

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)

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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 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.

Methods

In 2014, the author applied topology concept, finite-element engineering technique, and nonlinear algebra operations to develop a mathematical metabolism model which contains ten categories, including four basic output categories such as weight, glucose, BP, and other lab-tested data (lipid, ACR, and TSH), and six 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 four prediction models of weight and

glucose (FPG, PPG, and HbA1C), he has 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:

1. For CVD & Stroke: blood flow blockage of arteries due to diabetes and hyperlipidemia; and blood vessel rupture of arteries due to diabetes and hypertension.
2. For CKD: leakage from micro-blood vessels due to diabetes and hypertension.
3. 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 is rather difficult for him at the beginning since he is a professionally trained mathematician, physicist, and engineer, and lack of academic training in biology and chemistry.

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

1. Blockage and rupture of arteries in 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.
2. Kidney complications, including glucose, blood pressure, kidney, glomeruli, bladder, urinary tract, etc. We already know that two main causes of chronic kidney disease are diabetes and hypertension, which are responsible for up to two-thirds of the CKD cases.
3. Elevated glucose were 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 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 HbA1C in order 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.

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 three separate percentages of risk probability (i.e. medical-based, lifestyle-based, and MI-based, but these three 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 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 (GM) 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 three sets of simple formulas for his “Beta-cells %” calculation which are used in his calculation table (Figure 5).

- (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 of “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 other three datasets. After all, all of these data or curves are “relative”, not “absolute”, and for serving the porpoise of investigating their relative relationship.

Regarding these four prominent influential biomarkers, i.e. MI %, CVD risk %, CKD risk %, and Beta cells %, he further calculated three pairs of correlation coefficients using time-series method. Due to the small data volume in this study using “annualized” averaged 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 time-series method in order to conduct its analysis.

During the past 8+ years, all of 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

Figure 1 demonstrates the author’s overall metabolic conditions, including both MI and GHSU for the past eight and one-half years (1/1/2012 - 5/10/2020), along with the detailed descriptions of 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 development of metabolism model and his discipline on implementing the lifestyle management program.

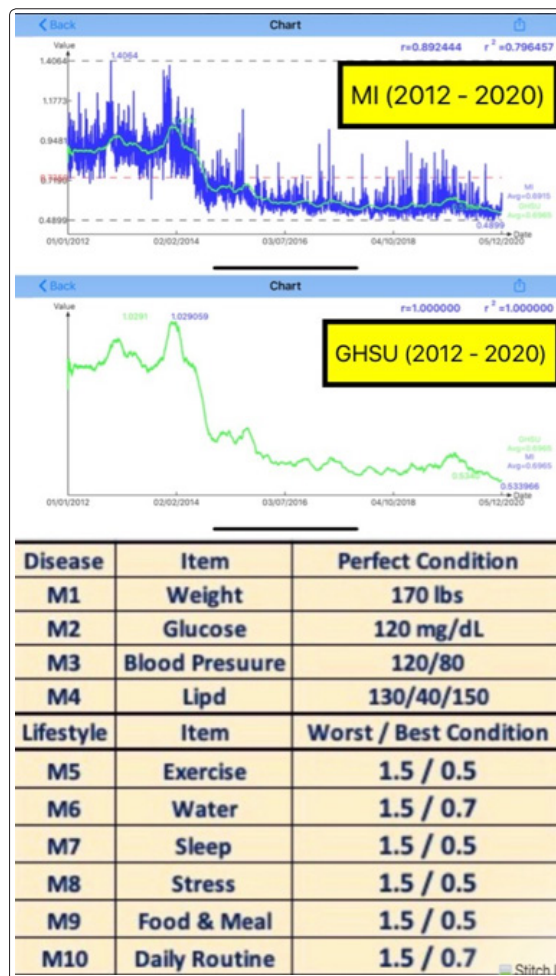


Figure 1: Metabolism Index (2012-2020) & measurement standards (M1-M10)

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 of 2014 is the turning point.

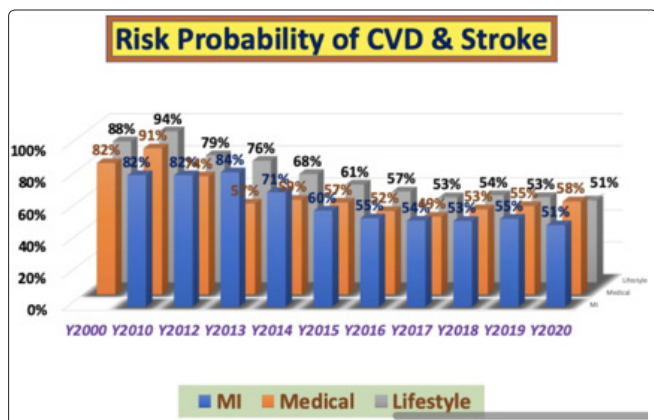


Figure 2: Probability Risk % of having a CVD or Stroke (2012-2020)

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 of 2013 as the turning point.

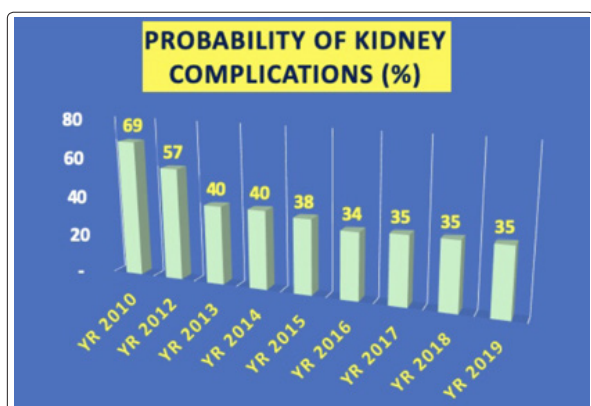


Figure 3: Probability Risk % of having a CKD (2012-2020)

Figure 4 depicts a table which lists all of numerical values of MI %, CVD risk %, CKD risk %, and Beta cells %.

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 %, & Beta cells (2012-2020)

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

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

Furthermore, in Figure 6, three additional interesting discoveries are observed:

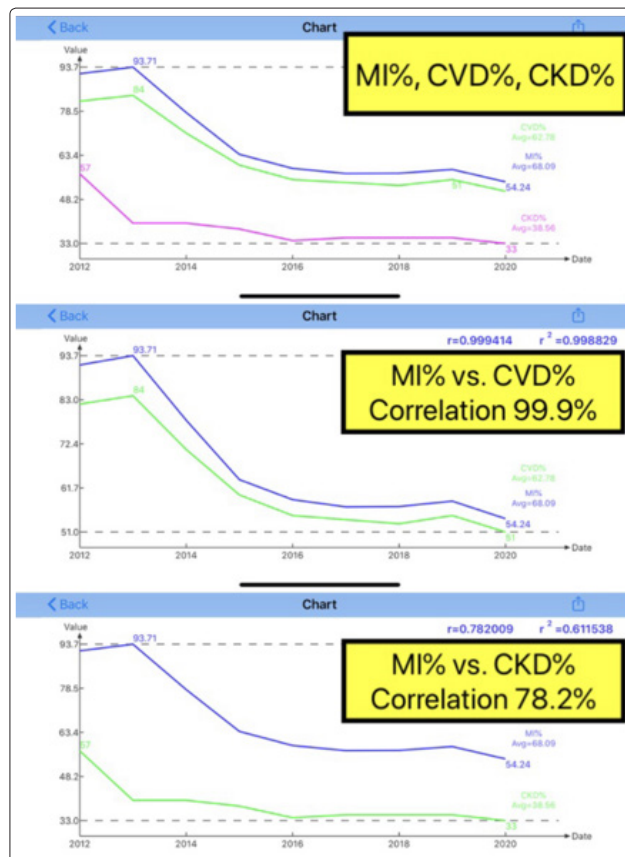


Figure 6: Correlation of MI % vs. CVD %, MI % vs. CKD %, and MI % vs. Beta cells (2012-2020)

(A) 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 causing the risk probabilities of having both heart/brain and kidney complications.

(B) He further delves into the question of why there is a gap between these two correlation coefficients. It is obvious that the 99.9% of the damage on the heart and brain's arteries is 21.7% higher than the 78.2% of the damage on the kidney's microvessels. This difference probably indicates a closer and stronger relationship between MI and CVD than MI and CKD. In order to provide more confirmation regarding this hypothesis, more clinical data analyses will be needed.

(C) 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 1.6% reduction rate per year which is close to his prior findings of 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”.

In Figure 7, we can see the combined four curves of these different cases.

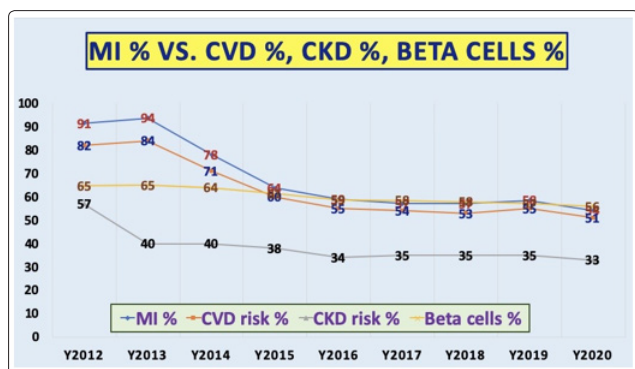


Figure 7: Four curves of MI %, CVD Risk %, CKD Risk %, Beta cells (2012-2020)

Conclusions

These annualized big data analytics using four 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 math-physical medicine approach (mathematics, physics, engineering modeling, and computer science), it can certainly attain similar conclusions without lengthy and expensive biochemical experiments performed in a laboratory [1-6].

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