Collated Distributions: A New Model for Infectious Disease Spread Using Early COVID-19 Data

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Abstract: A new modelling technique, Collated Distributions (CD), is presented, to model Infectious Disease Spread (IDS). This modeling shows that there are 3 probability distributions that one must determine to effectively manage public health, infectability, mortality and survival. Thus, leading to better understanding of Infectious Disease Spread (IDS). The Reproduction Model used in COVID-19 disease spread modeling is shown to be doubtful.

Collated Distributions have implications in many other fields of study such as dissemination of knowledge, and the rise and fall of civilizations.

Keywords: *Covid, Mortality, Survival, Disease, Health Policy.*

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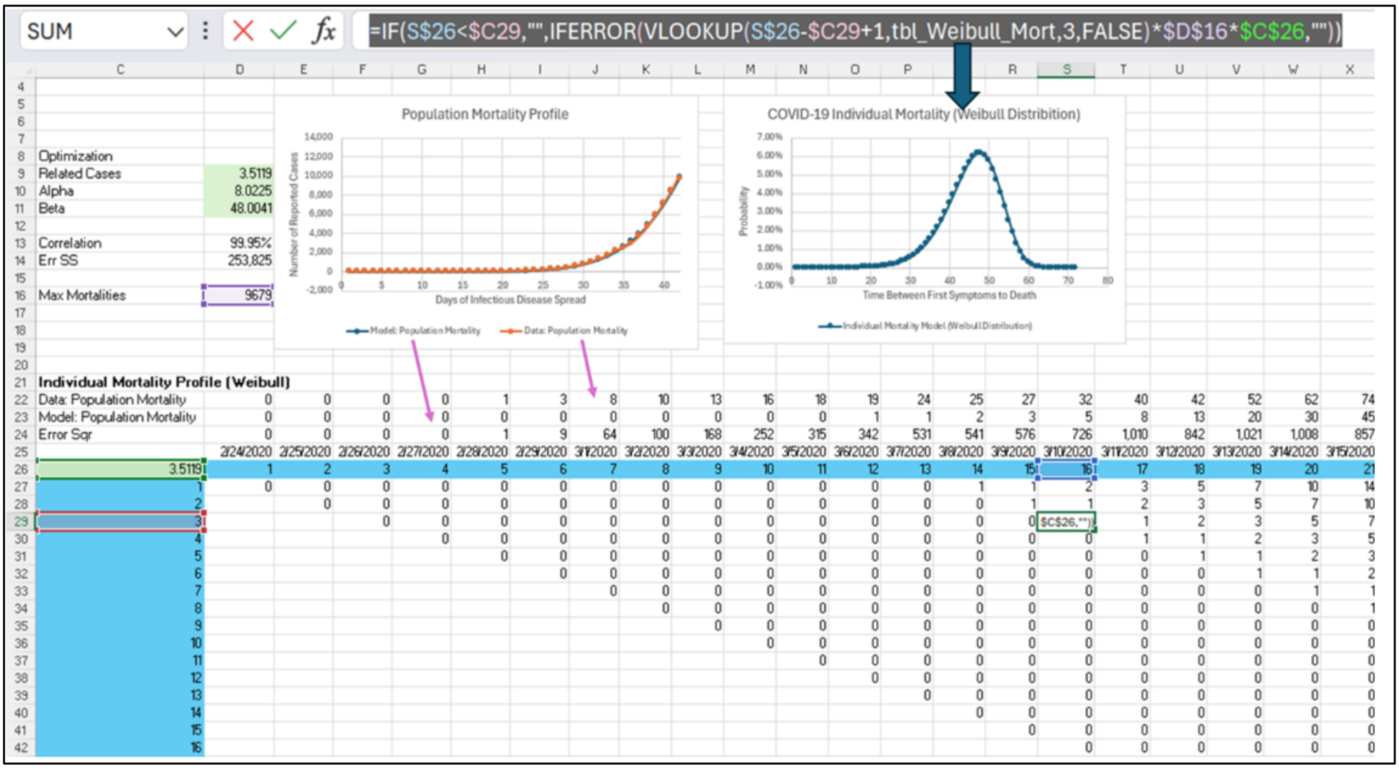
I. INTRODUCTION

The purpose of this paper is to provide (i) A means to formulating an informed opinion about future disease spread. And (ii) to understand at which stage in the disease lifecycle is a treatment effective. This is accomplished by characterizing an infectious disease using Collated Distributions [1 & 2] so that rapid development of health & treatment policies to counter the disease spread can be developed early as COVID-19 has shown us.

This paper explains why the authoritative models (reproduction models [3 & 4] such as that of the Washington University's Institute of Health Metrics & Evaluation, IHME, Reproduction model) used to manage health policy are doubtful. Though the mathematics of these models are accepted on a peer-reviewed consensus basis to be valid, but due to implicit assumptions, the statistical implementation is biased, and cannot be corrected. See Discussion section.

The term, context structure, is used as in many cases many models are required to construct a context structure that fits the characteristics of the context structure. Models are best fits for the data and are only as good as the data available. The data is only as good as the test accuracy (whether the tests are accurate or provide substantial false positives or false negatives). It is interaction between the models that gives the context structure its power to provide deep insights into disease spread, i.e. context structure tells much more than the data alone could.

Note, that (i) the Infectious Disease Spread (IDS) model presented here does not belong to any of the three know [5] model types, metapopulation model, cellular automaton model or gravity model, and (ii) The US COVID-19 data [6] used was from February 23rd, 2020 to April 5th, 2020 (early in the pandemic). See Table 1.


SUM
Population Mortality Profile
14,000
12,000
10,000
8,000
6,000
4,000
2,000
0
-2,000
Number of Reported Cases
0
5
10
15
20
25
30
35
40
Days of Infectious Disease Spread
Model: Population Mortality
Data: Population Mortality
COVID-19 Individual Mortality (Weibull Distribition)
7.00%
6.00%
5.00%
4.00%
3.00%
2.00%
1.00%
0.00%
-1.00%
Probability
0
10
20
30
40
50
60
70
80
Time Between First Symptoms to Death
Individual Mortality Model (Weibull Distribution)

```
=IF(S$26<$C29,"",IFERROR(VLOOKUP(S$26-$C29+1,tbl_Weibull_Mort,3,FALSE)*$D$16*$C$26,""))
```

	C	D	E	F	G	H	I	J	K	L	M	N	O	P	Q	R	S	T	U	V	W	X
8	Optimization																					
9	Related Cases	3.5119																				
10	Alpha	8.0225																				
11	Beta	48.0041																				
12																						
13	Correlation	99.95%																				
14	Err SS	253,825																				
15																						
16	Max Mortalities	9679																				
21	**Individual Mortality Profile (Weibull)**																					
22	Data: Population Mortality	0	0	0	0	1	3	8	10	13	16	18	19	24	25	27	32	40	42	52	62	74
23	Model: Population Mortality	0	0	0	0	0	0	0	0	0	0	0	1	1	2	3	5	8	13	20	30	45
24	Error Sqr	0	0	0	0	1	9	64	100	168	252	315	342	531	541	576	726	1,010	842	1,021	1,008	857
25		2/24/2020	2/25/2020	2/26/2020	2/27/2020	2/28/2020	2/29/2020	3/1/2020	3/2/2020	3/3/2020	3/4/2020	3/5/2020	3/6/2020	3/7/2020	3/8/2020	3/9/2020	3/10/2020	3/11/2020	3/12/2020	3/13/2020	3/14/2020	3/15/2020
26	3.5119	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
27	1	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	2	3	5	7	10	14
28	2		0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	2	3	5	7	10
29	3			0	0	0	0	0	0	0	0	0	0	0	0	0	C26,""))	1	2	3	5	7
30	4				0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	2	3	5
31	5					0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	2	3
32	6						0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	2
33	7							0	0	0	0	0	0	0	0	0	0	0	0	0	1	1
34	8								0	0	0	0	0	0	0	0	0	0	0	0	0	1
35	9									0	0	0	0	0	0	0	0	0	0	0	0	0
36	10										0	0	0	0	0	0	0	0	0	0	0	0
37	11											0	0	0	0	0	0	0	0	0	0	0
38	12												0	0	0	0	0	0	0	0	0	0
39	13													0	0	0	0	0	0	0	0	0
40	14														0	0	0	0	0	0	0	0
41	15															0	0	0	0	0	0	0
42	16																0	0	0	0	0	0

Fig 1 Collated Distribution Model for Mortalities Using MS Excel

Table 1 US COVID-19 Data [6]

Day	Date	Infected	Cum	Deaths	Cum
0	2/23/2020	35	35	0	0
1	2/24/2020	18	53	0	0
2	2/25/2020	5	58	0	0
3	2/26/2020	2	60	0	0
4	2/27/2020	1	61	0	0
5	2/28/2020	6	67	1	1
6	2/29/2020	5	72	2	3
7	3/1/2020	22	94	5	8
8	3/2/2020	18	112	2	10
9	3/3/2020	22	134	3	13
10	3/4/2020	35	169	3	16
11	3/5/2020	71	240	2	18
12	3/6/2020	104	344	1	19
13	3/7/2020	116	460	5	24
14	3/8/2020	121	581	1	25
15	3/9/2020	176	757	2	27
16	3/10/2020	290	1047	5	32
17	3/11/2020	245	1292	8	40
18	3/12/2020	424	1716	2	42
19	3/13/2020	532	2248	10	52
20	3/14/2020	724	2972	10	62
21	3/15/2020	707	3679	12	74
22	3/16/2020	965	4644	27	101
23	3/17/2020	1454	6098	23	124
24	3/18/2020	2567	8665	31	155
25	3/19/2020	5438	14103	56	211
26	3/20/2020	5489	19592	67	278
27	3/21/2020	7169	26761	73	351
28	3/22/2020	8336	35097	122	473
29	3/23/2020	9739	44836	128	601

30	3/24/2020	10523	55359	205	806
31	3/25/2020	13890	69249	259	1065
32	3/26/2020	16755	86004	257	1322
33	3/27/2020	18747	104751	404	1726
34	3/28/2020	19722	124473	479	2205
35	3/29/2020	17939	142412	312	2517
36	3/30/2020	21801	164213	489	3006
37	3/31/2020	25599	189812	753	3759
38	4/1/2020	26429	216241	960	4719
39	4/2/2020	29420	245661	1232	5951
40	4/3/2020	32500	278161	1257	7208
41	4/4/2020	34128	312289	1324	8532
42	4/5/2020	25827	338116	1147	9679

➢ *What are Collated Distributions?*

Fig. 1 depicts a set of individual probability distributions collated into a matrix, where the x-axis or rows are individual probability distribution of events by age, days, or relevant time periods, of events. Each row or cohort starting at the next time-period or day. The y-axis or columns are the next time periods cases per the case probabilistic behavior. At the top is the sum population rows for that time period.

Each row or cohort can be considered as a spontaneous set of cases. For example, as I recall, Google's Android phone COVID-19 tracking did not produce any useful results. Consider a person walking at 4 mph along a city street, with a wind speed of 10 mph, i.e., the relative wind speed ranges from 6 to 14 mph. Let assume an exhale of 2 seconds. In 2 seconds, the exhale could have travelled between 15 and 36 feet. Or if the Google tracking was between phones at most 6 feet apart, the results would definitely be inconclusive, unless people were inside a closed ventilated building. Therefore, it is not possible to realistically model spontaneous infections. Thus, each cohort are spontaneous infections for the next time periods.

Thus, Collated Distributions are a good reasonable approach to modeling IDS.

➢ *Collated Distribution Infectious Disease Spread (CD-IDS) Basics*

The Collated Distribution Infectious Disease Spread (CD-IDS) model presented in this paper is different other IDS models [5]. See Fig. 2. It is based on the axiom that infectious diseases can be characterize by 3 probability distributions, Individual Infectiousness Profile, Individual Mortality Profile, and Individual Survival Profile (Survival is defined as Infected – Mortalities) derived from a minimum of 2 data sets consisting of new infected, and new deaths. To determine the 3 individual probability distributions (profiles), the starting distributions was obtained after researching [7-10] possible distributions in 2020 publications.

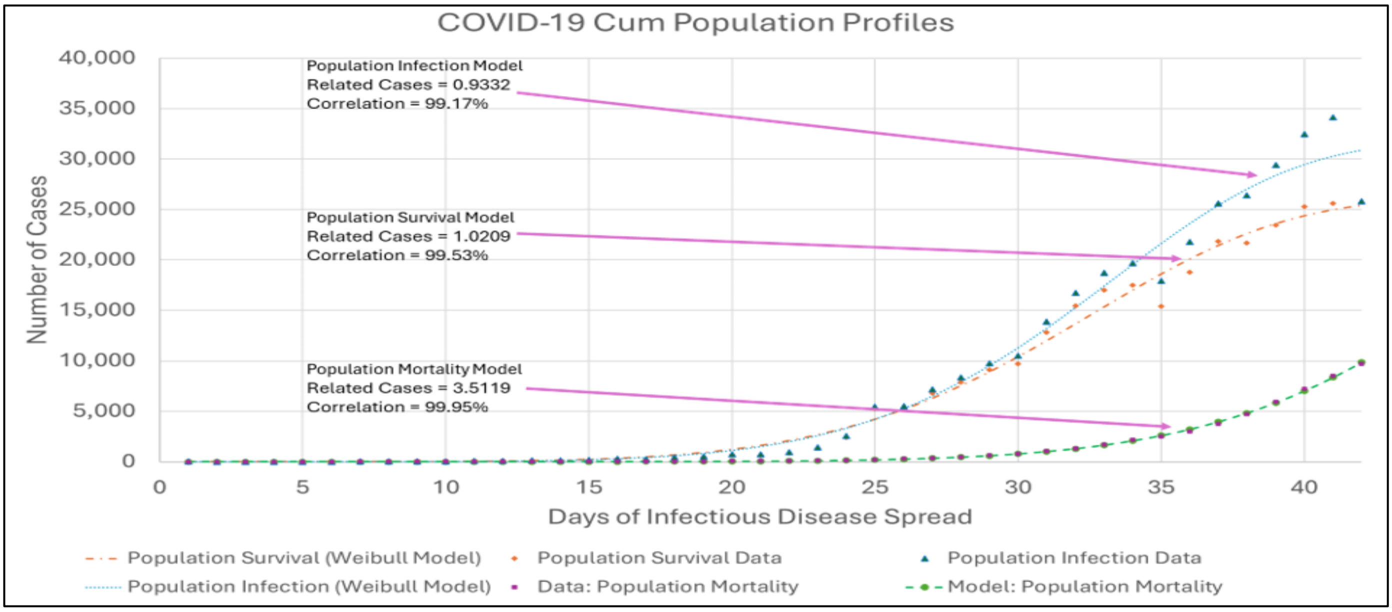


Fig 2 COVID-19 Population Profiles, Infection, Survival & Mortality Between Jan 24, 2020 to Apr 5, 2020

This CD-IDS model uses a new class of solutions, Collated Distributions [1 & 2], that the population's cumulative cases are derived from the sum of the single individual distribution of cases (1). A rudimentary version was first pioneered [11 & 12] in the financial services industry, but Collated Distributions are significantly different from these with the use of a row single distribution that "reflects" in the columns. The modeled cases at day j, $\hat{n}_j$ is given by,

$$\hat{n}_j = N\sum_i\left(P_{i,j}\right) \qquad \text{N is cell D16 in Fig. 1} \qquad (1)$$

Where j = day or column of IDS (row 26 in Fig.1), i = cohort or row (column C in Fig. 1), $P_{i,j}$ is the probability of case, and N is the number of cases as of the last day of IDS. See Fig. 1. Thus, the Population for a specific case type (infection, survival, or mortality) up to day j, is given by,

$$\widehat{N}_j = \sum_j \hat{n}_j \qquad \text{row 23 in Fig. 1} \qquad (2)$$

Given,

$$\hat{n}_{i,j} = R_C N P_{i,j} \qquad \text{cells D21 to X42 in Fig. 1} \qquad (3)$$

The Related Cases, R_C, cell C26 or D5 in Fig. 1, is the number of cases associated with each probability and converts this probability matrix $P_{i,j}$ into a population count matrix $\hat{n}_{i,j}$. R_C is the number of related cases modeled to provide a best fit. For Individual Mortality R_C = 3.519 related cases that best fits the data.

II. METHOD

In this study, Wilcoxon Regression [13, 14] was used, as regression results can lead to technique breakdown, and provide good Goodness-Of-Fit measures but biased modeling 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).

Wilcoxon Regression [13, 14] 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 of the sum of squared error SS_E (cell D14 of Fig. 1) between each data point j of value N_j and model value $\widehat{N}_j$, by changing the value of the R_C, α and β (cells D9, D10 & D11, respectively, in Fig.1),

$$Min(SS_E) = Min\left(\sum_j\left(N_j - \widehat{N}_j\right)^2\right) \qquad \text{cell D14 in Fig. 1} \qquad (4)$$

In this study, MS Excel's GRG Nonlinear was used to find the minimum sum of squared error SS_E**.** Weibull model is defined as a named table, tbl_Weibull_Mort, whose α and β (cells D10 & D11, respectively, in Fig.1) are the changing variables for the Weibull Distribution.

Note, there are two a necessary check, (i) the Survival distribution cannot start before the Infection distribution, and (ii) the Mortality distribution cannot start before the Infection distribution.

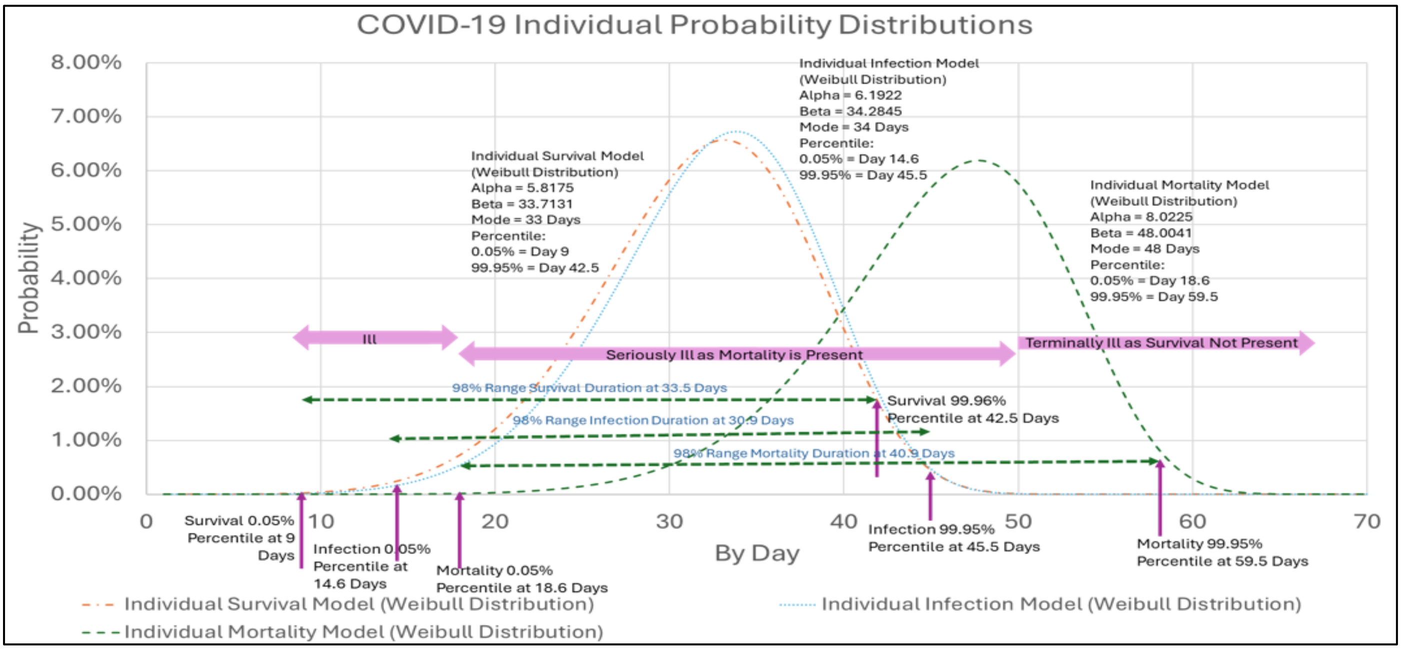


Fig 3 COVID-19 Individual Probabilities Prior to the Introduction of US COVID-19 Health Policies

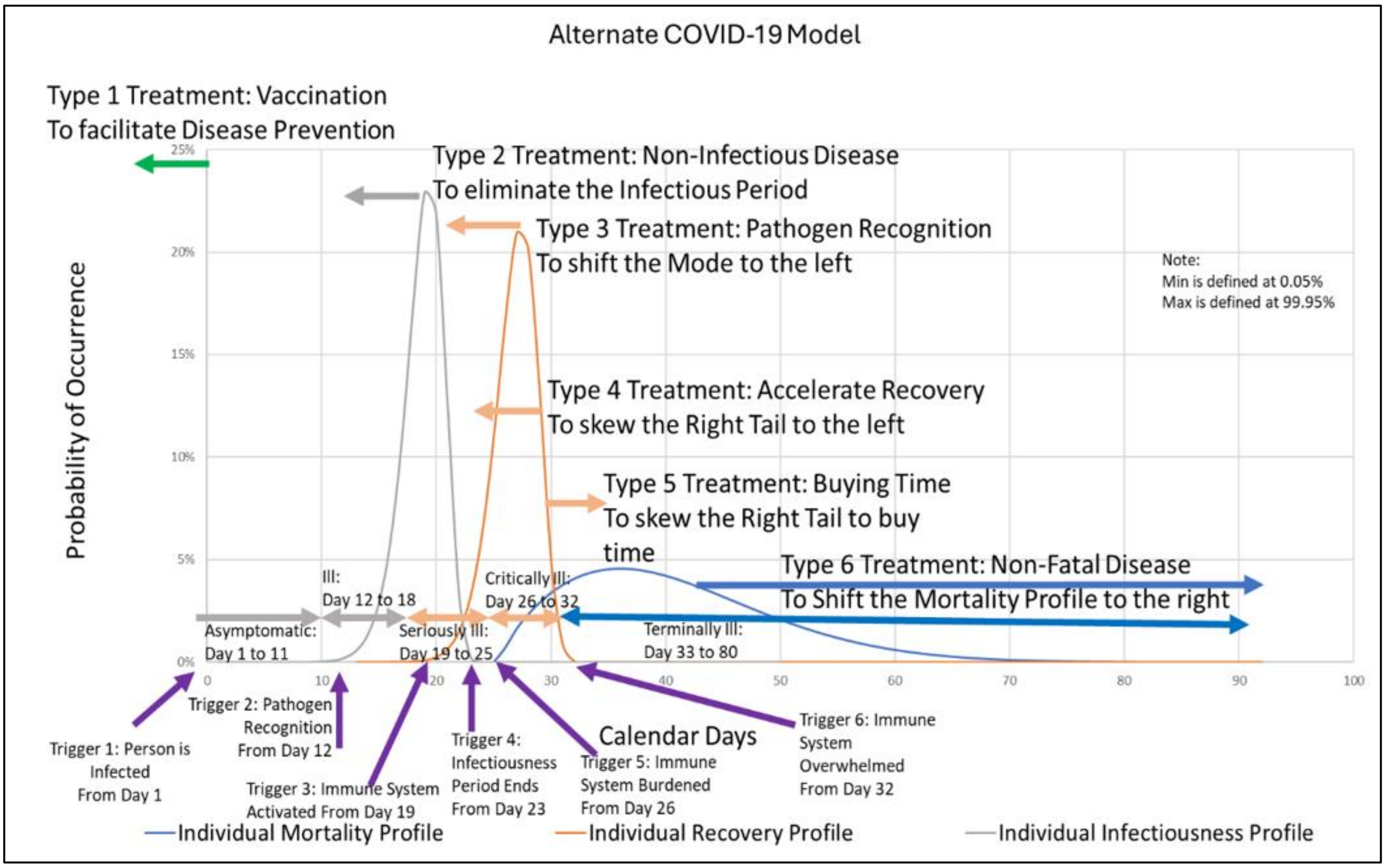


Fig 4 An Alternate COVID-19 Model (2020)

III. DISCUSSION

➢ *Discussion: Interpretation*

A key part of any analysis is the interpretation of the results. Fig. 3 shows the Individual Infections, Survival & Mortality probability density functions, and depicts the 0.05 & 99.95 percentiles of cum probabilities.

- Mortality ranges between day 18.6 to day 59.5 or a range of 40.9 days.
- Infections ranges between day 14.6 to day 45.5 or a range of 30.9 days.
- Survival ranges between day 9 and day 42.5 or a range of 33.5 days.

Both the Individual Infection & Survival overlap each other. That is a patient is responding to the infection almost immediately. And that it takes about 9 to 15 days for the viral load to become infectious to others. With infections being active for 31 days, would suggest that, if necessary, an effective quarantine period should be 31 days.

Fig. 3 shows a proposed disease stages,

- Ill, defined as Mortality not being present.
- Seriously Ill, defined as Mortality being present.
- Terminally Ill, defined as Survival not being present.

These disease stages' durations are based on the 0.05 & 99.95 percentiles because human lives are at stake but could be changed to other percentiles based on experience in the field.

Fig. 4 depicts an alternate model based on a more complex modeling (but the author lost this model after his PC crashed). However, it is a good example of the interpretation of results. From a statistical perspective, it shows (i) the three individual probability distributions (infection, survival / recovery & mortality) (ii) the disease stages, (iii) the 6 triggers present at each disease stage, and (iv) how to determine a cure strategy i.e. what is the intention of the research to find a cure?

➢ *Discussion: Critique*

The IHME model is essentially a variation of current epidemiological theory [15] (5),

$$I_{final} = N - (N - I_0)e^{\left(\frac{-R_0 I_{final}}{N}\right)} \tag{5}$$

Where *N* is the size of the susceptible population, I_0 the number of primary cases infected, and I_{final} the expected final size of an outbreak of an infectious disease. If $R_0>1$, each primary infection will, on average, generate more than one secondary infection. If $R_0<1$, each primary case will, on average, fail to replace itself (although short chains of transmission are still possible) and each single introduction will lead to no more than a minor outbreak.

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The IHME model [15] (6) at its essence is the ln() function that has been found to be produce unacceptable forecast results [16]. It suffers from two major problems.

The cumulative mortalities y^t_j at time t in location j in the IHME mortality model (6) is given by,

$$log\left(y_j^t\right) = \frac{p_j}{2}\left(1 + \frac{2}{\sqrt{\pi}}\int_0^{\alpha_j\left(t-\beta_j\right)} e^{\left(-\tau^2\right)} d\tau\right) + \varepsilon_{t,j} \qquad (6)$$

$$\varepsilon_{t,j} \sim N(0, V_t) \qquad (7)$$

Where level p controls the ultimate level, slope α controls speed of infection, and inflection β is the time at which the rate of change is maximal.

The first model problem. Even though the log model errors $\varepsilon_{t,j}$ are Normally distributed $N(0,V_t)$, this is the log of the data errors $\Delta_{t,j}$ with respect to the exponential function (8),

$$y_j^t = e^{\frac{p_j}{2}\left(1+\frac{2}{\sqrt{\pi}}\int_0^{\alpha_j\left(t-\beta_j\right)} e^{\left(-\tau^2\right)} d\tau\right)+\varepsilon_{t,j}} + \delta_{t,j} = \left(\varDelta_{t,j}\right) e^{\frac{p_j}{2}\left(1+\frac{2}{\sqrt{\pi}}\int_0^{\alpha_j\left(t-\beta_j\right)} e^{\left(-\tau^2\right)} d\tau\right)} + \delta_{t,j} \qquad (8)$$

That is,

$$\varDelta_{t,j} = e^{\varepsilon_{t,j}} \neq N(0, \sigma) \qquad (9)$$

Where σ is some standard deviation value and $\delta_{t,j}$ is the true model errors of the true model of population statistic. (8) shows that $\Delta_{t,j}$ acts as a multiplier distorting y^t_j, the statistic one is interested in. For example, for $n = 42$ days, mean $\bar{y}_t$ of the known infected (dependent variable) is 8,050 but mean of this dependent variable as a ln() function $\sum[\ln(y_t)]/n$ is 6.6059 which is 739.4, or,

$$\bar{y}_t \neq e^{\sum ln(y_t)/n} \qquad (10)$$

There is a very big difference between 8,050 and 739.4. Thus, the minimization of the error sum of squares of the transformed data is not the same as minimization of error sum of squares of the data.

Heteroscedasticity [16] is present when the model (dependent) errors exhibit a monotonic behavior with respect to the dependent variable i.e. errors get larger or smaller, even after excluding outliers or is some function of the dependent variable. This is due to (i) missing factors (variables) in the context structure, (ii) incorrectly modeled factors/independent variables and/or (iii) when an independent variable represents more than one underlying factor. Heteroscedasticity is a major problem [17] when the range of values of the dependent variable is very large, as is the case with modeling infectious disease spread and therefore the dire need for a different approach.

The second problem with the IHME's models is the use of cums. From the author's 40-years of working with data, cum models give extremely good fit because cums cancel, not minimize, the noise in the data. However, cums can substantially bias the non-cum model results, the statistic one is interested in. That is, the cum model errors $\varDelta_{cum(t,j)}$ is,

$$\varDelta_{cum(t,j)} = N(0, \sigma) \qquad \text{where standard deviation } \sigma \to 0 \text{ as } t \to \infty \qquad (11)$$

Or as $t \to \infty$ the standard deviation of the model cum errors $\sigma \to 0$ as the errors cancel out. This is another form of heteroscedasticity. Therefore, the residual sum of squares $\to 0$ and thus, the F-Ratio skyrockets but the model usually gives misleading results.

IV. CONCLUSION

This paper has shown that it is possible to statistically determine statistical disease properties and treatment strategies. The Collated Distributions models should lead to a better understanding and parametrization of generic population spreads and therefore, make informed opinions on how public health management should be conducted and possibly which drug treatment is likely to succeed.

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