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Comparison of CVD/Stroke Risk Percentages Between the Pre-virus Period and Virus Period without the Glucose Fluctuations Factor Case versus with the Glucose Fluctuations Factor Case Using Three Different Energy Estimation Methods Based on GH-Method: Math-Physical Medicine (No. 432)

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Abstract

In this article, the author investigates the risk differences of having a CVD/stroke episode between the pre-virus period (623 days from 5/5/2018 to 1/18/2020) and the COVID-19 virus period (447 days from 1/18/2020 to 4/10/2021). Through the introduction of the glucose fluctuations (GF) influential factor into his developed risk assessment method, physical and engineering modeling, and mathematical equation to calculate the risk probability of having severe diabetic complications, especially for the macro-blood vessels (arteries) related diseases, such as stroke and cardiovascular diseases (CVD). In summary, by presenting the extra influential factor of GF associated energy via three different estimation methods into his existing CVD risk assessment model based on metabolism, his risk percentages between the pre-virus period and virus period are listed below in the format of pre-virus and virus: without GF: 56%, 51%, with GF-method 1: 60%, 56%, with GF-method 2: 62%, 54%, with GF-method 3: 62%, 54%. From the table above, we can draw the following conclusions: (1) Overall, the virus period has lower risk percentages than the pre-virus period (5% less for a case without GF and 4%-8% for three cases with GF). This means that the author's overall health conditions are better during the virus period. (2) Results from the GF-method 2 and GF-method 3 are similar due to the specific definitions for the two GF energy methods which are almost identical. The exceedingly small difference is a result of numerical truncations. (3) The GF-method 1 is based on the definition of GF energy where it is equal to the square of the GF magnitude. This is different from the other two methods which are derived from Fourier transform operation based on a time-domain (TD) function into a frequency domain (FD) function. Furthermore, basic physics says that the energy associated with a wave is directly proportional to the square of the wave amplitude (but not equal to the square of the wave amplitude). (4) In comparison between the case with GF vs. three cases without GF, the summarized risk percentage differences are listed below in the format of pre-virus and virus: method 1 vs. without: 4%, 5%, method 2 vs. without: 6%, 3%, method 3 vs. without: 6%, 3%. It is obvious that the GF brings in extra energies which damage internal organs, and therefore, increase the risks of having a CVD or stroke episode.

Keywords: Glucose fluctuations; Diabetes; Cardiovascular diseases; Stroke; Health

Abbreviations: GF: glucose fluctuations; CVD: cardiovascular diseases; TD: time-domain; FD: frequency domain; MPM: math-physical medicine; MI: metabolism index; GHSU: general health status unit; CKD: chronic kidney diseases; PPG: postprandial plasma glucose; HbA1C: hemoglobin A1c; CGM: continuous glucose monitoring; GV: glycemic variability; SMBG: self-monitored blood glucose; SD: standard deviation

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1. INTRODUCTION

In this article, the author investigates the risk differences of having a CVD/stroke episode between the pre-virus period (623 days from 5/5/2018 to 1/18/2020) and the COVID-19 virus period (447 days from 1/18/2020 to 4/10/2021). Through the introduction of the glucose fluctuations (GF) influential factor into his developed risk assessment method, physical and engineering modeling, and mathematical equation to calculate the risk probability of having severe diabetic complications, especially for the macro-blood vessels (arteries) related diseases, such as stroke and cardiovascular diseases (CVD).

2. METHODS

2.1 MPM background

To learn more about his developed GH-method: math-physical medicine (MPM) methodology, readers can read the following three papers selected from the published 400+ medical papers.

The first paper, No. 386 describes his MPM methodology in a general conceptual format[1]. The second paper, No. 387 outlines the history of his personalized diabetes research, various application tools, and the differences between the biochemical medicine (BCM) approach vs. the MPM approach[2]. The third paper, No. 397 depicts a general flow diagram containing ~10 key MPM research methods and different tools[3].

2.2 CVD/stroke risk model based on metabolism

In 2014, the author applied the topology concept, finite-element engineering technique, and non-linear algebra operations to develop a complex mathematical model of metabolism. This model contains 10 categories, including four output categories (weight, glucose, blood pressure, and lipids), and six input categories (food, water intake, exercise, sleep, stress, and routine life patterns). These 10 categories are comprised of approximately 500 detailed elements. He also defined two new parameters: metabolism index (MI), as the combined score of the above 10 metabolism categories and 500 elements along with the general health status unit (GHSU), as the 90-days moving average value of MI. Since 2012, he has collected more than 2 million data of his own biomedical conditions and personal lifestyle details.

Following the mathematical metabolism model, he further developed a series of models regarding diabetic complications which contain some detailed equations to predict his risk probabilities of having a stroke, CVD, chronic kidney diseases (CKD), and pancreatic beta-cells self-recovering assessment. These risk assessment models include a patient's baseline data including age, race, gender, family genetic history, medical history, and bad habits which contribute approximately 20% to the total risk. Furthermore, it also includes the following two major areas each with a 40% contribution:

(1) Medical conditions - individual M1 through M4 which include obesity, diabetes, hypertension, hyperlipidemia, and others. It should be emphasized here that diabetes (i.e., glucose) alone contributes about 20% of the total risk.
(2) Lifestyle details - individual M5 through M10 which affect medical conditions.

In addition, he also uses his defined two terms, MI and GHSU, as a combined score of M1 through M10 and 90-days moving average MI, for his calculation. Of course, all of these 10 metabolism factors (M1 through M10) are interrelated.

With this mathematical risk assessment model, he can obtain three separate risk probability percentages associated with each of the three calculations mentioned above. As a result, this model would offer a range of the risk probability predictions of having a CVD or stroke based on the patient's metabolic disorder conditions, unhealthy lifestyles, and the combined impact on the body.

2.3 The author's diabetes history

The author was a severe type 2 diabetes (T2DM) patient since 1996. 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 the albumin-

creatinine ratio (ACR) at 116 (high risk for CKD). 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. During 2015 and 2016, he developed four mathematical prediction models related to diabetes conditions: weight, postprandial plasma glucose (PPG), fasting plasma glucose (FPG), and HbA1C. By using his developed mathematical 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 had achieved excellent results on all fronts, especially his glucose control. However, during the pre-COVID period, including both 2018 and 2019, he traveled to ~50 international cities to attend 65+ medical conferences and made ~120 oral presentations. This hectic schedule inflicted damage to his diabetes control caused by stress, dining out frequently, post-meal exercise disruption, jet lag, along with the overall negative metabolic impact from the irregular life patterns; therefore, his glucose control was somewhat affected during the two-year traveling period of 2018-2019.

He started his self-quarantined life on 1/19/2020. By now, 4/10/2021, his weight was further reduced to ~165 lbs. (BMI 24.4) and his A1C was at 6.2% without any medication intervention or insulin injection. In fact, with the special COVID-19 quarantine lifestyle since early 2020, not only has he written 200 new research articles and published a total of 400 medical papers in various medical and engineering journals, but he has also achieved his best health conditions for the past 26 years. These achievements have resulted from his non-traveling, low-stress, and regular daily life routines. Of course, his in-depth knowledge of chronic diseases, sufficient practical lifestyle management experiences, and his own developed high-tech tools have also contributed to his excellent health improvements.

On 5/5/2018, he applied a continuous glucose monitoring (CGM) sensor device on his upper arm and checks his glucose measurements every 5 minutes for a total of 288 times each day. He has maintained the same measurement pattern to the present day.

During the past 11 years, he has continuously investigated, studied, and analyzed his collected ~2 million data regarding his health status, medical conditions, and lifestyle details. He applies his physics knowledge, engineering models, mathematical tools, and computer programming to conduct his medical research work. His entire medical research work is based on the aim of achieving high precision with quantitative proof in the biomedical findings, not just through linguistic expressions with qualitative words, vague statements, or complex medical terminologies.

His personal goal is aimed at saving his own life through research, and then helping family members and other patients through distributing his learned knowledge and experiences gained from his 11-years of medical research work to combat these chronic diseases and complications at the root cause level.

2.4 Other GV research work

There are many available articles regarding the subject of glycemic variability (GV), however, the author decides to include the following combined excerpt from two particular published articles[3-5]. These two references have cited a total of 114 published papers. In this way, readers do not have to search for key information from a long list of their cited reference articles. These papers focus on the comparison of many published GV articles and algorithm, method, and firmware design of a web-based app software for calculating GV values[3,4].

Here is the combined excerpt:

"Several pathophysiological mechanisms were reported, unifying the two primary mechanisms: excessive protein glycation end products and activation of oxidative stress, which causes vascular complications. Intermittent high blood glucose exposure,

rather than constant exposure to high blood glucose, has been shown to have deleterious effects in experimental studies. In the in-vitro experimental settings and in animal studies, glycemic fluctuations display a more deleterious effect on the parameters of CV risk, such as endothelial dysfunction. There is a significant association between GV and the increased incidence of hypoglycemia. Hypoglycemic events may trigger inflammation by inducing the release of inflammatory cytokines. Hypoglycemia also induces increased platelet and neutrophil activation. The sympathoadrenal response during hypoglycemia increases adrenaline secretion and may induce arrhythmias and increase the cardiac workload. Underlying endothelial dysfunction leading to decreased vasodilation may contribute to CV risk. Published studies have demonstrated that GV, particularly when associated with severe hypoglycemia, could be harmful not only to people with diabetes but also to non-diabetic patients in critical care settings. Overall, the pathophysiological evidence appears to be highly suggestive of GV being an important key determinant of vascular damage. Growing evidence indicates that significant GV, particularly when accompanied by hypoglycemia, can have a harmful effect not only on the onset and progression of diabetes complications but also in clinical conditions other than diabetes treated in intensive care units (ICUs). In addition to HbA1c, GV may have a predictive value for the development of T1DM complications. In insulin-treated T2DM, the relevance of GV varies according to the heterogeneity of the disease, the presence of residual insulin secretion, and insulin resistance. HbA1c is a poor predictor of hypoglycemic episodes because it only considers 8% of the likelihood of severe hypoglycemia; on the contrary, GV can account for an estimated 40% to 50% of future hypoglycemic episodes. HbA1c is a poor predictor of hypoglycemic risk, whereas GV is a strong predictor of hypoglycemic episodes. GV was an independent predictor of chronic diabetic complications, in addition to HbA1c. We should note that PPG and GV are not identical, even if they are closely related. The attention dedicated to GV is derived from the above evidence concerning its effects on oxidative stress and, from the latter, on chronic diabetes complications. Control of GV has been the focus of a number of interventional studies aimed at reducing this fluctuation. Diet and weight reduction are the first therapeutic instrument that can be used for reducing GV.

Despite the various formulas offered, simple and standard clinical tools for defining GV have yet to evolve and different indexes of GV should be used, depending on the metabolic profile of the studied population. Moreover, the absence of a uniformly accepted standard of how to estimate postprandial hyperglycemia and GV adds another challenge to this debate.

The majority of these studies have used time-averaged glucose values measured as HbA1c, an indicator of the degree of glycemic control, which is why HbA1c has become the reference parameter for therapies aimed at reducing the risk of complications from diabetes. Chronic hyperglycemia is almost universally assessed by HbA1c which has been shown to correlate closely with mean glucose levels over time, as determined by CGM. However, the relative contribution of postprandial glycemic excursions and fasting to overall hyperglycemia has been the subject of considerable debate. Monnier et al. suggested that the relative contributions of fasting and postprandial glucose differ according to the level of overall glycemic control. Fasting glucose concentrations present the most important contribution to hemoglobin glycosylation, whereas, at lower levels of HbA1c, the relative contribution of postprandial hyperglycemia becomes predominant. Collectively, GV is likely to be incompletely expressed by HbA1c, particularly in patients with good metabolic control.

GV is a physiological phenomenon that assumes an even more important dimension in the presence of diabetes because it not only contributes to increasing the mean blood glucose values but it also favors the development of chronic diabetes complications. It appears that GV is poised to become a future target parameter for optimal glycemic control over and above standard glycemic parameters, such as blood glucose and HbA1c. Avoiding both hyperglycemia and hypoglycemia by careful use of self-monitored blood glucose (SMBG) and the availability of new agents to correct hyperglycemia without inducing hypoglycemia is expected to reduce the burden of premature mortality and disabling CV events associated with diabetes mellitus.

However, defining GV remains a challenge primarily due to the difficulty of measuring it and the lack of consensus regarding the most optimal approach for patient management.

The risk of developing diabetes-related complications is related not only to long-term GV but may also be related to short-term glucose variability from peaks to nadirs. Oscillating glucose concentration may exert more deleterious effects than sustained chronic hyperglycemia on endothelial function and oxidative stress, two key players in the development and progression of CVD in diabetes. Percentages of hyperglycemia (levels between 126 and 180 mg/dl) and hypoglycemia (levels below 70.2 mg/dl) episodes should be used in the GV-related research. Mean amplitude of glycemic excursions (MAGE), together with mean and standard deviation (SD), is the most popular parameter for assessing GV and is calculated based on the arithmetic mean of differences between consecutive peaks and nadirs of differences greater than one SD of mean glycemia. It is designed to assess major glucose swings and exclude minor ones.

The features discouraging the use of GV as a parameter in clinical practice and trials are the difficulty of interpreting numerous parameters describing this phenomenon and a limited number of computational opportunities allowing rapid calculation of GV parameters in CGM data.

The UK Prospective Diabetes Study (UKPDS) showed that after an initial improvement, glycemic control continues to deteriorate despite the use of oral agents to enhance insulin secretion and reduce insulin resistance. This deterioration can be attributed to the progressive decline of ß-cell function. Even in subjects with well-controlled T2DM, 70% of the variability of A1C can be explained by abnormalities in postprandial glucose. Chronic sustained hyperglycemia has been shown to exert deleterious effects on the ß-cells and the vascular endothelium. Monnier et al. and Brownlee and Hirsch have recently emphasized that another component of dysglycemia, i.e., GV, is even more important than chronic sustained hyperglycemia in generating oxidative stress and contributing to the development of secondary diabetes complications. In-vivo studies have convincingly demonstrated that hyperglycemic spikes induce increased production of free radicals and various mediators of inflammation, leading to dysfunction of both the vascular endothelium and the pancreatic ß-cell[3]."

2.5 Glucose fluctuations (GF)

The concept and practice of GV have existed since the clinical usage of CGM devices to monitor severe diabetes patients and insulin treatments in hospitals. Many medical papers have been published on GV; however, there is no universally accepted formula or equation for generally accepted applications[6-8].

Defining GV remains a challenge primarily due to the difficulty of data collection with its associated data cleaning, processing, comprehension, and interpretation of the results by physicians and patients along with no consensus regarding the optimal approach for its clinical management. For example, the GV derivation involves the usage of SD from statistics[9]. Although SD is widely used, it has limitations because the assumption of measured glucose data is normally distributed (similar to a Gaussian distribution), which is typically not the case for bio-waves and medical data. Besides, many research articles use glucose data collected within a few days from hospitalized patients rather than the use of glucose data collected over a long period, such as years, from outpatients. The reason is that until recently, after 2016-2017, the SMBG devices became available to diabetes out-patients to collect their own glucose data at home, instead of in the hospitals or clinic centers[10]. However, the tasks of glucose data transfer from a CGM device to a computer and then the necessary follow-on tasks of data processing, data management, and data analysis still remain a challenge, particularly for out-patients. Due to the lack of professional training and academic knowledge in this domain, most patients and clinical physicians have encountered difficulties with these tasks. Data without careful cleaning and proper preparation would create a situation of garbage inputs result in garbage outputs which fit the common expression in the computer science industry of "garbage in and garbage out".

Based on the above-mentioned theoretical and technical viewpoints, the author decided

to conduct his study on just applying the basic concept of GV (i.e., glucose fluctuation between peak and nadir), and without touching certain terms or derived formulas described in some of those publications. However, he further combined the primary characteristics of wave theory, e.g., frequency, amplitude, and wavelength along with the concept of energy theory to include the estimated energy associated with the GF[11,12].

He re-named the GV as glucose fluctuations (GF) where GF equals the value of maximum glucose minus minimum glucose. Not only does the simpler definition and form of GF provide a straightforward interpretation and easier comprehension to be applied by both physicians and patients, but it also fully represents the meaning of GV. The word variability can involve and signify many various things to different people.

GV can be applied to many clinical cases of greater mortality for those in the intensive care units, increased rate and risk of diabetes complications, and postprandial beta-cell dysfunction (insulin health)[13,14].

3. RESULTS

3.1 Collected input data

The author has collected 288 glucose data per day (every 5 minutes) and extracted 96 glucose data per day (every 15 minutes) from the CGM sensor device then entered them into his computer software since 5/5/2018. During the past 1,070 days (from 5/5/2018 to 4/10/2020), he has collected 308,160 glucose data from his 5-minute model and utilized 102,720 glucose data from his 15-minute model for this specific analysis project.

For this study, he divided the big dataset into two periods: the pre-virus period of 623 days from 5/5/2018 to 1/18/2020 and the COVID-19 virus period of 447 days from 1/19/2020 to 4/10/2021. Therefore, in his energy method 2 involving the horizontal axis of days or frequency components, he needed to amplify the second virus period's results by a factor of (623/447 = 1.39) in order to have an equivalent energy estimation.

3.2 Three methods for estimated GF energy

The first method uses the square of Y-amplitude of GF (max-min of daily glucose) in the time-domain (TD).

The second method utilizes the Y-amplitude of GF in the frequency domain (FD).

The third method applies the Y-amplitude times the X-frequency components numbers to obtain the total area underneath the GF frequency curve in the FD.

Method 2 and method 3 would produce almost identical results. Method 1 would generate a similar energy curve's shape (i.e., with extremely high correlation coefficients) but there is some degree of numerical differences with both method 2 and method 3.

After obtaining the estimated energy data, they need to be normalized by a factor of 0.735 which is the dividing break-even line in his ready-developed metabolism model. The overall metabolism-based results continue to be the major players in his risk assessment models since disease-induced complications are based on the combination of chronic medical conditions and lifestyle details.

Finally, he uses 90% of the values calculated using the MI model without GF and 10% of the above-derived energy results using GF where he added them together as the final results of CVD/stroke risk probability percentage with GF influential factor inside.

3.3 Graphic diagrams of results

Figure 1 shows the data table of input values and calculated results using three different energy methods to estimate the energy associated with GF influential factor. The actual physical meaning of this rather long data table can be summarized by the following two conclusive figures.

Figure 2 depicts the calculated CVD risk diagram between without GF factor and with GF factors (based on three different energy methods) for both pre-virus and virus periods.

These three energy methods are method 1 (square of GF Y-magnitude in the TD), method 2 (GF Y-magnitude in the FD), and method 3 (GF area underneath the GF curve in the FD).

The key findings are the following:

(1) The risk of having a stroke or CVD over the pre-virus period is higher than the virus period (blue line of the pre-virus is higher than the orange line of virus).
(2) Results of method 2 is identical with method 3 (62% for pre-virus and 54% for virus).
(3) Risks for the models with three GF energy factor is higher than the model without GF energy factor i.e., purely based on metabolism.

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CVD Risk with GF inputs	Pre-Virus	Virus	Virus/Pre-Virus	Ideal Case
No. of Days/Frequencies	623	447	72%	
Avg Daily Glucose	131	117	89%	120
Glucose Fluctuation (Max-Min)	101	89	89%	80
Fluctuation / Glucose	77%	77%	99%	67%
CVD Risk with GF inputs (Energy 1)	Pre-Virus	Virus	Virus/Pre-Virus	Ideal Case
SQ of (Fluctuation/Glucose)	60%	59%	98%	44%
Eneegy of Glucose (SQ of Gluc)	17038	13600	80%	14400
Eneegy of Fluctuation (SQ of Fluc)	10179	7978	78%	6400
Fluc Eneegy /Glucose Energy	60%	59%	98%	44%
(Period/Ideal) of (Fluc.E/Gluc.E): (Eneegy 1)	1.3442	1.3199	98%	1.0000
Normalize to 0.735	0.988	0.970	98%	0.735
10% of Normaized Fluc Eneegy	0.099	0.097	98%	0.074
CVD Risk Probabilty %	0.5582	0.5123	92%	
90% of CVD Risk	0.5024	0.4611	92%	
!0% Fluc Energy + 90% CVD Risk	0.6012	0.5581	93%	
CVD Risk with GF inputs	2H18	1H19	Virus/Pre-Virus	Average
CVD Risk Changes (Energy 1)	4%	5%	107%	#DIV/0!
CVD Risk without GF (Energy 1)	56%	51%	92%	53.6%
CVD Risk with GF (Energy 1)	60%	56%	93%	#DIV/0!
CVD Risk with GF inputs (Energy 2)	Pre-Virus	Virus	Virus/Pre-Virus	Ideal Case
FD-Y magnitude: (GF Eneegy 2)	672	484	72%	425
Normalize to 0.735	1.163	0.837	72%	74%
10% of Normaized Fluc Eneegy	0.116	0.084	72%	7%
CVD Risk Probabilty %	0.5582	0.5123	92%	
90% of CVD Risk	0.5024	0.4611	92%	
!0% Fluc Energy + 90% CVD Risk	0.6186	0.5448	88%	
CVD Risk with GF inputs (Energy 2)	Pre-Virus	Virus	Virus/Pre-Virus	Ideal Case
CVD Risk Changes (Energy 2)	6%	3%	54%	4.6%
CVD Risk without GF (Energy 2)	56%	51%	92%	53.6%
CVD Risk with GF (Energy 2)	62%	54%	88%	58.2%
CVD Risk with GF inputs (Energy 3)	Pre-Virus	Virus	Virus/Pre-Virus	Ideal Case
Avg Daily Glucose	130	132	101%	120
Glucose Fluctuation (Max-Min)	97	98	101%	80
Fluctuation / Glucose	75%	74%	100%	67%
SQ of (Fluctuation/Glucose)	56%	55%	99%	44%
FD-Y magnitude	417968	215844	52%	42911
FD-Y magnitude (GF Eneegy 3)	417968	300830	72%	264158
Normalize to 0.735	1.1630	0.8370	72%	0.735
10% of Normaized Fluc Eneegy	0.1163	0.0837	72%	0.074
CVD Risk Probabilty %	0.5582	0.5123	92%	
90% of CVD Risk	0.5024	0.4611	92%	
!0% Fluc Energy + 90% CVD Risk	0.6187	0.5448	88%	
CVD Risk Changes (Energy 3)	6%	3%	54%	4.6%
CVD Risk without GF (Energy 3)	56%	51%	92%	53.5%
CVD Risk with GF (Energy 3)	62%	54%	88%	58.2%

Figure 1: Data table of CVD/stroke risk with GF using three different GF energy methods.

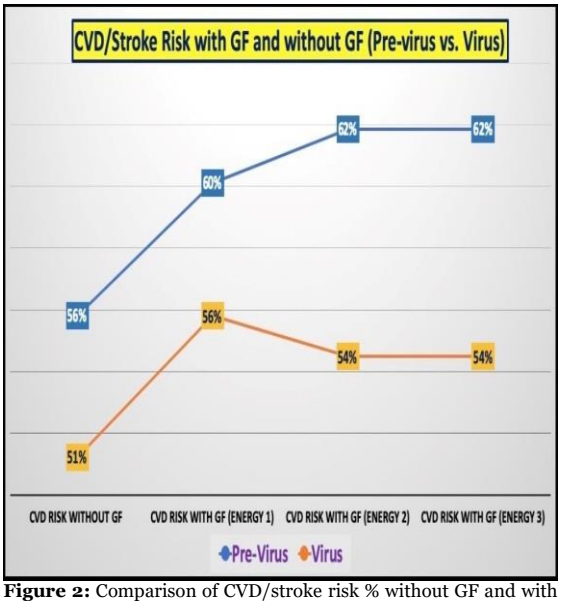

Figure 2: Comparison of CVD/stroke risk % without GF and with GF (using 3 energy methods 1, 2, and 3) for pre-virus vs. virus.

Figure 3 reveals the risk differences using the three different energy methods. The first observation is that all of the shown risk differences are nominal. The second observation is that the results from using method 2 of GF Y-axis magnitude and using method 3 of GF energy curve area are identical. The last observation is that using method 1, the risk percentage difference between the pre-virus and virus periods is only 1%, which means the square of the value of (fluctuation/glucose) are closely related to each other, 60% for pre-virus and 59% for the virus.

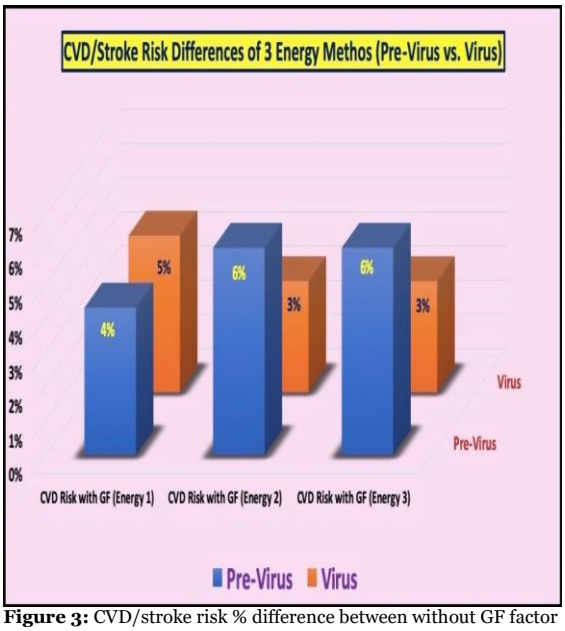

Figure 3: CVD/stroke risk % difference between without GF factor and with GF factor using methods 1, 2, and 3 for pre-virus vs. virus.

4. CONCLUSION

In summary, by presenting the extra influential factor of GF associated energy via three different estimation methods into his existing CVD risk assessment model based on metabolism, his risk percentages between the pre-virus period and virus period are listed below in the format of pre-virus and virus:

Without GF: 56%, 51%, with GF-method 1: 60%, 56%, with GF-method 2: 62%, 54%, with GF-method 3: 62%, 54%

From the table above, we can draw the following conclusion:

(1) Overall, the virus period has lower risk percentages than the pre-virus period (5% less for a case without GF and 4%-8% for three cases with GF). This means that the author's overall health conditions are better during the virus period.
(2) Results from the GF-method 2 and GF-method 3 are similar due to the specific definitions for the two GF energy methods which are almost identical. The exceedingly small difference is a result of numerical truncations.
(3) The GF-method 1 is based on the definition of GF energy where it is equal to the square of the GF magnitude. This is different from the other two methods which are derived from Fourier transform operation based on a TD function into a FD function. Furthermore, basic physics says that the energy associated with a wave is directly proportional to the square of the wave amplitude (but not equal to the square of the wave amplitude).
(4) In comparison between the case with GF vs. three cases without GF, the summarized risk percentage differences are listed below in the format of pre-virus and virus:

Method 1 vs. without: 4%, 5%, method 2 vs. without: 6%, 3%, method 3 vs. without: 6%, 3%

It is obvious that the GF brings in extra energies which damage internal organs, and therefore, increase the risks of having a CVD or stroke episode.

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