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Research Article

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A Summary Report of 25 Research Articles Utilizing Linear Elastic Glucose Theory Based on GH-Method: Math-Physical Medicine, LEGT Part 26 (No. 415)

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Abstract

Starting on 10/14/2020, the author wrote his first research note on linear elastic glucose theory (LEGT). Over the past 5 months, he has continuously conducted research on this subject, where he completed his research note, No. 414, LEGT Part 25 on 3/14/2021.

On 3/15/2021, he decided to take a break in order to work on a summary report based on his past 25 research articles of LEGT.

Introduction

Starting from 10/14/2020, the author wrote his first research note on linear elastic glucose theory (LEGT). For the past 5 months, he has continuously conducted research on this subject, where he completed his research note, No. 414, LEGT Part 25 on 3/14/2021.

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Methods

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

Background: his Diabetes Case

The author was a severe type 2 diabetes patient since 1996. He weighed 220 lb. (100 kg) at that time. By 2010, he still weighed 198 lb. with an average daily glucose of 250 mg/dL (HbA1C of 10%). During that year, his triglycerides reached to 1161 and albumin-creatinine ratio (ACR) at 116. He also suffered from five cardiac episodes within a decade. In 2010, three independent physicians warned him regarding his needs of kidney dialysis treatment and his future high risk of dying from his severe diabetic complications.

In 2010, he decided to self-study endocrinology, diabetes and food nutrition. During 2015 and 2016, he developed four prediction models related to diabetes conditions, i.e., weight, postprandial plasma glucose (PPG), fasting plasma glucose (FPG), and HbA1C (A1C). As a result, from using his developed mathematical metabolism index (MI) model and those four prediction tools, by 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), averaged finger glucose from 250 mg/dL to 120 mg/dL, and HbA1C from 10% to ~6.5%. One of his major accomplishments is that he no longer takes any diabetes medications since 12/8/2015.

In 2017, he had achieved excellent results on all fronts, especially glucose control. However, during the pre-COVID period of 2018 and 2019, he traveled to approximately 50+ international cities to attend 65+ medical conferences and made ~120 oral presentations. This hectic schedule inflicted damage to his diabetes control, through dinning out frequently, post-meal exercise disruption, jet lag, and along with the overall metabolism impact due to his irregular life patterns through a busy travel schedule; therefore, his glucose control was affected during this two-year period.

By 2020, his weight was further reduced to 165 lbs. (BMI 24.4) and his HbA1C was at 6.2% without any medications intervention or insulin injection. Actually, during 2020 with the special COVID-19 quarantined lifestyle, not only has he published approximately 400 medical papers in journals, but he has also achieved his best health conditions for the past 26 years. These good results are due to his non-traveling, low-stress, and regular

daily life routines. Of course, his rich chronic diseases knowledge, practical lifestyle management experiences, and his developed various high-tech tools also contribute to his excellent health status since 1/19/2020.

On 5/5/2018, he applied a continuous glucose monitoring (CGM) sensor device on his upper arm and checks his glucose measurements every 15 minutes for a total of ~96 times each day. He has maintained the same measurement pattern to present day. Therefore, during the past 11 years, he could study and analyze his collected ~2 million data regarding his health status, medical conditions, and lifestyle details. He applies his knowledge, models, and tools from mathematics, physics, engineering, and computer science to conduct his medical research work. His medical research work is based on the aims of achieving both "high precision" with "quantitative proof" in the medical findings, not just through linguistic expressions of qualitative words, vague statements, or complex terminologies.

Stress, Strain, & Young's Modulus

The following excerpts are from the internet public domain of Google and Wikipedia:

Strain - ε

Strain is the "deformation of a solid due to stress" - change in dimension divided by the original value of the dimension - and can be expressed as
ε = dL / L
where
ε = strain (m/m, in/in)
dL = elongation or compression (offset) of object (m, in)
L = length of object (m, in)

Stress - σ
Stress is force per unit area and can be expressed as
σ = F / A
where
σ = stress (lb./in2, psi)
F = applied force (lb.)
A = stress area of object (in2)

Stress includes tensile stress, compressible stress, shearing stress, etc.

E, Young's Modulus:It can be expressed as:
E = stress / strain = σ / ε

where
E = Young's Modulus of Elasticity was named after the 18th-century English physicist Thomas Young.

Elasticity:
Elasticity is a property of an object or material indicating how it will restore it to its original shape after distortion. A spring is an example of an elastic object - when stretched, it exerts a restoring force which tends to bring it back to its original length.

Young's modules in the above table are ranked from soft material (low E) to stiff material (higher E)."

Highlights of his Developed Linear Elastic Glucose Theory

The author self-studied internal medicine, including endocrinology and food nutrition over a 4-year period from 2010 to 2013. By the end of 2013, he has determined the primary path from lifestyle to metabolism and immunity, which are two sides of the same coin, to chronic diseases (via metabolism) and infectious diseases (defended by the immune system), and finally to all kinds of complications.

Therefore, in 2014, he spent the entire year to develop a mathematical model of metabolism using topology concept of mathematics and finite element method of engineering.

Starting from 2015, he started to develop his prediction model for post-prandial plasma glucose (PPG). He identified approximately 19 influential factors of PPG formation, where the health of pancreatic beta cells insulin secretion (quantity) and insulin resistance (quality) are the most important one. Once removing the biomedical factor (~50%-80% of contribution), the other 18 lifestyle details, ranging from ~20%-50%, contribute to the PPG formation. Taking a macro-view of PPG formation, the pancreatic beta cells accounts for 50% to 80% and diet of lifestyle provides ~37%-67%, while post-meal walking exercise contributes -17% (negative correlation). The author has collected more than 2 million data regarding his medical conditions (~10%) and lifestyle details (~90%) over the past decade. Using various GH-Method, math-physical medicine tools on the collected big data, *he has already developed an intuition of the existence for a linear relationship between his measure PPG and carbs/sugar intake amount with a coefficient around 1.8-2.5.*

In order to solve the puzzle of his predicted PPG, the first task was to identify an accurate way to estimate his carbs/sugar intake amount. Utilizing optical physics and artificial intelligence, he developed a computer software program to predict the carbs/sugar amount of his food or meals via a pictured food or meal with a prediction accuracy of >98%. He then applied this linear coefficient of 1.8-2.5 to multiply his estimated carbs/sugar amount to obtain his incremental PPG from food. This observed linear coefficient of 1.8-2.5 was his first identified GH-modulus, the GH.p-Modulus.

However, diet is the most complicated part of the puzzle. Actually, a predicted PPG is a nonlinear mathematical problem. In order to simplify his difficult task at hand, he broke this non-linear system into 3 simpler linear systems, which are the pancreatic beta cells' insulin, food and meals for glucose rising, and exercise for glucose declining.

In the early morning of 3/16/2016, he identified a strong correlation (~90% correlation coefficient) existing between body weight and fasting plasma glucose (FPG) by conducting an "out-of-box" experiment. Since both weight and FPG are outputs of the biomedical system, lifestyle details are inputs of the biomedical system. As a trained engineer, he was taught to seek for relationships between input and output, not just outputs only. In the following years of 2017-2020, he identified 7 to 8 different research angles to investigate his pancreatic beta cells "self-repair" situation. One angle was using the FPG data since there is no food or exercise occurring during sleep hours to confuse the equation; therefore, a long-term collected FPG record can serve as a reliable indicator of the pancreatic health state. As a result, he identified his second GH-Modulus, the GH.f-Modulus, to transform FPG into a baseline PPG.

Relatively speaking, exercise is a much simpler and easier subject to deal with. Based on his trial-and-error for the linear coefficient between the post-meal walking k-steps (1,000 steps) and change amount of PPG, he identified that PPG would be reduced by 5 to 6 mg/dL for every thousand steps (about 10 minutes of walking). Therefore, he chose -5 to -6 as his third GH-Modulus, the GH-w-Modulus.

By combining these three linear models together, he obtained a "pseudo-linear" model of his predicted PPG as shown in Figure 1 with a synthesized PPG wave from the collected 3,255 meals data over a period of 1,085 days from 5/5/2018 to 3/15/2021.

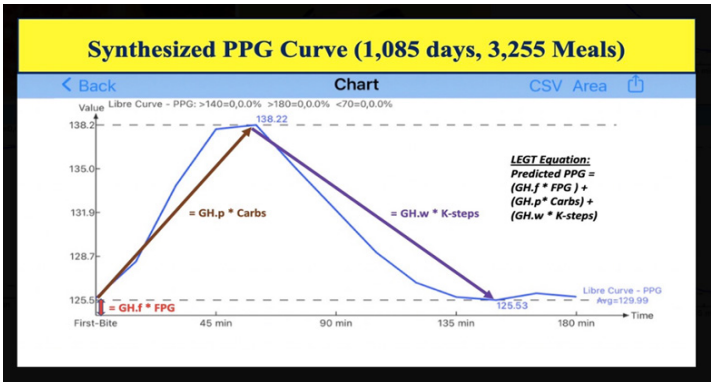


Figure 1: Three linear segments of a synthesized PPG wave using three GH-Moduli

If readers are interested in learning the step-by-step explanation of the predicted PPG equation using LEGT, they can read the author's published papers listed in References 4 through 28.

The LEGT equation is:

Predicted PPG= Baseline PPG + food induced incremental PPG + exercise induced incremental PPG
or,
Predicted PPG = (FPG * GH.f) + (Carbs/sugar * GH.p) + (post-meal walking k-steps * GH.w)

Based on the author's experiences on utilizing his GH-Modulus, the GH.f ranges between 0.6 to 1.0 (he uses 0.97 frequently for his own cases) and the GH.w of -5.0 for most cases. Therefore, the GH.p-Modulus is the most important variable which defines the food induced incremental PPG as follows:

Food induced Incremental PPG= GH.p * carbs/sugar or GH.p = incremental PPG / carbs

In comparison with Young's modulus equation:

E = stress / strain

where higher E (stiff material) under the same stress would result into less strain.

If we consider carbs/sugar intake similar to stress and incremental PPG similar to strain, then ***the biomedical GH.p-modulus and engineering E of Young's Modulus would have a "reciprocal" connection.***

Therefore, a higher E of Young's modulus value is equivalent to a lower GH.p-Modulus value. If a higher E (stiff material) under the same stress level would result in a lower strain. This is similar to a lower GH.p-Modulus under the same carbs/sugar intake amount which would result in a smaller amount of incremental PPG.

The above explanation provides an analogy of LEGT in biomedicine with the theory of elasticity in engineering.

Pancreatic Beta-Cells Study

Since 2019, the author focused on his continuous medical research work for the "self-recovery" of his pancreatic beta cells. He uses the term "self-recovery" because he has kept his carbs/sugar intake amount less than 15 grams per meal and his post-meal walking exercise more than 4,000 steps for the past five years. Since 12/8/2015, he has also ceased taking any diabetes medication, which is the strongest influential factor for the phenomena of glucose fluctuations. Therefore, his body is totally free of any external chemical intervention that may alter or interrupt the internal organ's biochemical process and reactions. Under this strict controlled lifestyle and environment, his damaged pancreatic beta cells must go through the self-repairing process in order to show any meaningful improvement signs of his diabetes conditions through glucose value changes. This is his chosen approach since 2016 that "fixing his diabetes conditions from their root causes via a stringent lifestyle management" instead of "using a tranquilizer to calm down the external symptom or behavior of his glucose problems".

Furthermore, during the FPG period, e.g., between 00:00 midnight through 07:00 next morning, glucose is not under any influence from external factors, mainly food and exercise. However, the FPG values would still fluctuate through the hours of sleep. Of course, there are some other factors, such as sleep conditions, stress, illness, or room environments that can alter FPG, which are secondary influential factors. The weight is the major influential factor of FPG which occupies ~85% of FPG formation. However, we also know that weight is highly correlated to insulin resistance phenomenon. The left-over and major influential factor of FPG formation is "insulin" which is produced by the pancreatic beta cells. Therefore, the ability to analyze and interpret FPG and then extend it to connect with PPG is important for understanding his situation of insulin resistance.

Results

The author has reviewed each of his previous 25 written LEGT papers with the emphasis on GH.p-Modulus behaviors.

At first, he wrote an important conclusion with certain supporting data, which are timespan period, patients or cases, key elements (carbs/sugar, FPG, PPG), and GH-moduli (GH.f, GH.p, GH.w) on paper for each part or each article (see Figure 2).

Figure 2: Hand-written notes (6 pages together)

The second step, he enters the hand-written information into a computer with some consistent formats (see Figure 3).

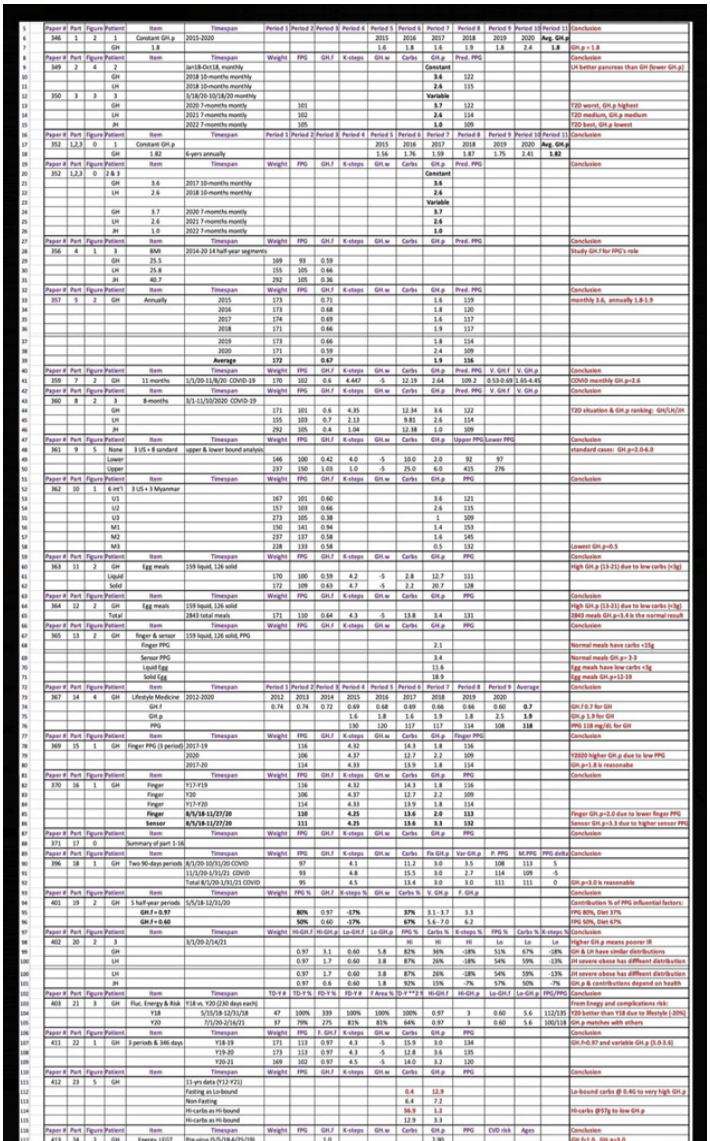
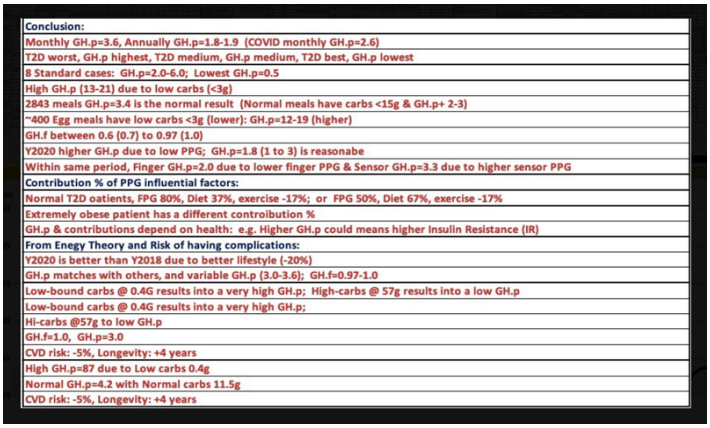


Figure 3: Typed notes in computer (4 pages together)

The third step, he assembles all of the observed conclusions from the 25 papers together (see Figure 4).



Conclusion:
Monthly GH.p=3.6, Annually GH.p=1.8-1.9 (COVID monthly GH.p=2.6)
T2D worst, GH.p highest, T2D medium, GH.p medium, T2D best, GH.p lowest
8 Standard cases: GH.p=2.0-6.0; Lowest GH.p=0.5
High GH.p (13-21) due to low carbs (<3g)
2843 meals GH.p=3.4 is the normal result (Normal meals have carbs <15g & GH.p+ 2-3)
~400 Egg meals have low carbs <3g (lower): GH.p=12-19 (higher)
GH.f between 0.6 (0.7) to 0.97 (1.0)
Y2020 higher GH.p due to low PPG; GH.p=1.8 (1 to 3) is reasonabe
Within same period, Finger GH.p=2.0 due to lower finger PPG & Sensor GH.p=3.3 due to higher sensor PPG
Contribution % of PPG influential factors:
Normal T2D oatients, FPG 80%, Diet 37%, exercise -17%; or FPG 50%, Diet 67%, exercise -17%
Extremely obese patient has a different controibution %
GH.p & contributions depend on health: e.g. Higher GH.p could means higher Insulin Resistance (IR)
From Enegy Theory and Risk of having complications:
Y2020 is better than Y2018 due to better lifestyle (-20%)
GH.p matches with others, and variable GH.p (3.0-3.6); GH.f=0.97-1.0
Low-bound carbs @ 0.4G results into a very high GH.p; High-carbs @ 57g results into a low GH.p
Low-bound carbs @ 0.4G results into a very high GH.p;
Hi-carbs @57g to low GH.p
GH.f=1.0, GH.p=3.0
CVD risk: -5%, Longevity: +4 years
High GH.p=87 due to Low carbs 0.4g
Normal GH.p=4.2 with Normal carbs 11.5g
CVD risk: -5%, Longevity: +4 years

Figure 4: Isolated conclusion statements from each part (1 page)

The last step, he interprets the isolated conclusion sentences and placed them into the Conclusion section of this research note, No. 415, Part 26 of LEGT.

Regarding the important figures, he has only selected the most important ones from over 100+ diagrams from the 25 papers in the Reference section. However, he decided not to include any of the figures into this article.

Conclusions

In summary, similar to Young's modulus of engineering materials, the GH.p-Modulus of medicine is dependent on the patient's overall metabolic conditions, including age, overall health conditions, medical diseases, types of food, amounts of carbs/sugar intake, and to some degree, the chosen time-window for one particular analysis. Nevertheless, its linear and elastic behaviors and characteristics are still quite similar to the Young's modulus of stress and strain in theory of elasticity of engineering. Some of long-term and severe diabetes patients with extreme cases of hyperglycemia may already enter into a "plastic" region, where the damage cannot be totally recovered. However, this is another research topic with academic interest for the author since all of his collected clinic glucose data to date are within the "elastic" region.

Here are the eight summarized key conclusions from his 25 written papers regarding LEGT:

1. For the GH.f-Modulus for pancreatic beta cells health state, he uses baseline PPG = GH.f * FPG where GH.f is between 0.97 and 1.0. He could also choose the equation of baseline PPG = GH.f * weight where GH.f is between 0.6 to 0.7 depends on individual patient's health status. For his own case, occasionally he uses 0.6 as his GH.f-modules since he can choose his baseline PPG = 0.6 * Weight = (0.97 or 1.0) * FPG where his

FPG in mg/dL is around 60% to 70% of his weight in lbs.

2. For the GH.w-Modulus for post-meal walking k-steps, he always uses -5.0 to -6.0 since it provides an accurate estimated PPG reduction due to exercise.
3. Based on his collected 6 clinical and 8 standard cases, for other non-severe type 2 diabetes (T2D) patients, he recommends a range of 1.0 to 6.0 as the GH.p-Modulus. For himself, he uses a range of 1.8 to 3.6 as his GH.p-Modulus. The above-mentioned recommendations are based on his extremities boundary analysis results which reached the heavy weight of >300 lbs. and high PPG level of >400 mg/dL.
4. His monthly GH.p is around 3-6 and his annual GH.p is around 2-4. However, if he eats low-carb meals, such as egg only (his ~400 egg meal experiments with <3 grams of carbs), his GH.p would go as high as 12-21. During his ~80 intermittent fasting days, his GH.p values could go as high as 87 due to his extremely low carbs of 0.4 gram (near zero) of drinking tea only. Theoretically, an infinitely large GH.p number multiplying with a zero carbs amount would still provide a zero result. Of course, this is just an extreme lower-bound case investigation. For his high-carb meals with an average of 57 grams per meal, his GH.p was as low as 1.2. This information would provide readers a realistic physical sense of the boundaries or ranges of GH.p-Modulus.
5. During a stabilized and healthy lifestyle in the COVID-19 period, his PPG was consistently lower than the other pre-virus periods, and his GH.p value could go as low as 0.6-1.8.
6. In order to compare multiple patients, generally speaking, the healthier patient (i.e., a less severe T2D patient) would have a lower GH.p value, and vice versa. Of course, this statement would hold true only when and if the other associated conditions are comparable or similar.
7. For normal T2D patients, their PPG contribution % are FPG 80%, diet 37%, exercise -17% for the case of GH.f =0.97; and FPG 50%, diet 67%, exercise -17% for the case of GH.f =0.60. For extremely obese patients, with a BMI greater than 40, their PPG factor's contribution % would be different from patients within a normal BMI range.
8. Another benefit of LEGT applications, from the author's case study, during the COVID-19 quarantine period, his risk probability of having a cardiovascular disease (CVD) or stroke is 5% lower than his pre-virus period. Similarly, his COVID period life expectancy is an additional 4 years longer compared to his pre-virus period. All of the two extended medical research work are based on his diabetes control via glucose prediction and continuous metabolism improvement program.

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