

The GH-Method

Viscoelastic Medicine theory (VMT #407): Relationships Between Cancer Risks and Five Influential Factors, Obesity, Diabetes, Combined Hypertension and Dyslipidemia, Diet, Exercise, Using Viscoplastic Energy Model of GH-Method: Math-Physical Medicine (No. 1009)

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Abstract

The author's investigation into the causes of various cancers extends beyond genetics, family history, and age, focusing on influential factors such as smoking, alcohol consumption, environmental elements, hormonal influences, radiation, air pollution, occupational hazards, inflammation, infections, obesity, diabetes, and poor dietary habits. These cancer contributors encompass lifestyle details, such as excessive food portions, poor diet quality, and physical inactivity. They are associated with many cancer types, including pancreatic, liver, colorectal, prostate, kidney, lung, and breast. In this article, the introduction section compiles some pathophysiological information and statistical data related to several serious cancers.

The author, a non-smoker who abstains from alcohol and illicit drugs, and has minimal exposure to environmental hazards, recognizes an inability to modify his physical age, genetic conditions, or family history. Therefore, he decided to focus on five controllable factors, namely body weight (m1), glucose levels (m2), blood pressure and blood lipids (m3 & m4), diet quality and quantity (m9), and physical activity (m5). He has been gathering personal health data since 2012, utilizing this information to analyze its impact on his risk of developing cancers. Notably, the data from the earlier years, specifically 2012 and 2013, includes a certain degree of estimated figures.

He adopted the space-domain viscoplastic medicine theory's energy method (SD-VMT) to conduct this cancer research.

In summary, the correlations between cancer risk and 5 inputs are very high, within the range of 84% and 93%.

His total SD-VMT energy is 248 with the following 5 energy contribution ratios: Obesity (m1) = 22%; Diabetes (m2) = 23%; BP and Lipid (m3 & m4) = 18%; Diet (m9) = 17%; Exercise (m5) = 19%

The time-zone energy distributions are: Y12-Y17 = 98%; Y18-Y23 = 2%.

Key message:

The author, diagnosed with severe Type 2 Diabetes in 1995, faced escalating health issues by 2010 (HbA1C at 11% with CVD and CKD). With a body weight of 220 lbs (BMI 32), he struggled with hypertension and dyslipidemia for more than ten years. His diet was marked by poor diet choices in both quantity and quality, and he also dealt with significant physical inactivity. The life-threatening situation in 2010 prompted him to overhaul his lifestyle drastically, to safeguard his health and life. As a result, his total energy within 2012 to 2017 accounted for 98%, whereas within 2018 to 2023, it accounted for merely 2%.

His overall cancer risk was primarily influenced by his diabetes (high glucose levels) at 23%, followed by obesity (high body weight) at 22%. His blood pressures, lipid levels, diet, and exercise had lesser but still notable impacts on his overall cancer risk, between 17% and 19%. However, to effectively reduce and maintain a healthy weight and glucose level, he had to continuously focus on improving his diet (both quantity and quality) and increasing his physical activity.

Keywords: Viscoelastic; Viscoplastic; Diabetes; Glucose; Biomarkers; Insulin; Hyperglycemia

Abbreviations: CGM: continuous glucose monitoring; T2D: type 2 diabetes; PPG: postprandial plasma glucose; FPG: fasting plasma glucose; SD: space-domain; VMT: viscoelastic medicine theory; FFT: Fast Fourier Transform; BMI: Body Mass Index; CKD: Chronic Kidney Disease

1. INTRODUCTION

The author's investigation into the causes of various cancers extends beyond genetics, family history, and age, focusing on influential factors such as smoking, alcohol consumption, environmental elements, hormonal influences, radiation, air pollution, occupational hazards, inflammation, infections, obesity, diabetes, and poor dietary habits. These cancer contributors encompass lifestyle details, such as excessive food portions, poor diet quality, and physical inactivity. They are associated with many cancer types, including pancreatic, liver, colorectal, prostate, kidney, lung, and breast. In this article, the introduction section compiles some pathophysiological information and statistical data related to several serious cancers.

The author, a non-smoker who abstains from alcohol and illicit drugs, and has minimal exposure to environmental hazards, recognizes an inability to modify his physical age, genetic conditions, or family history. Therefore, he decided to focus on five controllable factors, namely body weight (m1), glucose levels (m2), blood pressure and blood lipids (m3 & m4), diet quality and quantity (m9), and physical activity (m5). He has been gathering personal health data since 2012, utilizing this information to analyze its impact on his risk of developing cancers. Notably, the data from the earlier years, specifically 2012 and 2013, includes a certain degree of estimated figures.

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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, intending to optimize 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.

Most common cancer types and General causes for cancers:

The most common types of cancer in 2023 include:

- Breast Cancer: It is the most common cancer, with 300,590 new cases expected in the United States in 2023.
- Prostate Cancer: This is the most common cancer among men in the United States, with an estimated 288,300 new cases diagnosed in 2023.
- Lung Cancer: Lung and bronchus cancer are among the most common cancers worldwide and in the United States.
- Colorectal Cancer: Colorectal cancers, which include colon and rectal cancers, are commonly combined in statistics and are among the most prevalent types.

These cancers represent a significant portion of new cancer cases and are also major contributors to cancer-related deaths globally and in the United States. Breast, lung, prostate, and colorectal cancers together account for nearly half of all new cancer cases in the United States.

Cancer is a complex disease with multiple potential causes. Some of the general causes include:

- Genetic Factors: Certain types of cancer run in families, indicating a genetic predisposition.
- Lifestyle Factors: These include smoking, excessive alcohol consumption, poor diet, lack of physical activity, and obesity.
- Environmental Exposures: Exposure to certain chemicals, pollutants, and radiation can increase cancer risk.
- Infections: Certain infections can increase the risk of cancer, such as human papillomavirus (HPV) for cervical cancer and *Helicobacter pylori* for stomach cancer.
- Age: The risk of developing cancer generally increases with age due to the accumulation of risks over time and the natural ageing of cells.

• **Chronic Inflammation:** Long-term inflammation in the body can damage DNA and lead to cancer.

It's important to note that cancer is a multifactorial disease, meaning that usually, multiple factors contribute to its development. Regular screenings and adopting a healthy lifestyle can help in early detection and prevention.

Pancreatic cancer:

Pancreatic cancer, a complex and deadly disease, arises from a combination of genetic, environmental, and lifestyle factors. Here's a pathophysiological explanation along with statistical evidence regarding its causes:

• **Genetic Mutations:** The development of pancreatic cancer is often linked to mutations in specific genes. For instance, mutations in the KRAS gene are found in about 90% of pancreatic cancer cases. These mutations lead to uncontrolled cell growth and tumour formation.

• **Family History and Genetic Syndromes:** About 10% of pancreatic cancer cases are thought to be hereditary. Certain genetic syndromes, like hereditary breast and ovarian cancer syndrome (associated with BRCA1 and BRCA2 mutations), Lynch syndrome, and Familial Atypical Multiple Mole Melanoma (FAMMM) syndromes, increase the risk.

• **Smoking:** Smoking is a major risk factor, with studies showing that smokers are about twice as likely to develop pancreatic cancer compared to non-smokers. Approximately 20% to 30% of pancreatic adenocarcinomas (the most common type of pancreatic cancer) are believed to be caused by cigarette smoking.

• **Obesity and Diet:** Obesity increases the risk of pancreatic cancer by about 20%. A diet high in red and processed meats has also been linked to an increased risk, while a diet rich in fruits and vegetables may reduce the risk.

• **Chronic Pancreatitis:** Long-term inflammation of the pancreas, known as chronic pancreatitis, has been associated with an increased risk of developing pancreatic cancer.

• **Diabetes:** Pancreatic cancer is more common in people with diabetes. The

relationship is complex; while long-standing diabetes is a risk factor, pancreatic cancer can also cause diabetes.

• **Alcohol Use:** Heavy alcohol use can lead to chronic pancreatitis, which in turn increases the risk of pancreatic cancer.

• **Age, Gender, and Race:** The risk of pancreatic cancer increases with age, being more common in people over the age of 45. Men are slightly more likely to develop pancreatic cancer than women. Additionally, African Americans are at a higher risk compared to other racial groups.

• **Workplace Exposures:** Exposure to certain chemicals and toxins, particularly in the workplace, may increase the risk of pancreatic cancer.

Understanding these factors is crucial for early detection and prevention strategies. However, pancreatic cancer often presents late, when it is already advanced, making it one of the most challenging cancers to treat effectively.

Liver Cancer:

Liver cancer, specifically hepatocellular carcinoma (HCC), is a complex disease influenced by a variety of factors. Here's a pathophysiological explanation along with some statistical evidence regarding its causes:

• **Chronic Hepatitis B and C Infections:** These viral infections are the leading causes of HCC worldwide. Chronic infection leads to liver inflammation, fibrosis, and eventually cirrhosis, setting the stage for cancerous transformations. Statistically, chronic hepatitis B accounts for up to 50-55% of HCC cases globally, while hepatitis C contributes to around 25%.

• **Cirrhosis:** Regardless of the underlying cause (such as alcohol abuse, nonalcoholic steatohepatitis [NASH], or chronic viral hepatitis), cirrhosis is a significant risk factor for HCC. Cirrhosis represents the final stage of chronic liver disease, characterized by scarring and liver dysfunction, greatly enhancing the risk of developing liver cancer. About 80-90% of HCC cases occur in cirrhotic livers.

• **Aflatoxin Exposure:** Aflatoxins, produced by certain moulds found in improperly stored grains and nuts, are potent carcinogens.

Aflatoxin exposure is a significant risk factor, especially in parts of Africa and Asia. Long-term exposure can lead to mutations in the p53 tumour suppressor gene, increasing the risk of HCC.

- **Alcohol Consumption:** Chronic alcohol abuse is a well-established cause of cirrhosis and subsequent HCC. The risk correlates with the amount and duration of alcohol consumption.

- **Obesity and Nonalcoholic Fatty Liver Disease (NAFLD):** The rising prevalence of obesity and type 2 diabetes has led to an increase in NAFLD and its progressive form, NASH. These conditions can progress to cirrhosis and HCC. NAFLD-related HCC is becoming more common in Western countries.

- **Genetic Factors:** There are also genetic predispositions to liver cancer. Certain inherited liver diseases, like hemochromatosis and alpha-1 antitrypsin deficiency, increase the risk.

- **Environmental and Lifestyle Factors:** Chronic exposure to certain chemicals (like vinyl chloride and arsenic) and smoking can also increase liver cancer risk.

In terms of statistical evidence:

- Hepatitis B is responsible for 50-55% of HCC globally.

- Cirrhosis underlies 80-90% of HCC cases.

- NAFLD/NASH-related HCC is increasing in prevalence, particularly in Western countries.

It's important to note that these statistics can vary by region due to differences in risk factors prevalence, like hepatitis infection rates and dietary habits. Early detection and management of these risk factors are crucial in preventing the development of HCC.

Prostate Cancer:

Prostate cancer, like other forms of cancer, arises due to the uncontrolled growth and division of cells in the prostate gland. This complex condition involves multiple pathophysiological factors and its exact causes can be multifaceted. The following points summarize key aspects of its pathophysiology and statistical evidence regarding its causes:

- **Genetic Factors:** Mutations in certain genes, such as BRCA1 and BRCA2, increase the risk of prostate cancer. Men with a family history of prostate cancer have a higher risk, indicating a genetic predisposition.

- **Hormonal Influences:** Androgens, particularly testosterone, play a significant role in prostate growth and health. The growth of prostate cancer is often androgen-dependent, which is why treatments often target androgen signalling.

- **Age:** Prostate cancer risk increases with age, particularly after the age of 50. It's rare in men under 40, but the incidence rises sharply in older age groups.

- **Ethnicity and Geographic Variations:** African-American men have a higher incidence of prostate cancer and are more likely to have aggressive forms of the disease. The reasons for these disparities are not fully understood but are believed to involve a combination of genetic, environmental, and lifestyle factors.

- **Diet and Lifestyle:** A diet, particularly a diet high in red meat and high-fat dairy products, may increase the risk of prostate cancer. Conversely, diets rich in fruits and vegetables may lower the risk.

- **Obesity:** Obesity has been linked with an increased risk of developing more aggressive forms of prostate cancer.

- **Inflammation and Infections:** Chronic inflammation of the prostate (prostatitis) may increase the risk of prostate cancer, though the evidence is not definitive.

- **Environmental Factors:** Exposure to certain chemicals and toxins might increase the risk, but clear evidence is lacking.

Statistically, prostate cancer is one of the most common types of cancer in men. However, many men diagnosed with prostate cancer do not die from it; many live with the disease for years without experiencing serious symptoms. The survival rate for prostate cancer is relatively high compared to other cancers, especially when diagnosed at an early stage. Understanding the pathophysiology and statistical trends of prostate cancer is crucial for developing prevention strategies, improving diagnostic methods, and creating effective treatments. However, due to its complex nature, research

is ongoing to better understand all the factors involved in the development and progression of this disease.

Colorectal cancer:

Colorectal cancer, like many cancers, arises from a complex interplay of genetic, environmental, and lifestyle factors. Here's a pathophysiological explanation along with some statistical evidence of its causes:

- **Genetic Factors:** About 5-10% of colorectal cancers are due to inherited genetic mutations. The most common syndromes are Lynch syndrome and familial adenomatous polyposis (FAP). Individuals with these conditions have a significantly higher risk of developing colorectal cancer.
- **Environmental Factors:** Environmental influences, particularly diet, play a significant role. Diets high in red and processed meats have been linked to an increased risk of colorectal cancer. In contrast, diets rich in fruits, vegetables, and whole grains are thought to have a protective effect.
- **Lifestyle Factors:** Lifestyle choices such as physical inactivity, obesity, smoking, and heavy alcohol use have been associated with an increased risk of developing colorectal cancer.
- **Age:** The risk increases with age, with over 90% of cases diagnosed in individuals aged 50 and older.
- **Other Factors:** Chronic inflammatory conditions of the colon, such as ulcerative colitis and Crohn's disease, can increase the risk.

In terms of statistical evidence:

- **Diet:** Studies have shown that individuals who consume a high amount of red and processed meats have a 20-30% higher risk of developing colorectal cancer compared to those who eat little to no red or processed meat.
- **Obesity:** Obese individuals have a 30% higher risk of developing colorectal cancer compared to those with normal weight.
- **Smoking and Alcohol:** Smokers are about 20% more likely to develop colorectal cancer than non-smokers. Heavy alcohol use also increases the risk.

It's important to note that these statistics can vary based on the population studied and other factors. Regular screening and healthy lifestyle choices are key to reducing the risk of colorectal cancer.

Kidney cancer:

Kidney cancer, also known as renal cancer, is a complex disease influenced by a variety of pathophysiological factors and has certain statistical trends regarding its causes. Here's an overview:

Pathophysiological Explanation:

- **Genetic Mutations:** Certain genetic mutations can lead to uncontrolled cell growth in the kidney, resulting in cancer. These mutations can be inherited or acquired over a person's lifetime.
- **Von Hippel-Lindau (VHL) Disease:** This inherited disorder is associated with a higher risk of developing clear cell renal cell carcinoma, a common type of kidney cancer.
- **Environmental Factors:** Exposure to certain substances, like asbestos, cadmium, some herbicides, and organic solvents, particularly trichloroethylene, is linked to an increased risk of kidney cancer.
- **Smoking:** Tobacco use significantly increases the risk. The harmful substances in tobacco can damage the kidneys, leading to cancer.
- **Obesity:** Excess body weight may cause changes in hormone levels, which could increase the risk of kidney cancer.
- **High Blood Pressure (Hypertension):** There's an observed correlation between hypertension and an increased risk of kidney cancer, though the exact mechanism is not fully understood.
- **Chronic Kidney Disease and Dialysis:** Long-term kidney problems and dialysis treatment can lead to the development of cysts in the kidneys, which can potentially turn cancerous.

Statistical Evidence:

- **Prevalence:** Kidney cancer is among the 10 most common cancers in both men and women.

• **Gender:** Statistically, it is more common in men than in women.

• **Age Factor:** The risk increases with age, most commonly diagnosed in people aged 60-70 years.

• **Ethnicity and Geography:** Certain ethnicities and geographical locations show varying incidences, suggesting a combination of genetic and environmental factors.

• **Survival Rates:** The survival rates for kidney cancer vary based on the stage at diagnosis, with earlier detection generally leading to better outcomes.

• **Lifestyle Factors:** Statistical data also point to lifestyle factors, like diet and physical activity, influencing the risk of developing kidney cancer.

• **Incidence and Mortality Rates:** These rates vary globally, influenced by factors such as healthcare access, environmental exposures, and lifestyle choices.

Understanding these pathophysiological mechanisms and statistical trends helps in the prevention, early detection, and treatment of kidney cancer. However, it's important to note that every individual case can vary significantly.

Lung cancer:

Lung cancer, one of the most common types of cancer worldwide, arises due to a complex interplay of genetic, environmental, and lifestyle factors. Here's a pathophysiological explanation and some statistical evidence regarding its causes:

• **Smoking:** The primary cause of lung cancer is smoking, which accounts for about 85% of cases. Tobacco smoke contains carcinogens that damage the DNA in lung cells. Over time, this damage can lead to cancer. Non-smokers exposed to secondhand smoke are also at increased risk.

• **Radon Exposure:** Radon, a naturally occurring radioactive gas, is the second leading cause of lung cancer. It can accumulate in buildings, particularly in basements. Long-term exposure to high levels of radon can lead to lung cancer.

• **Occupational Exposure:** Certain occupations expose workers to carcinogens like asbestos, arsenic, diesel exhaust, and

some forms of silica and chromium. These exposures are significant risk factors, especially in industrial settings.

• **Air Pollution:** Long-term exposure to air pollutants, particularly fine particulate matter, is associated with a modest increase in the risk of lung cancer.

• **Genetic Factors:** Genetic predisposition plays a role, especially in non-smokers. Mutations in certain genes can increase susceptibility to lung cancer.

Statistically, lung cancer is more prevalent in older individuals, with the majority of cases diagnosed in people aged 65 or older. Men are more likely to develop lung cancer than women, although the gap is narrowing due to changes in smoking habits. Lung cancer rates also vary by race and ethnicity, influenced by factors like smoking prevalence and access to healthcare.

In conclusion, while smoking remains the most significant risk factor, lung cancer's etiology is multifactorial, involving a combination of environmental exposures and genetic predisposition. Public health measures, like smoking cessation programs and reducing occupational and environmental exposures, play a key role in prevention.

Breast cancer:

Breast cancer is a complex disease with various pathophysiological mechanisms and risk factors. Here's a detailed explanation along with some statistical evidence:

• **Hormonal Factors:** Estrogen and progesterone, female hormones, play a significant role in breast cancer development. Prolonged exposure to these hormones, either due to early menstruation, late menopause, or hormone replacement therapy, increases the risk. Statistically, women who experience menopause after the age of 55 are at a higher risk compared to those who experience it earlier.

• **Genetic Mutations:** BRCA1 and BRCA2 gene mutations are known to significantly increase the risk of breast cancer. Women with a BRCA1 mutation have about a 55-65% lifetime risk of developing breast cancer, while those with a BRCA2 mutation have about a 45% risk.

- **Family History:** Women with a close relative (mother, sister, or daughter) who had breast cancer have a doubled risk of developing the disease compared to those without a family history.

- **Age:** The risk of breast cancer increases with age. Statistics show that the majority of breast cancer cases occur in women over the age of 50.

- **Lifestyle Factors:** Obesity, especially after menopause, physical inactivity, and alcohol consumption are associated with an increased risk of breast cancer. Women who consume 2-3 alcoholic drinks per day have a 20% higher risk compared to non-drinkers.

- **Reproductive History:** Women who have their first pregnancy after the age of 30 or who never have a full-term pregnancy are at a higher risk. Breastfeeding for a total of one year or longer slightly reduces the risk.

- **Radiation Exposure:** Exposure to ionizing radiation, especially during puberty, increases the risk of developing breast cancer later in life.

- **Diet and Environmental Factors:** Though not conclusively proven, high-fat diets and exposure to certain chemicals and pollutants might contribute to the risk.

It's crucial to understand that these factors can interact in complex ways, and having one or more risk factors doesn't necessarily mean a woman will develop breast cancer. Regular screening and lifestyle modifications can help in early detection and risk reduction.

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 the 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 has been 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 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 travelled 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 travelling 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-travelling, 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 checked 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 lengths 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 labour-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 glucose 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 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) influence 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 labour work or exercise, the glucose level will decrease. As a result, the exercise burns off the energy, 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 behaviours 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 have 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, 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).

2. RESULTS

Figure 1 shows data table, inputs and SD-VMT energy output diagram.

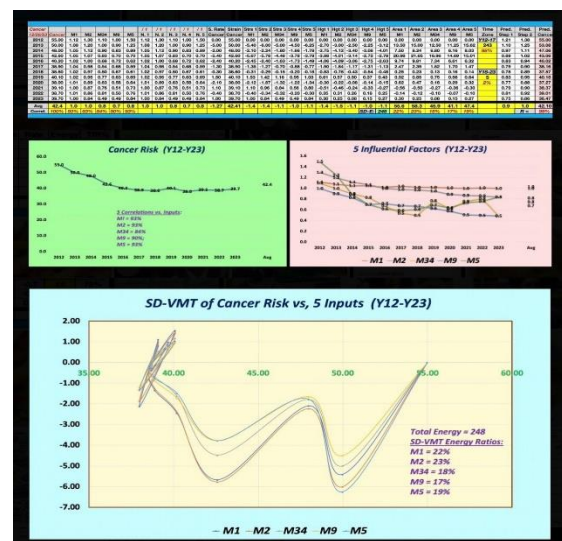


Figure 1. Data table, inputs, and SD-VMT energy output diagram.

3. CONCLUSION

In summary, the correlations between cancer risk and 5 inputs are very high, within the range of 84% and 93%.

His total SD-VMT energy is 248 with the following 5 energy contribution ratios:

- Obesity (m1) = 22%;
- Diabetes (m2) = 23%;
- BP and Lipid (m3 & m4) = 18%
- Diet (m9) = 17%
- Exercise (m5) = 19%

The time-zone energy distributions are:

- Y12-Y17 = 98%
- Y18-Y23 = 2%.

Key message:

The author, diagnosed with severe Type 2 Diabetes in 1995, faced escalating health issues by 2010 (HbA1C at 11% with CVD and CKD). With a body weight of 220 lbs (BMI 32), he struggled with hypertension and dyslipidemia for more than ten years. His diet was marked by poor diet choices in both quantity and quality, and he also dealt with significant physical inactivity. The life-threatening situation in 2010 prompted him to overhaul his lifestyle drastically, to safeguard his health and life. As a result, his

total energy within 2012 to 2017 accounted for 98%, whereas within 2018 to 2023, it accounted for merely 2%.

His overall cancer risk was primarily influenced by his diabetes (high glucose levels) at 23%, followed by obesity (high body weight) at 22%. His blood pressures, lipid levels, diet, and exercise had lesser but still notable impacts on his overall cancer risk, between 17% and 19%. However, to effectively reduce and maintain a healthy weight and glucose level, he had to continuously focus on improving his diet (both quantity and quality) and increasing his physical activity.

4. REFERENCES

For editing purposes, the majority of the references in this paper, which are self-references, have been removed from 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, 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.

Viscoelastic and Viscoplastic Glucose Theory Application in Medicine

Gerald C. Hsu

