, the author found two moderate-strong statistical correlations and two low correlations from his four input biomarkers compared to his TSH output dataset:

- **ACR (kidney): 58%**
- **Glucose (diabetes): 55%**
- **Blood pressures: 30%**
- **Cholesterols: 14%**

Using a Viscoplastic energy analysis research approach, the author determined the following four contribution ratios from his four input biomarkers on his TSH output:

- **ACR energy: 18%**
- **Glucose energy: 31%**
- **Blood pressures energy: 29%**
- **Cholesterols energy: 23%**

These ratios indicate that the primary inputs of diabetes and hypertension collectively influence TSH around 60%, more than the secondary inputs of cholesterols and ACR, which collectively influence around 40%. **This split of 60 versus 40 aligns with the author's earlier finding and definition of the "Da Vinci Code of Medicine."**



1. Introduction

In 2010, the author faced significant health challenges reflected in following key biomarkers:

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diseases), and various cancers. His ultimate objective has been longevity with happiness.

His recent health data in 2023 shows significant improvements:

- **Body weight: 162 lbs (73.6 kg) with BMI: 24**

- **Daily averaged glucose: 100**

(**HbA1C: 6.1%**)

- **TSH: 1.5**

- **ACR: 14.5**

- **Normalized blood pressure: 95%**

- **Normalized cholesterol: 63% - Triglycerides: 102**

What transpired over the past 14 years, from 2010 to 2023, regarding these important biomarkers? Notably, meaningful data was absent for 2010-2011, with the author's useful datasets spanning from 2012 to 2023.

A few Japanese scientists published a research paper on July 10, 2023 which connecting thyroid health to kidney conditions, diabetes, and cholesterol, based on 200 hospital patients over a period of 2 years. Inspired by aforementioned article, the author of this paper decides to conduct a similar analysis based on his own data collected from 2012 to 2023 (over 12 years) using a unique math-physical engineering research method.

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. Thyroid stimulating hormone (TSH)

Normal values of thyroid-stimulating hormone (TSH) are from **0.4 to 4.0 mIU/L** for those with no symptoms of an under- or over-active thyroid.

For example, when TSH value is 5.0 generally indicate hypothyroidism rather than hyperthyroidism. TSH is produced by the pituitary gland and helps regulate the production of thyroid hormones by the thyroid gland. In hypothyroidism, the thyroid gland is underactive and produces insufficient amounts of thyroid hormones, resulting in elevated TSH levels as the pituitary gland attempts to stimulate the thyroid gland to produce more hormones.

Conversely, in hyperthyroidism, the thyroid gland is overactive and produces excessive amounts of thyroid hormones, leading to decreased TSH levels as the pituitary gland reduces its production to try to regulate the thyroid hormones.

1.3. Pathophysiological explanations of relationships between hypothyroidism (high TSH) and ACR of kidney biomarker

The relationship between hypothyroidism (elevated TSH) and increased albumin-to-creatinine ratio (ACR), a kidney

biomarker, may involve impaired renal function due to altered hemodynamics, inflammation, and oxidative stress associated with hypothyroidism.

Additionally, hypothyroidism can lead to changes in vascular permeability, potentially contributing to elevated ACR. However, the exact pathophysiological mechanisms are complex and may vary among individuals.

1.4. Pathophysiological explanations of relationships between hypothyroidism (high TSH) and glucose (HbA1C) of type 2 diabetes

The relationship between hypothyroidism (elevated TSH) and glucose control, as measured by HbA1c in type 2 diabetes, can be attributed to various factors.

Hypothyroidism may lead to insulin resistance, impair insulin secretion, and disrupt glucose metabolism. Additionally, it can contribute to weight gain and alterations in lipid profiles, further exacerbating insulin resistance. These interconnected mechanisms can collectively contribute to poor glycemic control and higher HbA1c levels in individuals with both hypothyroidism and type 2 diabetes. It is important to note that the impact can vary among individuals.

1.5. Pathophysiological explanations of relationships between hypothyroidism (high TSH) and cholesterol

Hypothyroidism (elevated TSH) is associated with changes in lipid metabolism, leading to alterations in cholesterol levels. The thyroid hormones, particularly T3, play a crucial role in regulating lipid synthesis, breakdown, and clearance. In hypothyroidism, the reduced thyroid hormone activity can result in decreased expression of hepatic LDL receptors, leading to impaired clearance of low-density lipoprotein (LDL) cholesterol. Additionally, there may be an increase in cholesterol synthesis and triglyceride levels.

Moreover, hypothyroidism is linked to alterations in the composition of lipoproteins, including an increase in the proportion of more atherogenic small, dense LDL particles. These changes collectively contribute to elevated levels of total cholesterol and LDL cholesterol in individuals with hypothyroidism. It is important to note that the impact can vary among individuals.

1.6. Pathophysiological explanations of relationships between hypothyroidism (high TSH) and blood pressure (hypertension)

The relationship between hypothyroidism (elevated TSH levels) and hypertension is complex. Hypothyroidism can lead to increased systemic vascular resistance and decreased cardiac output, contributing to elevated blood pressure. Additionally, impaired nitric oxide synthesis and endothelial dysfunction may further contribute to hypertension in individuals with hypothyroidism. Regular monitoring and management of thyroid function are crucial for addressing associated cardiovascular risks.

1.7. An excerpt of a Japanese thyroid study report

Thyroid function, glycemic control, and diabetic nephropathy in patients with type 2 diabetes over 24 months: prospective

observational study By Hiroshi Iwakura etc. at Wakayama Medical University, Japan Published online July 10, 2023

A 653-words excerpt of aforementioned paper: “The higher prevalence of thyroid dysfunction in type 1 diabetes patients has been well established, whereas it is a matter of debate whether that is also observed in type 2 diabetes patients. This study was conducted to reveal whether higher prevalence of thyroid dysfunction is observed in patients with type 2 diabetes.

We examined thyroid functions and thyroid autoantibodies in 200 patients with type 2 diabetes and 225 controls, with 24 months follow up for those with type 2 diabetes.

In the follow-up observation, we found no significant correlation between basal thyrotropin (TSH), fT3, fT4 or fT3/fT4 ratio with the amounts of changes of HbA1c levels at 12 or 24 months after the basal measurements. There was a negative relationship between TSH levels and eGFR at baseline measurements, but TSH levels did not seem to predict future decline of eGFR levels. No relationship was observed between urine albumin/ creatinine levels and thyroid function.

Basal thyroid function did not predict future diabetes control or renal function within 24 months of followup.

According to the Japan Thyroid Association, the prevalence of thyroid disease including thyroid nodules is estimated to be between 5 and 7 million (approximately 4 to 5.5% of the general population) in Japan. Diabetes is also a highly prevalent disease, with estimated prevalence in Japan is 18.7% in men and 9.3% in women according to the National Health and Nutrition

Examination Survey 2018. **Due to such high prevalence of these two disorders, a number of patients are likely to have both diabetes and thyroid dysfunction concurrently.**

The prevalence of thyroid dysfunction has been widely reported to be high in patients with type 2 diabetes, but others argue that the incidence of thyroid dysfunction is not dissimilar to that of the general population.

There are no known reports that directly compare the prevalence of thyroid dysfunction in patients with and without type 2 diabetes in Japan.

In this study, the prevalence of thyroid dysfunction in patients with type 2 diabetes are compared with that in control subjects without diabetes.

Included in this study were 203 patients with type 2 diabetes (127 males and 76 females) and 225 nondiabetic control subjects (115 males and 110 females).

Patients with type 2 diabetes were significantly older than control subjects and had significantly higher weight, BMI, systolic blood pressure, HbA1c, fasting plasma glucose, serum insulin, CPR, and triglyceride levels and significantly lower height, diastolic blood pressure, serum total cholesterol, HDL cholesterol, and LDL-cholesterol levels. A significantly higher percentage of patients with type 2 diabetes were on medication for hypertension and dyslipidemia. Most patient with type 2 diabetes (88%) were treated with anti-diabetic drugs including insulin (23%).

	Control	T2DM
Characteristics		
Male	115	124
Female	110	76
Total	225	
Age	51.0 ± 11.2	68.3 ± 10.3**
Height(cm)	164.0 ± 8.6	161.3 ± 9.0**
Weight (kg)	62.4 ± 12.6	68.8 ± 45.9*
BMI (kg/m ²)	23.1 ± 3.5	26.3 ± 17.1**
Systolic BP (mmHg)	125.0 ± 16.3	133.9 ± 19.3**
Diastolic BP (mmHg)	75.4 ± 11.4	73 ± 11.6*
HbA1c(%)	5.4 ± 0.3	7.2 ± 0.8**
FPG (mg/dl)	91.6 ± 8.5	147.9 ± 43.4*
IRI (μU/ml)	6.8 ± 3.8	17.6 ± 32.3**
CPR (ng/ml)	1.5 ± 0.6	2.5 ± 1.6**
T-CHO (mg/ dl)	217.7 ± 41.3	190.7 ± 36.2**
TG (mg/dl)	106.7 ± 86.8	138.3 ± 88.2**
HDL (mg/dl)	68.9 ± 15.6	54 ± 14.1**
LDL (mg/dl)	124.5 ± 32.9	107.2 ± 26.8*

Medications				
	number	(%)	number	(%)
For hypertension				
any	28	12,4	104	52.0**
ARB	16	7,1	79	39.5**
ACEI	0	0	5	1.5*
CaB	24	10,7	65	32.5**
α-blocker	1	0,4	5	2,5
β-blocker	4	1,8	4	2,0
diuretics	1	0,4	5	2,5
For dyslipidemia				
any	14	6,2	79	39.5*
statins	11	4,9	73	36.5*
ezetimib	1	0,4	5	2,5
fibrate	1	0,4	5	2,5
vitamin E	2	0,9	0	0
For diabetes				
any	-	-	176	88
DPPIVI	-	-	116	56,9
biguanide	-	-	91	45,5
SU	-	-	69	34,5
αGI	-	-	30	15
SGLT2	-	-	16	8
TZD	-	-	5	2,5
glinide	-	-	5	2,5
insulin	-	-	46	23
GLP1	-	-	7	3,5

Table 1: Baseline Characteristics and Medications

Serum TSH levels of patients with type 2 diabetes were not significantly different from those of non-diabetic controls.

Normal reference range TSH: 0.61–4.23 μIU/ml

At baseline measurements, TSH levels were negatively correlated with eGFR levels ($r=-0.29$, $P<0.001$).

2. Discussion

In this study of thyroid function in patients with type 2 diabetes and controls, there were no differences in the rate of patients with thyroid dysfunction. **The prevalence of thyroid disease has been widely reported to be high in patients with type 2 diabetes.** Although some studies did not strictly exclude patients with type 1 diabetes, others lacked control subjects. **After excluding these reports, just one report clearly demonstrates that the higher prevalence of thyroid dysfunction among patients with type 2 diabetes than in controls, 7.3% vs. 2.9%. Our findings are more consistent with those of several other studies that showed no significant differences in the prevalence of thyroid dysfunction between patients with type 2 diabetes and controls.**

3. Conclusion

There were no differences in prevalence of thyroid dysfunction

and thyroid autoantibodies between patients with type 2 diabetes and controls. Basal thyroid function did not predict future diabetes control or renal function within a 24-month period.”

4. Methods

4.1. 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.

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.

4.2. 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.*

*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)*.

5. Results

Figure 1 shows data table, TD analysis results and SD-VMT analysis results.

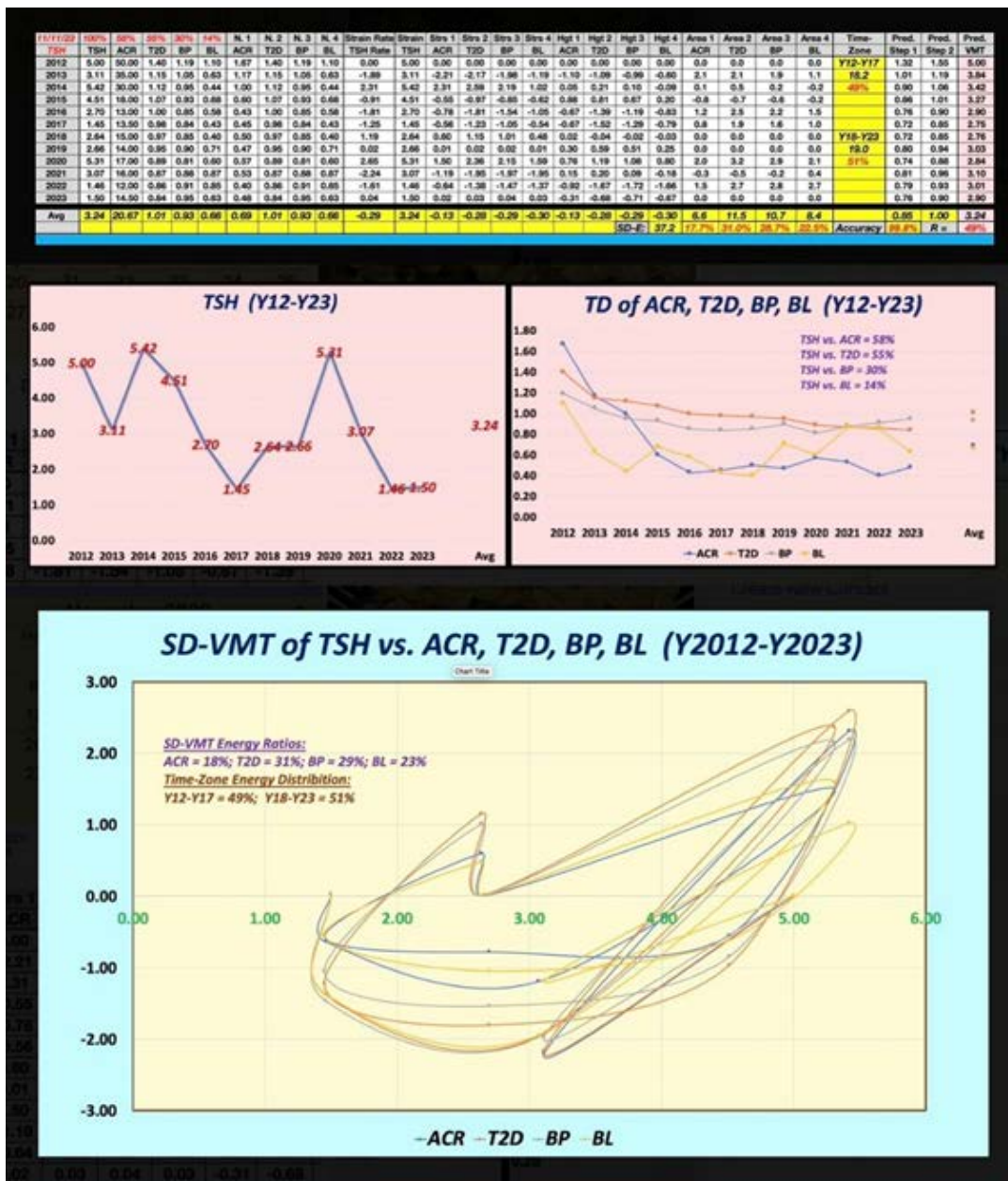


Figure 1: Data table, TD analysis results and SD-VMT analysis results

5.1. Conclusions

In summary, the author found two moderate-strong statistical correlations and two low correlations from his four input biomarkers compared to his TSH output dataset:

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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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