

Research Article

Open Access

Changes in Estimated Health State of Pancreatic Beta Cells Over Eleven Years Using the GH-Method: Math-Physical Medicine

Gerald C Hsu

EclaireMD Foundation, USA

***Corresponding author**

Gerald C Hsu, EclaireMD Foundation, USA. E-mail: g.hsu@eclairermd.com

Received: June 16, 2020; Accepted: June 22, 2020; Published: July 01, 2020

Introduction

This article addresses the author's change of health state for his pancreatic beta cells over the past 11 years (2009-2019) using the GH-Method: math-physical medicine approach.

Methods

The methodology of analyzing pancreatic beta cells is described in the author's article reference number 103.

Glucose contains many useful pieces of information regarding the human body. It is up to research scientists to utilize effective methods to discover them. Due to his lack of formal training in biology and chemistry, the author adopted a combined methodology of observing physical phenomena, identifying interrelationships, predicting outcomes via mathematical equations, conducting big data analytics, and building artificial intelligence (AI) algorithms to discover many hidden biomedical facts of the body.

In mechanical and structural engineering, when a system possesses its inherent characteristics (i.e. steady state), but under a variety of dynamic external forces (i.e. stimulators), its responsive outcomes are different and complicated. If we can remove influences from external stimulators, then its steady state conditions will be shown.

By applying wave and energy theories along with signal processing techniques, the author identified 19 influential factors of postprandial plasma glucose (PPG). Next, he developed an AI-based Glucometer to predict PPG using only eight factors. Finally, he further simplified his glucose model by developing a two-factors based linear equation of PPG formation.

In order to find the "steady state" or "original state" of PPG, he removed all of the external influential factors; however, one must have a full understanding of the details regarding those influential factors. Then, the body's natural glucose conditions will appear on the surface. At this "original steady state", the natural level of glucose production is actually the combined effect of liver

glucose production and pancreas glucose regulation (glycogen from alpha cells and insulin from beta cells). Among those three elements, the beta cell's health state is the most critical one for controlling diabetes. Using this approach through finger-piercing obtained glucose data (i.e. finger glucose), we will get a lower bound of the pancreatic relative health state (i.e. relative degree of damage of beta cells).

The author's previous papers have demonstrated his sensor glucose values via a continuous blood glucose monitoring (CBGM) device is higher than his finger glucose. Using mathematical modeling, he can extend from his finger glucose to build up a sensor glucose triangular model (i.e. OHCA model, see reference paper number 90). The baseline of this OHCA triangle indicates the glucose during "pre-periods", i.e. both pre-meal and pre-bed periods. During pre-periods, our body is not under any significant external stimulator's influence. The associated glucose values represent the upper bound of the relative health state of pancreatic beta cells.

By using these two values, lower and upper bounds, we can observe the changes of relative health state of pancreas over time. This is how he constructed his pancreatic health state diagram over a period of eleven years (Figure 2). From this dynamic time-series chart, a diabetes patient's relative degree of damage on his/her pancreatic beta cells and its moving pattern can be observed clearly, providing a better understanding of the diabetes patient's basic biomedical conditions.

Results

As shown in Figure 1, the author's HbA1C prominent changes and diabetes complications developmental stages are listed as follows:

8% in 2000 (Cardiac episodes)
9% in 2009 (Foot ulcer)
10% in 2010 (Renal complications)
~6.6% during 2011-2019 (Stabilized)

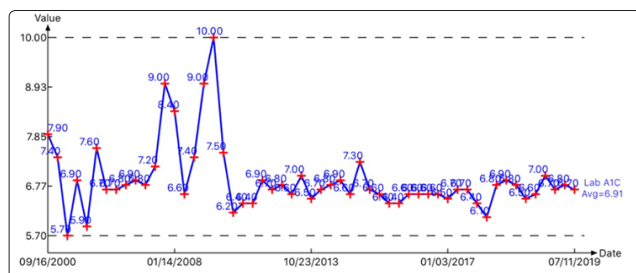


Figure 1: HbA1C lab-test results from 2000 through 2019

As shown in Figure 2, his prominent health state of pancreas beta cells are listed in the format of (upper bound / lower bound)

2009: 236 / 209 (88%)
 2010: 267 / 237 (100%)
 2011: 152 / 134 (57%)
 2016: 134 / 115 (50%)
 2019: 128 / 114 (48%)

Regarding the data integrity, it should be noted that only a few lab tested A1C converted glucose values were available for the period of 2009-2011. Finger glucose (4 times per day) were available during the period of 2012-2019 and a simulated OHCA model was developed for period of 2/1/2012-5/4/2018. The actual sensor glucose values (74 measurements per day) were available during 5/5/2018 - 8/11/2019; therefore, a realistic and non-simulated OHCA model is available during this CBGM measured sensor glucose period.

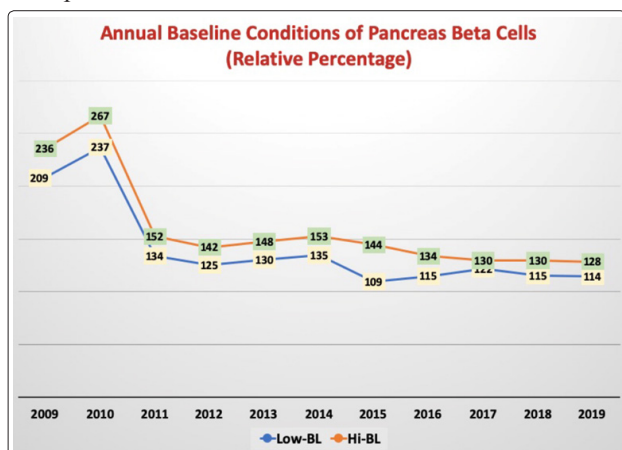


Figure 2: The author's baseline conditions from 2009 through 2019

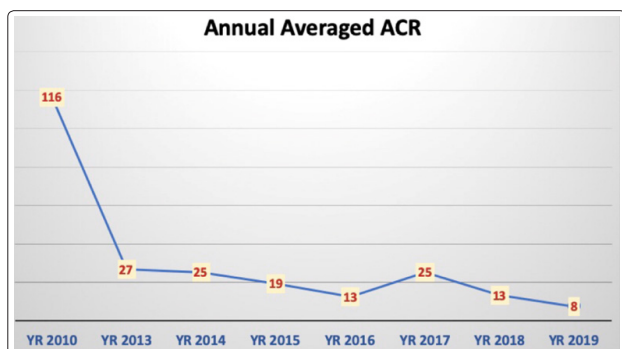


Figure 3: History of ACR

Conclusion

This paper illustrates that the author's relative health state of the pancreatic beta cells improved approximately 50% between 2010 (the worst year) and 2019 (the best year). The improvement of the pancreatic health state is not as dramatic and instant as the HbA1C progress; however, due to the author's stringent and persistent lifestyle management over the past nine years, his pancreatic health state improved accordingly. Hyperglycemia is the root cause of diabetes and its many complications, while the pancreatic beta cell's malfunction has been considered as the major cause of type 2 diabetes. Medical professionals working with this metabolic disease must possess a thorough understanding and deep knowledge of "glucose" in order to effectively control it.

The author had a similar self-recovery experience on his renal conditions. His albumin-to-creatinine ratio (ACR) was 116 mg/g in 2010, then down to 8 mg/g in 2018 due to his dramatic lifestyle change (see Figure 3). Therefore, the author feels that the beta cell's damage may be able to self-repair or restore itself to a certain degree via a stringent lifestyle management.

The following excerpt provides an interesting insight:

"The longevity and turnover of human beta cells is unknown in rodents <1 year old, a half-life of 30 days is estimated. Human beta cells, unlike those of young rodents, are long-lived. Intracellular lipofuscin body (LB) proportions in type 2 diabetes and obesity suggest that little adaptive change occurs in the adult human beta cell population, which is largely established by age 20 years" [1-4].

Most organ cells go through many lifecycles during a person's lifespan. If a person lives his life in a healthy way for a long period of time, those positive influences may be able to affect or improve the future generation of certain new cell's genetic formation. By continuing this kind of positive influence and progressive modification, some body cells may be able to self-repair the damage or restore back to its original health state. However, this process may take a long duration and require a persistent healthy lifestyle effort in order to observe some positive changes. The above statement is the author's personal theory or hypothesis. To prove his theory or verify his hypothesis, additional effective methodologies and persistent research will be required.

References

1. Hsu Gerald C (2018) Using Math-Physical Medicine to Control T2D via Metabolism Monitoring and Glucose Predictions. Journal of Endocrinology and Diabetes 1: 1-6.
2. Hsu Gerald C (2018) Using Signal Processing Techniques to Predict PPG for T2D. International Journal of Diabetes & Metabolic Disorders 3: 1-3.
3. Hsu Gerald C (2018) Using Math-Physical Medicine and Artificial Intelligence Technology to Manage Lifestyle and Control Metabolic Conditions of T2D. International Journal of Diabetes & Its Complications 2: 1-7.
4. Cnop M, Hughes SJ, Igoillo-Esteve M, Hoppa MB, Sayyed F, et al. (2010) The long lifespan and low turnover of human islet beta cells estimated by mathematical modeling of lipofuscin accumulation." Diabetologia 53: 321-330.

Copyright: ©2020 Gerald C Hsu. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.