

Relationship Between Metabolism and Risk of Cardiovascular Disease and Stroke, Risk of Chronic Kidney Disease, and Probability of Pancreatic Beta Cells Self-Recovery Using GH-Method: Math-Physical Medicine (No. 259)

Hsu GC*

eclaireMD Foundation, USA

***Correspondence:** Gerald C Hsu, eclaireMD Foundation, USA

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Keywords: metabolism, cardiovascular disease, stroke, kidney, pancreatic beta cells

Abbreviations: MPM: math-physical medicine; CVD: cardiovascular disease; CKD: chronic kidney disease; MI: metabolism index; GHSU: general health status unit; FPG: fasting plasma glucose; PPG: postprandial plasma glucose; HbA1C: hemoglobin A1C

Introduction

The author uses GH-method: math-physical medicine (MPM) approach to investigate his risk probability on metabolic disorder induced cardiovascular disease (CVD), stroke, or chronic kidney disease (CKD), as well as the probability of pancreatic beta cells self-recovery. He addresses the damages caused by metabolic disorders affecting arteries and micro-vessels in terms of blockage, rupture, or leakage along with the probability assessment of pancreatic beta cells self-recovery. Furthermore, he uses mathematical correlations to distinguish the weighted impact by metabolism on heart, brain, kidney, and pancreas [1].

Methods

In 2014, the author applied topology concept, finite-element engineering technique, and non-linear algebra operations to develop a mathematical metabolism model that contains 10 categories, including 4 basic output categories such as weight, glucose, BP, and other lab-tested data (lipid, ACR, and TSH), and 6 basic input categories such as food, water drinking, exercise, sleep, stress, routine life patterns and safety measures, with approximately 500 detailed elements.

He further defined a new parameter, metabolism index (MI), as the combined score of the above 10 metabolism categories and 500 elements. He also defined another term, general health status unit (GHSU), as the 90-days moving average value of MI for indicating the trend of metabolism. This MI value is continuously and dynamically calculated whenever the patient has encountered some condition changes and their relevant data were collected regarding his medical conditions and lifestyle details. He has identified a mathematical normalized “break-even line” at 0.735 (73.5%) to separate his metabolism conditions between healthy (below 0.735) and unhealthy (above 0.735).

He started to collect his above mentioned personal detailed data on 1/1/2012. Thus far, he has collected and stored ~2 million data of his own body health and personal lifestyle.

Through his developed 4 prediction models of weight and glucose (FPG, PPG, and HbA1C), he had successfully reduced his glucose level from 280 mg/dL (A1C 10%) in 2010 to 113 mg/dL (A1C 6.4%) in 2020. It should be noted that, for the period of 2016–2020, he did not take any diabetes drugs or insulin injections.

In 2017–2018, he developed two similar but rather different mathematical models to calculate risk probability of having a CVD/stroke and CKD, respectively.

In 2019–2020, he further conducted a special research on probability and improvement rate of his pancreatic beta cells self-recovery situation.

At first, he built a baseline model, including genetic factors such as steady state and unchangeable conditions (race, gender, family history, and personal medical history), semi-permanent factors such as weight and waistline, and bad habits such as hard to change conditions (smoking, alcohol drinking, and illicit drugs).

Next, he developed a risk probability calculation model for estimating the following three scenarios:

- For CVD and stroke: blood flow blockage of arteries due to diabetes and hyperlipidemia; and blood vessel rupture of arteries due to diabetes and hypertension.
- For CKD: leakage from micro-blood vessels due to diabetes and hypertension.
- For pancreatic beta cells: this is a quite different subject since it involves hormone production capability and capacity, mainly insulin which are not the same as his research work on heart/brain and kidney complications. CVD and CKD dealt with physically observed phenomena, such as blood vessel's structural damage or weakening by high glucose or artery rupture by high blood pressure. He finally found a way to cut into the problem of "beta cells insulin" through "glucose" phenomena. Beta cells structure with insulin production is a kind of "black box" problem which was rather difficult for him at the beginning since he is a professionally trained mathematician, physicist, and engineer, and lacks academic training in biology and chemistry.

Finally, he applied his collected several hundred thousand data of medical conditions regarding 4 basic chronic diseases and more than 1 million data of lifestyle details [2], from the past eight and a half years (2012–2020), to calculate their combined contribution to cause the following situations related to CVD, stroke, CKD, and pancreatic beta cells:

- Blockage and rupture of arteries in the heart or brain, including situations of CVD, CAD, CHD due to diabetes, hypertension, and hyperlipidemia. We already know that more than 50% of heart diseases or stroke patients also have different chronic diseases due to their metabolic disorders.
- Kidney complications, including glucose, blood pressure, kidney, glomeruli, bladder, urinary tract, etc.
- We already know that the two main causes of chronic kidney disease are diabetes and hypertension, which are responsible for up to two-thirds of the CKD cases.
- Elevated glucose was caused by insufficient insulin production or insulin resistance of damaged pancreatic beta cells. Once he removed the element of medications out from the equation, he started to notice the changes of fasting plasma glucose (FPG) are directly related to the health state of the pancreas since both diet and exercise play no rules in FPG formation. He then further investigated changes due to both postprandial plasma glucose (PPG) and hemoglobin A1C (HbA1C) to figure out the possible changes and ranges of both functionality (*i.e.*, insulin resistance) and production amount (*i.e.*, insufficient insulin) of pancreatic beta cells [3–6].

For CVD and CKD, his calculation results are further divided into the following three groups:

- A. Medical conditions (individual M1 through M4: *i.e.*, weight, glucose, blood pressure, lipid, and ACR).

- B. Lifestyle details (individual M5 through M10).
- C. MI scores (a combined score of M1 through M10).

With these mathematical risk probability assessment models, he can obtain 3 separate percentages of risk probability (*i.e.*, medical-based, lifestyle-based, and MI-based, but these 3 results are quite close to each other) to offer a range of the risk prediction of having cardiovascular diseases, stroke, or chronic kidney diseases resulted from metabolic disorders, unhealthy lifestyles, and their combined impact on the human body.

For self-recovery of pancreatic beta cells, he examined the annual change rate of FPG, PPG, and HbA1C, (its outcome, “glucose” only). However, there is a data reliability issue associated with existing medical testing and measurement community, including finger-piercing glucose devices, continuous glucose monitoring (CGM) sensor devices, and lab-tested HbA1C devices and process. Sometimes, their output data’s deviation could be in the range of 25% or more. Therefore, the author decided to derive the following 3 sets of simple formulas for his “beta cells %” calculation which are used in his calculation table.

- 1) Average glucose = (FPG + PPG) / 2
- 2) Average HbA1C = (Lab A1C + math A1C) / 2
- 3) Beta cell % = (Average glucose + (Average HbA1C * 17.1931)) / 4; where 17.1931 is the glucose conversion factor he has used in his mathematical daily predicted HbA1C value. The reason for “dividing by 4” is for a purpose of better viewing for his data or curve that it would give a final number within the general data range of the other 3 datasets. Afterall, all these data or curves are “relative”, not “absolute”, and for serving the porpoise of investigating their relative relationship.

Regarding these 4 prominent influential biomarkers, *i.e.*, MI %, CVD risk %, CKD risk %, and beta cells %, he further calculated 3 pairs of correlation coefficients using the time-series method. Due to the small data volume in this study using “annualized” average data, he cannot apply the powerful spatial analysis method. Spatial analysis method can provide an accurate and clear picture of data relationship pattern and moving trend; however, it also requires a bigger size of collected data than the time-series method to conduct its analysis.

During the past 8+ years, all his measurements of weight, glucose, and blood pressure were performed using home-based devices. However, to obtain his tested data of HbA1C, albumin, creatinine, and albumin-creatinine ratio (ACR), these were done in a laboratory or hospital.

Finally, it should be noted here that the risk probability percentages and pancreatic beta cells self-recovery rate are expressed on a “relative” scale, not on an “absolute” scale.

Results

The figure (Figure 1) demonstrates the author’s overall metabolic conditions, including both MI and GHSU for the past eight and a half years (1/1/2012–5/10/2020), along with the detailed descriptions of the MI category’s measurement standards. Both his MI and GHSU were > 73.5% during 2012–2014 (unhealthy) and < 73.5% during 2014–2020 (healthy). In 2014, his health greatly improved due to his knowledge gained from the development of the metabolism model and his discipline in implementing the lifestyle management program.

The figure (Figure 2) shows his risk probability % of having a CVD or stroke (heart or brain). It is obvious that his CVD risk % is decreasing from 82% in 2012 gradually down to 51% in 2020 with a linear decreasing speed of 4.3% per year. The year 2014 is the turning point.

The figure (Figure 3) depicts his risk probability % of having a CKD (kidney). His CKD risk % is decreasing from 69% in 2012 gradually down to 35% in 2018 through 2020 with a linear decreasing speed of 5.8% per year. The year 2013 as the turning point.

The figure (Figure 4) depicts a table that lists all the numerical values of MI %, CVD risk %, CKD risk %, and beta cells %.

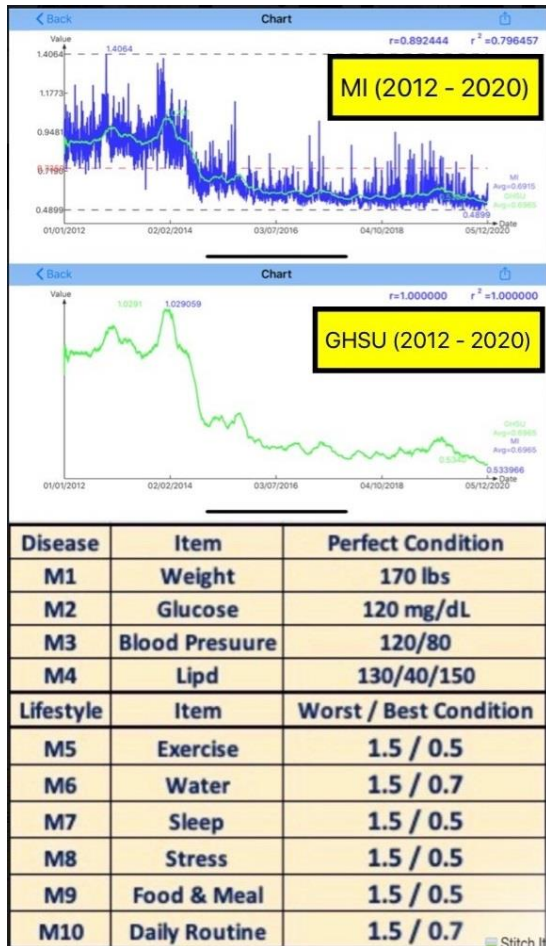


Figure 1: Metabolism index (2012–2020) and measurement standards (M1–M10).

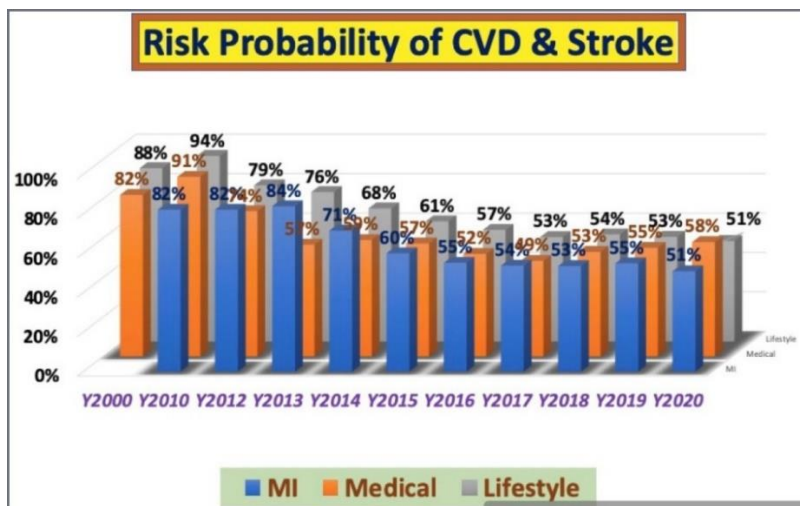


Figure 2: Probability risk % of having a CVD or stroke (2012–2020).

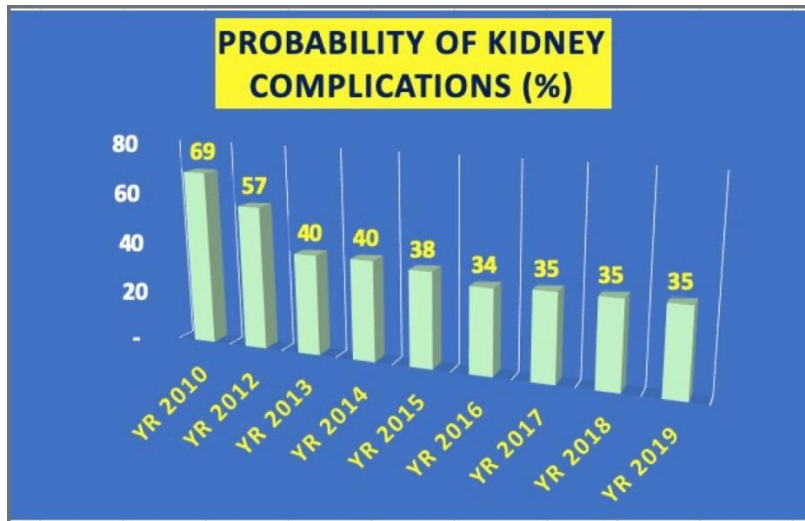


Figure 3: Probability risk % of having a CKD (2012–2020).

| Year | MI % | CVD risk % | CKD risk % | Beta cells % |
|-------|------|------------|------------|--------------|
| Y2012 | 91 | 82 | 57 | 65 |
| Y2013 | 94 | 84 | 40 | 65 |
| Y2014 | 78 | 71 | 40 | 64 |
| Y2015 | 64 | 60 | 38 | 61 |
| Y2016 | 59 | 55 | 34 | 59 |
| Y2017 | 57 | 54 | 35 | 58 |
| Y2018 | 57 | 53 | 35 | 58 |
| Y2019 | 58 | 55 | 35 | 57 |
| Y2020 | 54 | 51 | 33 | 56 |

Figure 4: Table of MI %, CVD risk %, CKD risk %, and beta cells (2012–2020).

The figure (Figure 5) shows the calculation table of beta cells % from both glucose and HbA1C.

Furthermore, in the figure (Figure 6), three additional interesting discoveries are observed:

- The correlation between MI % and CVD % is 99.9% while the correlation between MI % and CKD % is 78.2%. Both correlations are quite high (greater than 50%) which indicates that metabolism conditions indeed cause the risk probabilities of having both heart/brain and kidney complications.
- He further delves into the question of why there is a gap between these two correlation coefficients. It is obvious that 99.9% of the damage on the heart and brain’s arteries is 21.7% higher than 78.2% of the damage on the kidney’s micro-vessels. This difference probably indicates a closer and stronger relationship between MI and CVD than MI and CKD. To provide more confirmation regarding this hypothesis, more clinical data analyses will be needed.
- The correlation between MI % and beta cells % is 95.1%, but the actual difference between the beta cell value of 65 in 2012 and 56 in 2020 is only 13.85% over 8.5 years. This difference provides a 1.6% reduction rate per year which is close to his prior findings of the possible self-recovery rate of pancreatic beta cells around 2.3% per year. The small difference of 0.7% is due to many simplification steps used in beta cells % formation in this paper with its main objectives being “trend” and “relationship”.

| Year | Beta cells % | Lab A1C | Daily A1C | Avg A1C | FPG | PPG | Avg Glucose |
|-------|--------------|---------|-----------|---------|-----|-----|-------------|
| Y2012 | 65 | 6.9 | 7.37 | 7.1 | 139 | 128 | 136 |
| Y2013 | 65 | 6.9 | 7.70 | 7.3 | 135 | 133 | 135 |
| Y2014 | 64 | 6.8 | 7.82 | 7.3 | 128 | 137 | 130 |
| Y2015 | 61 | 6.6 | 7.59 | 7.1 | 121 | 130 | 123 |
| Y2016 | 59 | 6.6 | 6.95 | 6.8 | 117 | 120 | 118 |
| Y2017 | 58 | 6.5 | 6.84 | 6.7 | 120 | 117 | 119 |
| Y2018 | 58 | 6.8 | 6.78 | 6.8 | 114 | 117 | 115 |
| Y2019 | 57 | 6.7 | 6.67 | 6.7 | 113 | 115 | 114 |
| Y2020 | 56 | 6.5 | 6.54 | 6.5 | 112 | 111 | 112 |

Figure 5: Calculation table of beta cells from both average glucose and average HbA1C.

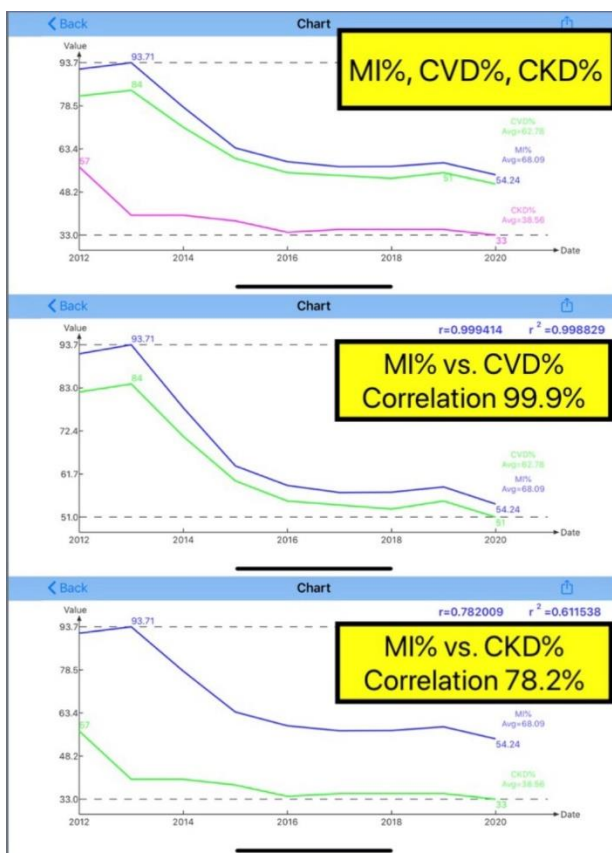


Figure 6: Correlation of MI % vs CVD %, MI % vs CKD %, and MI % vs beta cells (2012–2020).

In the figure (Figure 7), we can see the combined 4 curves of these different cases.

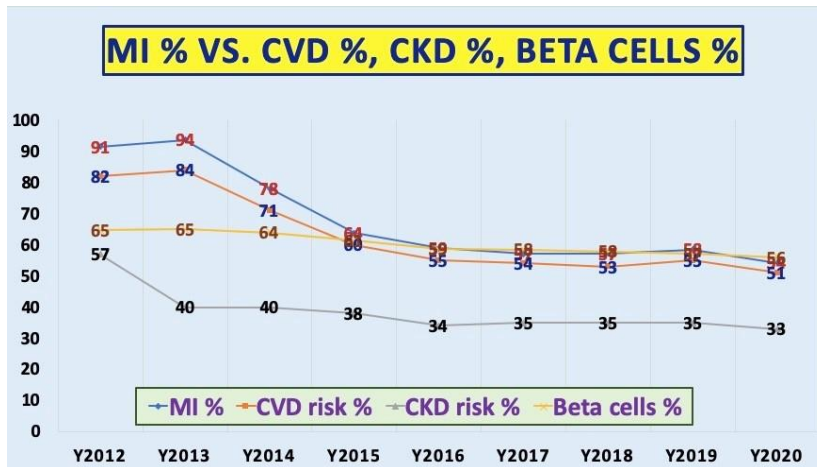


Figure 7: Four curves of MI %, CVD risk %, CKD risk %, beta cells (2012–2020).

Conclusion

These annualized big data analytics using 4 different sophisticated mathematical models for MI, CVD, CKD, and pancreatic beta cells have demonstrated the close relationships between metabolism and two major chronic disease induced complications, CVD/stroke (heart/brain) and CKD (kidney), as well as the beta cells self-recovery rate. By using the GH-method: MPM approach (mathematics, physics, engineering modeling, and computer science), it can certainly attain similar conclusions without lengthy and expensive biochemical experiments performed in a laboratory.

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