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Viscoelastic Medicine theory (VMT #373): Relationships of Alzheimer's disease risks and five inputs, WHR, BMI, eAG, Diet, and exercise, using viscoplastic energy model of GH-Method: math-physical medicine (No. 974)

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Abstract

More than 6 million individuals in the U.S. have Alzheimer's disease (AD), this number is projected to reach 13 million by 2050, constituting about 3% of the anticipated U.S. population of 388 million in 2050, according to the Alzheimer's Association.

Motivated by research results presented at the annual meeting of the Radiological Society of North America on 11/27/2023, the author decided to explore his Alzheimer's disease risk in connection to his values of BMI and WHR. This exploration involves integrating three additional factors: averaged glucose, diet score (considering both food portion and meal quality), and exercise (daily walking steps).

Over the past 12 years (2013-2023), the author's food portion decreased from 125% to 49%, with 100% denoting his "normal" portion before 2010. His food quality score declined from 90% to 50% (a lower percentage indicates improvement). Walking steps increased from 7.5k (116%) in 2013 to 18.5k (60%) in 2018, later decreasing to 12.7k (86%) in 2023 due to age-related discomfort resulting from excessive walking.

In summary, this study explores the author's annual Alzheimer's disease risks associated with these five selected inputs. Conventional statistical analysis reveals strong correlations (90% to 93%) with four inputs, and with a moderately high correlation of 67% with walking steps: - AD vs. WHR: 92%; - AD vs. BMI: 91%; - AD vs. eAG: 93%; - AD vs. Diet: 90%; - AD vs. Steps: 67%.

Correlation provides insights into the similarity between two waveforms. Despite these curve similarities, their impacts on the outcome of AD risks remain unclear.

The space-domain viscoplastic energy (SD-VMT) method reveals five different energy associations: Energy from WHR: 22%; Energy from BMI: 24%; Energy from eAG: 23%; Energy from Diet: 16%; Energy from Steps: 15%.

Energy contributions reveal nearly equal influences from three primary factors, namely body weight (BMI), waist-to-hip ratio (visceral fat), and diabetes glucose conditions at 22% to 23%, while diet and exercise serve as secondary influential factors at 15% to 16%.

The distribution of time-zone energy indicates a more substantial impact in the earlier period (2013-2016), with a 74% influence on his overall AD risk compared to the recent period (2017-2024), which has only a 26% impact. This arises from lifestyle details serving as influential factors of metabolic disorders.

Key Message:

Three metabolic disorder-related factors, such as body weight, visceral fat ratio, and glucose of type 2 diabetes, exert a greater influence at 22%-23% each (totaling 69%) on the author's AD risks. Two lifestyle factors of diet and exercise contribute 15% to 16% each (totaling 31%). These findings align with the author's established Da-Vinci Ratio of Medicine (70 to 30 split). Despite no diagnosed signs of dementia, this AD research provides valuable insights into the author's health maintenance. program from a preventive medicine perspective.

Keywords: Viscoelastic; Viscoplastic; Diabetes; Glucose; Insulin; Hyperglycemia; Dementia; Body Mass Index

Abbreviations: CGM: continuous glucose monitoring; eAG: estimated average glucose; T2D: type 2 diabetes; PPG: postprandial plasma glucose; FPG: fasting plasma glucose; SD: space-domain; VMT: viscoelastic medicine theory; FFT: Fast Fourier Transform

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1. INTRODUCTION

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Over the past 12 years (2013-2023), the author's food portion decreased from 125% to 49%, with 100% denoting his "normal" portion before 2010. His food quality score declined from 90% to 50% (a lower percentage indicates improvement). Walking steps increased from 7.5k (116%) in 2013 to 18.5k (60%) in 2018, later decreasing to 12.7k (86%) in 2023 due to age-related discomfort resulting from excessive walking.

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.

Pathophysiological explanations of relationship between Alzheimer's diseases versus, BMI and WHR:

The relationship between Alzheimer's disease (AD), Body Mass Index (BMI), and Waist-to-Hip Ratio (WHR) involves complex interactions. Elevated BMI is associated with increased AD risk, possibly due to insulin resistance and inflammation linked to obesity. WHR, reflecting central adiposity, may contribute to AD risk by influencing insulin sensitivity and promoting inflammation. Neuroinflammation, insulin dysregulation, and vascular changes are potential pathophysiological links. However, the exact mechanisms are still being researched, and individual variations exist.

Pathophysiological explanations of relationship between Alzheimer's diseases versus averaged glucose values of diabetes conditions:

The relationship between Alzheimer's disease (AD) and averaged glucose levels in diabetes is multifaceted. Diabetes, especially uncontrolled, can lead to hyperglycemia and insulin resistance. These conditions may contribute to AD pathogenesis through several mechanisms. Elevated glucose levels can promote oxidative stress, inflammation, and the formation of advanced glycation end products, all of which are implicated in neurodegenerative processes. Additionally, insulin resistance may affect brain function, influencing amyloid beta processing and tau phosphorylation, hallmark features of AD. The intricate interplay between diabetes and AD involves intricate molecular pathways that continue to be explored in research.

Pathophysiological explanations of relationship between Alzheimer's diseases versus diet and exercise:

The relationship between Alzheimer's disease (AD) and diet/exercise involves various pathophysiological factors. Regular physical activity is associated with improved brain health, promoting neuroplasticity and reducing inflammation, which may mitigate AD risk. A healthy diet, particularly rich in antioxidants, omega-3 fatty acids, and low in saturated fats, can support cognitive function and reduce oxidative stress.

Exercise influences factors like insulin sensitivity and neurotrophic factors, which are crucial for brain health. On the dietary side, nutrients like antioxidants counteract oxidative damage, while omega-3 fatty acids contribute to neuronal structure.

Conversely, a sedentary lifestyle and poor diet may contribute to conditions like obesity and diabetes, which are linked to an increased risk of AD. Chronic inflammation and insulin resistance are potential bridges between unhealthy lifestyle choices and AD pathogenesis. Adopting a lifestyle with regular exercise and a balanced diet can be

protective against AD by positively impacting these interconnected pathways.

Regarding Alzheimer's diseases, in comparison of BMI/WHR versus diet/exercise, which category is more important?

The relationship between Alzheimer's disease (AD), BMI/WHR, and diet/exercise is complex, and it's challenging to definitively say which category is more important. Both BMI/WHR and lifestyle factors like diet and exercise play significant roles, often interacting synergistically.

Maintaining a healthy weight (reflected in BMI and WHR) is associated with reduced AD risk, likely due to factors such as inflammation and insulin sensitivity. However, lifestyle choices, including a balanced diet and regular exercise, also contribute to overall brain health and can impact AD risk through mechanisms like neuroplasticity, oxidative stress reduction, and inflammation control.

In essence, a holistic approach that combines maintaining a healthy weight with a nutritious diet and regular exercise is generally recommended for reducing the risk of Alzheimer's disease and promoting overall well-being. Individual variations and genetic factors also contribute to the complexity of this relationship.

What is neuroplasticity?

Neuroplasticity refers to the brain's ability to reorganize itself by forming new neural connections throughout life. It involves the adaptation of neural networks through learning and experience. This process allows the brain to compensate for injury, adjust to new situations, and optimize functionality. Neuroplasticity is a fundamental property of the brain that underlies learning, memory, and recovery from various neurological conditions.

What is oxidative stress and damage?

Oxidative stress is a condition characterized by an imbalance between the production of reactive oxygen species (ROS) and the ability of the body to neutralize or repair the resulting damage. ROS, including free radicals, are highly reactive molecules that can interact with and damage cellular structures such as proteins, lipids, and DNA. When the production of ROS overwhelms the body's antioxidant defenses, oxidative stress occurs.

Oxidative damage refers to the harm caused to cells and biomolecules by these reactive oxygen species. This damage can contribute to various health issues, including neurodegenerative diseases like Alzheimer's. The brain, being particularly susceptible to oxidative stress, can experience disruptions in cellular function and contribute to the progression of conditions associated with aging and neurodegeneration.

Inflammation and Alzheimer's disease:

Inflammation is believed to play a significant role in the development and progression of Alzheimer's disease (AD). Chronic inflammation in the brain, often referred to as neuroinflammation, is characterized by the activation of immune cells and the release of pro-inflammatory molecules.

In the context of AD, the brain's immune cells, such as microglia, become activated, leading to the release of cytokines and other inflammatory mediators. While inflammation is a normal part of the body's immune response, chronic or excessive inflammation in the brain can contribute to neuronal damage and the formation of amyloid plaques and neurofibrillary tangles, which are hallmark features of AD.

The exact relationship between inflammation and AD is complex and is an active area of research. Some theories suggest that inflammation may be both a cause and a consequence of AD pathology, creating a vicious cycle that contributes to disease progression. Controlling inflammation is a potential target for therapeutic interventions in AD.

Which foods contain antioxidants and anti-inflammatory compounds?

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

Fruits:
- Berries (blueberries, strawberries, raspberries)
- Citrus fruits (oranges, grapefruits)
- Cherries

- Apples
- Grapes

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

Nuts and Seeds:
- Almonds
- Walnuts
- Chia seeds
- Flaxseeds

Herbs and Spices:
- Turmeric
- Ginger
- Garlic
- Cinnamon
- Rosemary

Fatty Fish
- Salmon
- Mackerel
- Sardines
- Trout

Green Tea:
- Contains polyphenols with antioxidant and anti-inflammatory properties.

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

Olive Oil:
- Extra virgin olive oil contains polyphenols and has anti-inflammatory effects.

Whole Grains:
- Quinoa
- Brown rice
- Oats

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.

Recent article of Alzheimer's disease and visceral fat:

A type of belly fat may be linked to increased risk of developing Alzheimer's MRI scans show that visceral fat, which can accumulate around the organs of even people at healthy BMIs, is associated with changes in the brain.

By Linda Carroll
11/28/2023

People who have large amounts of fat stored around their organs as they age may be at higher risk of developing Alzheimer's disease, a new study suggests.

This type of fat isn't necessarily reflected in a high body mass index.

Visceral fat, which can accumulate around the organs of even people at healthy BMIs, is associated with changes in the brain potentially decades before any symptoms of cognitive decline are seen, according to the study presented at the annual meeting of the Radiological Society of North America on Monday (11/27/2023).

Visceral fat has previously been associated with systemic inflammation — which occurs when the immune system is constantly turned up even when there is no threat to health — and higher levels of insulin, both of which are thought to be involved in the development of Alzheimer's, according to the study's senior author, Dr. Cyrus Raji, a neuroradiologist at the Washington University School of Medicine in St. Louis.

"We need to move beyond traditional conceptions of body fat, like BMI, and really look at the specifics of how fat is distributed to understand the health risks," Raji said.

While it takes an MRI scan of the abdomen to confirm a person has visceral fat, there can be signs, Raji said.

According to Raji, signs that you may have accumulated fat around your organs include:

- A waist that is larger than the hips.
- Blood sugar that is high enough for a diagnosis of diabetes or prediabetes.

More than 6 million people in the U.S. live with Alzheimer's disease, according to the Alzheimer's Association. The group estimates that by 2050 that number will rise to 13 million (about 3% of US population of 388 million in 2050).

To take a closer look at the potential impact of visceral fat on the risk of Alzheimer's, Raji and his colleagues analyzed data from 54 cognitively healthy volunteers ages 40 to 60 who had average BMIs of 32. A BMI of 30 or higher is considered obese, according to the Centers for Disease Control and Prevention. The researchers measured a host of health parameters, including insulin and blood sugar levels. Using MRI scans, they assessed the amount of fat just under the skin, as well as what was surrounding the organs. MRIs were also used to measure the thickness of the cortex — the outer layer of the brain responsible for functions such as speech, perception, long-term memory and judgment — which becomes thinner as Alzheimer's progresses. PET scans were used in a subset of participants to determine whether two proteins associated with Alzheimer's — tau and amyloid — were at higher levels.

When the researchers analyzed the fat measurements and the brain scans together, they found that participants with more visceral fat had larger accumulations of amyloid in their brains, suggesting that they might be at higher risk of developing Alzheimer's disease.

Prior research has shown that inflammation and high levels of insulin, which can block the proteins that break down amyloid in the brain, are linked with visceral fat, Raji said.

Because the earliest development of Alzheimer's in the brain can begin as much as 20 years before the first symptoms appear, the researchers plan to study the potential long-term impact of visceral fat by following up on the study's participants.

"That's why we started with a population at midlife," Raji said. "We want to see how it might play a role in the development of Alzheimer's, and that will give us an idea of the best window for effective interventions."

The best way to lose visceral fat is through exercise, especially aerobic exercise, Raji said.

It's not yet known whether getting rid of visceral fat can reverse its impact on the brain.

Dr. Mary Ellen Koran wasn't surprised by the findings.

"Since we already know that visceral fat is linked to so many bad health outcomes, including those involving the heart, it makes sense that it's also linked to poor brain health," said Koran, an Alzheimer's imaging specialist and an assistant professor of radiology and biological sciences at the Vanderbilt University Medical Center and School of Medicine. "But it's important that we do studies like this to define that link."

While it's more likely that an obese person will have both types of fat, thin people can also have visceral fat but not realize it.

Koran said she's seen "really skinny people who don't have much subcutaneous fat but a bunch of fat around their organs." Still, Koran doesn't recommend that people hoping to protect their brains get scanned for visceral fat until more research confirms the link.

While the number of people in the study is small, "it's helpful that the researchers are looking at a younger group of people," said Alzheimer's specialist Dr. Joel Salinas, a neurologist at NYU Langone Health and chief medical officer at the Isaac Health Online Memory Clinic.

Knowing who is at risk will allow people to start treatments earlier, Salinas said. "You want to stop the fire before the building burns down."

Dr. Borna Bonakdarpour, a neurology researcher at Northwestern University's Feinberg School of Medicine, says the study doesn't prove that visceral fat causes damage to the brain.

"It may simply be a marker for poor health," Bonakdarpour said. "We know that people who don't exercise or have a poor diet are at risk for developing Alzheimer's."

Before people start asking for abdominal scans to look for visceral fat, "we would need a much larger study," said Dr. Fanny Elahi, a neurologist and Alzheimer's researcher at the Icahn School of Medicine at Mount Sinai. "This is a very, very small study."

1.2 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 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.

Energy theory:

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

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

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

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

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

Time-dependent output strain and stress of (viscous input*output rate):

Hooke's law of linear elasticity is expressed as:

Strain (ε: epsilon) = Stress (σ: sigma) / Young's modulus (E)

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

PPG (strain) = carbs/sugar (stress) * GH.p-Modulus (a positive number) + post-meal walking k-steps * GH.w-Modulus (a negative number)

where GH.p-Modulus is 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, 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 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, 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 shows data table, Time-domain curves and SD-VMT energies.

3. CONCLUSION

In summary, this study explores the author's annual Alzheimer's disease risks associated with these five selected inputs. Conventional statistical analysis reveals strong correlations (90% to 93%) with four inputs, and with a moderately high correlation of 67% with walking steps.

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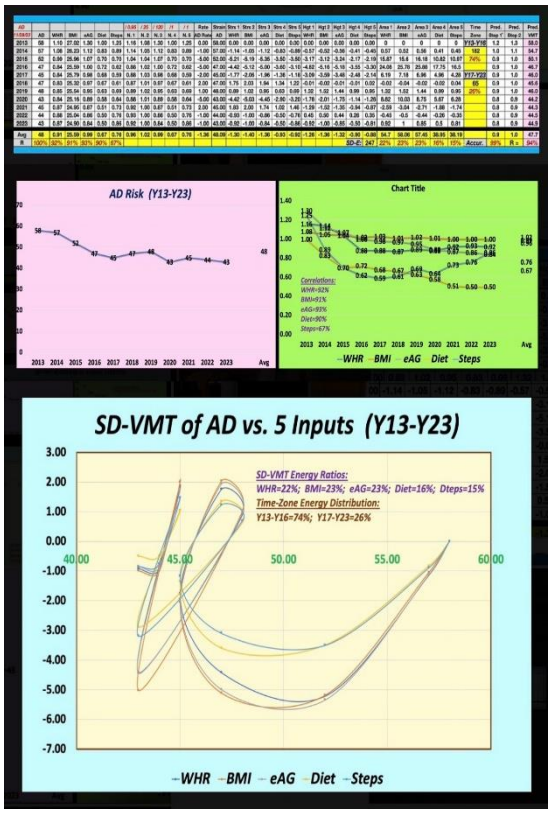

Figure 1: Data table, Time-domain curves and SD-VMT energies.

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(visceral fat), and diabetes glucose conditions at 22% to 23%, while diet and exercise serve as secondary influential factors at 15% to 16%.

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4. 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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Viscoelastic and Viscoplastic Glucose Theory Application in Medicine

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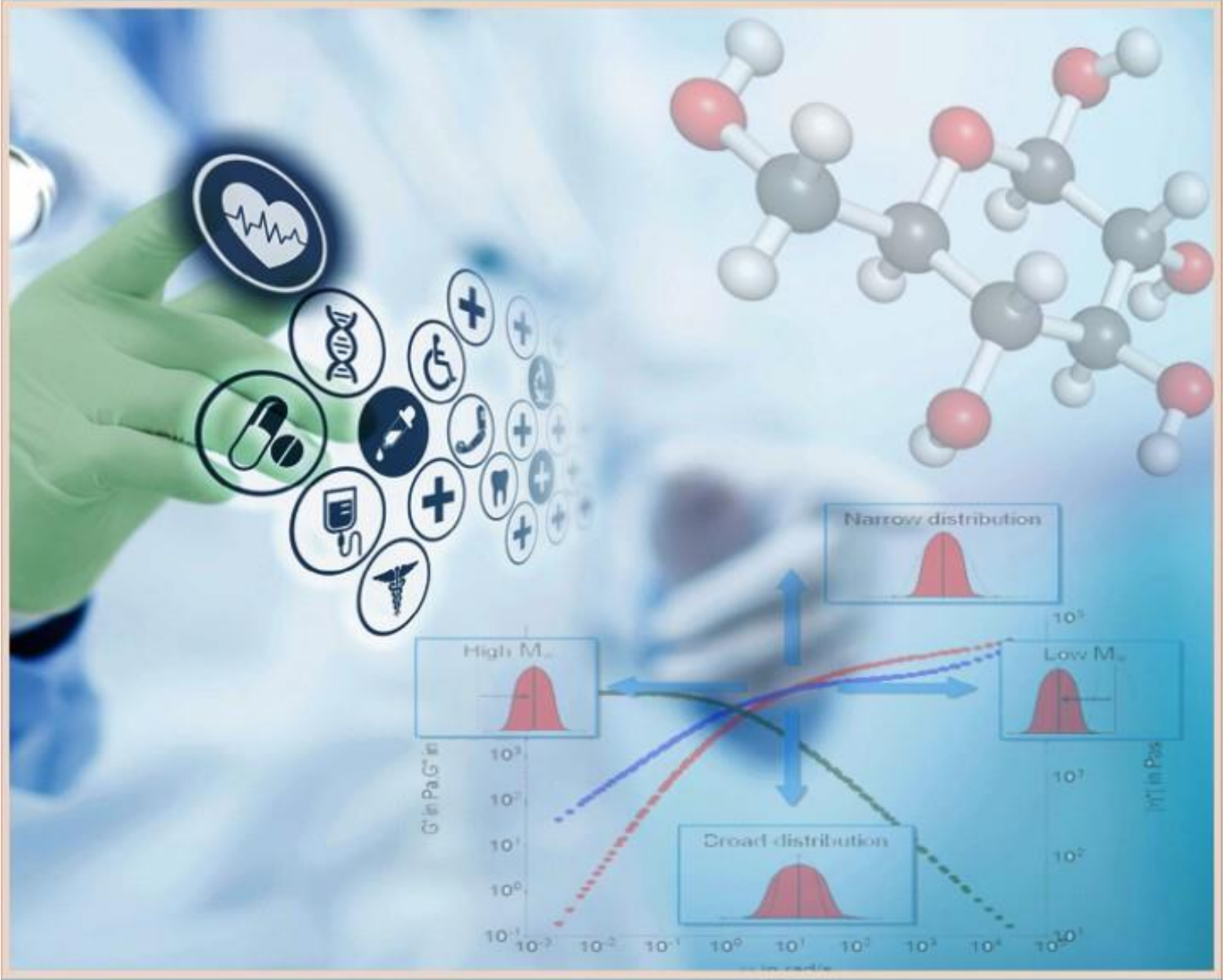

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