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Introduction
In this paper, the author describes his hypothesis on the “self-recovery” of partial insulin regeneration capacity of the pancreatic beta cells from a type 2 diabetes (T2D) patient (himself), using his collected big data of postprandial plasma glucose (PPG) including Finger piercing PPG and Synthesized PPG via Sensor PPG pattern, fasting plasma glucose (FPG) including Finger piercing FPG, and HbA1C for a period of six years from 1/1/2014 to 12/31/2019.

Methods
Background
The author has had T2D for 25 years and took various diabetes medications to control his elevated glucose levels starting in 1998. For the last 20 years, he has suffered from many complications from his diabetes, including five cardiac episodes, foot ulcer, bladder infection, renal complications, diabetic retinopathy, and hypothyroidism; however, he did not have a stroke.

In 2013, he started different stages to reduce the dosages from his three prescribed diabetes medications. On 12/8/2015, he finally ceased his last one, the classic metformin HCL. For more than four years, his body has been free of any medications.

Since then, he has completely relied on a stringent lifestyle management program to control his diabetes conditions. As a result, his HbA1C has been reduced from 10% in 2010, while taking medications, to an average of 6.63% during 2016-2019 without any medication or use of insulin injection (Figure 1).

Thus far, he has kept ~2 million data of his own medical conditions and lifestyle details. He has also developed a sophisticated computer software by using big data analytics, artificial intelli-
By using a compound able to influence biological functions would show or expose some sort of physical phenomena in terms of their actions and chain reactions in the human body and organs. This explicitly shows the true status and severity of his existing T2D conditions in terms of insulin resistance or lack of insulin production supply. The second observation, from checking his massive data since 2014, his natural health state of pancreatic beta cells seems to be on a “slow” self-recovery path, even though its recovery rate might be on a small scale.

Other Research
Recently, he read an article online, “Diabetes: Can we teach the body to heal itself?” on Medical News Today, which was published on January 8, 2019.

Here is an excerpt:
“A new study by researchers from the University of Bergen in Norway, Maria Cohut, Ph.D. and Luiza Ghoul, suggests that, with just a small "push," we may be able to train the body to start producing adequate levels of insulin once more, on its own. The researchers were able, for the first time, to uncover some of the key mechanisms that allow cells to "switch" identity, looking specifically at pancreatic alpha- and beta-cells in a mouse model. They found that alpha-cells respond to complex signals they receive from neighboring cells in the context of beta-cell loss. Approximately 2 percent of alpha-cells can thus "reprogram" themselves and start producing insulin. By using a compound able to influence cell signaling in the pancreas, the researchers could boost the number of insulin-making cells by 5 percent.

The author’s research methodology is a “math-physical medicine” approach, rather than a “biochemical medicine” approach as used in the article above. Furthermore, he uses his own body, instead of a “mouse model” cited in Norway’s lab test.

Methodology
Math-physical medicine approach has three key steps of research methodology. For the first step, it starts with observing physical phenomena of some prominent biomedical characteristics from collected data. All of the biological and chemical work including their actions and chain reactions in the human body and organs would show or expose some sort of physical phenomena in terms of exterior appearances, because both biology and chemistry are applied physics. In the second step, he then forms a hypothesis from these specific physical observations based on physics theories or concepts. The third step, if possible, he utilizes existing mathematical equations, along with their given constraints, or derives his own mathematical equations and constraints based on both original physics concept and engineering models (engineering is applied physics and physics is applied mathematics), in order to verify or prove his hypothesis. Finally, once his hypothesis is proven by using his collected data, he can then apply these mathematical prediction equations to recreate future outcomes or reproduce the results based on future input data. It should be noted that computer science techniques, such as artificial intelligence and big data analytics, are only served as convenient and useful “tools” for his simulation model work and massive data processing.

This Research Work
This project started in July 2019 and ended in May 2020. During this 10-month period, he has already written 10 medical papers (No. 103, 108, 112, 133, 138, 241, 242, 243, and 244) regarding the subject of beta cells. In those referenced papers, he has described his suspicion and hypothesis regarding the probability of his pancreatic beta cells’ “self-recovery”. He will try to summarize his efforts on how he determined the annualized self-recovery rate of his damaged pancreatic beta cells due to either insufficient insulin production and/or insulin resistance.

Results
(A) Baseline PPG: Lower Bound
Applying signal processing technique of wave theory from electronics and earth science, the author was able to successfully decompose his PPG waveform into 19 sub-waves of carbs/sugar intake (39%) and post-meal walking exercise (41%) plus other 17 secondary factors and “left-over” or remaining insulin’s biological functionality (a total of ~20%). He developed a linear equation for a newly termed “Baseline PPG” which is further defined as follows:

Baseline PPG = Measured PPG + PPG Adjustment

Where PPG Adjustment = (diet *B - walking/1000 * C)
B and C are different multipliers

We can observe from Figure 2 that both increased PPG amount by food intake and decreased PPG amount by walking steps are almost equal and cancel each other for both periods (6/1/2015-5/3/2020 and 1/1/2017-5/3/2020). However, the PPG moving average curves still have noticeable variances due to some of remaining secondary factors, and mostly the “left-over” insulin functionalities of the pancreatic beta cells.
He then calculated these Baseline PPG values using the above equation and segregated the Baseline PPG data into annualized average Baseline PPG datasets for the 6-year period between 2014 and 2019 (Figure 3). Finally, he computed the baseline PPG’s change rate (%) of each year as well as the average change rates of these 6 years. For this lower-bound case of Baseline PPG, the annualized change rate is 1.5%.

Figure 3: Baseline PPG (minus adjustments) improvements (lower bound)

(B) Segment turning points PPG: Medium
During the period of ~5 years (June of 2015 through April of 2020), there are 7 “turning-points” PPG values (i.e. peaks or bottoms of a wave). By connecting these turning points and then calculating the decline rate of each segment, he was able to get the overall average dropping rate (see Figure 4). If we include the first PPG peak of 141 mg/dL, then the overall average-declining rate is 3.8%; and if we exclude the first peak, then the overall average-declining rate is 2.3%. For this medium case of Segment Turning point PPG, the annualized change rate is 2.3%. The author choose this conservative rate of 2.3% in order to remove the perturbation caused by the first peak data.)

Figure 4: Segment turning-points PPG improvements (medium)
(C) Synthesized PPG via OHCA Model: Upper Bound
The author has applied his created OHCA (Open, High, Close, and Average) Model of the CGM Sensor PPG data during 5/5/2018 to 4/5/2020 to build a hypothetical synthesized sensor PPG model for the pre-CGM period of 1/1/2014 - 5/4/2018 (see Figure 5). Since the average sensor PPG value is about 18% higher than Finger PPG value, this is the reason he created “synthesized PPG” value, which would serve as an upper bound of his research results. Following the same calculation scheme, we get a higher bound of PPG change rate result. This effect is due to the observation of CGM sensor monitoring the entire 3-hour time span of a PPG wave, while Finger PPG measuring around 2-hours after the first bite of food, usually catching a much lower PPG value. For this upper-bound case of Synthesized PPG via OHCA Model, the annualized change rate is 3.5% (see Figure 6).

(D) Combined Average PPG
When we add the above three PPG models change rates, we then get the combined average PPG change rate of 2.3% per year.

(E) FPG
By applying the signal processing technique similar to the PPG case, we can identify the most significant contribution factor of FPG is the body weight in the morning (> 90% of influence). The remaining 4 other secondary factors and “left-over” or “remaining” insulin functionalities contribute only ~10% or less. As shown in Figure 7, the correlation between the annualized body weights vs. annualized FPG is as high as 93%. The author notices that for the past 6 years, his weight dropped moderately from 177 lbs. in 2014 down to 173 lbs. (~ 2.3%) in 2019. However, his FPG decreased more noticeably from 128 mg/dL down to 113 mg/dL (~ 11.7%). This observation is another indirect proof that his pancreatic beta cells have been self-repairing over the past 6 years. For this FPG case, the FPG change rate is 2.3% per year.

Currently, we can see that both of his combined average PPG change rate and FPG change rate are at 2.3% per year (see Figure 8).
rate of 2.3% (PPG & FPG)

(F) HbA1C
He developed a mathematical model to predict his HbA1C level on daily basis. He utilized his past 4-moth glucoses data, including both FPG and PPG, and then assigned different weighting factors for each month input data. Combining with some other assumptions and adjustments, this daily HbA1C model becomes a “non-linear” mathematical model, which he named as the “N-2 model” inside of his computer software program. Due to this difficulty, it is not easy to decompose his HbA1C wave similar to his PPG wave using signal-processing technique. For this HbA1C case, the HbA1C change rate is 2.9% per year (see Figure 9), which is higher than 2.3% for both FPG and PPG cases.

Conclusion
The author has spent 10 months to research the self-recovery of his pancreatic beta cells via different entry-points and various methods. He has written a total of 10 medical articles regarding this specific subject [1-10]. Finally, after summarizing all of his past findings at different research stages, he has confirmed that, via his continuous and stringent lifestyle management efforts, he has seen a self-recovery rate of 2.3% to 2.9% per year. This percentage range is actually quite close to the cited Norwegian Lab’s mouse result of ~2% conversion rate from alpha cells into beta cells to produce insulin. Although this is only a moderate improvement, it is still promising. If his self-recovery rate is 2.3% to 2.9%, then for the past 6 to 7 years, his beta cells insulin functionalities have been repaired by 13% to 20%. This solid evidence of “glucose improvement” for nearly 7-years is a non-arguable fact. Most medical professionals stated that diabetes is an “irreversible” or “non-curable” disease. At least, the author has now proven that not only has he stopped the “deterioration” of his T2D conditions, but also he may have “reversed” his damaged pancreatic beta cells. I hope that by sharing his lifestyle program, research methods, and positive results, other T2D patients can also be encouraged to achieve similar positive results on their battles against diabetes [1-10].

Acknowledgement
Foremost, I would like to express my deep appreciation to my former professors: professor James Andrews at the University of Iowa, who helped develop my foundation in basic engineering and computer science, and Professor Norman Jones at the Massachusetts Institute of Technology, who taught me how to solve tough scientific problem through the right attitude and methodology.

References
medicine approach and various glucose data to investigate the health state of a type 2 diabetes patient’s pancreatic β-cells (No. 244).