Special Issue 5
2022 https://www.theghmethod.com

The GH-Method

Viscoelastic Medicine Theory (VMT #335): Multi-Tiered VMT Energy Method Uses Multiple Tiers of Input Causes to Predict Risk of Cancers Based on GH-Method: Math-Physical Medicine (No. 935)

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Abstract

Since January 2021, the author has been utilizing the space-domain viscoplastic energy model (SD-VMT) in his medical research work. With a focus on the VMT methodology, he has authored a total of 335 research papers so far. This article aims to investigate various diseases or symptoms using a multi-tiered VMT research approach that takes into account multiple influential factors. The author follows a specific research process, which involves the following three steps: 1. Body weight (BW) versus food quantity (food size), daily walking steps (exercise), and sleep quality score (sleep). 2. PPG (representation of T2D status) versus pancreatic beta cells health state (via FPG), carbs & sugar intake grams (carbs), and post-meal walking steps (steps). 3. Assessment of cancer's genetic, body weight (it changes with time), life-time bad habits, environmental factors and viral infections (gene & habits) which includes three major normalized input elements: genetic scores (constant since he can not change his gene or his family medical history), personal body weight and bad habits (where he does not smoke cigarettes, drink alcohol, or use illicit drugs), and environmental exposures or viral infections (where he does not have any of these factors). To assess the risk of cancers, the author conducted three VMT analyses, resulting in three predicted outputs for body weight (BW), type 2 diabetes (T2D), and gene with habits. These three predicted outputs were then further utilized in the second-tier VMT analysis for a VMT-based cancer risk assessment. Subsequently, the author compared the VMT-based Cancer risks with the metabolism index (MI)-based Cancer risk, evaluating prediction accuracy and correlation. This study employs a two-tiered SD-VMT model, analyzing interactions among multiple influential factors and disease symptoms. The data for this analysis was collected between January 1, 2015, and September 23, 2023. In summary, the analysis conducted in this study leads to three significant observations: 1. The VMT analysis reveals that the total energy ratios for BW, T2D, and gene with habits are approximately 35%, 34%, and 32% respectively, indicating a nearly equal distribution. 2. The energy distribution across time zones shows that 83% of the energy is allocated to the Y15-Y19 period, while 17% is allocated to the Y29-Y23 period, resulting in an 80/20 split. This suggests that the author's risk of developing cancers is significantly lower during the COVID period compared to the pre-COVID period. 3. The VMT-based cancer risks exhibit an average value that is equivalent to the MI-based cancer risk, with a prediction accuracy of 100%. However, there is a waveform correlation of 68% between VMT-predicted cancer risks and MI-based cancer risks. In conclusion, this multi-tiered VMT model offers a comprehensive understanding and presents a continuous roadmap of the relationship between lifestyle details, metabolic disorders, and the development of deadly cancers.

Keywords: Viscoelastic; Viscoplastic; Cancers; Body weight; Diabetes; Exercise

Abbreviations: MI: metabolism index; CVD: cardiovascular diseases; BW: body weight; CKD: chronic kidney diseases; T2D: type 2 diabetes; PPG: postprandial plasma glucose; FPG: fasting plasma glucose

Available online: 11 January 2024

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1. INTRODUCTION

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This article aims to investigate various diseases or symptoms using a multi-tiered VMT research approach that takes into account multiple influential factors. The author follows a specific research process, which involves the following three steps:

1. Body weight (BW) versus food quantity (food size), daily walking steps (exercise), and sleep quality score (sleep).

2. PPG (representation of T2D status) versus pancreatic beta cells health state (via FPG), carbs & sugar intake grams (carbs), and post-meal walking steps (steps).

3. Assessment of cancer's genetic, body weight (it changes with time), life-time bad habits, environmental factors and viral infections (gene & habits) which includes three major normalized input elements: genetic scores (constant since he can not change his gene or his family medical history), personal body weight and bad habits (where he does not smoke cigarettes, drink alcohol, or use illicit drugs), and environmental exposures or viral infections (where he does not have any of these factors).

To assess the risk of cancers, the author conducted three VMT analyses, resulting in three predicted outputs for body weight (BW), type 2 diabetes (T2D), and gene with habits. These three predicted outputs were then further utilized in the second-tier VMT analysis for a VMT-based cancer risk assessment.

Subsequently, the author compared the VMT-based Cancer risks with the metabolism index (MI)-based Cancer risk, evaluating prediction accuracy and correlation. This study employs a two-tiered SD-VMT model, analyzing interactions among multiple influential factors and disease symptoms. The data for this analysis was collected between January 1, 2015, and September 23, 2023.

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.

Notes from the author of this paper:

Upon reviewing the upcoming excerpts from other published articles, it becomes evident that these findings are predominantly conveyed using qualitative statements. On occasion, these statements include a limited number of numerical values, typically sourced from statistical data within epidemiological studies. However, a recurring deficiency among them is the lack of robust quantitative findings to underpin their qualitative conclusions. Consequently, the author of this paper has deliberately opted to leverage his familiar methodologies from mathematics, physics, and engineering fields in his medical research pursuits. This strategic choice is intended to yield substantial conclusions supported by sound proofs via quantitative data, effectively bridging the current gap in the realm of biomedical research.

Pathophysiological explanations and statistical data regarding cancers versus obesity and diabetes:

There are several pathophysiological explanations and statistical data linking cancers with obesity and diabetes:

1. Obesity and cancer

Obesity is a significant risk factor for various types of cancer. The underlying mechanisms include chronic inflammation, altered hormone levels (such as increased insulin and estrogen), impaired immune function, and oxidative stress. Adipose tissue produces several hormones and cytokines that can promote tumor growth and progression. According to statistical data, obesity is associated with an increased risk of developing cancers such as breast, colorectal, endometrial, kidney, liver, and pancreatic cancer.

2. Diabetes and cancer

Diabetes, particularly type 2 diabetes (T2D), has been consistently associated with an increased risk of certain cancers. The pathophysiological links between diabetes and cancer include hyperinsulinemia, insulin resistance, chronic hyperglycemia, and low-grade inflammation. These factors contribute to cellular proliferation, DNA damage, and impaired DNA repair mechanisms, increasing the likelihood of cancer development. Statistical data indicates that individuals with T2D have a higher risk of developing cancers, including liver, pancreatic, colorectal, breast, and bladder cancer.

3. Shared risk factors

Obesity and diabetes share common risk factors that contribute to their association with cancer. These risk factors include a sedentary lifestyle, unhealthy dietary habits (such as high-calorie and high-sugar diets), and genetic predisposition. These factors can lead to metabolic abnormalities, chronic inflammation, and hormonal disturbances, all of which play a role in cancer development.

4. Statistical data

Numerous studies and population-based analyses have demonstrated the significant impact of obesity and diabetes on cancer incidence and mortality. For instance, according to the World Cancer Research Fund (WCRF) and the American Institute for Cancer Research (AICR), excess body weight is estimated to be a contributing factor for approximately 20% of all cancer cases globally. Similarly, individuals with diabetes have been shown to have an increased risk of developing certain cancers. For example, a meta-analysis published in the Lancet Oncology reported that diabetes is associated with a 20% higher risk of developing cancer.

In conclusion, the pathophysiological links between cancers, obesity, and diabetes are well-established. Statistical data consistently demonstrates that individuals who are obese or have diabetes are at a higher risk of developing various types of cancers. This highlights the importance of maintaining a healthy weight, promoting physical activity, adopting a balanced diet, and effectively managing diabetes to reduce the risk of cancer development.

Pathophysiological explanations and statistical data regarding cancers versus genetic, environmental, smoking, drinking, illicit drug, viral infections, etc.:

There are several pathophysiological explanations and statistical data regarding cancers in relation to genetic factors, environmental exposures, smoking, alcohol consumption, illicit drug use, viral infections, and other risk factors. Here are some key points:

1. Genetic factors

Certain inherited genetic mutations can increase the risk of developing specific types of cancer. For example, mutations in BRCA1 and BRCA2 genes are associated with an increased risk of breast and ovarian cancer. Statistical data suggests that around 5-10% of all cancers are caused by hereditary genetic abnormalities.

2. Environmental exposures

Exposure to various environmental factors, such as chemicals, radiation, and pollutants, can contribute to the development of cancer. For example, asbestos exposure is linked to an increased risk of lung cancer, mesothelioma, and other cancers. Statistical data estimates that environmental factors contribute to approximately 15-20% of all cancer cases.

3. Smoking

Tobacco smoking is a significant risk factor for many types of cancer, including lung, oral, esophageal, pancreatic, bladder, and kidney cancer. Smoking is responsible for about 30% of all cancer deaths. Statistical data shows that smokers are more likely to develop and die from cancer compared to non-smokers.

4. Alcohol consumption

Regular and excessive alcohol consumption is associated with an increased risk of various cancers, including those of the mouth, throat, esophagus, liver, breast, and colorectal. The risk appears to be dose-dependent. Statistical data indicates that alcohol consumption accounts for an estimated 4% of all cancer cases globally.

5. Illicit drug use

Certain illicit drugs, such as tobacco mixed with marijuana (cannabis) and injected drugs, can have carcinogenic effects, primarily through the inhalation of smoke or exposure to contaminated needles. However, the direct link between illicit drug use and specific cancers is complex and can be confounded by other factors, such as lifestyle behaviors and shared risk factors.

6. Viral infections

Certain viral infections are known to increase the risk of developing certain types of cancer. For example, human papillomavirus (HPV) is associated with cervical, anal, and oropharyngeal cancers, while hepatitis B and C viruses are linked to liver cancer. Statistical data suggests that viral infections account for about 15-20% of all cancer cases worldwide.

It is important to note that these risk factors often interact with each other, and individual susceptibility may vary. Additionally, lifestyle modifications, such as tobacco cessation, limiting alcohol consumption, practicing safe sex, and getting vaccinated against viral infections (where available), can help reduce the risk of developing cancer associated with these factors. Regular screenings and detection are also crucial in improving outcomes.

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 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 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.

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 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.

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 reciprocal of Young's modulus E.

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

Stress
= viscosity factor (η: eta) * strain rate (dε/dt)

Where strain is expressed as Greek epsilon or ε.

In this article, in order to construct an "ellipse-like" diagram in a stress-strain space domain (e.g. "hysteresis loop") covering both the positive side and negative side of space, he has modified the definition of strain as follows:

Strain
= (body weight at certain specific time instant)

He also calculates his strain rate using the following formula:

Strain rate
= (body weight at next time instant) - (body weight at present time instant)

The risk probability % of developing into CVD, CKD, Cancer is calculated based on his developed metabolism index model (MI) in 2014. His MI value is calculated using inputs of 4 chronic conditions, i.e. weight, glucose, blood pressure, and lipids; and 6 lifestyle details, i.e. diet, drinking water, exercise, sleep, stress, and daily routines. These 10 metabolism categories further contain ~500 elements with millions of input data collected and processed since 2010. For individual deadly disease risk probability %, his mathematical model contains certain specific weighting factors for simulating certain risk percentages associated with different deadly diseases, such as metabolic disorder-induced CVD, stroke, kidney failure, cancers, dementia; artery damage in heart and brain, micro-vessel damage in kidney, and immunity-related infectious diseases, such as COVID death.

Some of explored deadly diseases and longevity characteristics using the viscoplastic medicine theory (VMT) include stress relaxation, creep, hysteresis loop, and material stiffness, damping effect based on time-dependent stress and strain which are different from his previous research findings using linear elastic glucose theory (LEGT) and nonlinear plastic glucose theory (NPGT).

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 4 data tables.

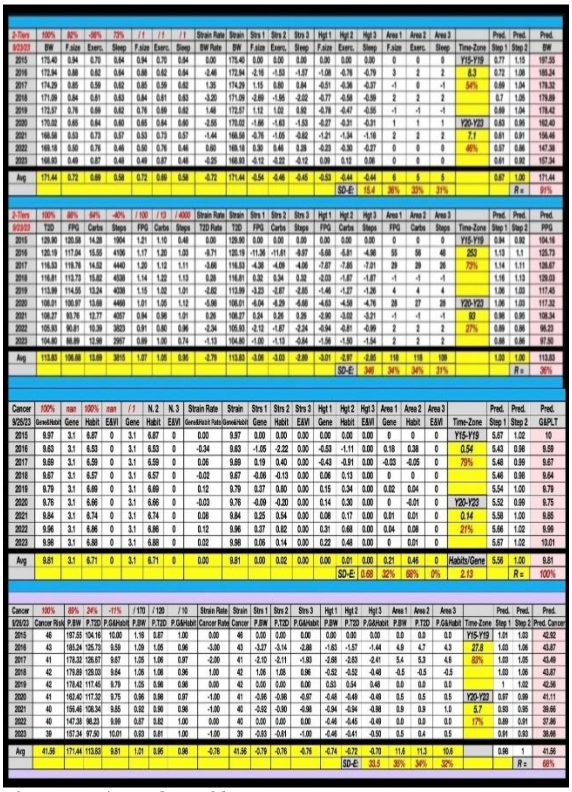

Figure 1: 4 input data tables.

Figure 2 shows 4 output diagrams.

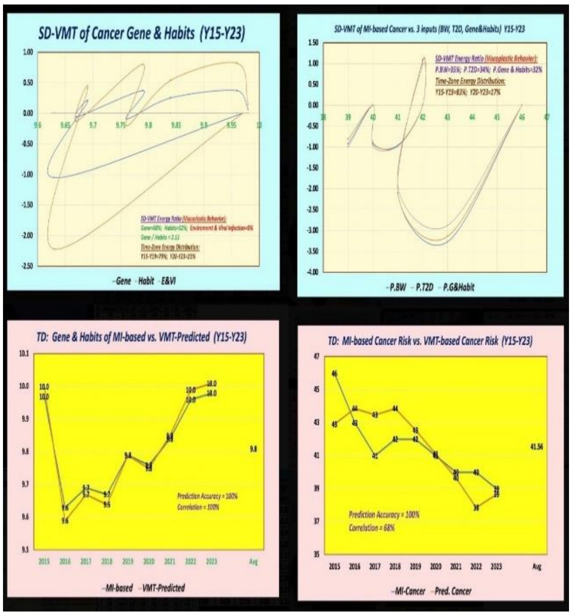

Figure 2: 4 output diagrams.

4. CONCLUSION

In summary, the analysis conducted in this study leads to three significant observations:

1. The VMT analysis reveals that the total energy ratios for BW, T2D, and gene with habits are approximately 35%, 34%, and 32% respectively, indicating a nearly equal distribution.

2. The energy distribution across time zones shows that 83% of the energy is allocated to the Y15-Y19 period, while 17% is allocated to the Y29-Y23 period, resulting in an 80/20 split. This suggests that the author's risk of developing cancers is significantly lower during the COVID period compared to the pre-COVID period.

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In conclusion, this multi-tiered VMT model offers a comprehensive understanding and presents a continuous roadmap of the relationship between lifestyle details, metabolic disorders, and the development of deadly cancers.

5. 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.

Special Issue | Issue No. 5

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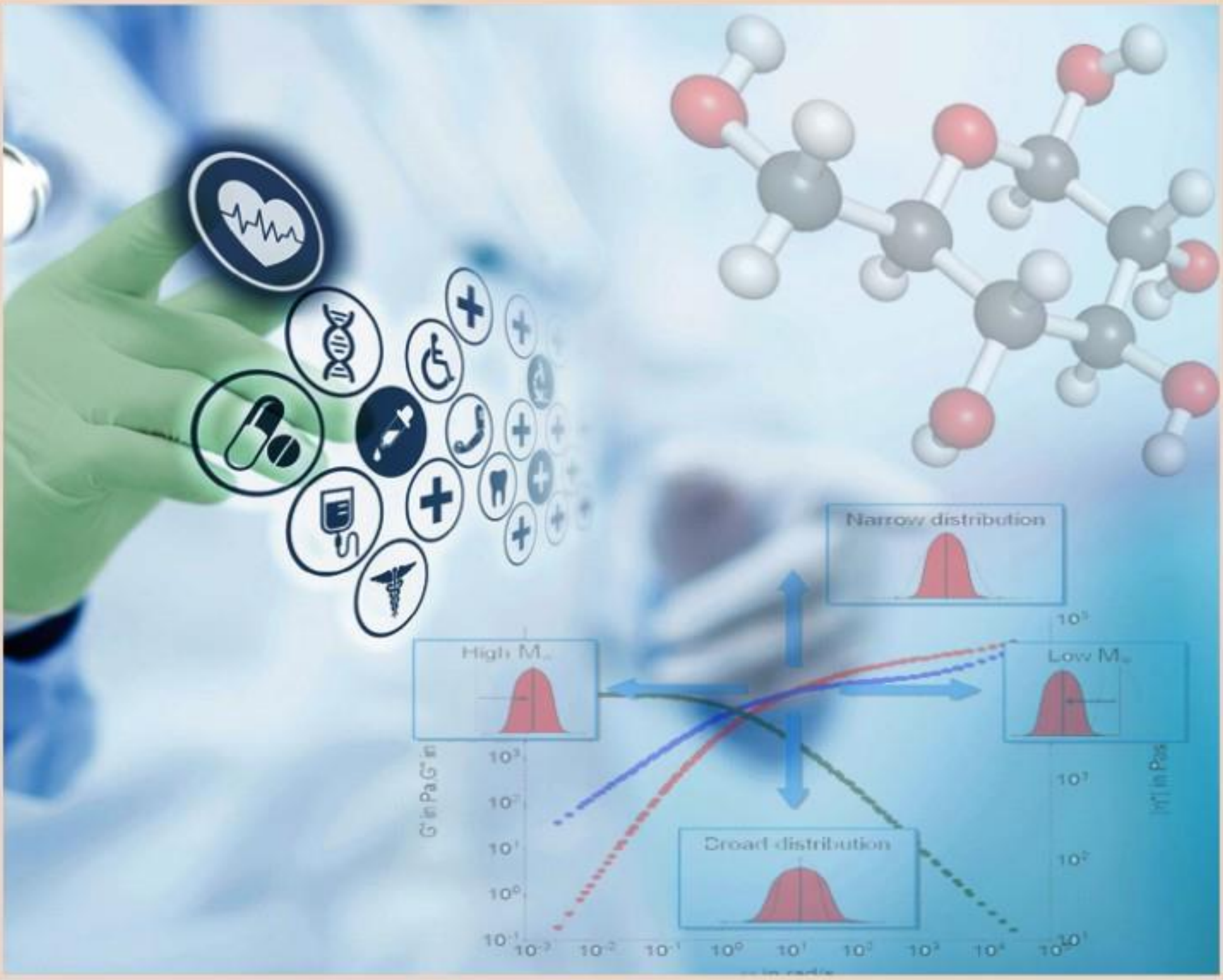

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