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Research Article

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Using Starchy Food to Investigate Insulin Resistance State of Pancreatic Beta Cells Based on Viscoplastic Energy Model of GH-Method: Math-Physical Medicine (No. 1047, VMT #445)

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Abstract

Pathophysiologically, insulin resistance (IR) results in decreased glucose uptake by cells and a failure to regulate the liver's overproduction of glucose, leading to elevated fasting plasma glucose (FPG) levels. Additionally, due to IR, the body is unable to efficiently clear the postprandial glucose (PPG) surge from the blood. Obesity is often linked with insulin resistance, creating a cycle where IR can lead to body weight (BW) gain, and excessive BW, in turn, exacerbates IR.

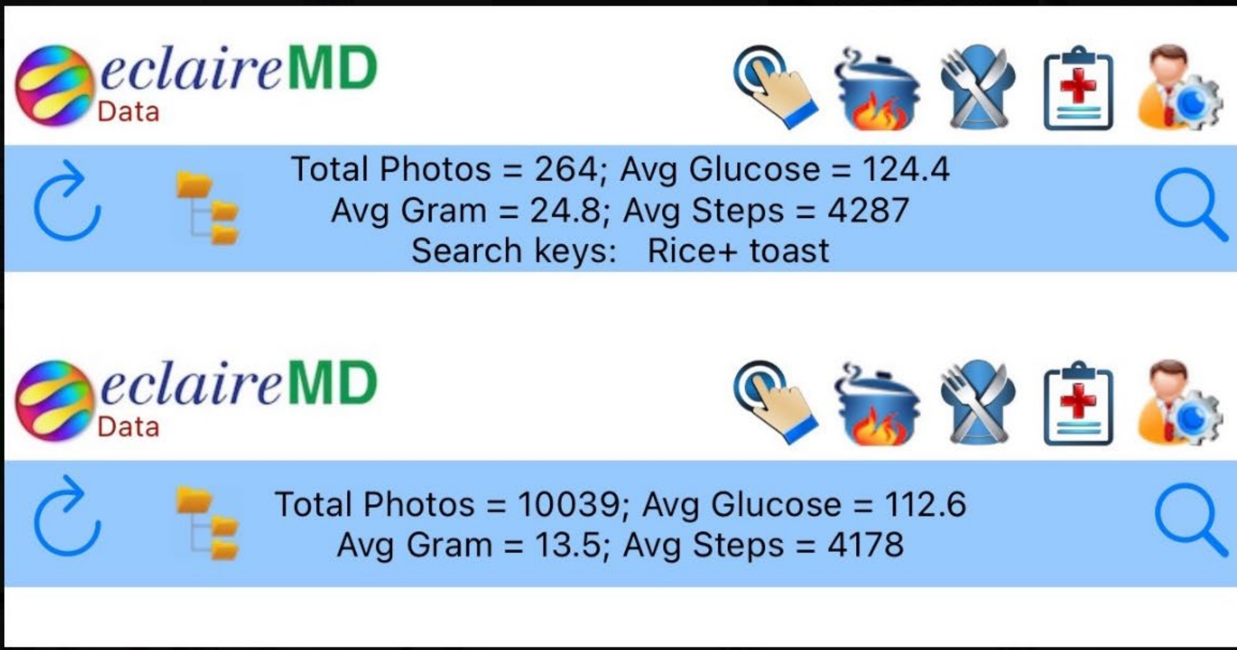
The author's previous research work using the math-physical medicine (MPM) method has already indicated that FPG levels in the early morning are a relatively accurate indicator of IR severity. Moreover, FPG also contributes to approximately 70% to 80% of the PPG level, acting as its baseline, while the remaining 20% to 30% of PPG is primarily influenced by carbohydrate and sugar intake and post-meal physical activity. Based on data collected from 1/1/2015 to 3/28/2024, a high correlation of 83% between body weight (BW) and FPG has also been established.

In this study, the author isolated 264 meals (2.6%) containing "starchy food," such as rice and toast, from a total of 10,039 meals (100%) over nine years (May 1, 2015, to March 28, 2024) for a space-domain Viscoplastic medicine energy (SD-VMT) analysis. This analysis estimated his "linear annual IR% values" based on an average annual reduction rate of 2.8%, from 100% in 2015 to 72% in 2024, with input values of FPG, PPG, and BW from selected meals with starchy foods.

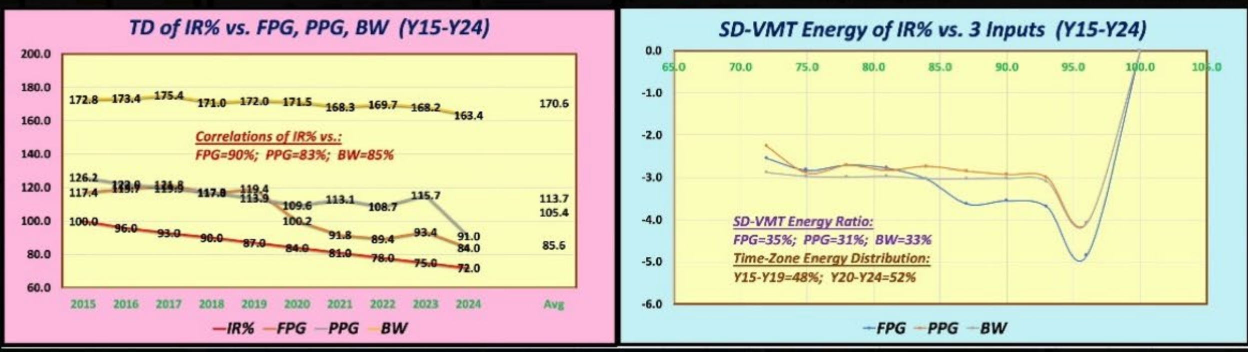
In summary, t*here are three major findings:*
First, the analysis showed three high correlations between IR% and three associated biomarkers:
- FPG: 90%
- PPG: 83%
- BW: 85%

Second, the SD-VMT energy ratios of three biomarkers versus IR% were:
- FPG: 35% (highest)
- PPG: 31% (lowest)
- BW: 33% (middle)
These SD-VMT energy ratios aligned ver well with aforementioned pathophysiological explanations of IR and these three biomarkers.
Third, the VMT time-zone energy distributions were:
- 2015-2019: 48%
- 2020-2024: 52%
This suggests that the IR self-repair and recovery rate is nearly linear on an annual basis over this 10-years period.

Key Message
Over a nearly 10-year span from 2015 to 2024, the author's pancreatic beta cells exhibited a self-repair and recovery rate of 3.8% per year, indicating that it took nearly a decade to self-recover 38% of his damaged insulin-producing capacity of the pancreatic beta cells.



3/29/24					/ 99	/ 120	/ 170	S. Rate	Strain	Stres 1	Stres 2	Stres 3	Hgt 1	Hgt 2	Hgt 3	Area 1	Area 2	Area 3	
IR	IR%	FPG	PPG	BW	FPG	PPG	BW	IR%	IR%	FPG	PPG	BW	FPG	PPG	BW	FPG	PPG	BW	Time-Zone
2015	100.0	117.4	126.2	172.8	1.2	1.1	1.0	0.0	100.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	Y15-Y19
2016	96.0	119.7	122.0	173.4	1.2	1.0	1.0	-4.0	96.0	-4.8	-4.1	-4.1	-2.4	-2.0	-2.0	9.7	8.1	8.2	117.5
2017	93.0	121.8	119.9	175.4	1.2	1.0	1.0	-3.0	93.0	-3.7	-3.0	-3.1	-4.3	-3.5	-3.6	12.8	10.6	10.8	48%
2018	90.0	117.0	117.3	171.0	1.2	1.0	1.0	-3.0	90.0	-3.6	-2.9	-3.0	-3.6	-3.0	-3.1	10.9	8.9	9.2	
2019	87.0	119.4	113.9	172.0	1.2	1.0	1.0	-3.0	87.0	-3.6	-2.9	-3.0	-3.6	-2.9	-3.0	10.8	8.7	9.1	
2020	84.0	100.2	109.6	171.5	1.0	0.9	1.0	-3.0	84.0	-3.0	-2.7	-3.0	-3.3	-2.8	-3.0	10.0	8.4	9.1	Y20-Y24
2021	81.0	91.8	113.1	168.3	0.9	0.9	1.0	-3.0	81.0	-2.8	-2.8	-3.0	-2.9	-2.8	-3.0	8.7	8.4	9.0	129.3
2022	78.0	89.4	108.7	169.7	0.9	0.9	1.0	-3.0	78.0	-2.7	-2.7	-3.0	-2.7	-2.8	-3.0	8.2	8.3	9.0	52%
2023	75.0	93.4	115.7	168.2	0.9	1.0	1.0	-3.0	75.0	-2.8	-2.9	-3.0	-2.8	-2.8	-3.0	8.3	8.4	8.9	
2024	72.0	84.0	91.0	163.4	0.9	0.8	1.0	-3.0	72.0	-2.6	-2.3	-2.9	-2.7	-2.6	-2.9	8.1	7.8	8.8	
Avg	85.6	105.4	113.7	170.6	1.1	1.0	1.0	-2.8	85.6	-3.0	-2.6	-2.8	-2.8	-2.5	-2.7	87.4	77.5	81.9	
Correl.	100%	90%	83%	85%										SD-E:	246.8	35%	31%	33%	
Diff: Max-Min		37.8	35.2																
Difference / 10		3.8	3.5																



Viscoelastic Medicine theory (VMT #445):

1. Introduction

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In this study, the author isolated 264 meals (2.6%) containing "starchy food," such as rice and toast, from a total of 10,039 meals (100%) over nine years (May 1, 2015, to March 28, 2024) for a space-domain Viscoplastic medicine energy (SD-VMT) analysis. This analysis estimated his “linear annual IR% values” based on an average annual reduction rate of 2.8%, from 100% in 2015 to 72% in 2024, with input values of FPG, PPG, and BW from selected meals with starchy foods.

2. Biomedical and Engineering or Technical Information

The following sections contain excerpts and concise information on meticulously reviewed by the author of this paper. The author has adopted this approach as an alternative to including a conventional reference list at the end of this document, with the intention of optimizing his valuable research time. It is essential to clarify that these sections do not constitute part of the author's original contribution but have been included to aid the author in his future reviews and offer valuable insights to other readers with an interest in these subjects.

3. Pathophysiological Explanation of Insulin Resistance Versus FPG, PPG and Body Weight

Insulin resistance is a pathophysiological condition where the body's cells become less responsive to the hormone insulin, which is crucial for regulating blood glucose levels. This resistance impacts fasting plasma glucose (FPG), postprandial plasma glucose (PPG), and body weight in several ways:

1. **Insulin Resistance and Fasting Plasma Glucose (FPG):**
 - In a healthy individual, insulin facilitates the uptake of glucose into cells, reducing blood glucose levels. In insulin resistance, the effectiveness of insulin is diminished, leading to reduced glucose uptake by cells.
 - The liver, which normally reduces glucose production in response to insulin, continues to release glucose into the bloodstream despite elevated insulin levels.
 - As a result, blood glucose levels remain high, leading to increased FPG levels, a common indicator used to diagnose diabetes.

2. **Insulin Resistance and Postprandial Plasma Glucose (PPG):**
 - After eating, glucose levels in the blood rise, prompting the pancreas to release insulin to help cells absorb the glucose.
 - In the case of insulin resistance, this post-meal glucose surge is not efficiently cleared from the blood due to the body's reduced sensitivity to insulin.
 - This results in elevated PPG levels, reflecting how the body manages glucose after meals and indicating the severity of insulin resistance.

3. **Insulin Resistance and Body Weight:**
 - Insulin resistance is often associated with obesity, particularly the accumulation of excess visceral fat around the abdomen. This fat type is metabolically active and releases fatty acids, inflammatory cytokines, and other substances that exacerbate insulin resistance.
 - Conversely, increased body weight, especially fat mass, can lead to a greater degree of insulin resistance. The exact mechanism is not fully understood but involves the disruption of insulin signaling pathways due to factors released by excess fat tissue.
 - This bidirectional relationship forms a cycle where insulin resistance can lead to weight gain, and excess body weight further aggravates insulin resistance.

Overall, the pathophysiology of insulin resistance involves complex interactions between metabolic pathways that regulate glucose and lipid metabolism, and its impact on FPG, PPG, and body weight highlights the interconnected nature of metabolic processes in the body.

4. MPM Background

To learn more about his developed GH-Method: math-physical medicine (MPM) methodology, readers can read the following three papers selected from his published 760+ papers.

The first paper, No. 386 (Reference 1) describes his MPM methodology in a general conceptual format. The second paper, No. 387 (Reference 2) outlines the history of his personalized diabetes research, various application tools, and the differences between biochemical medicine (BCM) approach versus the MPM approach. The third paper, No. 397 (Reference 3) depicts a general flow diagram containing ~10 key MPM research methods and different tools.

5. The Author’s Diabetes History

The author was a severe T2D patient since 1995. He weighed 220 lb. (100 kg) at that time. By 2010, he still weighed 198 lb. with an average daily glucose of 250 mg/dL (HbA1C at 10%). During that year, his triglycerides reached 1161 (high risk for CVD and stroke) and his albumin-creatinine ratio (ACR) at 116 (high risk for chronic kidney disease). He also suffered from five cardiac episodes within a decade. In 2010, three independent physicians warned him regarding the need for kidney dialysis treatment and the future high risk of dying from his severe diabetic complications.

In 2010, he decided to self-study endocrinology with an emphasis on diabetes and food nutrition. He spent the entire year of 2014 to develop a metabolism index (MI) mathematical model. During 2015 and 2016, he developed four mathematical prediction models related to diabetes conditions: weight, PPG, fasting plasma glucose (FPG), and HbA1C (A1C). Through using his developed mathematical metabolism index (MI) model and the other four glucose prediction tools, by the end of 2016, his weight was reduced from 220 lbs. (100 kg) to 176 lbs. (89 kg), waistline from 44 inches (112 cm) to 33 inches (84 cm), average finger-piercing glucose from 250 mg/dL to 120 mg/dL, and A1C from 10% to ~6.5%. One of his major accomplishments is that he no longer takes any diabetes-related medications since 12/8/2015.

In 2017, he achieved excellent results on all fronts, especially his glucose control. However, during the pre-COVID period, including both 2018 and 2019, he traveled to ~50 international cities to attend 65+ medical conferences and made ~120 oral presentations. This hectic schedule inflicted damage to his diabetes control caused by stress, dining out frequently, post-meal exercise disruption, and jet lag, along with the overall negative metabolic impact from the irregular life patterns; therefore, his glucose control was somewhat affected during the two-year traveling period of 2018-2019.

He started his COVID-19 self-quarantined life on 1/19/2020. By 10/16/2022, his weight was further reduced to ~164 lbs. (BMI 24.22) and his A1C was at 6.0% without any medication intervention or insulin injection. In fact, with the special COVID-19 quarantine lifestyle since early 2020, not only has he written and published ~500 new research articles in various medical and engineering journals, but he has also achieved his best health conditions for the past 27 years. These achievements have resulted from his non-traveling, low-stress, and regular daily life routines. Of course, his in-depth knowledge of chronic diseases, sufficient practical lifestyle management experiences, and his own developed high-tech tools have also contributed to his excellent health improvements.

On 5/5/2018, he applied a continuous glucose monitoring (CGM) sensor device on his upper arm and checks his glucose measurements every 5 minutes for a total of 288 times each day. Furthermore, he extracted the 5-minute intervals from every 15-minute interval for a total of 96 glucose data each day stored in his computer software.

Through the author's medical research work over 40,000 hours and read over 4,000 published medical papers online in the past 13 years, he discovered and became convinced that good life habits of not smoking, moderate or no alcohol intake, avoiding illicit drugs; along with eating the right food with well-balanced nutrition, persistent exercise, having a sufficient and good quality of sleep, reducing all kinds of unnecessary stress, maintaining a regular daily life routine contribute to the risk reduction of having many diseases, including CVD, stroke, kidney problems, micro blood vessels issues, peripheral nervous system problems, and even cancers and dementia. In addition, a long-term healthy lifestyle can even "repair" some damaged internal organs, with different required time-length depending on the particular organ's cell lifespan. For example, he has "self-repaired" about 35% of his damaged pancreatic beta cells during the past 10 years.

6. Energy Theory

The human body and organs have around 37 trillion live cells which are composed of different organic cells that require energy infusion from glucose carried by red blood cells; and energy consumption from labor-work or exercise. When the residual energy (resulting from the plastic glucose scenario) is stored inside our bodies, it will cause different degrees of damage or influence to many of our internal organs.

According to physics, energies associated with the glucose waves are proportional to the square of the glucose amplitude. The residual energies from elevated glucoses are circulating inside the body via blood vessels which then impact all of the internal organs to cause different degrees of damage or influence, e.g. diabetic complications. Elevated glucose (hyperglycemia) causes damage to the structural integrity of blood vessels. When it combines with both hypertension (rupture of arteries) and hyperlipidemia (blockage of arteries), CVD or Stroke happens. Similarly, many other deadly diseases could result from these excessive energies which would finally shorten our lifespan. For an example, the combination of hyperglycemia and hypertension would cause micro-blood vessel's leakage in kidney systems which is one of the major cause of CKD.

The author then applied Fast Fourier Transform (FFT) operations to convert the input wave from a time domain into a frequency domain. The y-axis amplitude values in the frequency domain indicate the proportional energy levels associated with each different frequency component of input occurrence. ***Both output symptom value (i.e. strain amplitude in the time domain) and output symptom fluctuation rate (i.e. the strain rate and strain frequency) are influencing the energy level (i.e. the Y-amplitude in the frequency domain).***

Currently, many people live a sedentary lifestyle and lack sufficient exercise to burn off the energy influx which causes them to become overweight or obese. Being overweight and having obesity leads to a variety of chronic diseases, particularly diabetes. In addition, many types of processed food add unnecessary ingredients and harmful chemicals that are toxic to the bodies, which lead to the development of many other deadly diseases, such as cancers. For example, ~85% of worldwide diabetes patients are overweight, and ~75% of patients with cardiac illnesses or surgeries have diabetes conditions.

In engineering analysis, when the load is applied to the structure, it bends or twists, i.e. deform; however, when the load is removed, it will either be restored to its original shape (i.e, elastic case) or remain in a deformed shape (i.e. plastic case). In a biomedical system, the glucose level will increase after eating carbohydrates or sugar from food; therefore, the carbohydrates and sugar function as the energy supply. After having labor work or exercise, the glucose level will decrease. As a result, the exercise burns off the energy, which is similar to load removal in the engineering case. In the biomedical case, both processes of energy influx and energy dissipation take some time which is not as simple and quick as the structural load removal in the engineering case. Therefore, the age difference and 3 input behaviors are "dynamic" in nature, i.e. time-dependent. *This time-dependent nature leads to a "viscoelastic or viscoplastic" situation.* ***For the author's case, it is "viscoplastic" since most of his biomarkers are continuously improved during the past 13-year time window.***

*Time-Dependent output Strain and Stress of (Viscous Input*Output Rate)*

Hooke's law of linear elasticity is expressed as:

Strain (ε: epsilon)
= Stress (σ: sigma) / Young's modulus (E)

For biomedical glucose application, his developed linear elastic glucose theory (LEGT) is expressed as:

PPG (strain) = carbs/sugar (stress) * GH.p-Modulus (a positive number) + post-meal walking k-steps * GH.w-Modulus (a negative number)

Where GH.p-Modulus is reciprocal of Young's modulus E.

However, in viscoelasticity or viscoplasticity theory, the stress is expressed as:

Stress
= viscosity factor (η: eta) * strain rate (dε/dt)

Where strain is expressed as Greek epsilon or ε.

In this article, in order to construct an "ellipse-like" diagram in a stress-strain space domain (e.g. "hysteresis loop") covering both the positive side and negative side of space, he has modified the definition of strain as follows:

Strain
= (body weight at certain specific time instant)

He also calculates his strain rate using the following formula:

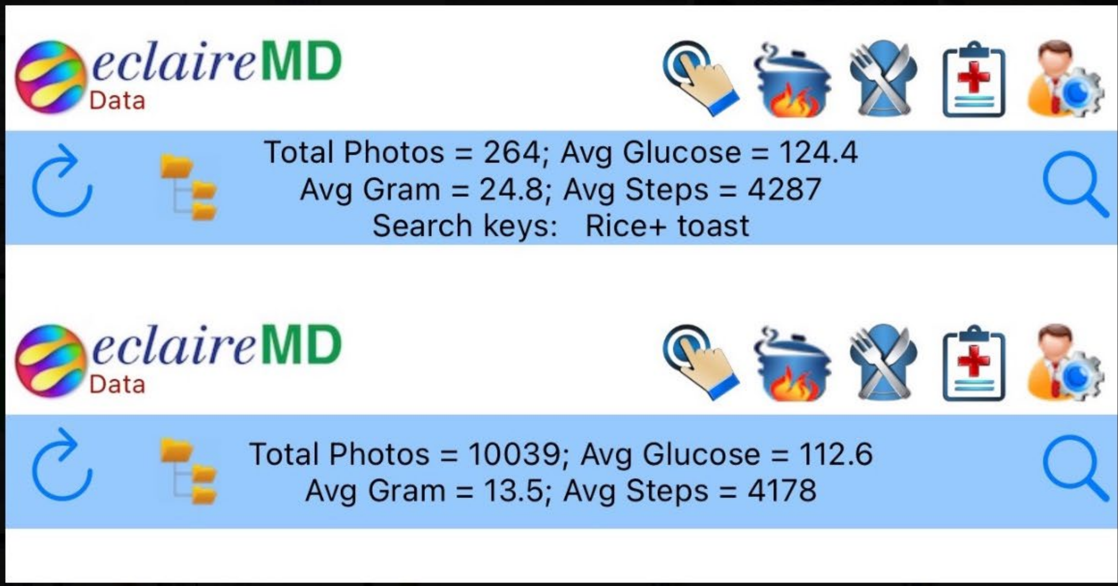
Strain rate
= (body weight at next time instant) - (body weight at present time instant)

The risk probability % of developing into CVD, CKD, Cancer is calculated based on his developed metabolism index model (MI) in 2014. His MI value is calculated using inputs of 4 chronic conditions, i.e. weight, glucose, blood pressure, and lipids; and 6 lifestyle details, i.e. diet, drinking water, exercise, sleep, stress, and daily routines. These 10 metabolism categories further contain ~500 elements with millions of input data collected and processed since 2010. For individual deadly disease risk probability %, his mathematical model contains certain specific weighting factors for simulating certain risk percentages associated with different deadly diseases, such as metabolic disorder-induced CVD, stroke, kidney failure, cancers, dementia; artery damage in heart and brain, micro-vessel damage in kidney, and immunity-related infectious diseases, such as COVID death.

Some of explored deadly diseases and longevity characteristics using the ***viscoplastic medicine theory (VMT)*** include stress relaxation, creep, hysteresis loop, and material stiffness, damping effect ***based on time-dependent stress and strain*** which are different from his previous research findings using ***linear elastic glucose theory (LEGT) and nonlinear plastic glucose theory (NPGT).***

7. Results

Figure 1 shows Data table, TD and SD results.



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2017	93.0	121.8	119.9	175.4	1.2	1.0	1.0	-3.0	93.0	-3.7	-3.0	-3.1	-4.3	-3.5	-3.6	12.8	10.6	10.8	48%
2018	90.0	117.0	117.3	171.0	1.2	1.0	1.0	-3.0	90.0	-3.6	-2.9	-3.0	-3.6	-3.0	-3.1	10.9	8.9	9.2	
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Avg	85.6	105.4	113.7	170.6	1.1	1.0	1.0	-2.8	85.6	-3.0	-2.6	-2.8	-2.8	-2.5	-2.7	87.4	77.5	81.9	
Correl.	100%	90%	83%	85%										SD-E:	246.8	35%	31%	33%	
Diff: Max-Min		37.8	35.2																
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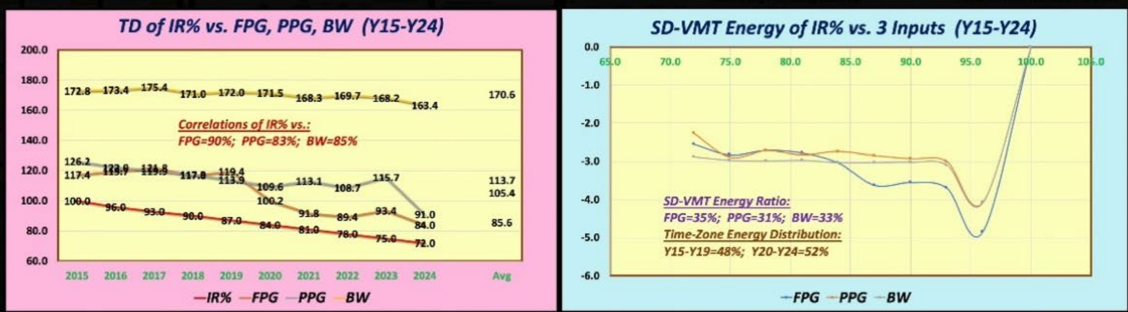

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References

For editing purposes, majority of the references in this paper, which are self-references, have been removed for this article. Only references from other authors' published sources remain. The bibliography of the author’s original self-references can be viewed at www.eclairemd.com.

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