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A Study on Weight, a Brain Stimulator, and Fasting Plasma Glucose Production, Simulation Model, using GH-Method: Math-Physical Medicine

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Introduction
In this paper, the author presents his hypothesis of the role the brain plays in glucose production, particularly the simulation model of fasting plasma glucose (FPG) production, and the body weight functioning as the brain's stimulator.

Methods
The author has been a type 2 diabetes (T2D) patient for over 25 years. During the first 20 years, he was relying heavily on medications to control his disease symptoms until they worsen in 2010 when most of his severe complications appeared including five cardiac episodes, kidney complications, bladder infection, foot ulcer, thyroid, and retinal problems. By that time, the medications seemed to stop working. As a result, he focused on his lifestyle issues, especially diet and exercise, in controlling his postprandial plasma glucose (PPG) since it contributes around 75% to 85% of HbA1C formation.

In November of 2016, he flew to Honolulu and stayed in Hawaii for a while. He suddenly noticed his persistently elevated FPG values in the morning. During the following four months, he exhausted all avenues based on his knowledge to examine what went wrong with his body. On March 17, 2017 at 3am, he had a dream to delve into the relationships among output factors, instead of relationships between input categories and output categories as he was educated and trained as an engineer for 40 years. After four hours of running a computer software to examine the relationships among glucose, lipids, blood pressure, and weight, he identified that his increased body weight was the reason of his elevated FPG! Since then, for the past 2.5 years, he has continuously researched this subject and provided additional proofs of this astonishing relationship.

Results
Among all of the human internal organs, only the brain has the power of cognition, decision-making, and issuing an order capabilities. The brain instructs the liver to produce glucose and the pancreatic alpha cells to produce glucagon to raise glucose level if it is too low and beta cells to produce insulin to reduce glucose level if it is too high. In other words, the liver and pancreas are merely working machines for the master, the brain.

In the scope of FPG, what is the stimulator for the brain to instruct liver producing glucose and at what appropriate level of glucose? During our sleep time (other than the continuous operations of the internal organs and somewhat natural sweating, vaporization, nighttime urination), our body lacks the heavy physical activities such as eating, drinking, and exercising. It is the author's hypothesis that our brain knows our body weight level and situation continuously and then used this vital information as the yardstick to decide how much glucose level our body needs.

Based on this hypothesis, the author tried to prove these available physical characteristics of our biomedical phenomenon via some mathematical and computational tools.

As the first evidence, Figure 1 shows that the 77% high correlation coefficient exists between weight and FPG from a time-series analysis using data from 1/1/2014 through 10/18/2019 (a total of 2,116 days in ~5.5 years). Furthermore, a spatial analysis diagram in the lower part of Figure 1 also depicts a skewed cucumber shape of these data sets between weight and FPG without time factor. It indicates that when body weight increases or decreases, the FPG changes upward or downward accordingly.
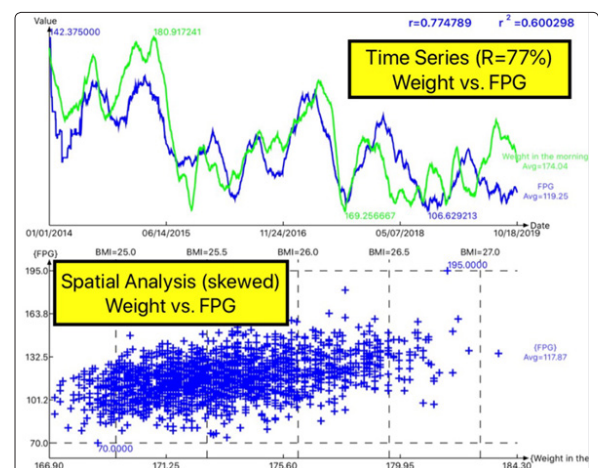

Figure 1: High correlation between weight and FPG

Based on this finding, the author developed a prediction model for FPG by using weight as its major input (~80%) and a cold weather temperature (FPG drops 0.3 mg/dL for every degree of weather temperature drop when it is below 67 degrees Fahrenheit) as its secondary factor due to "hibernation" (~10%).

Figure 2 displays the extremely high correlation of 99.97% existing between predicted FPG and measured FPG by using data from 5/5/2018 through 10/18/2019 (a total of 532 days). The reason he selected this time period is due to his use of a "dual and parallel measurements using both continuous glucose monitoring sensor device and finger-piercing with test strip". Although the patterns have low similarity between Finger FPG and Sensor FPG, but their averaged FPG value deviations is within 0.9% to 1.2% (113 mg/dL for Sensor FPG and 112 mg/dL for Finger FPG).
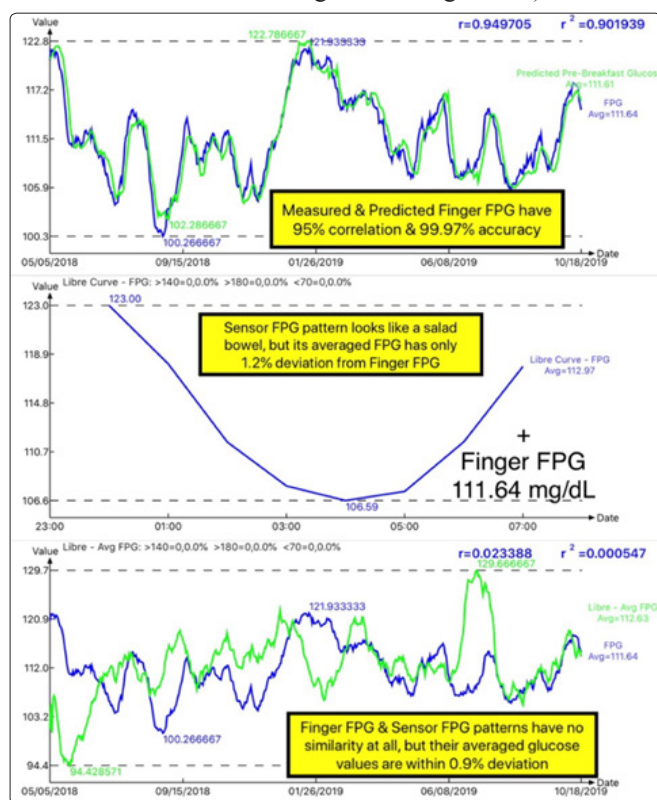

Figure 2: Predicted and measured FPG and results between finger FPG and sensor FPG

Figure 3 shows both of his annual averaged weight and annual averaged FPG from 2012 to 2019. It should be noted that both of them are declining with time. In other words, while his weight reduced from 189 lbs. to 173 lbs., his FPG also decreased from 135 mg/dL to 113 mg/dL. This FPG reduction indeed contributes around 20% of his overall A1C reduction from 10% in 2010 with 198 lbs. weight down to ~6.5% in 2019 with 172 lbs. weight.
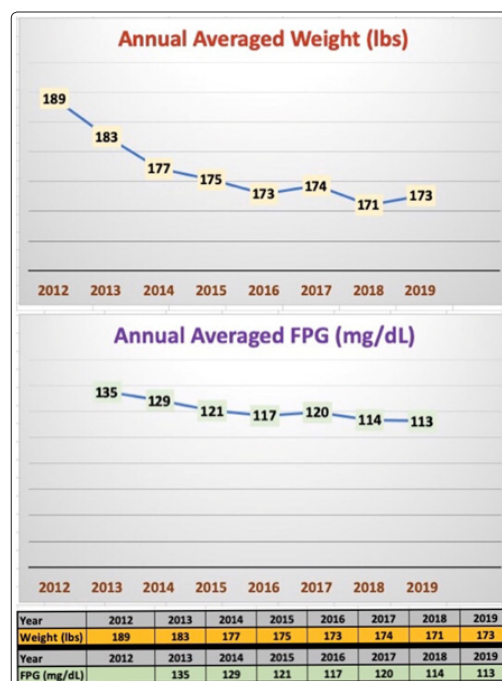

Year	2012	2013	2014	2015	2016	2017	2018	2019
Weight (lbs)	189	183	177	175	173	174	171	173
Year	2012	2013	2014	2015	2016	2017	2018	2019
FPG (mg/dL)		135	129	121	117	120	114	113

Figure 3: Annualized weight and FPG

In summary, the findings and proof of FPG level based on body weight is extremely accurate and valuable enough for him to develop FPG simulation or prediction model based on this hypothesis.

Conclusion

Body weight serves as a stimulator to the brain in making decisions on producing FPG and its appropriate amount in early mornings. The FPG prediction model is merely a mathematical simulation model to explain the complex operation of FPG production as instructed by the brain to communicate with the organs of liver and pancreas.

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