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Viscoelastic Medicine Theory (VMT #284): The Pathophysiological Illustration of Dementia Risk versus Four Influential Factors of Body Weight, Glucose, Blood Pressure, and Blood Lipids Complimented with a Space-Domain Viscoplastic Energy Analysis Based on GH-Method: Math-Physical Medicine (No.884)

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Abstract

Dementia is a complex and multifactorial disease that primarily affects older individuals, typically those above the age of 65. Among the six recognized dementia types, Alzheimer's disease is the most prevalent, accounting for approximately 60-70% of all cases. The causes of dementia are equally intricate. It is widely accepted that obesity (m1) and three chronic metabolic disorders - diabetes (m2), hypertension (m3), and hyperlipidemia (m4) - either individually or collectively, influence the risk of developing dementia conditions. In this study, the author assesses the risk percentages of developing dementia with an emphasis on blood lipids, using the following equation: Dementia Risk = Metabolism Index (MI) * 0.7 + Normalized Lipids (M4) * 0.1 + (Triglycerides / 150) * 0.1 + (Age / 65) * 0.1. The Metabolism Index (MI) is determined through a mathematical model that incorporates 10 categories (4 chronic diseases and 6 lifestyle factors) with 500 elements. This model employs topology, nonlinear algebra, geometric algebra, engineering finite element method, and big data analytics to evaluate an individual's overall health condition and the potential health risks posed by internal organ failure (metabolism related) and external infectious diseases (immunity). In summary, the findings of this study on predicted dementia risk reveal three main points: A. Time-domain analysis indicates a decline in dementia risk, starting at over 100% in 2012 (when his TG level was at 1161 with worsening conditions of both T2D and hypertension) and gradually decreasing to 87% in 2013 and further down to 62% by 2023. B. SD-VMT energy analysis highlights the degree of influence of various factors: His T2D (glucose m2) contributes 30%, which is greater than his obesity (body weight m1) contribution of 27%, and slightly higher than his hypertension (blood pressure m3) contribution of 26%. His hyperlipidemia (blood lipids m4) is the smallest contributor at 16%. C. Time-zone analysis in SD demonstrates that the period from Y12 to Y14 accounts for 96% of his dementia risk, indicating that if he had continued experiencing poor health conditions during that time, it would have been almost certain for him to develop dementia. The period from Y15 to Y19 contributes 4% of the risk, while the COVID period from Y20 to Y23 carries no dementia risk. Overall, these three analyses from TD, SD-VGT, and SD-TZ collectively suggest that he had the highest dementia risk around a decade ago, but currently, he does not have any dementia risk (in this COVID period).

Keywords: Viscoelastic; Viscoplastic; Dementia; Body weight; Glucose; Blood pressure; Blood lipids; Hyperglycemia

Abbreviations: MI: metabolism index; T2D: type 2 diabetes; PPG: postprandial plasma glucose; FPG: fasting plasma glucose; SD: space-domain

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1. INTRODUCTION

Dementia is a complex and multifactorial disease that primarily affects older individuals, typically those above the age of 65. Among the six recognized dementia types, Alzheimer's disease is the most prevalent, accounting for approximately 60-70% of all cases.

The causes of dementia are equally intricate. It is widely accepted that obesity (m1) and three chronic metabolic disorders - diabetes (m2), hypertension (m3), and hyperlipidemia (m4) - either individually or collectively, influence the risk of developing dementia conditions.

In this study, the author assesses the risk percentages of developing dementia with an emphasis on blood lipids, using the following equation:

Dementia Risk
= Metabolism Index (MI) * 0.7 + Normalized Lipids (M4) * 0.1 + (Triglycerides / 150) * 0.1 + (Age / 65) * 0.1

The Metabolism Index (MI) is determined through a mathematical model that incorporates 10 categories (4 chronic diseases and 6 lifestyle factors) with 500 elements. This model employs topology, nonlinear algebra, geometric algebra, engineering finite element method, and big data analytics to evaluate an individual's overall health condition and the potential health risks posed by internal organ failure (metabolism related) and external infectious diseases (immunity).

1.1 Pathophysiological illustration of dementia

Dementia is a broad term used to describe a group of neurological disorders characterized by cognitive decline and impairment in memory, thinking, behavior, and the ability to perform daily activities. The pathophysiology of dementia varies depending on the specific underlying cause. Here are pathophysiological explanations for some different types of dementia:

1. Alzheimer's disease (AD): AD is the most common cause of dementia. This is the most common type of dementia, accounting for the majority of cases. It is characterized by the buildup of plaques and tangles in the brain, leading to the progressive degeneration of brain cells. In AD, two hallmark brain abnormalities are observed: the accumulation of beta-amyloid plaques and the formation of neurofibrillary tangles. These plaques and tangles disrupt normal communication between neurons, leading to neuronal damage, inflammation, and progressive loss of brain tissue and function. Alzheimer's disease is the most common cause of dementia. Estimates vary, but it is generally believed that Alzheimer's disease accounts for approximately 60-70% of all cases of dementia. This means that the majority of individuals diagnosed with dementia have Alzheimer's disease as the underlying cause. However, it is important to note that there are other causes of dementia. The exact percentage of Alzheimer's disease within the spectrum of dementia may vary based on different population studies and diagnostic criteria used.

2. Vascular dementia: This type of dementia is caused by reduced blood flow to the brain, typically due to blockages or damage in blood vessels, often due to small strokes or other vascular conditions that damage blood vessels in the brain. Reduced blood flow can result from conditions like atherosclerosis (buildup of plaques in arteries) or strokes. The impaired blood supply leads to brain cell death and the development of cognitive deficits.

3. Lewy body dementia (LBD): This type of dementia often has symptoms similar to both Alzheimer's disease and Parkinson's disease. LBD is characterized by the presence of Lewy bodies deposits or abnormal protein clumps, in brain cells. These Lewy bodies primarily affect areas of the brain that regulate thinking, memory, and movement. They disrupt the function of neurotransmitters like dopamine, acetylcholine, and serotonin, leading to cognitive decline, motor symptoms, and widespread disruption of brain circuits.

4. Frontotemporal dementia (FTD): This type of dementia is caused by the damage and degeneration of nerve cells of the frontal and temporal lobes of the brain which can lead to changes in behavior, personality, and language abilities. . This degeneration is associated with abnormal accumulation of

specific proteins, including tau or TDP-43. The affected brain regions control behavior, personality, language, and decision-making. The damage results in changes in behavior, personality, and language skills.

5. Parkinson's disease dementia (PDD): People with Parkinson's disease may eventually develop dementia. It is characterized by cognitive decline, along with movement and motor symptoms associated with Parkinson's disease. In other words, people with long-standing Parkinson's disease may develop dementia as the condition progresses. The underlying process involves the buildup of Lewy bodies in the brain, similar to LBD. The degeneration and dysfunction of dopaminergic neurons in the substantia nigra, a brain region involved in movement control, also play a role in Parkinson's dementia.

6. Mixed or other causes: This refers to a combination of different types of dementia, most commonly Alzheimer's disease and vascular dementia. There are still other less common causes of dementia, including Huntington's disease, Creutzfeldt-Jakob disease, and traumatic brain injury. Each has its own unique pathophysiology involving genetic mutations, abnormal protein accumulation, or brain damage.

Above-listed are just a few examples, and there are other less common types of dementia as well. It is important to note that each type of dementia may have unique characteristics, progression, and underlying causes. It is important to note that these explanations provide a simplified overview of the pathophysiological mechanisms underlying dementia. The complexity and interplay of various factors involved in each type of dementia are still areas of active research.

1.2 Known causes for developing into dementia

There are several known causes and risk factors associated with the development of dementia. Some common causes include:

1. Age: Advanced age is the most significant risk factor for developing dementia. The risk of dementia increases significantly after the age of 65.

2. Alzheimer's disease: Alzheimer’s disease is the most common cause of dementia. It involves the accumulation of abnormal protein deposits, such as beta-amyloid plaques and tau tangles, in the brain.

3. Vascular factors: Conditions that affect the blood vessels, including high blood pressure, high cholesterol, diabetes, and heart disease, can increase the risk of developing vascular dementia.

4. Lewy body disease: Lewy body dementia is caused by the presence of Lewy bodies, abnormal protein deposits, in the brain. It shares similarities with both Alzheimer's disease and Parkinson's disease.

5. Genetics: Some forms of dementia have a genetic component. For example, early-onset familial Alzheimer's disease is caused by specific gene mutations that are passed down within families.

6. Head trauma: Significant head injuries, especially those resulting in loss of consciousness, have been associated with an increased risk of developing dementia later in life.

7. Down syndrome: Individuals with Down syndrome have a higher risk of developing Alzheimer's disease.

8. Lifestyle factors: Certain lifestyle factors can increase the risk of dementia, including smoking, excessive alcohol consumption, poor diet, lack of physical activity, and social isolation.

It is important to note that some cases of dementia may have a combination of causes, and in many cases, the exact cause or causes of dementia may not be fully understood. Research is still ongoing to better understand the various factors contributing to the development of dementia.

1.3 Relationship between dementia and obesity

There is growing evidence to suggest that obesity is linked to a higher risk of developing dementia. Multiple studies have found that obesity in midlife is associated with an increased risk of dementia later in life. Here are some key points regarding the relationship between obesity and dementia:

1. Increased risk: Obesity, especially in midlife, has been associated with a higher risk of developing dementia, including Alzheimer's disease and vascular dementia.

2. Vascular damage: Obesity is known to contribute to the development of various cardiovascular risk factors, such as high blood pressure, high cholesterol, and type 2 diabetes. These conditions can lead to vascular damage, reduced blood flow to the brain, and an increased risk of vascular dementia.

3. Chronic inflammation: Obesity is characterized by a state of chronic low-grade inflammation in the body. This inflammation can have detrimental effects on the brain and may contribute to the development of dementia.

4. Insulin resistance: Obesity is often accompanied by insulin resistance, a condition in which cells become less responsive to the effects of insulin. Insulin resistance has been associated with cognitive decline and an increased risk of dementia.

5. Brain structure and function: Obesity has been linked to structural and functional changes in the brain, such as reduced volume in certain regions, altered white matter integrity, and impaired cognitive abilities. These changes may increase the susceptibility to dementia.

6. Other risk factors: Obesity is often associated with other lifestyle factors that can contribute to an increased risk of dementia, such as poor diet, lack of physical activity, and cardiovascular diseases.

However, it is important to note that the relationship between obesity and dementia is complex and multifactorial. Not all individuals with obesity will develop dementia, and there are various other factors that contribute to the development of the disease. Maintaining a healthy weight, adopting a balanced diet, engaging in regular physical activity, and managing associated risk factors may help reduce the risk of dementia.

1.4 Cholesterol fluctuations and triglyceride levels influence dementia risk

Studies have shown that high cholesterol and triglyceride levels, as well as fluctuations in these levels over time, are associated with an increased risk of cognitive decline and the development of dementia, particularly vascular dementia.

Fluctuating levels of cholesterol and triglycerides can lead to blood vessel damage, including the narrowing and hardening of arteries, which can impair blood flow to the brain. This reduced blood flow and potential vascular damage can contribute to the development of dementia.

However, the relationship between fluctuating cholesterol and/or high triglyceride levels and dementia is complex, and further research is needed to better understand the mechanisms involved. Additionally, managing cholesterol and triglyceride levels through a healthy lifestyle, diet, and medication, when necessary, may help reduce the risk of dementia.

The relationship between dementia and triglycerides particularly, which are a type of fat found in the blood, is still being studied and understood. Here are some known points regarding their relationship:

1. Vascular dementia: Elevated triglyceride levels have been associated with an increased risk of vascular dementia. Vascular dementia is caused by reduced blood flow to the brain due to damaged or blocked blood vessels. High triglyceride levels can contribute to the development of atherosclerosis, a condition characterized by the buildup of fatty plaques in the arteries, which can impair blood flow to the brain.

2. Alzheimer's disease: Certain studies have already examined the link between triglyceride levels and Alzheimer's disease, the most common form of dementia. While the evidence is less clear compared to vascular dementia, some studies have found associations between high triglyceride levels and an increased risk of Alzheimer's disease or cognitive decline.

3. Insulin resistance: Triglyceride levels can be affected by insulin resistance, a condition

in which cells become less responsive to the effects of insulin. Insulin resistance is commonly associated with obesity and metabolic syndrome. Both insulin resistance and metabolic syndrome have been linked to an increased risk of dementia.

4. Inflammation and oxidative stress: High triglyceride levels can contribute to inflammation and oxidative stress, which are thought to play a role in the pathogenesis of dementia. Chronic inflammation and oxidative stress can damage brain cells and lead to cognitive decline.

It's worth noting that the relationship between triglycerides and dementia is complex and may be influenced by various factors, including genetics, other cardiovascular risk factors, and lifestyle choices. Further research is needed to fully understand the impact of triglyceride levels on dementia risk. Managing triglyceride levels through a healthy diet, regular exercise, weight control, and medication when necessary, may help reduce the risk of vascular-related dementia.

1.5 Relationship and contribution percentages of obesity, diabetes, hypertension and hyperlipidemia on risk of developing into dementia

The relationship between dementia and obesity, diabetes, hypertension, and hyperlipidemia is complex and multifaceted. While these conditions are known to be risk factors for dementia, the specific contribution percentages and to dementia risks can vary.

1. Obesity: Obesity is associated with an increased risk of developing dementia, particularly Alzheimer's disease and vascular dementia. However, it is challenging to determine the exact weight percentage of dementia cases attributed to obesity alone, as obesity often coexists with other risk factors such as diabetes, hypertension, and hyperlipidemia.

2. Diabetes: Diabetes is a recognized risk factor for dementia. Studies suggest that individuals with diabetes have an increased risk of developing all-cause dementia, including Alzheimer's disease. The weight percentage of dementia cases attributable to diabetes is estimated to be around 10-15%.

3. Hypertension: High blood pressure, or hypertension, is a well-known risk factor for vascular dementia. It can lead to damage to blood vessels in the brain, impairing blood flow and increasing the risk of cognitive decline. The weight percentage of dementia cases linked to hypertension is estimated to be around 15-20%.

4. Hyperlipidemia: High cholesterol, or hyperlipidemia, particularly high levels of LDL (low-density lipoprotein) cholesterol, has been associated with an increased risk of Alzheimer's disease and vascular dementia. However, it is challenging to determine a specific weight percentage for hyperlipidemia as a risk factor for dementia, as it often coexists with other conditions like obesity, diabetes, and hypertension.

It's important to note that these percentages are approximate and can vary based on numerous factors such as the population studied, research methodology, and diagnostic criteria used. Additionally, the presence of multiple risk factors in an individual can amplify the overall risk of developing dementia. Managing these risk factors through lifestyle modifications, medication, and other interventions can help reduce the risk of dementia.

2. METHODS

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

2.2 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 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 developing 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 has no longer taken 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 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 of over 40,000 hours and reading 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.

2.3 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 circulate 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 example, the combination of hyperglycemia and hypertension would cause micro-blood vessel leakage in the kidney systems which is one of the major causes 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. deforms; 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, 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.

2.4 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 the 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, 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 a 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)

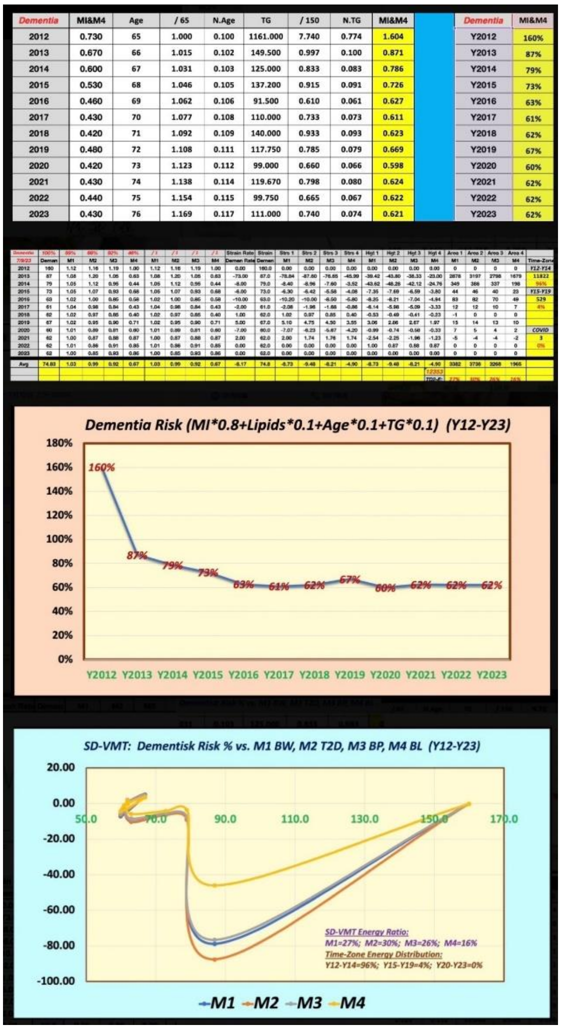
The risk probability % of developing into CVD, CKD, and Cancer is calculated based on his developed metabolism index model (MI) in 2014. His MI value is calculated using inputs of 4 chronic conditions, i.e. weight, glucose, blood pressure, and lipids; and 6 lifestyle details, i.e. diet, drinking water, 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).

Note: For a more detailed description, please refer to the "consolidated method" section which is given at the beginning of the special issue.

3. RESULTS

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



Dementia	MI&M4	Age	/ 65	N.Age	TG	/ 150	N.TG	MI&M4		Dementia	MI&M4
2012	0.730	65	1.000	0.100	1161.000	7.740	0.774	1.604		Y2012	160%
2013	0.670	66	1.015	0.102	149.500	0.997	0.100	0.871		Y2013	87%
2014	0.600	67	1.031	0.103	125.000	0.833	0.083	0.786		Y2014	79%
2015	0.530	68	1.046	0.105	137.200	0.915	0.091	0.726		Y2015	73%
2016	0.460	69	1.062	0.106	91.500	0.610	0.061	0.627		Y2016	63%
2017	0.430	70	1.077	0.108	110.000	0.733	0.073	0.611		Y2017	61%
2018	0.420	71	1.092	0.109	140.000	0.933	0.093	0.623		Y2018	62%
2019	0.480	72	1.108	0.111	117.750	0.785	0.079	0.669		Y2019	67%
2020	0.420	73	1.123	0.112	99.000	0.660	0.066	0.598		Y2020	60%
2021	0.430	74	1.138	0.114	119.670	0.798	0.080	0.624		Y2021	62%
2022	0.440	75	1.154	0.115	99.750	0.665	0.067	0.622		Y2022	62%
2023	0.430	76	1.169	0.117	111.000	0.740	0.074	0.621		Y2023	62%

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

4. CONCLUSION

In summary, the findings of this study on predicted dementia risk reveal three main points:

A. Time-domain analysis indicates a decline in dementia risk, starting at over 100% in 2012 (when his TG level was at 1161 with worsening conditions of both T2D and hypertension) and gradually decreasing to 87% in 2013 and further down to 62% by 2023.

B. SD-VMT energy analysis highlights the degree of influence of various factors: His T2D (glucose m2) contributes 30%, which is greater than his obesity (body weight m1) contribution of 27%, and slightly higher than his hypertension (blood pressure m3) contribution of 26%. His hyperlipidemia (blood lipids m4) is the smallest contributor at 16%.

C. Time-zone analysis in SD demonstrates that the period from Y12 to Y14 accounts for 96% of his dementia risk, indicating that if he had continued experiencing poor health conditions during that time, it would have been almost certain for him to develop dementia. The period from Y15 to Y19 contributes 4% of the risk, while the COVID period from Y20 to Y23 carries no dementia risk.

Overall, these three analyses from TD, SD-VGT, and SD-TZ collectively suggest that he had the highest dementia risk around a decade ago, but currently, he does not have any dementia risk (in this COVID period).

5. REFERENCES

For editing purposes, the majority of the references in this paper, which are self-references, have been removed. 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, 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 three published special editions from the following three specific journals:

(1) Special Issue. The GH-Method. (https://www.theghmethod.com).

(2) Journal of Applied Material Science & Engineering Research (contact: Catherine).

(3) Advances in Bioengineering and Biomedical Science Research (contact: Sony Hazi).

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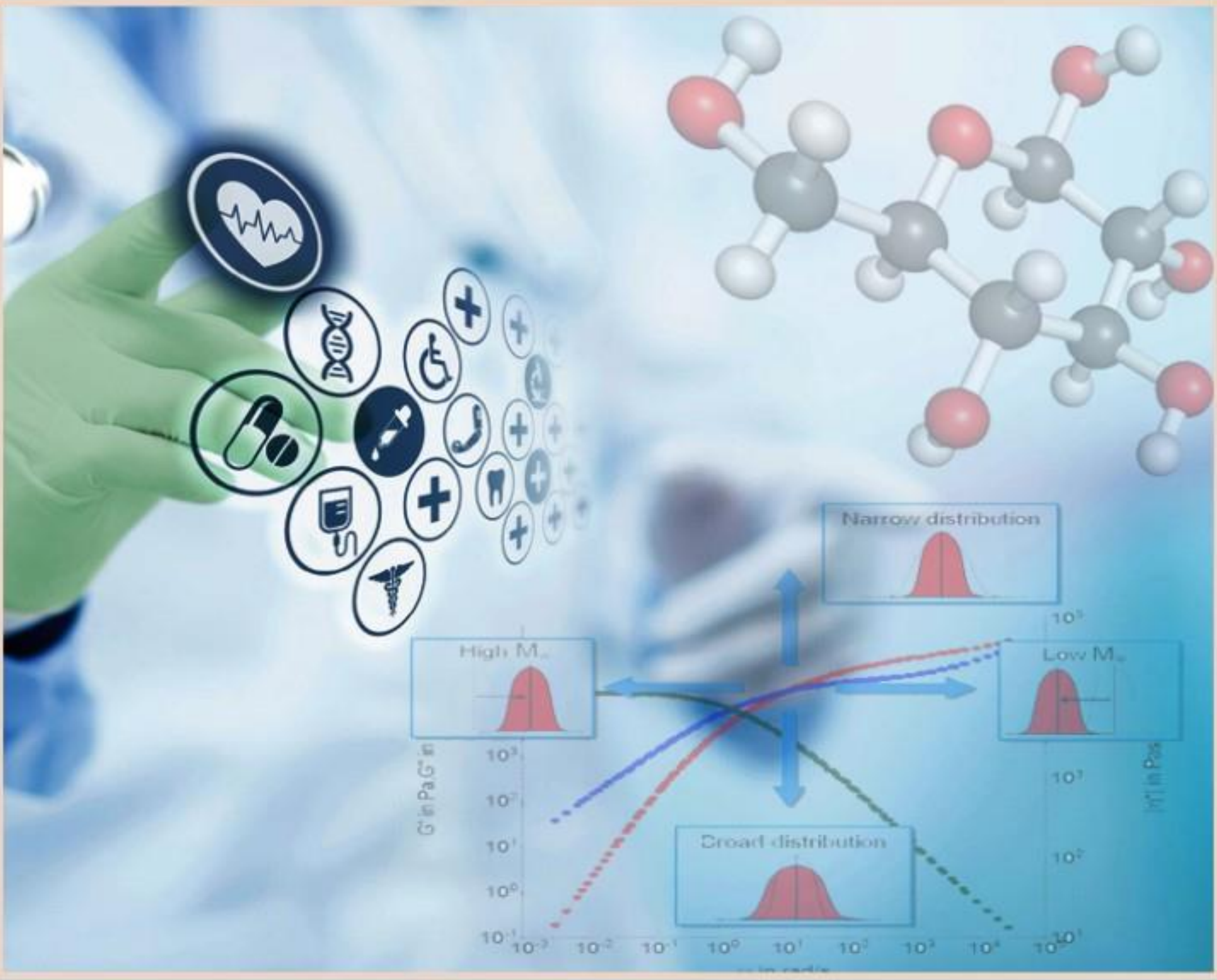

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