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Comparing a Type 2 Diabetic to Non-Diabetics' Blood Glucose Levels

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Abstract: A statistical approach to modeling Medical Time Series (MTS) using Blood Glucose Levels (BGL) is provided, for the purpose of inferring the characteristics of control theory feedback & feedforward mechanisms underlying Type-II diabetes (T2D). From this perspective, the multiple mechanisms identified may then be related back to the known pathways of T2D in further studies for their probative value in establishing the dominant process(es) in blood glucose level (BGL) dysregulation.

This study identified several BGL regulatory characteristics as stable statistical distributions. When non-diabetics' (ND) BGL is compared to the BGL of a T2D patient with Multiple Sclerosis (MSD), this preliminary study identifies a lack of 'Fine Control' and a lack of 'Gross Control' of BGL. Fine Control is the rate of change of BGL within ±2.75 mg/dL/min and Gross Control is when this rate of change is > |30.0| mg/dL/min.

In ND, the BGL probability distribution is Gamma (α = 43.3663, β = 10.9212), confirming BGL control/regulatory processes that maintain the normal BGL range. This Gamma establishes a characteristic sensitivity and responsiveness of ND BLG regulation based on a sample of 201 ND patients which can be used as a reference for other studies. In the sample T2D patient, the BGL probability distribution is also Gamma (α = 10.921, β = 12.4693) but has lowered mode, a distribution that is widened and shifted toward high glucose.

Inferred from this change in Gamma is a lowered 'sensitivity' to small decreases in BGL demand, compared to the 'sensitivity' to small increases in BGL demand, identifying a 'sensitivity asymmetry'. This small difference in 'sensitivity', and therefore in the body's BGL regulatory response, is shown by simulation to produce sustained high BGL. Sensitivity in this context does not imply a dominant pathological or regulatory mechanism in T2D but is a statistical characterization of T2D. Therefore, the statistical sensitivity does not by itself distinguish between histological defects, such as for example the fate of pancreatic beta cells, and non-histological factors, for example the impact of SNO-CoA-assisted nitrosylase.

A diurnal property of BGL was identified. BGL is either in an insulin dominance phase or a glucagon dominance phase, with macro level insulin dominance between 8:30 pm to 6:30 am and macro level glucagon dominance between 6:30 am to 8:30 pm. At the micro level this phase dominance is observed in the periods between measurements.

Keywords: *Type 2 Diabetic, Blood Glucose, Multiple Sclerosis, Stress.*

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I. INTRODUCTION

Diabetes [1], often described as chronic high blood sugar, is a disease identified as the dysregulation of blood glucose levels (BGL) outside of the boundaries shown in Table 1, [2]. Additionally [1], it is often described in terms of the presence of insulin resistance, or [3] "inability of insulin to accomplish its action". Statistical analysis was used to identify an alternate view.

Table 1 Adult Diabetic Condition in terms of Blood Glucose Levels (BGL)

Diabetic Condition	Fasting Plasma Glucose (FPG)	Plasma Glucose 2 hours after 75 g Glucose Load (2hPG)
Diabetic	>= 126 mg/dL	>= 200 mg/dL
Non-Diabetic	< 110 mg/dL	< 140 mg/dL

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Defining a medical time series (MTS) as medical data that has been collected over a sufficiently long period, a major objective of this paper is to detail how MTS should be analyzed. Current time series analyses [4] is well researched and focused on forecasting future outcomes.

Unlike current time series techniques, the purpose of MTS analyses is to determine the operating characteristics of the underlying process so that one can discover and address abnormalities in these processes as presented in this paper. One could build MTS forecasting models once the current pathways [5] are converted into feedback and feedforward loops.

How one analyses MTS is dependent upon the data being collected. For example, with Multiple Sclerosis [6] one could establish the relationship between emotional distress and symptom sets and propose a disease meta model. In another study, CGM [7] was used to study postprandial glucose, and variations between individuals to determine an analytical framework for glycemic subphenotypes.

In this study, the MTS for non-diabetics from publicly available studies is compared with the limited MTS from a Type 2 diabetic wearing a continuous monitoring device (FreeStyle Libre II).

The statistical method to determine best fit probability distributions is Wilcoxon Regression [8] based on the Wilcoxon 2-Sample Test (see Methodology: Statistical Methods section).

➢ *The Findings of this Initial Study Include:*

- *Autonomic Regulation:*

BGL is being altered over a vast expanse of tissues at any moment, and the analysis confirms autonomic regulation of BGL in both Non-Diabetics (ND) and Type 2 Diabetics (T2D). In ND, the body has a sophisticated control/regulatory system that returns BGL to a safe range at any instant, while able to radically alter BGL to satisfy the body's demands. Thus, both lowering BGL when there is excess BGL and increasing BGL when there is a deficiency of BGL are going on at variable "pump rates" (rate of change of BGL) all the time.

- *Diurnal Phase Dominance:*

This paper proposes BGL is either in an insulin dominance phase or a glucagon dominance phase. At a macro level, insulin dominance occurs between 8:30 pm to 6:30 am while glucagon dominance occurs from 6:30 am to 8:30 pm. At the micro level this phase dominance is observed in the time between measurements i.e. BGL can increase or decrease between measurements. This is true in both ND and T2D patients.

- *Sensitivity:*

It may be inferred from the graphs that the dysregulation of blood sugar in T2D may be caused by a loss of sensitivity in the body's innate regulation of blood glucose levels.

- *Symmetry:*

The statistical analysis indicates that in non-diabetics, the lowering and raising of BGL is ongoing and 'symmetrical' in response to the body's autonomic regulatory needs and the required BGL at that moment.

- *Symmetry of Regulation in ND:*

The statistical analysis indicates that in ND, the lowering and raising of BGL is 'symmetrical' in response to the body's autonomic regulation of BGL, indicating that the body can as effectively down-regulate BGL, as it can up-regulate BGL at any moment. The symmetry is lost in T2D.

- *Asymmetry of Regulation in T2D:*

In T2D, there is a lowered sensitivity to small decreases in BGL demand, compared to the sensitivity to small increases in BGL demand, identifying a 'sensitivity asymmetry'. This small difference in sensitivity, and therefore in the body's BGL regulatory response, is shown to produce sustained high BGL.

- *Indeterminacy of Measurement Versus Response:*

The data does not distinguish whether BGL dysregulation is due to the inability to measure BGL as accurately as in ND or the inability to respond to small changes required to decrease BGL. Under control theory, measurement would be considered an input, and response would be an output. However, it will be shown that for this T2D patient, is able to measuere BGLs but not respond correctly.

- *Control/Regulation of BGL:*

The regulation of BGL is achieved by altering the rate of change of BGL. That is the rate of change, Pump (1), is the primary mechanism for BGL regulation. Pump (1) is defined as the change in BGL (mg/dL/min) between two consecutive measurements over the time between these two measurements. Similarly, Pump (10) another mechanism to determine maximum and minimum BGL gradients with respect to time over 10 measurements (about an hour) i.e. using the minimum and maximum BGLs observed over 10 measurements.

- *Statistical Definition of T2D:*

This suggest that high BGL is a symptom of a subtler cause, that of the sensitivity asymmetry. That is, T2D is asymmetric BGL regulation sensitivity that is evidenced as high BGL.

- *Shifting Shapes:*

The analysis of the hourly BGL Distributions shows that the shapes of these distributions change over a 24-hour period. One infers that BGL processes change over this time period in a consistent manner. Therefore, care is required when using statistical techniques that assume a stable underlying distribution. By elimination the Gamma distribution is the best general fit.

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II. METHODOLOGY

➢ *Data & Metrics Required*

Preliminary findings are based on statistical comparison of a non-diabetic (ND) population [9] represented by 385,292 data points (mean & std dev of time between measurements is 6 mins & 48 mins, respectively), contrasted with Continuous Glucose Monitoring (CGM) of one Type-2 diabetic patient's blood sugar representing 19,419 data points (mean & std dev of time between measurements is 15 mins & 155 mins, respectively). This patient has Multiple Sclerosis and is being treated for diabetes or Multiple Sclerosis Diabetic (MSD). The Type-2 diabetic data was captured by a wearable Freestyle Libre II blood sugar with readings downloaded to an iPhone, representing 6 months of data capture. The findings provide justification for more extensive exploration with a larger Type-2 diabetic sample population.

➢ *The Analyses are Performed & Metrics Required are*

- *Diurnal Analysis:*

The statistical behavior of BGL over a 24-hour period, and includes 1, 2 and 3 standard deviations from the mean. It is based on the standard deviations for that specific hour of the day or night as the daytime "active" and nighttime "passive" metabolic processes would not be the same.

- *BGL Probability Distributions:*

Probability distributions of the measured BGL (mg/dL) over the entire data, consisting of 19,419 MSD and 385,292 ND raw data points.

- *Change (1):*

Change in BGL (mg/dL) between two consecutive measurements. "(1)" represents one measurement apart. Both probability distributions and diurnal behavior are documented.

- *Pump (1):*

Change in BGL (mg/dL/min) between two consecutive measurements over the time between these two measurements. "(1): represents one measurement apart over the time taken between these consecutive measurements. Both probability distributions and diurnal behavior are documented.

- *Pump (10):*

To determine the steepest negative and positive gradients in BGL as BGL is always changing, i.e. increasing, or decreasing. Change in BGL (mg/dL/min) between maximum and minimum BGL within 10 consecutive measurements. This produces positive (+ve) and negative(-ve) gradients when minimum is earlier or later than maximum, respectively. Both probability distributions and diurnal behavior are documented.

➢ *Methodology: Statistical Methods*

There are many statistical algorithms that can be used to determine the best fit of the data to a model. The most common are the variations on multivariate regression, with or without transformations, accompanied by a goodness-of-fit (GOF) metrics such as R^2, p-levels, correlations, to name a few. In this study, Wilcoxon Regression [8] was used, as regression results can be misleading if two conditions are not met;

- The errors must be Normally distributed.
- Heteroscedasticity must not be present, or errors are correlated to some factors (usually x-axis factor).

The author's experience suggests that these two conditions are not often met, thus it does not matter what the value of the GOF metric is, if the technique breaks down, the results are no longer valid.

Wilcoxon Regression [8] based on the Wilcoxon Two-Sample Test, is less sensitive to technique breakdown as it does not assume Normality. It can be summarized as the minimization (1) of the sum of squared error SS_E between each histogram i of data value y_i (2) or the number of data points n_i in bar i over the total data points $n_T = \sum n_i$ and the estimated model value $\hat{y}_i$ fitted to this data as;

$$Min(SS_E) = Min(\sum_i (y_i - \hat{y}_i)^2) \quad (1)$$

$$y_i = \frac{n_i}{n_T} \quad (2)$$

Note, don't forget to offset x_i to middle of the bar i and if you are using MS Excel, to remove from your data set, y_i which do not have values i.e. are null. The optimization problem then, is to determine the parameter values of the $\hat{y}_i$ function to minimize SS_E. Since each histogram bar has equal weights, this technique gives a better fit for distribution tails than regression. Regression weighs the data by the frequency n_i of the data which is much greater around the mode than at the tails. If the Wilcoxon Regression fit is not as good as expected, due to Type 2 errors, one can re-weight the histogram bars i by n_i or (3),

$$Min(SS_E) = Min(\sum_i n_i (y_i - \hat{y}_i)^2) \quad (3)$$

There are two additional considerations. (a) The number of bars in a histogram can bias the determination of the function. For example, with 5 bars, the data appears to fit many different distribution, and with 1,000 bars, that data appears to be a uniform distribution.

The "blind" fitting (without visual inspection or a theoretical basis) of data to a model can result in many different types of models fitting the data. Usually, about 25 bars per histogram would be sufficient, but for this large non-diabetic (ND) and diabetic (MSD) BGL data, histogram of 281 bars were required to correctly model the tails.

The second consideration (b) is having a good histogram representation of the data and the fitted model. The number of bars selected for a histogram alters the height of the model distribution. Less bars increases the number of data points per bar, and vice versa. To account for the number of bars in a histogram, the Histogram Compression H_C parameter is introduced. For gamma distribution (4), H_C is

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introduced in (5) to account for the changing heights because of the different number of bars.

$$\hat{y}_i = \frac{x_i^{\alpha-1} e^{-\beta x_i} \beta^{\alpha}}{\Gamma(\alpha)} \quad (4)$$

$$\hat{y}_i = H_C \frac{x_i^{\alpha-1} e^{-\beta x_i} \beta^{\alpha}}{\Gamma(\alpha)} \quad (5)$$

The minimization of (1) or (3) given (5) gives the best value for the distribution parameters, with the shape parameter, α and the rate parameter, β. The Histogram Compression H_C parameter can then be discarded as it is an artifact of the number of bars and not of the data. In this study, MS Excel's GRG Non-Linear Solver was used to determine the minimum SS_E by determining H_C, α, and β required. See Figure 5 as an example as to how well the models fit the actual data.

III. PRIMARY RESULTS OF THE STATISTICAL ANALYSES

This section documents the primary results of this blood glucose study. The non-diabetic data was provided by [9] JAEB Center For Health Research (https://public.jaeb.org/dataset/559). It is based on 201 patient data comprising 385,292 raw data points. The diabetic data was provided by a Multiple Sclerosis patient who is diabetic (MSD). It is based on 1 patient's data comprising 19,419 raw data points.

Of the 40 results the 6 most important are presented here. The full results are available here https://drive.google.com/file/d/1PO5iDuI9yYffbPwJDzdNUcx0jSgJnP_T/view?usp=sharing

➢ *BGL Probability Distributions*

Table 2 summarizes the differences between ND BGL and MSD BGL. See Figures 1. The BGL distributions obey a Gamma Distribution, but the ND & MSD Gamma Distributions are not the same as confirmed by the distribution parameters, 98% operating ranges and min and max observed BGL values.

Table 2 Comparison of ND & MSD Blood Glucose Levels (BGL): BGL Levels

Parameter	ND BGL	MSD BGL
Gamma, α	43.3663	2.2247
Gamma, β	10.9212	12.4693
24-Hour Weighted Average (mg/dL)	99.01	139.78
98% Range (mg/dL)	65.69 < BGL < 133.79	59.00 < BGL < 250.00
Minimum Observed (mg/dL)	38.50	52.00
Maximum Observed (mg/dL)	273.50	356.00
% of Time Hypoglycemic (<70mg/dL)	2.54%	0.0000071%
% of Time Hyperglycemic (>240 mg/dL)	3.30%	1.46%

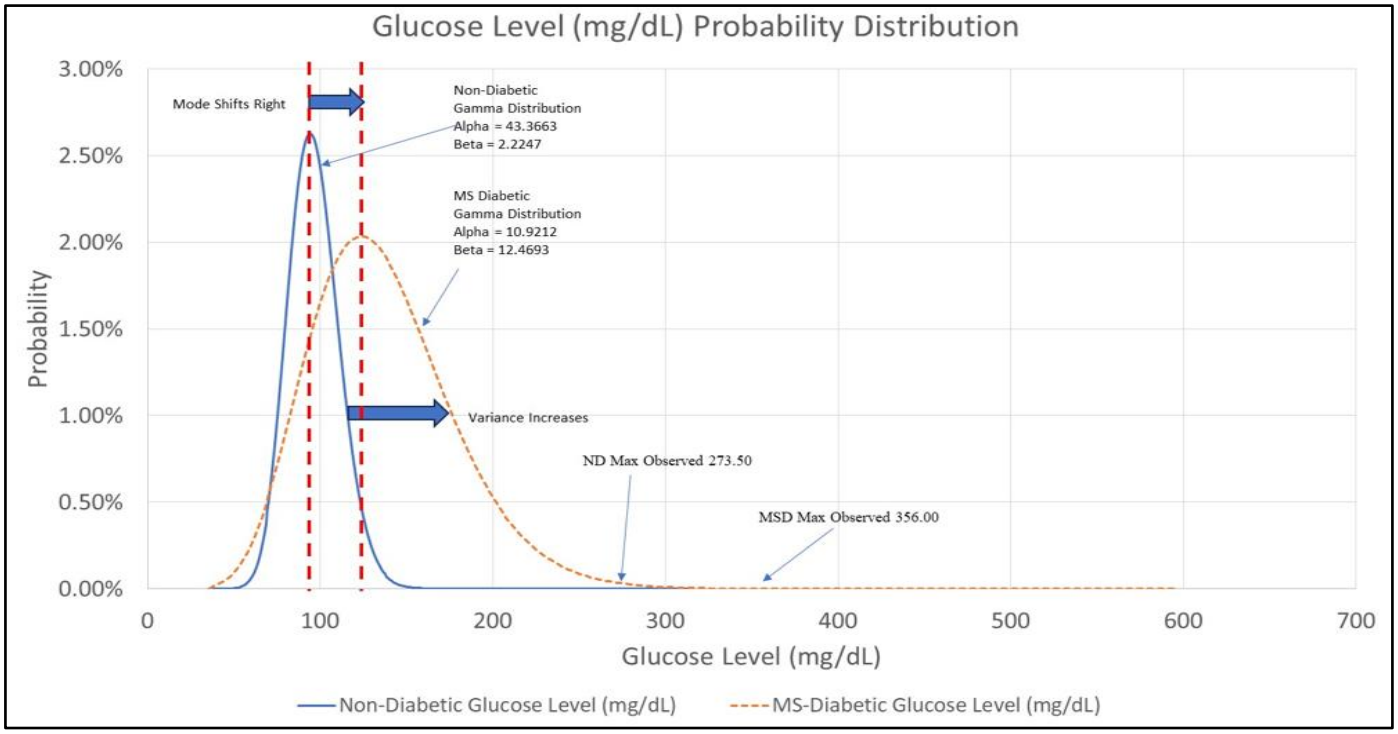

Fig 1 ND & MSD BGL Gamma Distributions

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One observes that even non-diabetics experience hypoglycemia and hyperglycemia. Even though treatment is better than no treatment, some Type 2 Diabetic patients still do not have sufficient control of their BGL.

➢ *Diurnal BGL*

Table 3 summarizes the key difference in the Diurnal BGL between ND BGL & MSD BGL and shown in Figures 2.

Table 3 Comparison of ND & MSD Diurnal BGL

Parameter	ND BGL	MSD BGL
Crest (mg/dL at time)	104.3 at 10:30 pm	(i) 160.34 at 10:30 am (ii) 159.87 at 8:30 pm
Trough (mg/dL at time)	93.5 at 6:30 am	93.5 at 6:30 am
Smallest observed maximum BGL (mg/dL)	174.50 at 6:30 am	258.00 at 3:30 pm
Largest observed maximum BGL (mg/dL)	273.50 at 6:30 pm	356.50 at 11:30 am
Minimum observed BGL (mg/dL)	38.5 throughout the day	52.00 to 70.00
Low mode of the hourly BGL distribution	84.50 at 10:30 am	96.00 at 6:30 am
High mode of the hourly BGL distribution	101.50 at 10:30 pm	174.00 at 6:30 pm

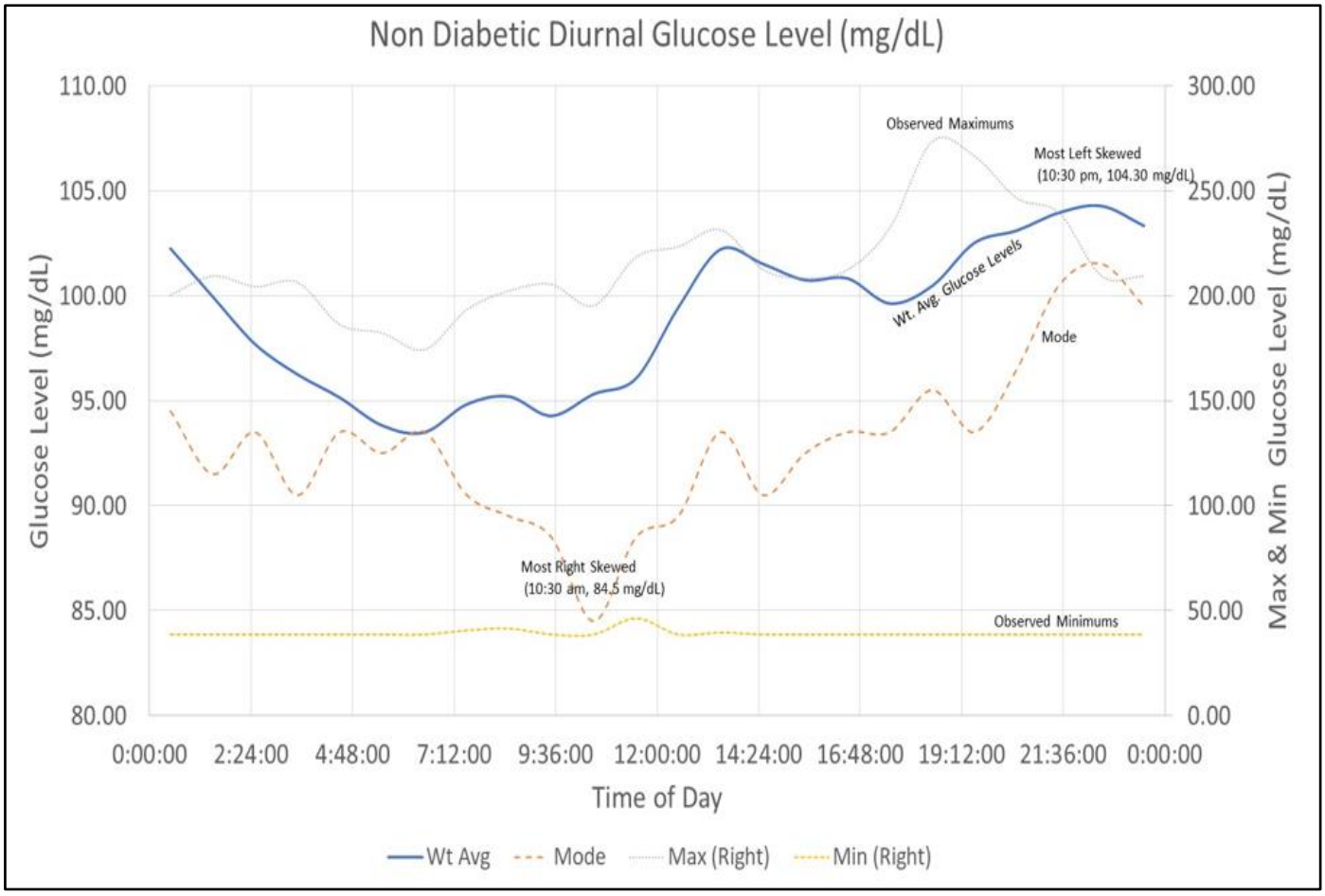

Fig 2 ND Diurnal BGL

Fig. 2, shows that the shape of the hourly ND BGL. That the hourly BGL probability distributions changes over a 24-hour period. For non-diabetics the wt. average low is early morning (6:30 am) at 93.5 mg/dL and the wt. average high is late evening (10:30 pm) at 104.30 mg/dL. It is, most right skewed at 10:30 am (84.50 mg/dL) and most left skewed at 10:30 pm (101.50 mg/dL). Fig. 3 shows that the mode of the hourly MSD BGL distribution shifts to the right in the daytime. It is most right skewed at 6:30 am (96.00 mg/dL) and most left skewed at 6:30 pm (174.0 mg/dL). The MSD wt. average morning (6:30 am) low of 107.67 mg/dL and remains high after 9:00 am between 103.45 and 160.34 mg/dL. That is MSD high BGL occurs between 9:00 am and 11:30 pm and that the BGL regulation is active but offset. Is this because digestion is active?

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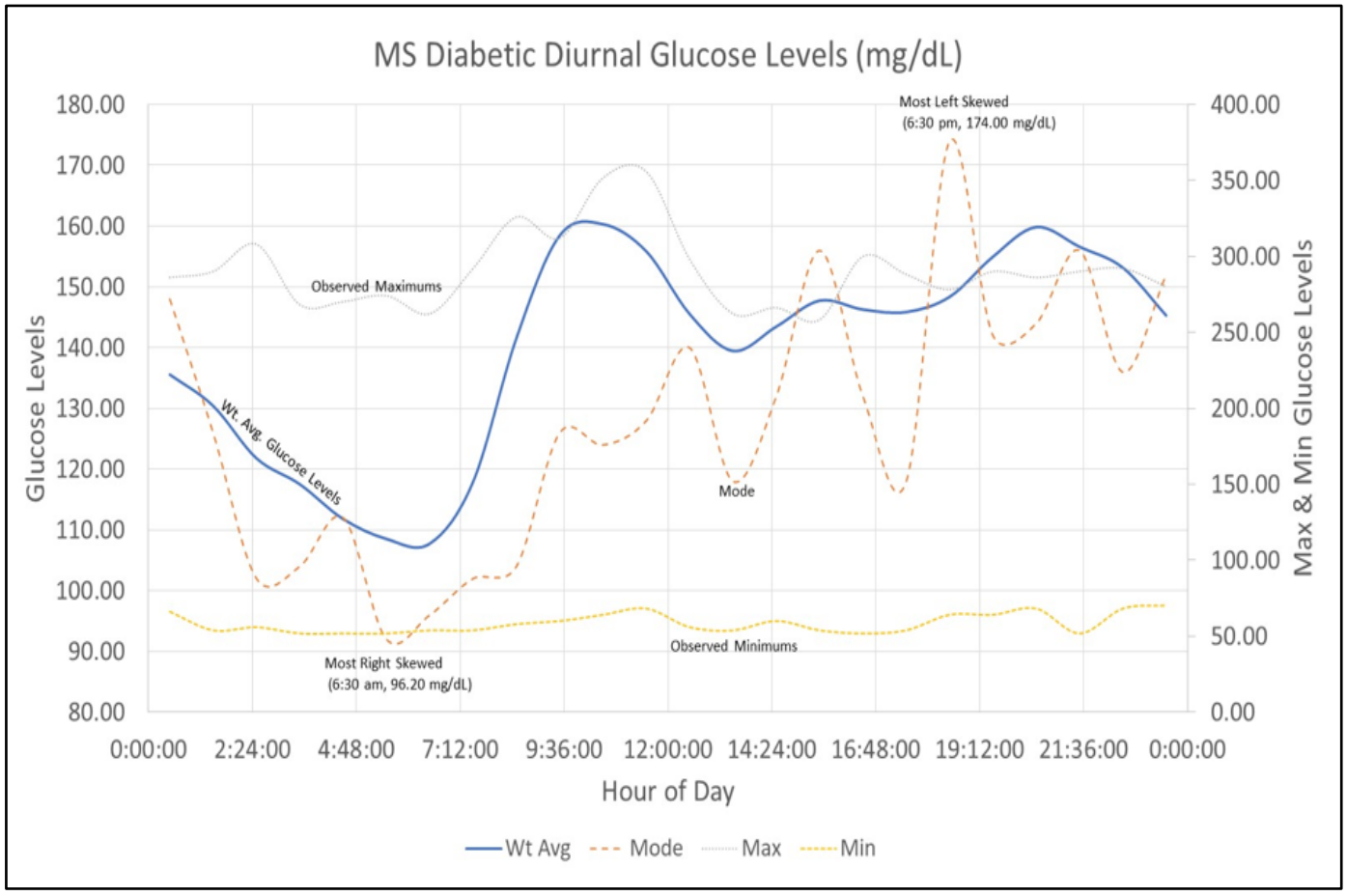

Fig 3 MSD Diurnal BGL

Other diabetics' data shows inverted diurnals (not shown in the data used), where the high is in the morning and the low is in the evening. More investigation of diabetic BGL is required. Both Figures 2 & 3 show that the shape of the hourly BGL probability distributions changes over a 24-hour period as the mode changes with respect to the wt. average.

➢ *Change (1) Observations*

Table 4 summarizes the consecutive change in BGL as two different processes, increase (+ve) and decrease (-ve) in BGL.

Table 4 Comparison of ND & MSD Change (1) BGL

Parameter	ND BGL	MSD BGL
(+) Gamma, α	0.7417	0.9172
(+) Gamma, β	4.0041	8.9509
(-) Gamma, α	0.8282	1.1079
(-) Gamma, β	3.5493	6.6401
98% Change (1) Range (mg/dL)	-14.00 < BGL < +14.00	-34.00 < BGL < +34.00
Minimum Observed Change (1) (mg/dL)	-97.50	-130.50
Maximum Observed Change (1) (mg/dL)	96.50	83.50

Fig. 4 shows the probability distribution of the change in consecutive ND BGL and MSD BGL measurements. For ND the (+ve) changes are similar if not the same as (-ve) changes. That is, the non-diabetic's BGL regulation is symmetrical in both increases and decreases in BGL and occurs all the time almost concurrently. This implies that the body's BGL regulation is realized primarily by continuous small changes to BGL.

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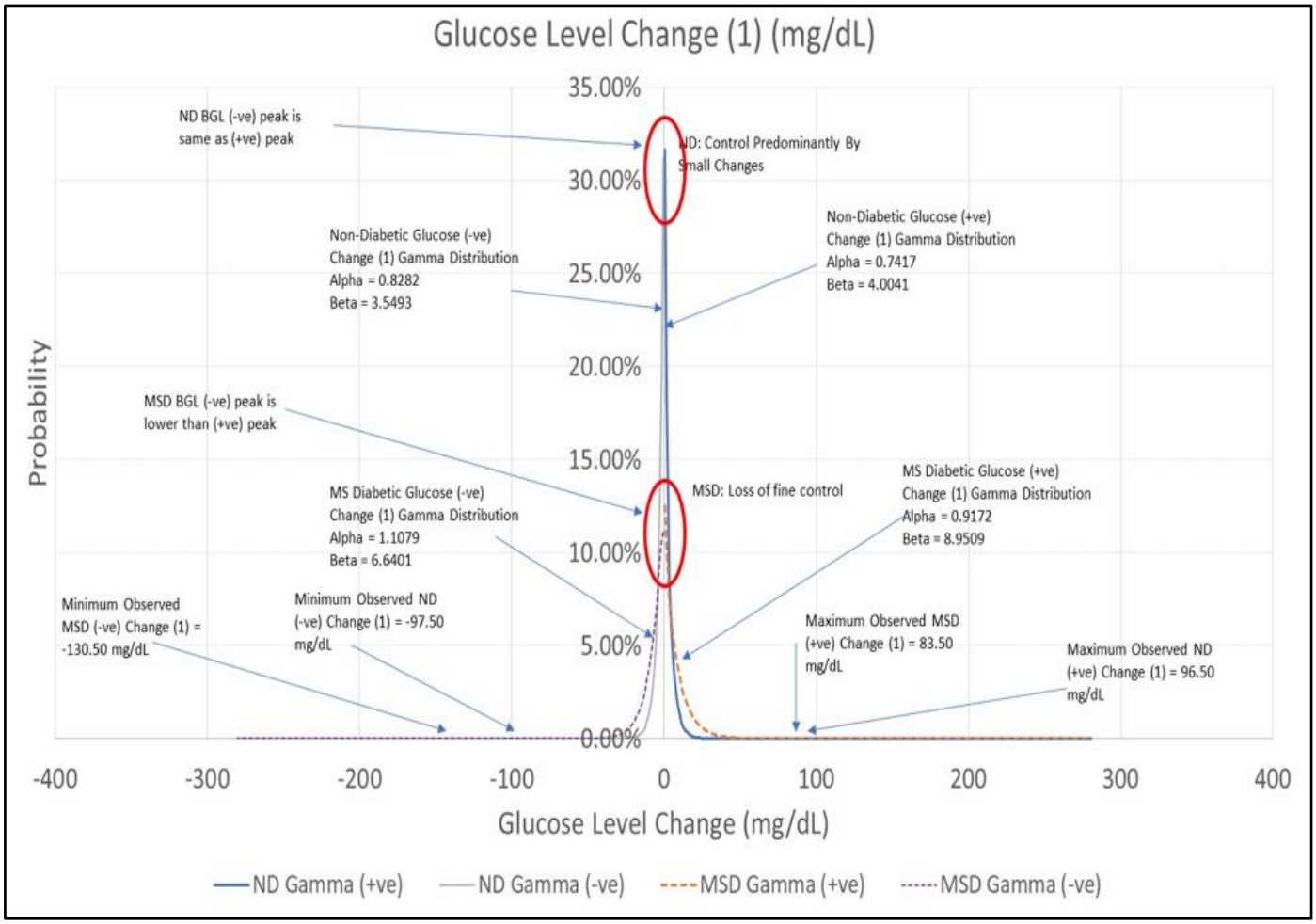

Fig 4 ND & MSD BGL (+ve) & (-ve) Change (1) Probability Distributions

However, for MSD (+ve) changes are not like (-ve) changes. Noting that the (-ve) is lower than the (+ve) close to 0 mg/dL. The maximum and minimum observed changes in MSD BGL are 83.5 mg/dL and -130.5 mg/dL, respectively, and occurs less than 2% of the time. That is, there is substantially more variation in MSD BGL than in ND BGL or reduced BGL control. A Monte Carlo model using Change (1) Gamma distributions shows that without some form of feedback control, the small difference between (+ve) and (-ve) Change (1) is sufficient to cause MSD BGL to approach 3,500 mg/dL after 10,000 measurements, i.e. a primary source of high BGL.

➢ *Pump (1) Observations*

Pump (1) Rate is the change in consecutive BGL measurements divided by the time between consecutive measurements. This is the BGL gradient with respect to time or the first derivative. Table 5 summarizes the key differences between ND and MSD BGL Pump (1).

Table 5 Comparison of ND & MSD Blood Glucose Levels (BGL): Pump (1)

Parameter	ND BGL	MSD BGL
(+) Gamma, α	0.8424	0.8776
(+) Gamma, β	0.8425	0.7730
(-) Gamma, α	0.9291	0.7333
(-) Gamma, β	0.7570	0.7299
98% Pump (1) Range (mg/dL/min)	-2.75 < Pump (1) < +2.75	-4.95 < Pump (1) < +4.95
Minimum Observed Pump (1) (mg/dL/min)	-65.38	-36.97
Maximum Observed Pump (1) (mg/dL/min)	69.38	+33.97

Fig. 5 depicts the probability distribution of (+ve) and (-ve) Pump (1) for ND & MSD BGL. These are the rates (mg/dL/min) of how quickly the BGL regulation can add or subtract glucose from or into the blood.

98% of the time the ND rate is within ±2.75 mg/dL/min. This range is defined as Fine Control. 98% of the time the MSD rate is within ±4.95 mg/dL/min. These ranges provide quantitative boundaries to effect T2D management. Note that non-diabetics have much more frequent small changes in BGL than the MS diabetic.

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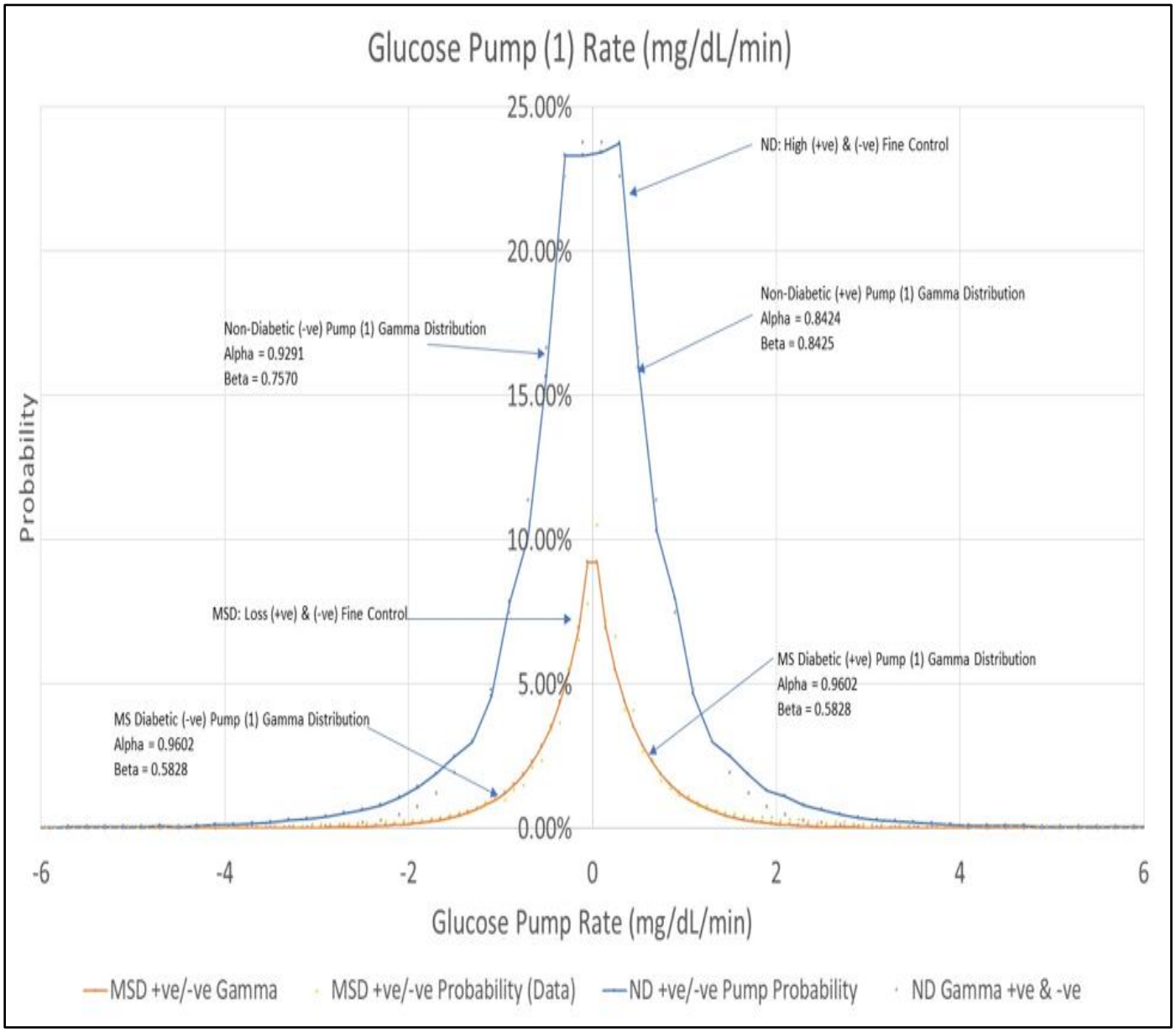

Fig 5 ND & MSD BGL (+ve) & (-ve) Pump (1) Probability Distributions

➢ *Pump (10) Observations*

Table 5 summarizes the key differences between ND and MSD BGL Pump (10) Rates and presented graphically in Figure 6 & 7. Pump (10) Rates determine the maximum and minimum BGL gradients over time or the "extreme case" pump rates the BGL control system can realize, i.e. what can the body realize if it had to? The same MS Excel model was used on all, (+ve), (-ve) for ND & MSD, four data sets.

Table 5 Comparison of ND & MSD (+ve) & (-ve) Pump (10) BGL

Parameter	ND BGL	MSD BGL
(+) Gamma, α	1.3608	1.2471
(+) Gamma, β	0.2673	0.3218
(-) Gamma, α	1.0618	1.4757
(-) Gamma, β	0.2208	0.3202
98% Pump (10) Range (mg/dL/min)	-1.085 < Pump (10) < +2.415	-1.1205 < Pump (10) < +1.6605
Minimum Observed Pump (10) (mg/dL/min)	-6.1800	-2.4688
Maximum Observed Pump (10) (mg/dL/min)	+8.0268	14.000

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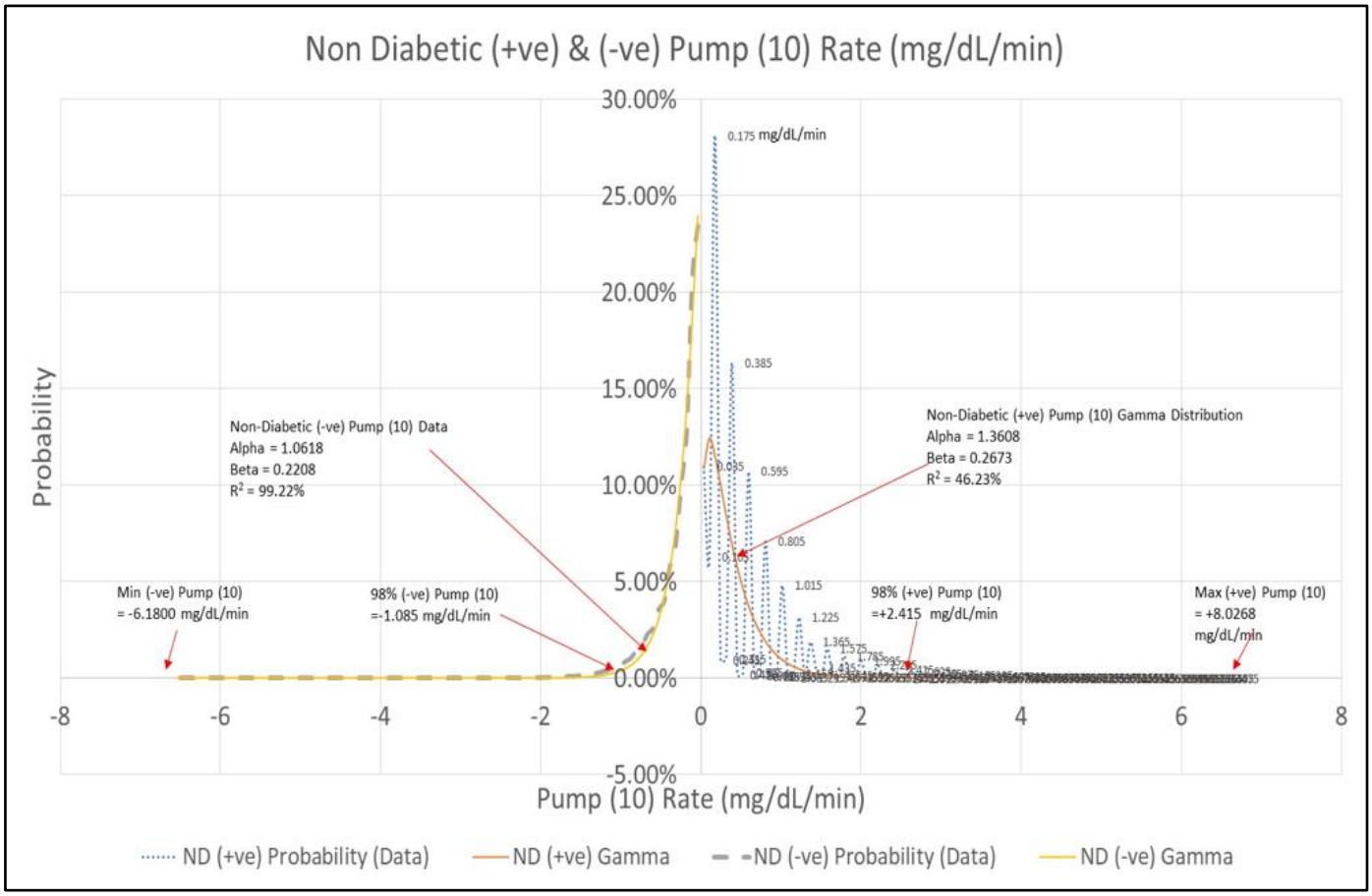

Fig 6 ND BGL (+ve) & (-ve) Pump (10) Probability Distributions

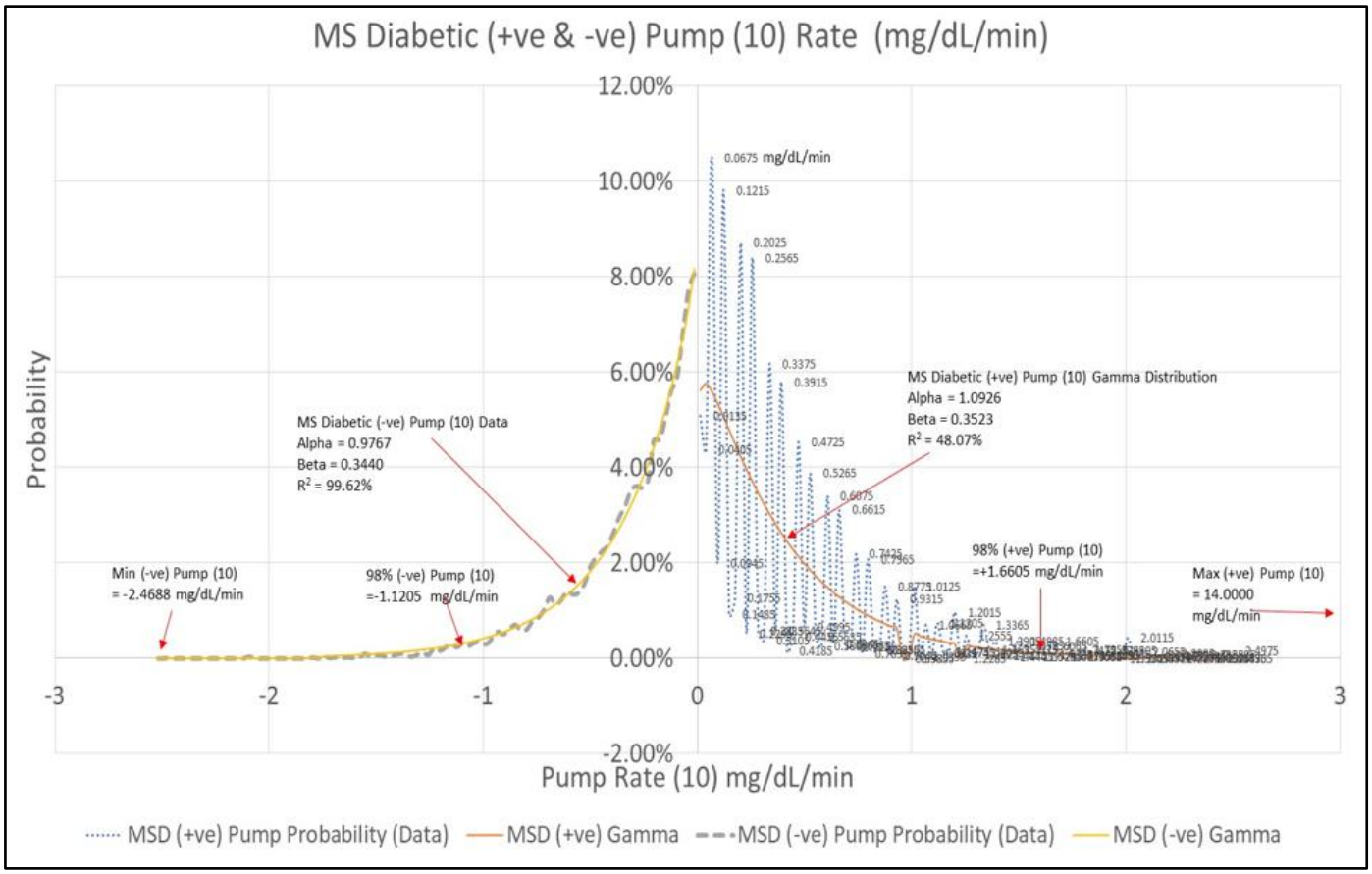

Fig 7 MSD BGL (+ve) & (-ve) Pump (10) Probability Distributions

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Figures 6 & 7 shows that some (+ve) Pump Rates peaks are more likely than others. Oscillations like these are not random processes but in response to an undetermined signals or stimuli. However, (-ve) Pump Rates are more like exponential processes with alpha ≈ 1. One interpretation is that the BGL control system does not know how much BGL is required. If more BGL is required there are subsequent bursts of BGL. The BGL control system then allows the (-ve) Pump Rates to reduce BGLs back to "normal".

IV. MEASUREMENT VERSUS CONTROL

In the Introduction, 7. Indeterminacy of measurement versus response, it was noted that it is possible to distinguish input measurement as opposed to output response in the BGL regulation.

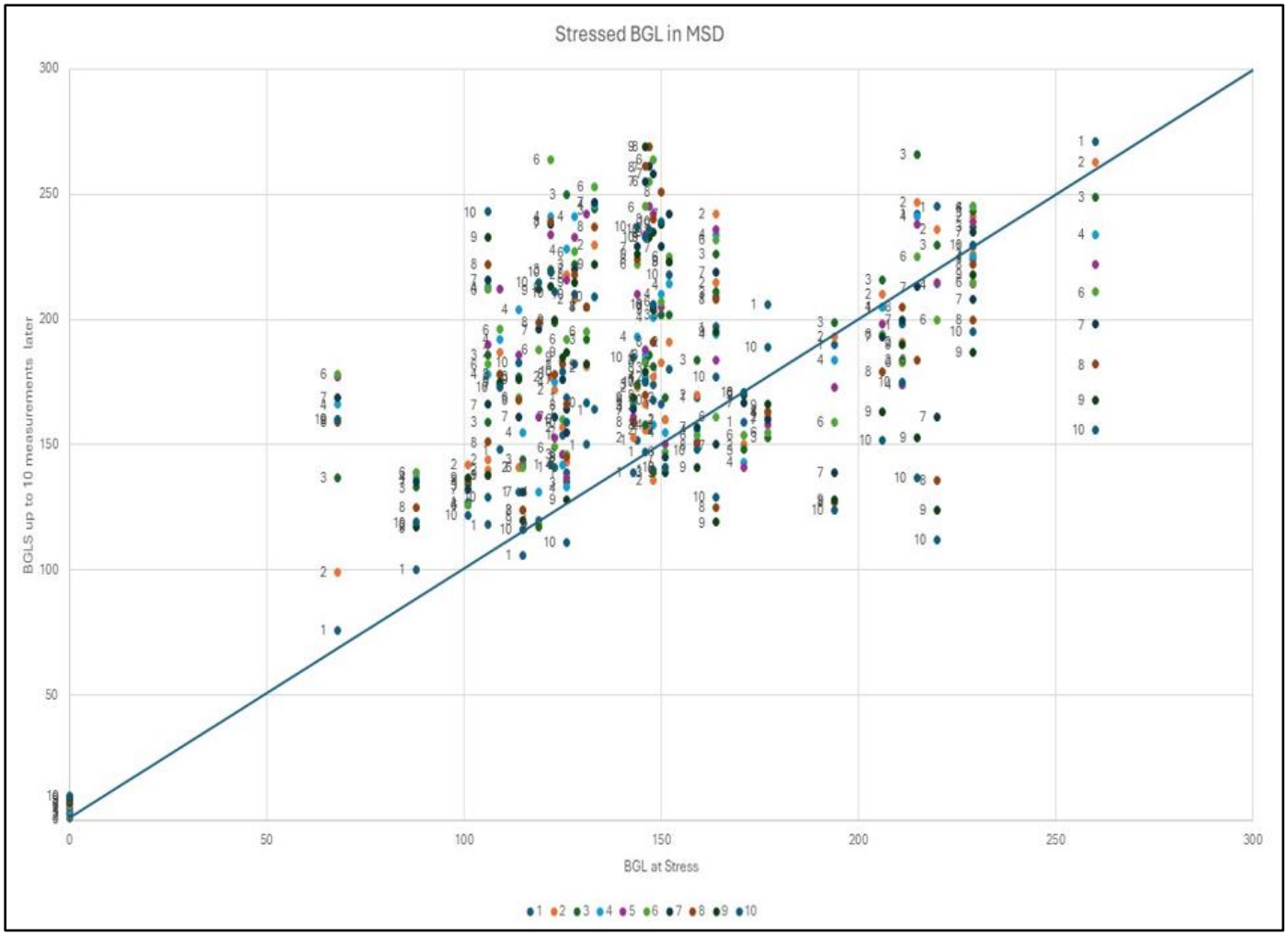

Fig 8 MSD BGL at Stress Versus 10 CGM Measurements (About an Hour) Later.

Fig. 8 shows the BGL of the T2D patient at emotional distress (1 to 10, with 10 being the worst) and about an hour later. This data is from the [6] data. The Fig. 8 shows that in general, when emotional distress is experienced;

- If BGL is low, then the BGL is raised.
- If BGL is high, then BGL remains similar an hour later or even decreases.

This suggested that for this T2D patient, the BGL regulation system is able to determine or measure BGLs and respond accordingly. However, there is no obvious relationship between BGL and emotional distress. One possible inference is that output regulation is not functioning correctly.

V. DISCUSSION

➢ *Comparisons*

Figures 1 & 2 show how the MSD BGL probability distribution has shifted to the right with increased variance. That is, there is a reduction in the diabetic's ability to control BGL even with treatment. Figure1 shows that there are two problems;

- Starts at a higher level: The lowest wt. avg. BGL of this MSD is 107.67 mg/dL compared to ND's 93.48 mg/dL, both at 6:30 am.
- In T2D, Fig. 3, the largest range of dysregulation occurs between 6:30 am and 10:30 am, after which the wt. average BGL remains elevated but somewhat stable.

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Fig. 4 depicts the (-ve) & (+ve) probability distributions of the Change (1) of both ND & MSD BGLs on the same graph. Non-diabetic control of BGL is frequently characterized by small ±14.0 mg/dL consecutive changes compared to the diabetic of ±34.0 mg/dL. That is, the diabetic regulation of BGL is not as sensitive to the body's BGL needs as the non-diabetics.

Fig. 5, shows that this diabetic's ability to lower BGL is substantially less than raising BGL, as MSD BGL (-ve) Pump (1) has more frequent smaller changes than MSD BGL (+ve) by approximately a third of the time (7.18% versus 20.82%).

There are a lot of excellent research into BGL regulation pathway [10, 11] but what is required is to determine when these turn on or off. In summary, one infers that a diabetic's BGL is high because a diabetic is unable to (i) make small reductions in BGL as frequently as is required to match the frequency of small increases, and (ii) from Fig. 7 the diabetic's ability to make large increases in BGL is not matched by similar large decreases.

VI. CONCLUSION

Statistical modeling with Medical Time Series (MTS) of BGL provides valuable insights into type-II diabetes (T2D) processes when compared to non-diabetics. Multiple mechanisms within BGL regulation can be identified that may then be related back to the known pathways of T2D in further studies for their probative value in establishing the dominant process(es) in blood glucose level (BGL) dysregulation on a patient-by-patient basis. Statistical characterization of a T2D patient's BGL regulation can potentially be used in disease management.

This study confirms that BGL regulation is a sophisticated process with feedback and feedforward loops and not just pathways, that maintain robust control of BGLs. The analysis indicates that the dysregulation of blood sugar in T2D may be caused by a loss of sensitivity in the body's innate regulation of BGL.

The statistical analysis indicates that in ND, the lowering and raising of BGL is 'symmetrical' in response to the body's autonomic regulation of BGL, indicating that the body can as effectively down-regulate BGL, as it can up-regulate BGL at any moment. The symmetry is lost in T2D. In T2D, there is a lowered sensitivity to small decreases in BGL demand, compared to the sensitivity to small increases in BGL demand, identifying a 'sensitivity asymmetry'. This small difference in sensitivity, and therefore in the body's BGL regulatory response, is shown to produce sustained high BGL.

A diurnal phase dominance is noted. At the macro level, insulin dominance occurs between 8:30 pm to 6:30 am while glucagon dominance occurs from 6:30 am to 8:30 pm. At the micro level this phase dominance is observed in the time between measurements i.e. BGL can increase or decrease between measurements. This is true in both ND and T2D patients.

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