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Viscoelastic or Viscoplastic Glucose Theory (VGT #66): Relative Risk Probability of Developing Ovarian and/or Uterine Cancers in Female Patients Using a Type 2 Diabetes and Obesity Patient Collected Data Over 12+ Years from 1/1/2010 to 4/23/2022 with 5 Gender-Independent Generic Cancer Risk Factors, i.e., Obesity, Diabetes, Lipids, Diet, and Exercise to Validate the Suitability of VGT Tool for Gynecological Cancer Study Based on GH-Method: Math-Physical Medicine (No. 655)

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Keywords: Viscoelastic; Viscoplastic; Ovarian cancer; Uterine cancer; Type 2 diabetes; Obesity; Diet; Exercise; Fasting plasma glucose; Postprandial plasma glucose

Abbreviations: T2D: type 2 diabetes; SD: space domain; TD: time domain; PPG: postprandial plasma glucose; FPG: fasting plasma glucose; MPM: math-physical medicine

1. INTRODUCTION

A family member of the author was recently diagnosed with ovarian and uterine cancer which piqued his interest in understanding more about this subject. To date, he has read numerous published articles regarding this topic. Although the majority of these research papers utilize statistical data analysis tools to draw their observed conclusions, they indeed identify certain risk factors related to ovarian and uterine cancers. As a result, he has learned that there are three general categories of risk factors related to these gynecological cancers.

The first risk category is the gender-dependent factors without any collected data to support this analysis. Here are the following 11 risk factors which are not covered in this article.

- things that affect female hormone level
- use of an intrauterine device (IUD)
- previous gynecological cancers, e.g. breast cancer
- previous radiation therapy
- having children later or never having a full-term pregnancy (after age 35)
- taking hormone therapy after menopause
- using fertility treatment
- pregnancies, breastfeeding, and taking birth control medications (lowering risk)
- increased number of menstrual cycles
- infertility
- certain medication effects

The second risk category is gender-independent but non-modifiable factors. Here are the 3 risk factors that are also not covered in this study:

- aging
- race
- family history

The third risk category is gender independent and modifiable by the patient which uses the author-collected data to support the study of

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developing ovarian cancer and/or uterine cancer. Here are these 5 cancer risk factors:

- obesity or being overweight
- diabetes conditions
- lipids
- diet
- exercise

The author is a male patient with multiple chronic diseases and is not licensed to treat patients. Therefore, he does not collect any data from female patients in this domain. He had no choice but to exclude those important gender-dependent cancer risk factors from this study. However, he is a 27-year veteran of type 2 diabetes (T2D), who also has hypertension, hyperlipidemia, obesity, and heart diseases, he has collected nearly 3 million data related to his metabolism conditions and various chronic diseases. Since he has accumulated plenty of data related to the gender-independent cancer risk factors, especially obesity, T2D, lipids, diet, and exercise, these still are useful resources for studying the risk probability of developing ovarian and/or uterine cancer in women. At a minimum, it may offer a partial picture of these two cancer cases. He then decided to create a hypothetical case covering a female cancer patient and apply his data to investigate these 5 gender-independent gynecological cancer risk factors. Next, he will analyze the risk of having ovarian and uterine cancers using obesity or being overweight via body weight, diabetes via HbA1C or glucoses, diet via a combined score of food quality and quantity, and exercise via daily walking steps.

Thus far, most of the author's published medical papers are based on his collected biomarker data from his own body over the past 12+ years. His research work is based on quantitative analysis of the collected data using a math-physical preventive medicine research methodology, not using a biochemical-based and statistical data analysis approach. In other words, he describes his observed biophysical phenomena using 10 numerical digits instead of the normal biochemical phenomena using 26 English alphabet letters due to the lack of training in biology and chemistry. Based on his past 13-year of self-study and intensive research on internal medicine and food nutrition, he has observed that most biomedical problems follow the basic law and principles of physics which can be easily analyzed and interpreted using many mathematical branches from the foundation level and using engineering modeling techniques from the application level.

1.1 Background information

In the following section, the author of this article has outlined some key information from 7 of his studied papers regarding gynecological oncology and then listed them in the Introduction section. The selected information sources have been referenced within this section and will not be listed in the Reference section again.

"Gynecological Cancer:

(1) American Cancer Society

Many factors affect the risk of developing endometrial cancer, including:

- Obesity
- Things that affect hormone levels, like taking estrogen after menopause, birth control pills, or tamoxifen; the number of menstrual cycles (over a lifetime), pregnancy, certain ovarian tumors, and polycystic ovarian syndrome (PCOS)
- Use of an intrauterine device (IUD)
- Age
- Diet and exercise
- Type 2 diabetes
- Family history (having close relatives with endometrial or colorectal cancer)
- Having had breast or ovarian cancer in the past
- Having had endometrial hyperplasia in the past
- Treatment with radiation therapy to the pelvis to treat another cancer

Some of these like pregnancy, birth control pills, and the use of an intrauterine device are linked to a lower risk of endometrial cancer, while many are linked to a higher risk.

(2) Ovarian Cancer Risk Factors

https://www.cancer.org/cancer/ovarian-cancer/causes-risks-prevention/risk-factors.html#

A risk factor is anything that increases your chance of getting a disease like cancer. Different cancers have different risk factors. Some risk factors, like smoking, can be changed. Others, like a person's age or family history, can't be changed.

But having a risk factor, or even many does not mean that you will get the disease. And some people who get the disease may not have any known risk factors. Researchers have discovered several risk factors that might increase a woman's chance of developing epithelial ovarian cancer.

Factors that increase your risk of ovarian cancers, such as getting older, being overweight, or being obese.

Obesity has been linked to a higher risk of developing many cancers. The current information available for ovarian cancer risk and obesity is not clear. Obese women (those with a body mass index [BMI] of at least 30) probably have a higher risk of developing ovarian cancer, but not necessarily the most aggressive types, such as high-grade serous cancers. Obesity may also negatively affect the overall survival of a woman with ovarian cancer.

Smoking

Smoking doesn't increase the risk of ovarian cancer overall, but it is linked to an increased risk for the mucinous type
Having children later or never having a full-term pregnancy
Taking hormone therapy after menopause
Having a family history of ovarian cancer, breast cancer, or colorectal cancer
Having a family cancer syndrome
Using fertility treatment
Having had breast cancer
Factors with unclear effects on ovarian cancer risk
Androgens
Talcum powder
Pregnancy and breastfeeding
Birth control

Diet

Some studies have shown a reduced rate of ovarian cancer in women who ate a diet high in vegetables or a low-fat diet, but other studies disagree. The American Cancer Society recommends following a healthy eating pattern that includes plenty of fruits, vegetables, and whole grains, and that limits or avoids red and processed meats, sugary drinks, and highly processed foods. Even though the effect of these dietary recommendations on ovarian cancer risk remains uncertain, following them can help prevent several other diseases, including some other types of cancer.

(2) Cancer Treatment Centers of America:

Risk factors for uterine cancer
by Maurie Markman, MD, President, Medicine & Science at CTCA, MARCH 21, 2022.

Each year, more than 60,000 women are diagnosed with endometrial cancer or uterine sarcoma, the two primary types of uterine cancer. No one knows if or when uterine cancer will develop, so it is important to understand the risk factors of the disease.

What causes uterine cancer?

Uterine cancer forms when the DNA in cells in the uterus mutates, disabling functions that control cell division and growth. In most cases, cancer cells in the uterus are found in the endometrium, the inner lining of the uterus. This is called endometrial cancer. While the exact cause of a woman's uterine cancer may not be known, certain risk factors are strongly linked to the disease, including obesity and high blood sugar.

Uterine sarcoma and endometrial cancer have different risk factors. Having one risk factor for cancers of the uterus, or even several, doesn't mean you will get the disease. Likewise, having no common risk factors doesn't mean you will not develop cancer.

Known risk factors for uterine cancer include:

General

Age: Most women diagnosed with endometrial cancer are older than age 50 and have gone through menopause. Sixty years old is the average age at diagnosis, according to the American Society of Clinical Oncology (ASCO).

Increased number of menstrual cycles: Women who have had more menstrual cycles

in their lifetime have an increased endometrial cancer risk. This includes those who started their periods before age 12 and who went through menopause after age 50.

No pregnancies: Researchers are investigating why pregnancy seems to reduce the risk of endometrial cancer. During pregnancy, a woman's hormonal balance shifts, with her body producing more progesterone and less estrogen.

Infertility: Irregular menstrual cycles and infertility also may cause imbalances in estrogen and progesterone levels, which may increase the risk of endometrial cancer.

Body

Obesity: Fat tissues tend to produce higher levels of estrogen, particularly after menopause, which increases the endometrial cancer risk for older, overweight women. Women who have a higher body mass index (BMI) have an elevated risk of endometrial cancer. As many as 70 percent of uterine cancer cases are thought to be due, in part, to obesity, according to the ASCO.

Metabolic syndrome: This syndrome occurs when a specific set of conditions develop at the same time, such as extra fat around the abdomen, high blood sugar, high blood pressure, high levels of triglycerides, and low levels of high-density lipoproteins in the blood.

Endometrial hyperplasia: This condition occurs when a buildup of cells and glandular structures causes a thickening in the endometrium (the lining of the uterus). There are several types of endometrial hyperplasia, including:

- Simple hyperplasia
- Complex hyperplasia
- Simple atypical hyperplasia
- Complex atypical hyperplasia

Hyperplasia is not cancer, but the condition may increase the risk of developing cancer, depending on the type of hyperplasia, whether the cells in the endometrium have become abnormal, and other factors. Endometrial hyperplasia most often occurs in women after menopause and may be caused by an imbalance of excess estrogen without progesterone. Obesity, other medical conditions, and family history may increase the risk of developing hyperplasia.

Prior pelvic radiation therapy: Patients who have undergone radiation therapy in the pelvic area for cancer in the past may face an increased risk of developing endometrial cancer. Radiation may damage the DNA within cells, which may increase the odds that these cells turn into cancer.

Breast or ovarian cancer: Breast, ovarian, and endometrial cancer share many of the same risk factors, especially those related to diet, hormones, and reproduction. As a result, breast or ovarian cancer survivors may have an elevated risk of developing endometrial cancer.

Hypothyroidism: This causes a decrease in the production of important hormones and has been linked to endometrial cancer. Studies have found that endometrial cancer patients have higher rates of hypothyroidism and that endometrial cancer sometimes seems to be linked to a past diagnosis with thyroid disease. However, more research is needed to confirm the association.

Race: Uterine cancer is more common among white women compared with other races and ethnicities. While Black women develop uterine cancer less often than white women overall, Black women are more likely to have advanced uterine cancer at the time of diagnosis. Aggressive uterine tumors are also more likely to develop among Black and Hispanic women.

Diabetes: Endometrial cancer is more common among women with type 2 diabetes—about two times more common than the average population, according to the ACS. However, it's unclear whether diabetes itself is linked to endometrial cancer or whether the association between diabetes and obesity is responsible for the increased risk.

Ovarian tumors: A type of ovarian cancer tumor called a granulosa cell tumor may produce estrogen that stimulates the growth of the uterine lining, which may lead to endometrial cancer. Sometimes, women may experience abnormal bleeding related to endometrial cancer that leads them to discover an ovarian tumor.

(3) Gynecological Cancers:

University of Michigan
Comprehensive Cancer Center
What Every Woman Should Know About Gynecologic Cancer & What is gynecologic cancer?
R. Kevin Reynolds, MD
The George W. Morley Professor & Chief, Division of Gyn Oncology University of Michigan
Ann Arbor, MI

Cancer is a disease where cells grow and spread without control. Gynecologic cancers begin in the female reproductive organs. The most common gynecologic cancers are endometrial cancer, ovarian cancer and cervical cancer. Less common gynecologic cancers involve vulva, Fallopian tube, uterine wall (sarcoma), vagina, and placenta (pregnancy tissue: molar pregnancy).

What causes endometrial cancer?

Endometrial cancer is the most common gynecologic cancer: one out of every 40 women will develop endometrial cancer. It is caused by too much estrogen, a hormone normally present in women. The most common cause of the excess estrogen is being overweight: fat cells actually produce estrogen. Another cause of excess estrogen is medication such as tamoxifen (often prescribed for breast cancer treatment) or some forms of prescribed estrogen hormone therapy (unopposed estrogen).

How is endometrial cancer detected?

Almost all endometrial cancer is detected when a woman notices vaginal bleeding after her menopause or irregular bleeding before her menopause. If bleeding occurs, a woman should contact her doctor so that appropriate testing can be performed. This usually includes an endometrial biopsy, a brief, slightly crampy test, performed in the office. Fortunately, most endometrial cancers are detected before spread to other parts of the body occurs.

Is endometrial cancer treatable?

Yes! Most women with endometrial cancer will undergo surgery including hysterectomy (removal of the uterus) in addition to removal of ovaries and lymph nodes. In most cases, if biopsies taken at the time of surgery show that the cancer has not spread, no other treatment is needed. If cancer has spread to other areas, then additional treatment with radiation is usually needed. Most women with endometrial cancer will be cured if they receive appropriate treatment.

How is ovarian cancer different from endometrial cancer?

Ovarian cancer is less common than endometrial cancer, but it is a more deadly type of cancer. More women die from ovarian cancer than all other forms of gynecologic cancer combined. Unlike endometrial cancer, ovarian cancer is often not detected until it has already spread to other parts of the body.

Who gets ovarian cancer?

One out of every 60 women will develop ovarian cancer in her lifetime. There are 2 different groups of women who may be at risk for ovarian cancer. The majority of ovarian cancers develop in women who have many ovulations (egg release from the ovary) during their lifetime. Bearing children and use of birth control pills both reduce the likelihood of ovarian cancer because they reduce the number of lifetime ovulations. About 10% of women who develop ovarian cancer have inherited a gene from either parent that increases the risk. There are at least 6 genes that can cause inherited ovarian cancer, including BRCA-1, BRCA-2, MSH, MLH, PMS-1, and PMS-2. Warning signs that a gene abnormality may be present in a woman's family include: Multiple family members with breast cancer, ovarian cancer or colon cancer.

Early age of cancer in affected relatives.

A woman should inform her doctor if she is aware of a family history of cancer, Tests are available that can detect abnormal genes and if they are detected, prophylactic removal of the ovaries may greatly reduce the risk of ovarian cancer.

How is ovarian cancer detected?

Warning signs of ovarian cancer include abdominal discomfort, loss of appetite, nausea, bloating, gas, constipation, and frequent urination, among others. These signs usually do not occur until the ovarian

cancer has already spread. Early ovarian cancer usually does not cause any symptoms, which is why this is often referred to as a "silent disease". There are no reliable tests yet available to detect early ovarian cancer.

Tests that may be very useful in some, but not all, situations include ultrasound (sound waves that provide a visual image of the ovary) and tumor marker blood tests (CA-125, CA 19-9, CEA, AFP, and others). None of these tests are perfect, and inappropriate use of them can actually cause more harm than good. There are a number of new tests in development including a proteomics test (Ovacheck) and new markers (LPA). It is too soon to know whether these tests will improve our ability to detect early ovarian cancer.

How is ovarian cancer treated?

Surgery is almost always the first step for treatment. For most women, this will include a hysterectomy with removal of ovaries, lymph nodes and the omentum (a pad for other organs in the abdomen). In young women, some types of ovarian cancer can be treated with less aggressive surgery to preserve fertility. Following surgery, the exact type of ovarian cancer and the extent of spread (stage), if any, are determined. Some early ovarian cancers require only surgery for treatment. The majority of women with ovarian cancer will need chemotherapy, which is medication designed to kill cancer cells to reduce the risk of cancer recurring.

Is ovarian cancer curable?

With modern surgical techniques and chemotherapy, ovarian cancer without spread at the time of diagnosis can be "cured" in up to 95% of women. Unfortunately, at least 70% of ovarian cancers are detected after the spread has occurred. For these women, treatment has an 80% chance of "remission", meaning disappearance or shrinkage of disease. The disease of this advanced extent has a high likelihood of relapse, anywhere from months to years after treatment. We can often successfully treat relapses and attain another remission, but we are not able to permanently "cure" ovarian cancer that has relapsed.

What is being done to improve ovarian cancer treatment?

Clinical trials allow experimental treatments to be attempted in controlled settings to protect the safety of patients. Research centers nationwide are developing new treatments including Immunotherapy (Ovarex, vaccines), Gene therapy< Biological therapy, and New chemotherapy treatment. It takes a great deal of time to improve treatment of ovarian cancer, but progress is being made.

The quality of life for women with ovarian cancer is improving. Chemotherapy no longer causes severe nausea, vomiting, or exhaustion.

Resources

University of Michigan
Department of ObGyn
Appointments
American Cancer Society Women's Cancer Network National Institutes of Health Society of Gynecologic Oncologists
www.med.umich.edu/obgyn/gynonc/more.htm
734-647-8906
www.cancer.org
www.wcn.org (locate a obgyn oncologist)
www.nci.nih.gov (listing of clinical trials)
www.sgo.org

Resources

University of Michigan		
Department of ObGyn	www.med.umich.edu/obgyn/gynonc/more.htm	
Appointments	734-647-8906	
American Cancer Society	www.cancer.org	
Women's Cancer Network	www.wcn.org	(locate a gyn oncologist)
National Institutes of Health	www.nci.nih.gov	(listing of clinical trials)
Society of Gynecologic Oncologists	www.sgo.org	

(4) Synchronous Primary Ovarian and Endometrial Cancers: A Series of Cases and a Review of Literature
Sylwia Dębska-Szmich, Urszula Czernek, [...], and Piotr Potemski

Conclusions

Synchronous ovarian and uterine cancers are predominant among patients with multiple gynecological cancers. The synchronous cancers are usually diagnosed at an earlier stage, have lower grading and prognosis is better when compared to a single advanced cancer.

(5) Gynecology Oncology

Diabetes Mellitus and Ovarian Cancer: More Complex than Just Increasing Risk
Monjri M. Shah, Britt K. Erickson, [...], and Charles A. Leath, III
Additional article information

Objective

Diabetes mellitus (DM) is a risk factor for endometrial cancer and is associated with poorer outcomes in breast and colon cancers. This association is less clear in epithelial ovarian cancer (EOC). We sought to examine the effect of DM on progression-free (PFS) and overall survival (OS) in women with EOC.

Conclusions

EOC patients with DM have poorer survival than patients without diabetes; this association is independent of obesity. Metformin use did not affect outcomes. The pathophysiology of this observation requires more inquiry.

(6) Association Between Diabetes Mellitus and Subsequent Ovarian Cancer in Women
A systematic review and meta-analysis of cohort studies
Wang, Lihua MS; Wang, Liangliang MS*; Zhang, Jing MS; Wang, Beibei MS; Liu, Hongli MS
Editor(s): Chang., Jongwha
Medicine: April 2017 - Volume 96 - Issue 16 - p e6396
doi: 10.1097/MD.0000000000006396

Abstract

Epidemiologic studies have suggested that diabetes mellitus (DM) might be associated with the risk of ovarian cancer; however, the results have been inconsistent. This study aimed to determine the relationship between DM and the incidence of ovarian cancer on the basis of cohort studies.

Relevant studies from PubMed, Embase, and the Cochrane Library until September 2016 were collected. The summary risk ratio (RR) was used as the effect measure in a random-effects model. The subgroup analysis indicated a higher incidence of ovarian cancer in patients with DM in studies published after 2010, studies not conducted in Europe or the United States, studies that did not adjust for body mass index or smoking status, and studies with lower Newcastle–Ottawa Scale scores.

The present findings indicated that DM is a risk factor for ovarian cancer, and future large-scale epidemiologic studies should be performed to evaluate this relation in specific populations.

Introduction

Ovarian cancer is the 5th leading cause of death among malignancies and accounted for approximately 240,000 cases and 150,000 deaths in 2012 worldwide.[1] Nearly 2/3 of cases are diagnosed in their advanced stages or unstaged and just 30% for 5-year survival rate for these patients.[2,3] The survival rates are poor, owing to the lack of effective screening strategies.[4] This emphasizes the need to focus on identifying risk factors, to reduce the risk of ovarian cancer. Diabetes mellitus (DM) is a growing global pandemic affecting approximately 3.0% to 4.0% of adults worldwide.[10] Systematic reviews and meta-analyses have already evaluated the risk of cancer incidence at different sites.[11–15] A previous study indicated that women with DM have a moderately increased risk of developing ovarian cancer.[16] However, traditional case-control studies were included in the previous study, which is less strong than cohort studies, and the findings of stratified analyses were affected by differences in study design. Furthermore, whether this relation differs according to the characteristics of participants remains unclear.

Several prospective cohort studies that explored the relationship between DM and ovarian cancer risk have already been published.[17–29] Several studies suggested that DM is associated with an elevated risk of ovarian cancer,[19,28] whereas other studies showed no evidence for this association.[17,18,20–27,29] Clarifying any potential correlation between DM and ovarian cancer, which has not been definitively established, is particularly important in the general population. We, therefore, attempted a comprehensive examination of the available cohort studies to determine the association between DM and the incidence of ovarian cancer.

Conclusions

The present meta-analysis demonstrated that women with DM had an increased risk of developing ovarian cancer, especially among Asians. To lower the risk of ovarian cancer, any potential risk factors need to be investigated, to allow early diagnosis and treatment. Additional epidemiologic studies about this relationship in specific populations need to be further conducted.

(7) Association of Lipid Metabolism with Ovarian Cancer

M. Tania, MS, M.A. Khan, MS, and Y. Song, PhD

Abstract

Defects in lipid metabolism are linked to several diseases, among which atherosclerosis, hypertension, obesity, and diabetes are the most important. Although cancer is chiefly a genetic disease, dietary lipid intake and metabolism are related to some cancer risks, including the risk for ovarian cancer. Higher intake of dietary lipids, systemic lipid metabolism malfunction, and abnormal serum lipid levels are somehow related to ovarian cancer. Over expression of some lipid metabolic enzymes are also found in ovarian cancer. In this review article, we summarize the relationships between lipid intake, lipid metabolism, and ovarian cancer.

1. INTRODUCTION

Lipids are the major macromolecules essential for various biologic functions, including energy production, signaling, and cell growth and division. Defects in lipid metabolism are associated with several diseases, among which atherosclerosis, hypertension, obesity, diabetes, and cancer are the most important 1. Among the factors that contribute to the appearance of cancer, diet has a fundamental role, and lipids are the main components that have been related to increases in the incidence of cancerous diseases, particularly breast, colorectal, ovarian, and prostate cancers 2. Data from animal studies have shown that some lipids have different effects on cancer risk than do others; for example, the omega-6 family of unsaturated fatty acids enhances tumour growth, whereas the omega-3 family delays or reduces tumour development 3.

Cholesterol in tissue and blood has consistently been found to have a prime role in the pathogenesis of coronary artery disease, but an association of cholesterol with cancers such as colorectal and breast cancer and leukemia has also been reported 4,5. The cholesterol content of cell membranes is tightly regulated, and this process of regulation involves the uptake of cholesterol-rich low-density lipoprotein (ldl). However, interestingly, cholesterol accumulation has been reported in various solid tumours, especially oral and prostate cancers 6,7. In addition, cholesterol metabolism is dysregulated in many malignancies, including myeloid leukemia and lung and breast cancers 7–10. Although a high level of serum triglycerides (tgs) does not appear to be mechanically involved in the development of most cancers, reduction of serum tgs and intensive surveillance with total colonoscopy in colon cancer may have benefits in men with hypertriglyceridemia 11.

Ovarian cancer is a neoplastic growth arising from various parts of the ovary, mainly the outer lining and the Fallopian tube. Ovarian cancer is the fifth leading cause of death from cancer in women and the leading cause of gynecologic cancer death 19. Dietary lipids, malfunctions of systemic lipid metabolism, and abnormal serum lipid profiles are all associated with ovarian cancer in multiple ways.

2. DIETARY LIPIDS AND THEIR METABOLISM IN OVARIAN CANCER

Several case-control and cohort studies have found positive associations between ovarian cancer and an intake of foods with high levels of saturated fats or cholesterol, such as red meat, eggs, and dairy products 20–22. Pan et al. 23 reported that ovarian cancer risk is positively associated with higher consumption of dietary cholesterol and eggs, and inversely associated with a higher intake of vegetables overall and cruciferous vegetables and with supplementation of vitamin E, beta-carotene, and vitamin B complex. High consumption of fats may increase circulating estrogen levels, thus increasing the possibility of cell damage and proliferation that is responsible for cancerous growth 24. Risch et al. 19 suggested that dietary cholesterol may influence the risk of ovarian cancer through elevated circulating estrogen or progesterone. The repeated

rupture of the follicle associated with ovulation is believed to expose the ovarian epithelium to hormones in the surrounding fluid; high estrogen concentrations may increase the likelihood of tumour development 25. However, Bertone et al. 24 found that the association of fat-rich food intake and ovarian cancer risk was not significant, although an increase in risk with frequent intake of eggs was observed. A weakly positive, but nonlinear association was observed for saturated fat intake and ovarian cancer risk in an Italian case–control study 26 in which intake of monounsaturated and polyunsaturated fatty acids was inversely correlated with ovarian cancer.

Numerous investigations have demonstrated altered systemic lipid metabolism in cancer patients and aberrant lipid utilization by tumour cells. The most common measure of altered systemic lipid metabolism in these individuals is hyperlipidemia. In a study by Taylor et al. 27 of peritoneal fluid from ovarian cancer patients and control subjects, isolation and analysis of lipids revealed four consistently altered lipid parameters in the cancer patients: elevated monoglycerides, diglycerides, and free fatty acids, and decreased triacylglycerides. Memon et al. 28 found an inverse relationship between total serum cholesterol and increased incidence of ovarian tumours in pre- and postmenopausal Pakistani women. In another study 29, oxidized ldl in serum was found to be higher in breast and ovarian cancer patients than in control subjects, but total cholesterol and high-density lipoprotein showed no such association. One nested case–control study in the United States found that women with a higher serum cholesterol level had an increased risk of ovarian cancer as compared with women who had a lower cholesterol level 30. A study by Pirozzo et al. 31 reported that cholesterol from eggs was associated with an increased risk of ovarian cancer, but that cholesterol from other sources was not. Those authors suggested that the association was not with the cholesterol in the eggs, speculating that it could be with the highly lipophilic organochlorine residues. However, an involvement of ldl with cancer was also supported by a recent study in which phytosterol and stanol consumption reduced blood levels of ldl cholesterol and lowered not only cancer risk but also cardiovascular disease risk 32.

A clear correlation between tg metabolism and ovarian cancer has not yet been reported. Li et al. 33 reported elevated tg levels (32%) in ovarian cancer patients, but previously, Ostroumova et al. 34 reported lower tg levels.

4. SUMMARY

As is the case for most complicated diseases, lipid intake and defective lipid metabolism are somehow involved in cancer. Ovarian cancer is the most dangerous disease of the female reproductive system, and so understanding its pathophysiology and therapeutics is a major concern. More and more research is being focused in this field to evaluate the appropriate dietary status of lipids, to manipulate lipid metabolism in the quest for a better life without ovarian cancer, and to discover new therapeutic strategies. More specifically, fas, lpa, and atx should receive closer scrutiny. Targeting fas, lpas, lpa receptors, and the enzymes of lpa metabolism may be the future of successful cancer therapy—especially atx, which may be a novel target for cancer therapy because blockage of this enzyme inhibits lpa production."

From above-quoted seven papers, diabetes has been identified either directly related to or inconclusive about its relationship with ovarian and uterine cancers; however, obesity, type 2 diabetes (T2D), and lipids have been mentioned as known possible risk factors. As we know, diet and exercise are directly related to obesity, T2D, and hyperlipidemia; therefore, the author can safely draw a picture which includes several "modifiable influential factors of these two gynecological cancers" with their respective 5 weighting factors as depicted below:

Obesity or being overweight: 30%
Diabetes (HbA1C): 25%
Lipids (combined LDL, HDL, TG): 15%
Diet including both quantity and quality of food and meals: 15%
Daily walking exercise: 15%

As a result, his defined relative ovarian and uterine cancer risk can be expressed as below:

Relative ovarian and uterine cancer risk %
= obesity * 30% + T2D * 25% + lipids * 15% + diet *15% + exercise * 15%

The author's stringent lifestyle management efforts during the past 12+ years, including both diet and exercise, are directly beneficial to his weight reduction, glucose control, and metabolism improvements. It is necessary to provide a brief description of his health history. His historic data can very well be happening to any female patients since those 5 risk factors are gender-independent. The important objective from using his generic data is to prove and quantify the relationship between these risk factors and gynecological cancers.

The author was diagnosed with T2D in 1997 with a random glucose check at a 300 mg/dL level; however, his T2D condition most likely began earlier. He suffered his first two chest pain episodes in 1993-1994 and three more heart episodes until 2007. His primary physician informed him that he had diabetic kidney issues in 2010. He then consulted with two more clinical doctors who advised him to immediately start insulin injections and kidney dialysis. This was his wake-up call. He then decided to save his life by conducting his study and research on food nutrition and chronic diseases that same year. His health profile in 2010 was: body weight at 220 lbs. (BMI 32), average glucose at 280 mg/dL, fasting plasma glucose (FPG) in the early morning at 180 mg/dL, lab-tested HbA1C at 10%, triglycerides at 1160 mg/dL (target: <150 mg/dL), and his ACR at 116 (target: <30). In addition, by 2010, he has also suffered a total of 5 heart episodes, foot ulcer, hypothyroidism, diabetic retinopathy, etc.

Over the past 13 years, he has made significant lifestyle changes. For example, he consumes less than 20 grams of carbohydrates and sugar per meal, stops eating processed food, reduces his food quantity by 50%, walks 6-7 miles or 10-11 kilometers daily, sleeps 7-8 hours each night, and avoids stress as much as possible. As a matter of fact, he has never drunk alcohol, smoked cigarettes, or used any illicit drugs in his life.

As of April 10, 2022, his health profile for the first 3 months of 2022 was: body weight at 169 lbs. (BMI 24.95), daily average glucose at 106 mg/dL, FPG in the early morning at 94 mg/dL, lab-tested A1C at 5.8%, triglycerides at 108, and ACR at 16. A significant accomplishment since he discontinued taking 3 different kinds of diabetes medications on 12/8/2015.

2. METHODS

To offer a simple explanation to readers who do not have a physics or engineering background, the author includes a brief excerpt from Wikipedia regarding the description of basic concepts for elasticity and plasticity theories, viscoelasticity, and viscoplasticity theories from the disciplines of engineering and physics, and his developed metabolism index (MI) Model in this method section.

2.1 Relationships between biomedical causes and biomedical symptoms

As a mathematician/engineer over 40 years and now conducting his medical research work for the past 13 years, the author has discovered that people frequently seek answers, illustrations, or explanations for the relationships between the input variable (force applied on a structure or cause of a disease) and output variable (deformation of a structure or symptom of a disease). However, the multiple relationships between input and output could be expressed with many different matrix formats of 1 x 1, 1 x n, m x 1, or m x n (m or n means different multiple variables). In addition to these described mathematical complications, the output resulting from one or more inputs can also become an input of another output, which is a symptom of certain causes that can become a cause of another different symptom. This phenomenon is indeed a complex scenario with "chain effects". In fact, both engineering and biomedical complications are fundamentally mathematical problems that correlate or conform with many inherent physical laws or principles. In his medical research work, he has encountered more than 100 different sets of biomarkers with almost equal or more amounts of causes (or input variables) and symptoms (or output variables).

Since December of 2021, the author applied theories of viscoelasticity and viscoplasticity (VGT) from physics and engineering disciplines to investigate more than 60 sets of input/output biomarkers, including nearly 10 sets of cancer cases. The purpose is to identify certain hidden relationships between certain output biomarkers, such as cancer risk, and

its corresponding multiple inputs, such as glucose, blood pressure, blood lipids, obesity or overweight, and metabolism index of 6 lifestyle details and 4 chronic diseases. In this study, the hidden biophysical behaviors and possible inter-relationships among the output symptom and multiple input causes are "time-dependent" and change from time to time. These important time-dependency characteristics provide insight into the cancer risk's moving pattern. It also controls the cancer risk curve shape, the associated energy created, stored, or burned inside during the process of stress up-loading (moving upward or increasing) and stress down-loading (moving downward or decreasing) of the input biomarkers with the output biomarker of cancer risk %. This VGT application emphasizes the time-dependency characteristics of involved variables. In the medical field, most biomarkers are time-dependent since body organ cells are organic in nature and change all of the time. Incidentally, VGT can generate a stress-strain curve or cause-symptom curve, known as a "hysteresis loop" in physics, in which area size can also be used to estimate the relative energy created, stored, or burned during the process of uploading (e.g., increasing glucose) and unloading (e.g., decreasing body weight) over the timespan of the cancer risk %. He calls this relative energy the "VGT energy".

It should be emphasized here that both cancer risk % and its associated VGT energy are estimated relative values, not "absolute" values.

The following defined stress and strain equations are used to establish the VGT stress-strain diagram in a space domain (SD):

VGT strain
= ε (symptom)
= individual symptom at the present time

VGT stress
= σ (based on the change rate of strain, symptom, multiplying with one or more viscosity factors or influential factors)
= η * (dε/dt)
= η * (d-strain/d-time)
= (viscosity factor η using normalized factor at present time) * (symptom at present time - symptom at a previous time)

Where the strain is the cancer risk percentage and the stress is his cancer risk change rate multiplied by several chosen input biomarkers as the individual viscosity factor. In his VGT studies, sometimes, he carefully selects certain normalization factors for each input biomarker, respectively. The normalization factors are the dividing lines between a healthy state and an unhealthy state. For example, 170 lbs. for body weight, 6.0 for HbA1C, 120 mg/dL for glucose, 180 mg/dL for hyperglycemia, 73.5% for overall MI score, and 10,000 steps for daily walking exercise, etc.

2.2 Elasticity, plasticity, viscoelasticity, and viscoplasticity (LEGT & VGT)

The Difference Between Elastic Materials and Viscoelastic Materials (from "Soborthans, innovating shock and vibration solutions").

What are elastic materials?

Elasticity is the tendency of solid materials to return to their original shape after forces are applied on them. When the forces are removed, the object will return to its initial shape and size if the material is elastic.

Medical analogy: The medical application is when cause or risk factors are reduced or removed, the symptoms of certain disease would be improved or ceased.

What are viscous materials?

Viscosity is a measure of a fluid's resistance to flow. A fluid with large viscosity resists motion. A fluid with low viscosity flows. For example, water flows more easily than syrup because it has a lower viscosity. High viscosity materials might include honey, syrups, or gels – generally things that resist flow. Water is a low viscosity material, as it flows readily. Viscous materials are thick or sticky or adhesive. Since heating reduces viscosity, these materials don't flow easily. For example, warm syrup flows more easily than cold.

What is viscoelastic?

Viscoelasticity is the property of materials that exhibit both viscous and elastic characteristics when undergoing deformation. Synthetic polymers, wood, and

human tissue, as well as metals at high temperature, display significant viscoelastic effects. In some applications, even a small viscoelastic response can be significant.

Medical analogy: Viscoelastic behavior means material has "time-dependent" characters. Biomedical data, i.e. biomarkers, are time-dependent due to body cells are organic which changes with time constantly.

Elastic behavior versus viscoelastic behavior

The difference between elastic materials and viscoelastic materials is that viscoelastic materials have a viscosity factor and the elastic ones don't. Because viscoelastic materials have the viscosity factor, they have a strain rate dependent on time. Purely elastic materials do not dissipate energy (heat) when a load is applied, then removed; however, a viscoelastic substance does.

Medical analogy: Most of the biomarkers display time-dependency; therefore they have both change-rate of time and viscosity factor behaviors. Viscoelastic biomarkers do dissipate energy when a cause force is applied on it.

The following brief introductions are excerpts from Wikipedia:

"Elasticity (physics):

The physical property is when materials or objects return to their original shape after deformation.

In physics and materials science, elasticity is the ability of a body to resist a distorting influence and to return to its original size and shape when that influence or force is removed. Solid objects will deform when adequate loads are applied to them; if the material is elastic, the object will return to its initial shape and size after removal. This is in contrast to plasticity, in which the object fails to do so and instead remains in its deformed state.

Hooke's law states that the force required to deform elastic objects should be directly proportional to the distance of deformation, regardless of how large that distance becomes. This is known as perfect elasticity, in which a given object will return to its original shape no matter how strongly it is deformed. This is an ideal concept only; most materials that possess elasticity in practice remain purely elastic only up to very small deformations, after which plastic (permanent) deformation occurs.

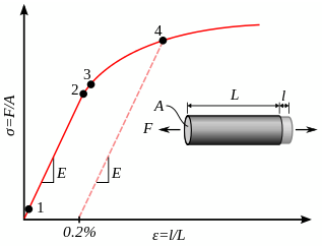
In engineering, the elasticity of a material is quantified by the elastic modulus such as the Young's modulus, bulk modulus or shear modulus which measure the amount of stress needed to achieve a unit of strain; a higher modulus indicates that the material is harder to deform. The material's elastic limit or yield strength is the maximum stress that can arise before the onset of plastic deformation.

Medical analogy: The elastic behavior analogy in medicine can be expressed by the metal rod analogy for the postprandial plasma glucose (PPG). Consuming carbohydrates and/or sugar acts like a tensile force to stretch a metal rod longer, while post-meal exercise acts like a compressive force to suppress a metal rod shorter. If lacking food consumption and exercise, the metal rod (analogy of PPG) will remain its original length, for a non-diabetes or less severe type 2 diabetes (T2D) patient.

Plasticity (physics):

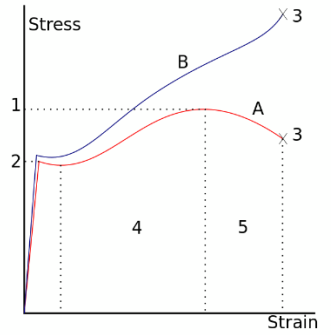
Deformation of a solid material undergoing non-reversible changes of shape in response to applied forces.

In physics and materials science, plasticity, also known as plastic deformation, is the ability of a solid material to undergo permanent deformation, a non-reversible change of shape in response to applied forces. For example, a solid piece of metal being bent or pounded into a new shape displays plasticity as permanent changes occur within the material itself. In engineering, the transition from elastic behavior to plastic behavior is known as yielding. Plastic deformation is observed in most materials, particularly metals, soils, rocks, concrete, and foams.



Stress-strain curve showing typical yield behavior for nonferrous alloys.

1. True elastic limit
2. Proportionality limit
3. Elastic limit
4. Offset yield strength



A stress-strain curve typical of structural steel.

- 1: Ultimate strength
- 2: Yield strength (yield point)
- 3: Rupture
- 4: Strain hardening region
- 5: Necking region
- A: Apparent stress (F/A0)
- B: Actual stress (F/A)

For many ductile metals, tensile loading applied to a sample will cause it to behave in an elastic manner. Each increment of load is accompanied by a proportional increment in extension. When the load is removed, the piece returns to its original size. However, once the load exceeds a threshold – the yield strength – the extension increases more rapidly than in the elastic region; now when the load is removed, some degree of extension will remain.

Medical analogy: A plastic behavior analogy in medicine is the PPG level of a severe T2D patient. Even consuming a smaller amount of carbs/sugar, the patient's PPG will rise sharply which cannot be totally brought down to a healthy PPG level even with a significant amount of exercise. This means the PPG level has exceeded its "elastic limit" and entering into a "plastic range".

Viscoelasticity:

Property of materials with both viscous and elastic characteristics under deformation.

In materials science and continuum mechanics, viscoelasticity is the property of materials that exhibit both viscous and elastic characteristics when undergoing deformation. Viscous materials, like water, resist shear flow and strain linearly with time when a stress is applied. Elastic materials strain when stretched and immediately return to their original state once the stress is removed.

Viscoelastic materials have elements of both of these properties and, as such, exhibit time-dependent strain. Whereas elasticity is usually the result of bond stretching along crystallographic planes in an ordered solid, viscosity is the result of the diffusion of atoms or molecules inside an amorphous material.

In the nineteenth century, physicists such as Maxwell, Boltzmann, and Kelvin researched and experimented with creep and recovery of glasses, metals, and rubbers. Viscoelasticity was further examined in the late twentieth century when synthetic polymers were engineered and used in a variety of applications. Viscoelasticity calculations depend heavily on the viscosity variable, η. The inverse of η is also known as fluidity, φ. The value of either can be derived as a function of temperature or as a given value (i.e. for a dashpot).

Depending on the change of strain rate versus stress inside a material, the viscosity can be categorized as having a linear, non-linear, or plastic response. In addition, when the stress is independent of this strain rate, the material exhibits plastic deformation. Many viscoelastic materials exhibit rubber-like behaviors explained by the thermodynamic theory of polymer elasticity.

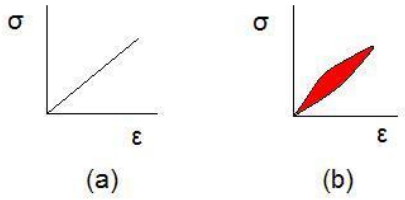
Cracking occurs when the strain is applied quickly and outside of the elastic limit. Ligaments and tendons are viscoelastic, so the extent of the potential damage to them depends both on the rate of the change of their length as well as on the force applied.

A viscoelastic material has the following properties:

- hysteresis is seen in the stress-strain curve
- stress relaxation occurs: step constant strain causes decreasing stress

- creep occurs: step constant stress causes increasing strain
- its stiffness depends on the strain rate or the stress rate.

Elastic versus viscoelastic behavior:



Stress-strain curves for a purely elastic material (a) and a viscoelastic material (b). The red area is a hysteresis loop and shows the amount of energy lost (as heat) in a loading and unloading cycle. It is equal to $\oint \sigma d\varepsilon$ where σ is stress and ε is strain. In other words, the hysteresis loop area represents the amount of energy during the loading and unloading process.

Unlike purely elastic substances, a viscoelastic substance has an elastic component and a viscous component. The viscosity of a viscoelastic substance gives the substance a strain rate dependence on time. Purely elastic materials do not dissipate energy (heat) when a load is applied, then removed. However, a viscoelastic substance dissipates energy when a load is applied, then removed. Hysteresis is observed in the stress-strain curve, with the area of the loop being equal to the energy lost during the loading cycle. Since viscosity is the resistance to thermally activated plastic deformation, a viscous material will lose energy through a loading cycle. Plastic deformation results in lost energy, which is uncharacteristic of a purely elastic material's reaction to a loading cycle.

Viscoplasticity:

Viscoplasticity is a theory in continuum mechanics that describes the rate-dependent inelastic behavior of solids. Rate-dependence in this context means that the deformation of the material depends on the rate at which loads are applied. The inelastic behavior that is the subject of viscoplasticity is plastic deformation which means that the material undergoes unrecoverable deformations when a load level is reached. Rate-dependent plasticity is important for transient plasticity calculations. The main difference between rate-independent plastic and viscoplastic material models is that the latter exhibit not only permanent deformations after the application of loads but continue to undergo a creep flow as a function of time under the influence of the applied load.

Medical analogy: In viscoelastic or viscoplastic analysis, the stress component equals the strain change rate of time multiplying with the viscosity factor, or

Stress (σ)
= strain (ε) change rate * viscosity factor (η)
= $d\varepsilon/dt * \eta$

The hysteresis loop area
= the integrated area of stress (σ) and strain (ε) curve
= $\oint \sigma d\varepsilon$

2.3 Metabolism index (MI) model

This model was developed in Y2014 by the author using the topology concept, nonlinear algebra, geometric algebra, and engineering finite element method. In summary, the human body metabolism is a complex mathematical problem with a matrix format of m causes by n symptoms, plus sometimes, one symptom or many symptoms would be turned into causes of another symptom.

This MI model contains 10 specific categories, including 4 output categories of medical conditions (body weight, glucose, blood pressure, and lipids), and 6 input categories of lifestyle details (food quantity and quality, drinking water intake, physical exercise, sleep, stress, and daily life routines). These 10 categories are comprised of approximately 500 detailed elements. He has also defined two new resulting parameters: the metabolism index or MI, as the combined score of the above 10 metabolism categories and 500 elements using his developed algorithm, along with the general health status unit (GHSU), as the 90-day moving average value of MI.

A physical analogy of this complex mathematical metabolism model is similar to "using multiple nails that are encircled by many rubber bands". For example, at first, we hammer 10 nails into a piece of flat wood with an initial shape of a circle, then take 3,628,800 (=10!) rubber bands to encircle the

nails, including all 10 nails. These ~3.6 million rubber bands (i.e. big number of relationships) indicate the possible relationships existing among these 10 nails (i.e. 10 original metabolism data). Some rubber bands encircle 2 nails or 3 nails and so on until the last rubber band encircles all of these 10 nails together (no rubber band to encircle a single nail is allowed). Now, if we move any one of the nails outward (i.e., moving away from the center of the nail circle), then this moving action would create some internal tension inside the encircled rubber band. Moving one nail "outward" means one of these ten metabolism categories is becoming "unhealthy" which would cause some stress to our body. Of course, we can also move some or all of the 10 nails outward at the same time, but with different moving scales. If we can measure the summation of the internal tension created in the affected rubber bands, then this summarized tension force is equivalent to the metabolism value of human health. The higher tension means a higher metabolism value which creates an unhealthy situation. The author uses the above-described scenario of moving nails and their encircled rubber bands to explain his developed mathematical metabolism model of human health.

During 2010 and 2011, the author collected sparse biomarker data, but from the beginning of 2012, he has been gathering his body weight and finger-piercing glucose values each day. More complete data collection started in Y2015. In addition, he accumulates medical conditions data including BP, heart rate (HR), and blood lipids along with lifestyle details (LD). Since 2020, he has added the daily body temperature (BT) and blood oxygen level (SPO2) due to his concerns about being exposed to COVID-19. Based on the collected big data of biomarkers, he further organized them into two main groups. The first is the medical conditions group (MC) with 4 categories: weight, glucose, BP, and blood lipids. The second is the lifestyle details group (LD) with 6 categories: food & diet, exercise, water intake, sleep, stress, and daily routines. At first, he calculated a unique combined daily score for each of the 10 categories within the MC and LD groups. The combined scores of the 2 groups, 10 categories, and 500+ detailed elements constitute an overall "metabolism index (MI) model". It includes the root-causes of 6 major lifestyle inputs and symptoms from 4 lifestyle-induced rudimentary chronic diseases, i.e. obesity, diabetes, hypertension, and hyperlipidemia. Therefore, the MI model, especially its 4 chronic disease conditions, can be used as the foundation and building block for his additional research work that can expand into various complications associated with different organs, such as cancer.

Of course, the same methodology can be extended to the study of many other medical complications, such as various heart problems (CVD & CHD), stroke, neuropathy, hypothyroidism, diabetic constipation, diabetic skin fungal infection, various cancers, and dementia.

In general, some genetic conditions and lifetime unhealthy habits, which include tobacco smoking, alcohol drinking, and illicit drug use, account for approximately 15% to 25% of the root-cause of chronic diseases and their complications, as well as cancers and dementia.

His calculated risk probability % for CKD, CVD, DR, stroke, and various cancers have some differences in their root-cause variables, their associated weighting factors for each key cause, and certain biomedical interpretations and assumptions. Specifically, the CVD/Stroke risk includes two major scenarios that combine emphasized weighting factors, blood vessel blockage due to blood glucose and blood lipids, and blood vessel rupture caused by blood glucose and blood pressure. Some recent research work have identified the relationship between pancreatic cancer with hyperglycemia and insulin resistance phenomena of type 2 diabetes, and chronic inflammation. Some aggressive prostate cancers are linked with 5 types of bacteria. There is also an evidence of a relationship between BP and DR (Reference: BP control and DR, by R. Klein and BEK Klein from British Journal of Ophthalmology). The CKD risks include hyperglycemic damage to micro-blood vessels and nerves which causes protein leakage found in urine and waste deposit within the kidneys; therefore, it requires dialysis to remove waste products and excess fluids from the body. However, the cancer risk also consists of additional influences from environmental conditions, such as some improper medications, viral

infections, food pollution or poison, toxic chemical, radiation, air and water pollution, hormonal treatment, etc.

All of the above-mentioned diseases fall into the category of "symptoms" which are the outcomes of "root-causes" of genetic conditions, unhealthy lifestyles, and poor living environments.

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 two stress-strain diagrams of ovarian and uterine cancer risk % with 5 hysteresis loops via a VGT energy analysis in a space domain.

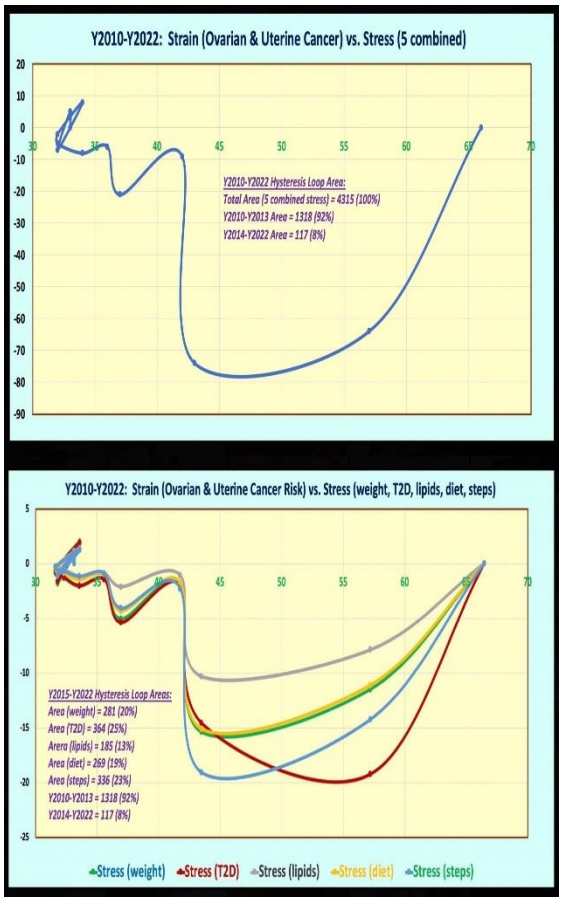
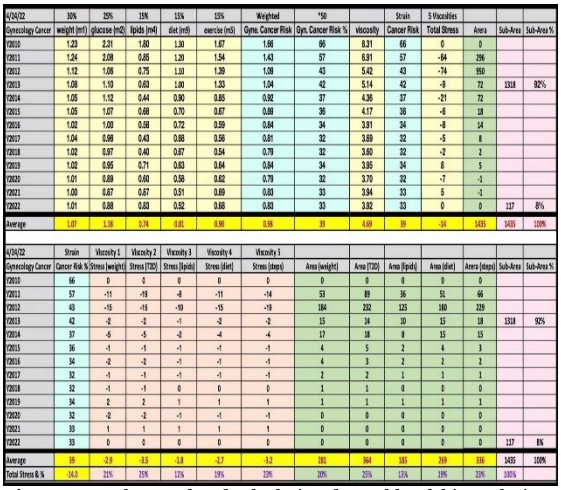


Figure 1: Two stress-strain diagrams of ovarian and uterine cancers risk % with 5 hysteresis loops via VGT analysis in a space-domain.

Figure 2 illustrates the background data and calculation data of this analysis.



4/24/22	30%	25%	15%	15%	15%	Weighted	*50		Strain	5 Viscosities			
Gynecology Cancer	weight (mt)	glucose (m2)	lipids (m4)	diet (m3)	exercise (m5)	Gyne. Cancer Risk	Gyn. Cancer Risk %	viscosity	Cancer Risk	Total Stress	Arera	Sub-Area	Sub-Area %
Y2010	1.23	2.21	1.60	1.30	1.67	1.66	66	8.31	66	0	0		
Y2011	1.24	2.08	0.85	1.20	1.54	1.43	57	6.91	57	-64	296		
Y2012	1.12	1.06	0.75	1.10	1.39	1.09	43	5.42	43	-74	950		
Y2013	1.08	1.10	0.63	1.00	1.33	1.04	42	5.14	42	-9	72	1318	92%
Y2014	1.05	1.12	0.44	0.90	0.85	0.92	37	4.36	37	-21	72		
Y2015	1.05	1.07	0.68	0.70	0.67	0.89	36	4.17	36	-8	18		
Y2016	1.02	1.00	0.58	0.72	0.59	0.84	34	3.91	34	-8	14		
Y2017	1.04	0.99	0.43	0.66	0.59	0.81	32	3.69	32	-5	8		
Y2018	1.02	0.97	0.40	0.67	0.54	0.79	32	3.60	32	-2	2		
Y2019	1.02	0.95	0.71	0.63	0.64	0.84	34	3.95	34	8	5		
Y2020	1.01	0.89	0.60	0.58	0.62	0.79	32	3.70	32	-7	-1		
Y2021	1.00	0.87	0.67	0.51	0.69	0.83	33	3.84	33	5	-1		
Y2022	1.01	0.88	0.63	0.52	0.68	0.83	33	3.92	33	0	0	117	8%
Average	1.07	1.38	0.74	0.81	0.90	0.98	39	4.69	39	-34	1435	1435	100%

4/24/22	Strain	Viscosity 1	Viscosity 2	Viscosity 3	Viscosity 4	Viscosity 5							
Gynecology Cancer	Cancer Risk %	Stress (weight)	Stress (T2D)	Stress (lipids)	Stress (diet)	Stress (steps)	Area (weight)	Area (T2D)	Area (lipids)	Area (diet)	Arera (steps)	Sub-Area	Sub-Area %
Y2010	66	0	0	0	0	0	0	0	0	0	0		
Y2011	57	-11	-19	-8	-11	-14	53	89	36	51	66		
Y2012	43	-15	-15	-10	-15	-19	184	232	125	180	229		
Y2013	42	-2	-2	-1	-2	-2	15	14	10	15	18	1318	92%
Y2014	37	-5	-5	-2	-4	-4	17	18	8	15	15		
Y2015	36	-1	-1	-1	-1	-1	4	5	2	4	3		
Y2016	34	-2	-2	-1	-1	-1	4	3	2	2	2		
Y2017	32	-1	-1	-1	-1	-1	2	2	1	1	1		
Y2018	32	-1	-1	0	0	0	1	1	0	0	0		
Y2019	34	2	2	1	1	1	1	1	1	1	1		
Y2020	32	-2	-2	-1	-1	-1	0	0	0	0	0		
Y2021	33	1	1	1	1	1	0	0	0	0	0		
Y2022	33	0	0	0	0	0	0	0	0	0	0	117	8%
Average	39	-2.9	-3.5	-1.8	-2.7	-3.2	281	364	185	269	336	1435	100%
Total Stress & %	-74.0	21%	25%	12%	19%	23%	20%	25%	13%	19%	23%	100%	

Figure 2: Background and calculation data table of this analysis.

4. CONCLUSION

The following four described biophysical characteristics have demonstrated certain biomedical behaviors of the hypothetical female patient's gynecological cancers (ovarian and uterine) risk probability under 5 specific risk factors, using the viscoelastic or viscoplastic energy (VGT) approach:

(1) From the x-axis value or the strain value on the stress-strain diagram, the ovarian and uterine cancers' relative risk % covers a range from the high-end of 66% in 2010 to the low-end of 33% in 2022. These gynecological cancer risks are only relative numbers, not absolute numbers. Nevertheless, from an overview angle and on a relative scale, this hypothetical female patient's gynecological cancer risk has been reduced year after year due to improvements in these 5 risk factors. The VGT analysis can provide a quantitative feeling regarding the situation.

(2) From the y-axis (stress) values and the associated hysteresis loop areas, we can see that both the stress values and the hysteresis loop areas (i.e. energies) for the period of Y2010-Y2013 are much larger than the period of Y2014-Y2022 (actually, 11.5 times larger). This fact indicates that, from the perspective of gynecological cancer risk, she is "healthier" during the recent 8+ years than her earlier 4 years; therefore, her gynecological cancer risks have been reduced accordingly due to the improvements made on these 5 risk factors.

(3) When we delve deeper into the comparisons among these 5 risk factors, we can further identify some additional details

regarding these "efforts versus results or causes versus symptoms" from each influential factor. Examples of those detailed observations are: T2D glucose contributes the most with 25% of the influence on her gynecological cancer risk; the contributions from the other four risk factors are listed in the order of exercise steps with 23%, the body weight of obesity diet with 20%, diet quantity and quality with 19% and blood lipids with 13%.

(4) In the comparison of the stress-strain diagrams between 5 combined stresses versus 5 individual stresses, the overall curve shapes are similar and comparable with each other. However, the combined diagram provides a clearer overview while the 5 individual diagrams provide a more detailed view regarding each risk factor's contribution to gynecological cancer risk.

In summary, the unique "time-dependency" characteristic of strain change rate (i.e., gynecological cancer risk change amount over time) can be presented via a VGT tool.

This ovarian and uterine cancers risk article has demonstrated how the author utilizes the VGT energy methodology from physics and engineering to construct and display the research results of a hypothetical female patient's risk perspective of developing ovarian and uterine cancers resulting from five gender-independent and modifiable risk factors.

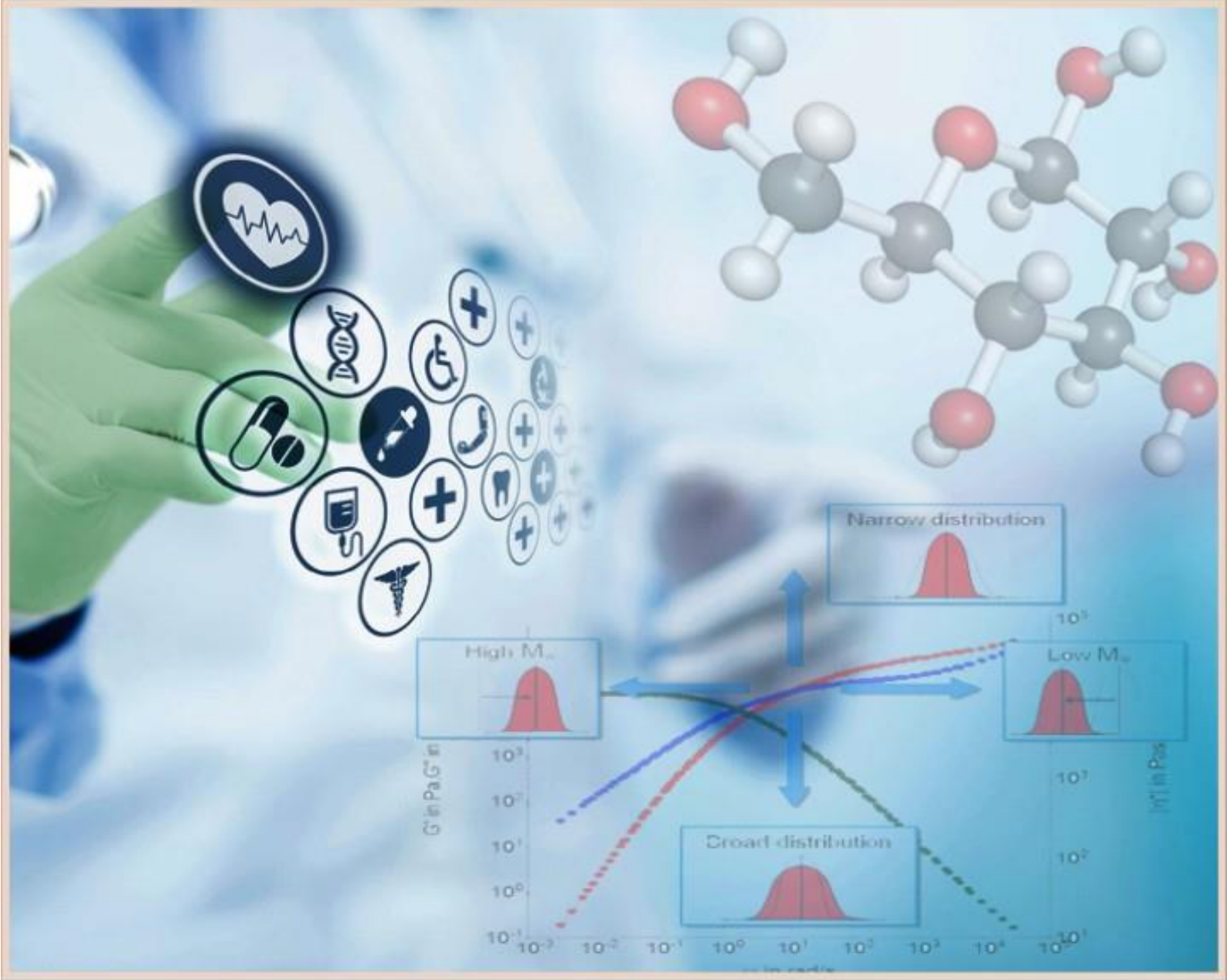
5. REFERENCES

For editing purposes, the majority of the references in this paper, which are self-references, have been removed. Only references from other authors' published sources remain. The bibliography of the author's original self-references can be viewed at www.eclairemd.com.

Special Issue | Issue No. 5 The GH-Method

Viscoelastic and Viscoplastic Glucose Theory Application in Medicine

Gerald C. Hsu



https://theghmethod.com

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