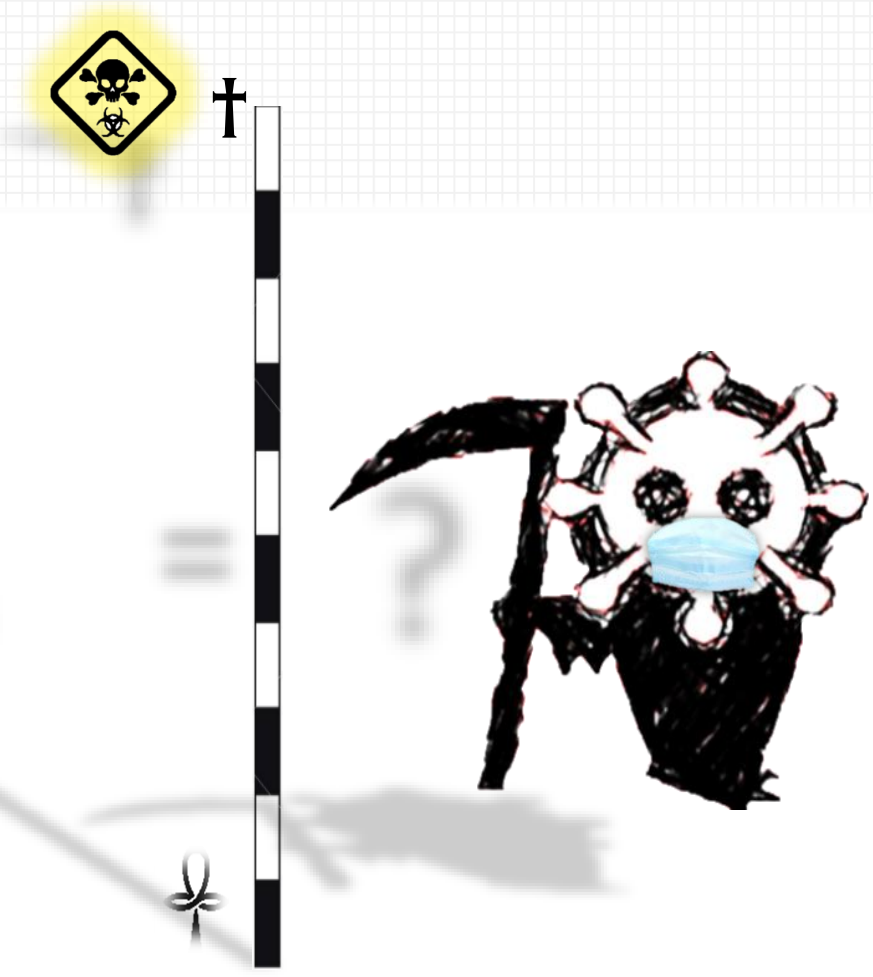
THANATOPHOBIA

The *quasi*-Metrological Determination of SARS-CoV-2 intrinsic Potential of Lethality

Demystifying the nocebo hypothesis
by a novel yet familiar approach
on statistical data science.



John S. Platanenbaum
Assistant to Jenna B. Huck - Regina Iustitiae
SANITAS CONSCIENTIARUM LTD.
GLOBAL IMPROVEMENT SPECIALISTS
sonni@celador.de

Dedicated to Anna-Meral and her dad.

THE PREAMBLE

Dear scientific community,

My dear fellow members of human kind.

The World we all live in has been a crazy one the longest time. The current global pandemic is not particularly helpful either for upholding sanity in general. Sometimes things appear illogical and counterintuitive. By times we wonder if it is the entire world that is crazy - or if in reality it is actually just us.

This is an invitation to contribute in a common struggle to

'make the world sane again'!

We invite every fellow human being - contemporary or in the future - to consider this their **personal duty to mankind** and give it a **try at least** whenever they see any chance to do so.

The Thought Experiment

Let us consider this a thought experiment. Any new information can be true or false. Also we do I imply that untrue information is or was spread by purpose or by anyone. Please do not take anything written here for a reason to disregard hygienic and social distancing regulations in our common effort to keep the pandemic under control. ***Probabilistically****, we have to assume that the results might be wrong. Therefore, nobody will be asked to believe anything. Please do not belive this document! What we are asking for is to* ***stop ignoring what we do not want to see.*** *Seeing is* ***believing.*** *Believing is not knowing. Not knowing is dangerous!*

I will provide a scenario which we could be in and we would be doing a couple of things very wrongly. I know, there are smarter and better qualified people out there who work very hard and undoubtedly, do a great job. However low the odds are, there is a chance we have run into a situation which we should definitely be aware of, in case we are.

Therefore, unless someone smarter than me confirms otherwise, consider this nothing more than a riddle for entertainment and educational purposes.

> ***"We are all agreed that your theory is crazy. The question which divides us is whether it is crazy enough to have a chance of being correct."***
> *— Niels Bohr*

Now, *have fun* and looking forward for an un**bias**ed, facts based and objective scientific debate.

∞ The Non-Credo Of Science

We are able to combine available information and come to conclusions and insights following a chain of logical causal contexts. Some of them may be wrong – in consequence of including values into our equations that *we believed* to be accurate. Sometimes we realize our equations give us implausible results despite our best efforts to double check all values and variables we add into them. And then we start questioning everything we see to refuse having to accept the fact that what *we believed to know* to be a fact was not actually knowledge but nothing more than our own conviction. It was faith. Unconfirmed assumptions, which we accept to be true – and so they become a true part of our "personal reality".

And even the smartest among us come up with new parameters and **cosmological constant**s to make the universe fit into our equations and to reflect what we believe in.

And here we have to consider thoroughly, if all the values we add into our equations to solve the problems we are confronted with, to be **purely based on facts**: on what we know and can confirm to be verifiably true.

∞ The Principle Of Scientific Secularity

With a vast majority of the world's population considering themselves members of a religious community, faith appears to be an elementary part of what makes us human. But believing something means we accept something to be a truth without asking for evidence. Believing means 'not knowing for sure'. And while it appears to be a human need to believe in something, science must purely be based on measurable and reproducible facts. For the part we cannot know, we have no choice but making assumptions, educated guesses.

We create theories based on mathematical methods that more or less exactly reflect what we can observe in reality. We introduce new variables into our equations to see if the results are plausible, consistent and deliver predictions that are in line with what we can detect and measure in reality. We create models and theories for complex issues. And as long as these models confirm what we expect them to do and we have no other way of knowing for sure, we have no choice but to rely on these models when we have to come up with decisions upon which we turn thoughts into ideas and ideas into actions and eventually, our actions shape reality. Hence, any information - data we have on hand and use to make decisions upon, that involve implications affecting continuation or erasure of human lives – **must be questioned and verified uninhibitedly**.

∞ The Sanctity Of the Socratic Method

No scientist shall feel required to remain silent, if (s)he has a rational thought supported by scientific facts. We must uphold an open mind-set, a culture of free and open exchange and communication of ideas, irrelevant if they reflect an unpopular opinion or question things, *we believe* to be unquestionable. Science must remain a place where meaningful ideas can flourish - where the status quo must always be questionable openly and freely! We do not have to accept every idea, but we cannot suppress those we dislike only because ***we believe*** in something else.

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I. ABSTRACT

The existence and significance of the placebo effect are commonly known and commonly accepted. Every new pharmaceutical drug has to demonstrate its effectiveness by showing better results in comparison to a placebo.

Contrary to the positive effects of the placebo there are studies showing evidence for the existence of the nocebo effect, which causes patients to develop negative symptoms (such as adverse effects) after reading the info leaflet even without taking the active substance. For both effects there are studies finding a rate of around 30 % of the patients to develop and describe a physical response.

For both effects the intensity of a response can spread over the entire spectrum. There are reports of extreme cases from terminal cancer patients with disappearing malign tumors (spontaneous regression or -remission) and patients prematurely dying at a time predicted based on a cancer stage miscalculation by their physician.

Most scientists agree that among those who die from COVID-19 there could be a fraction of people who actually die in result of the nocebo effect and not from their infection. If there is a chance something can happen, it's only a matter of probability and how often we roll the dice.

Therefore, the question is **not 'if'** but '**how many**' of them?

Could there be a way to distinguish and quantify this ratio? For the extreme case of spontaneous regressions(*) there are reports of incidence rates around 1:80.000 and 1:100.000. Should the current situation be representing a trigger for a nocebo response and we repeat the experiment billions of times, this must be visible in the numbers. If so, to what extent could media broadcastings, information management and protection measures against the virus themselves play a role during the pandemic? And what would need to be our learnings for the way we deal with patients and health related information in general and in terms of public communication during pandemics, now and in the future?

*) not necessarily indicated as placebo-caused but most cancer patients seek hope and believe in alternatives after being given up by conventional medicine, which would relate to believing in a placebo

II. INTRODUCTION

Placebo and nocebo effects are considered separate phenomena, but they could also be considered two opposite sides of the same phenomenon. To abstract the effect and release these phenomena from associations with a specific way of application and the context with good, bad, wanted or unwanted, we will refer to both phenomena as creabo effects.

1. The Creabo Effect

The creabo effect is based on the assumption, that placebo as well as nocebo affect are both based on the same principle:

Both are measurable effects, that cause a physical response - a change of subjective well-being, as well as visible symptomatics. Both acquire and unfold their effectuality depending on acceptance of a prophecy and conviction of its truth.

A. **The Prophecy**

In both cases, patients are provided with a prediction of outcomes to expect after an event or signal. If this "prophecy" is communicated in a credible and convincing way and from a trustworthy source, it is accepted as an undoubted fact. A set of expectations is created in accordance with the predictions. In earlier studies, this was referred to as injection, indicating an intentional process. Since we will be referring to unintended injections as well, this will generally be referred to as "**induction**"; The patient is induced with the prophecy.

B. **The Trigger**

For the prophecy to come true, there is a condition that needs to be fulfilled. In the case of a pill the patient himself has the choice to fulfil the condition at a time of their will. If an external notification is expected for the fulfilment of the prophecy to start, then the process starts upon registering it. The pill or the external event represent a trigger signal, that can be anything that is suitable to notify the patient about the point of time the prophecy will be considered started. Information is induced into the patients' mind, the prophecy is expected and waits for a trigger. This start-signal can be the ingestion of the classic pill resulting in both creabo effects (placebo or nocebo), or it could be an event, such as the first symptoms of a bad disease that was predicted. Upon reception of this trigger-signal the patient expects the induced predictions - the

prophecy - to become reality. This trigger appears to play an important role, as its credibility will affect a patients trust and confidence that the process has actually started.

C. The Credibility

For the prophecy to be accepted, we require credibility. In case of the placebo effect, we know that bigger pills statistically trigger a more intense placebo response. The confidence in the deadliness of the Corona virus is indubitable. The regulations and limitations resemble quite a big pill already and do not go unnoticed. The message is clear: if it were not absolutely dangerous, there would not be so much noise about it all ~~over~~ around the world. And in the world of science, there appears to be solid consensus. Nobody who expresses doubts would be taken serious but considered a fool.

2. The False Prophet

There is a lot we have still not understood; We are still not able to confidently quantify the deathrate, which usually was not a big issue earlier despite much smaller “sample sizes”. Also, it is easy to find lethality values year by year for the seasonal flu.
This is the worst pandemic in recent history. We have never had this much recorded data about any deadly pathogen and we have never been more advanced scientifically. Why is it so hard to quantify the risk this time?

A. The self-fulfilling Prophecy

> ***A self-fulfilling prophecy is the sociopsychological phenomenon*** *of someone "predicting" or expecting something, and this "prediction" or expectation* ***coming true simply because the person believes it will***[1] *and the person's resulting behaviors aligning to fulfill the belief. - Wikipedia*

Taken into account that creabo effects are driven by believing in something, this curious phenomenon seemingly can work through mechanisms, that go beyond influencing behaviour and can psychosomatically have an effect on our bodies and physical health as well.

B. The Doubt

We see people dying, which perceptioanally leaves no space for doubts. But are we really sure, that the prophecy about the deadliness of COVID-19 is even real? And if it were not; could it really become reality just because we believe in it?

C. The Schism

There is still a lot we have not understood yet but the strong polarization due to both sides seeing their interests under threat (health vs. freedom) led to tensions and even hostility between the two groups. Among those considering themselves educated, an attitude arose of feeling responsible to educate and instruct the often so-perceived "ignorant, irresponsible and simple-minded" who put the health of all of us and even our lives at risk.

While governmental paternalism and dictation of a behavioural codex, together with long-lasting constraints cause subjective impressions of coercion, suppression, inequality and injustice, they also lead to ever more aggravation of the polarization, compulsions of rebellion arise. Initially rather peaceful protests and demonstrations are now accompanied by anger, aggression, rage and occasionally also riots. Some of the footage of such protests show scenery which resemble combat battles. Further increase of tensions could lead to **further destabilization** and inherits the risk of escalation, possibly up to a level that could be referred to as a civil war - in one or more countries. And once two neighboring countries are in a civil war and the situation is difficult, the

3. The Psychosocial Trilemma

As nobody wants to be a fool and everyone is afraid of embarrassment and being ridiculed, would someone who actually knew better prefer to remain silent or even distrust their own logical conclusions and discard their findings?

There is still a lot we have not understood completely. Being the *casus belli*, COViD-19 and specifically the risks it exposes us to play a critical role. In spite of the not-fully-understood situation, the still-unconfirmed degree of lethality is what split the population, united each of

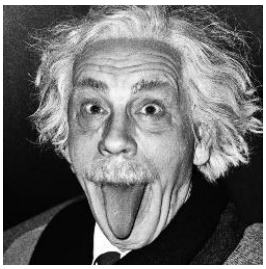
the two isolated groups, pursuing a common goal, based on a common belief and encouraged by their solidary. Mutual support and endorsement shared within each of the groups strengthens either sides feeling of righteousness. Both groups fight for what they believe in and to protect what they consider threatened by loss.
This polarization, combined with the fact that "the higher purpose" that both sides strive for and that, dividing both groups and bringing the members of each group closer together, has become something that resembles the status of a sanctuary.

It has become a pure matter of faith that caused the divide and that both groups identify themselves with. And nobody appears to be questioning their point of view anymore.

"The horizon of many people is a circle with a radius of zero.
They call this their point of view." - ***Albert Einstein***

Einstein was **absolute**lty right about this. And he is the very same man, who is best known for providing evidence that the point of view is <u>everything that matters</u>!? Isn't that, what **relativity** in its essence actually means?

And maybe there was the *self-fulling prophecy itself personally* at work, creating the best example and providing evidence for itselfs existence, when Einstein said:



"Two things are infinite: the universe and human stupidity;
and I'm not sure about the universe."

Because that might possibly be the most intelligent thing a human will <u>ever be able to say</u> about himself, the universe and everything. (Or maybe that is 42, depending on the question.)

4. The Critisizm

Nobody is questioning their point of view and no questioning at all appears to exist towards the causality. Of course, how could someone even hypothetically express any doubt, when evidently, there are people actually dying!? Seeing is believing! Researchers would waste their time, and even if they were to find something, they would be at risk to be heavily criticized or could even

damage their reputation and credibility as scientists, if they spoke it out. Their professional **existence is at risk**, creating fear and **inhibiting the exploration** of scientifically non-preclusive solutions.

Fear is a very natural feeling. It is a profound evolutionary heritage of our natural survival instinct. But it never is a nice feeling. And it should not be the main feeling that guides us in our lives. Neither individually, nor – and even much more importantly - in terms of human scientific progress. Could there be a forgotten Grail that nobody is searching for because *we believe* to have found the answers already? Or do we use it as a scapegoat, to distract from the fact that nobody really has the slightest idea what is going on here? Or is there even a hierarchy in the current system, that caused us to miss this chance in result of people being forced to remain silent because of social influence and repression from their next-higher hierarchy level?

How (un-)likely is it, that this could result in a global collective lock-down of the human mind?

A. The un-usual Suspect(s)

1. The primary suspect is COVID-19, of course, as the situation seems clear. The jury *believes to know* what they think:

The primary mass murderer must be the novel Corona virus, as there are meanwhile more than one million silent witnesses testifying that the virus was at the crime scene at the time of offense.

2. The secondary suspect, which the jury *believes to know* to be an excludably unlikely candidate and too unlikely to be seriously considered a serious suspect:

The infamous nocebo effect.

To identify and determine the degree of complicity, we will use the available data to forensically analyse and investigate to get further insights to put in context with our hypothesis:

There is a predicted/expected event upon which creabo effects start and the induced prediction is supposed to come into effect. There is no need for a pill, as the pill is inert anyway. What it resembles is a symbolic ritual to convince the patient that the induced prophecy has begun to

come into reality. The more convincing the ritual, the better it works. Bigger pills work better than smaller ones. Expensive looking ones work better than cheaper placebos. Studies have shown that repeated injection ⇒ repetitive induction, results in higher response rates.
In case of the nocebo effect in context with COVID-19, the infection itself would qualify as a trigger, once symptoms of an infection are noticed. A positive test would then convince those who were in hope it could still be something else. Hence, it would resemble a reminder, in case of uncertainty if the trigger signal was missed. So in case there is any problem with the credit and the mechanism is jammed, we reload and pull the trigger once again.

These are very credible and intense trigger signals which could potentially lead to even higher response rates than any environment a research study would be able to simulate.

And we do it on a global magnitude!

B. The Experiment

The Global Ring Study Experiment

The Situation we have currently, would qualify as ***a global nocebo experiment***. Technically, all factors required for such an unethical experiment are given. The collective induction of the **prophecy** takes place multi-channel and unintermittent. Once an infection is noticed, the infection (noticed symptoms) represents the **trigger** signal.
A positive test confirmation would be an additional push factor, like a reminder, a second try, repeating and emphasising the trigger signal, which usually was not included in earlier studies. The prophecy comes from those among the populations, who are expected to know best and therefore enjoy high amounts of advance **credit**: Scientists.
They are given billions for their research in vaccines by those who have, because these are missing out even more billions due to the lock-down of global economy, and so they spend more and more money, even the money they do not have yet, to keep what they believe to have, and when they do not have any more, they will have to overdraw their credit and ask those who still have and can give them credit to survive.

Hence, the setup of the experiment is ideal. The research is *very* well granted. And the prophecy is unequivocal.

Let the Experiment begin!

2) **The Null Hypothesis**

H_0: We hold the virus responsible *for all the deaths*, that we observe. If the virus kills a percentage of human samples, this percentage must be measurable. In a series of experiments, we should be able to determine this number. Our sample size is unmatched by any earlier studies. The expected results are supposed to be more than sufficiently significant**.**

C. The Casus Belli

A number of countries (participants in our global global study) are reporting a significant number of infections but close to zero casualties. The causality actually seems not to be quite unquestionable or at least not fully confirmable yet.

How can the same virus kill up to 20 % of hosts in one country and almost none somewhere else?

Objection: In dubio pro reo!

This question is actually very mysterious, while some smart people usually can supply a hypothesis that seems to be satisfactory enough to stop any further questioning.

D. The Defense-argument

How can all this be? The virus must be *innocent, in a significant number of cases* of the homicides, if not all of them.

3) **The Necessary Condition:**

If the virus is guilty of killing humans, this means **it must be doing it, independent of the location where we repeat the experiment!**

Therefore:

4) **The unconditional Expectation:**

For the deaths in result of the virus we expect **a baseline**, represented by **the minimal value we find over all countries**!

E.The Alternative Hypothesis

We expect a mutually exclusive dichotomous outcome:

H_a: Any additional deaths to that baseline must be the result of our cruelsome nocebo experiment.

Every country providing us with numbers performs the experiment locally, following strict directives in terms of induction of the prophecy. A homogenous information distribution system is deployed in every country, to make sure a maximum number of human samples can be reached by the prophecy so they can be included in the sample size. The induction is performed at our best efforts. The results are reported on a daily basis by every country contributing to our global ring study experiment.

5) The Theory

The news about the deadliness of the Corona virus arrived before the virus itself. Looking at the considerations above, we might be required to reflect about how a topic can become so undiscussable, in result of such strong polarization.

Even if the virus were harmless, which in the majority of infections it appears to be, a fraction of people who are susceptible to the nocebo effect would become seriously ill and some would die. Many infections go completely unnoticed or with only mild symptoms. But we know it can be absolutely deadly as well. From not even noticing to intense care and death, his makes an extremely wide range of severity which is quite untypical. There are a couple more confusing observations in context with COVID-19 and while many competent people work very hard to find solutions, there seems to be a lot of confusion as well.

6) The Goals and Objectives

Ten months after the Corona virus started making up so-perceived 50 % of the news and worries in our lives, there is additional and accumulated statistical data, which is publicly accessible.

In an attempt to gain more clarity and hopefully find something useful that can explain the observed irregularities, this data was analysed and (re-)interpreted.

III. METHODS

The method we will use in order to solve this global epidemio-psychosomatic mystery will focus on the lethality rate only, as there must be the answer which we look to find. We will use the statistical datasets available to transform the problem into something we are familiar with. Something every scientist should be familiar with, who ever had to generate significant and expressive data consistently. Particularly, the fellow biochemists among us and anyone else who has worked and is familiar with

The Measuring of Biological Samples:

1. The Methodo-Logical Assumptions

based on which we will work:

> ***"Measurement** is **the assignment of a number to a characteristic of an object** or event, which can be compared with other objects or events.**[1][2]"** - Wikipedia*

A. The Potential of Threat

To consider a virus dangerous we need to be able to quantify the danger we are exposed to by it. We must be able to put a number on it to be able to compare its potential danger against other risks. Elsewise, we have no scale to determine if there may be much higher risks we are already accepting. Risks are a matter of statistics. Statistical data is available and must contain the answer we search for.

B. The *quasi*-Metrological Approach

The risk we are exposed to by a pathogen is directly proportional to
the statistical probability **X**, that, in the event of a host organism

a: Me, humble representative of you and every individual specimen of our precious species - being infected with a specific strain of a pathogenic virus

b: COVID-19, will initiate a cascade of molecular and biological events on the last of which "A" would find his remaining "T" exponentially and irreversibly converging to 0, where **1** will be the choiceless result of

c: **Death.**

Therefore, if:

(0) $a + (b * X) = \mathbf{c}$

we have to know **X**!

In epidemiological context:

With A being the total population of potential host organisms, B represents the (number of) confirmed infections and C the number of dead in result of these infections. Defining A_1 as our initially healthy population (before B), and introducing A_2 for the number of the population that survive an infection by B (after B), we can note:

i. $A_1 + (B * X) = A_1 + C = A_2$

ii. $A_2 = A_1 + C.$ $\{ A_1 \geq A_2 \}$

Due to the number of casualties (C) being subtracted from the number of the initial total population (A_1), C cannot be a positive number:

$A_2 - A_1 = C$ $\{ Z \leq 0 \}$

(1) and (2) combined give us:

iii. $A_1 + (B * X) = C + A_1 = A_2$, $| -A_1$

iv. $B * X = C = A_2 - A_1$ $\{ C \leq 0 \mid$ thus, also: $(B * X) \leq 0 \}$

Solving this towards the deathrate, X:

v. $X = C / B$ $\{ C \leq 0 \mid B > 0 \} \Rightarrow \{ X \leq 0 \}$

⇒ X = (deaths / infected) , where X (deathrate) and Z (number of deaths) deduct from A_1 and hence, give negative values for C and X.

To eliminate the necessity for negative values, we introduce a new parameter for the Specific Lethality [Λ]:

vi. $\Lambda = -X = -Z / B$

vii. $\Lambda = |Z / B|$, with $|Z| \leq B$ and $B \in \mathbb{Z}^+$ (pos. integer) req.: $\{ 0 \leq \Lambda \leq 1 \}$

⇒ Therefore, Λ must *per definitio* be identical to $|X|$ and

The Case-Lethality-Rate (CLR):

viii. $\boldsymbol{\Lambda} \stackrel{\text{def}}{=} |\boldsymbol{X}| \equiv \boldsymbol{CLR}$ (?)

The Measurement

We are using the recorded statistical data to generate *quasi*-measurements probing for data points, determining the local lethality rates for every country. Considered as a set of measurements, measuring the specific lethality rate of a virus, from a mathematical perspective becomes very comparable to measuring expression levels of a protein, for instance. We can apply our familiar mathematical concepts for standard deviation, variance, significance and so forth.

2. The *quasi*-Metrological Principles

A. The Variability

Some deviation must always be expected as a result of limitations in the precision of the measurement tool (method) or the measured object itself is subject to influences or circumstances that cause the dimension we want wo measure to vary stronger than the precision of the tool would allow for. Even if very slightly, these deviations can have a huge impact on the precision scale we try to measure at.

In regression analysis, we have the probit model:

> *In statistics, a **probit model** is a type of regression where the dependent variable can take only two values, for example married or not married. The word is a portmanteau, coming from **prob**ability + un**it**.*[1] - *Wikipedia*

Every death case C represents a probit, similar to a qubit or a regular digital bit, that can have one out of two values. In this case:

0 = survival
1 = death

Due to various factors causing variability, the number of deaths per infected host organism will therefore be a matter of probability. The word itself contains an important detail:

The Probe-ability.

Every pair of numbers, case of death in relation to the registered infections, delivers a probit which will be considered a sub-measurement. In sum of many sub-measurements we expect a bell curve around a percentage value which is supposed to be determined.

The Uncertainty

At some point of the measurement we would be in a range where our signal that provided us with the "sensing" of the information gets indistinguishable from the background noise. That is the point where we lose certainty about if there is a signal or not. We are not able to sense (or measure) the existence of a signal any longer.

We cannot distinguish if yes or no, 1 or 0, up-spin or down-spin, true or false, real or not. That would be the limit of the sensitivity of the measuring method. We lose ability to probe for the measured information, when we lose certainty about the signal. That is what limits the primary dimensional resolution of our measurement.

But as long as our measurement gives us a clear signal**, <u>consistently and repeatably</u>,** we can be sure to make a valid measurement within a certain **tolerance range which we can determine mathematically**! And the more sub-measurements we collect, the more certain we can be. Hence, we should also see an increased significance.

B. The Significance

The Case Lethality Rates are calculated as described and displayed as a percentage ratio for every country that reported a significant number of confirmed infections ($N \geq 1.000$). While $N = 1.000$ would be a sample size rarely seen in artificial research studies and which is therefore expected to be large enough to deliver very significant results by itself already, we can still increase the minimal required sample size. to exclude uncertain data-points to enhance and improve the significance of the meta-analysis. Based on the sample size alone, we expect no bigger fluctuations among sample sub-sets. Therefore, occasional statistical deviations should not be expected to falsify or heavily distort our measurements.

If required, to enhance and improve the significance of the data for the meta-analysis and get more insight into observations, the minimum required sample size can be adjusted to exclude further data points that could possibly be far offs due to low sample sizes (unlikely) or other exceptional influences.

C. The Specificity

The illustrational Analogy:

If we try to measure the width of a table - which we do not know to be 2 m in width - with a tape rule, we should be reading something between 199.5 and 200.5 cm most of the times. We may get 197 cm or 203 cm sometimes, maybe 210 or 220 cm if we are measuring outdoors on a stormy day. No matter how untalented we may possibly be during use of the measurement tool, we would not expect to measure more than 300 cm or less than 100 cm.

> If the results of our measurement differ between measurements of the same object by *orders of magnitude*, **then our tool is obviously not suitable for the purpose which we intend to use it for.** If our measurements give us random values between 0 cm and 300 cm, we would suspect *there must be* ***something wrong with the tape rule,*** *and* ***not*** *with the table*.
>
> However, if the tool provides consistent results in a row of measurements at the same 'object', it is not naïve to assume that we could actually be measuring something. It is just not showing us what we wanted to see.

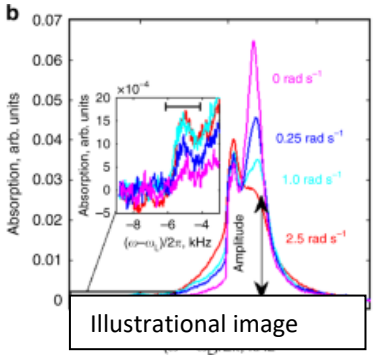
3. The Interference

A signal can during our measurement be influenced by another signal of homo- or heterogenic origin or type, causing amplification, cancellation, cross-talk, superimposition or any other kind of distortion to our (recorded) signal.

Given a resolution high enough, if they are independent phenomena influencing our measurement, we can mathematically separate the signals and analyse them independently.

The Distortions

There is always a risk of external factors interfering and influencing our signal. Since a quantitative measurement requires qualitative existence (!) and we seem to be measuring something else than we intended, we can conclude that we see a signal which is a reaction of our "detector" that must have



Illustrational image

been caused by something else that should be identifiable. Causality requires that every reaction must be caused by an action. actio = reactio.

"Something" must be there to cause an actio. Although it may not be what we were looking for, it could be useful, so we might want to try and understand what it is. Seeing is believing but believing means "not knowing". And not knowing can be very dangerous! Only once we understand it, we can start talking about "knowing" something.

4. The Qualification

Single (sub-)measurements can be far off but every additional measurement will improve our precision, as coincidental deviations from the measured value will be flattened/rounded/balanced out. The correct value will be **closest to the result we get most of the times** we repeat the measurement at the same object. Therefore, in result of many single measurements we expect to see Gaussian Normal Distribution. We should see a bell curve in the middle of which we expect to find our result.

The Bell Curve

In sum of all single (sub-)measurement results we expect an accumulation around the correct value, forming a bell curve. Comparable to the Schrödinger wave function, the bell curve represents the probability density of the expected sub-measurements and is the actual indicator verifying that our tool is a valid identification tool for the value we intend to measure. We can determine the precision of our instrument, we can fine tune and calibrate our measurement and optimize it according to our desired precision level.

The Error

In case we do not observe a bell curve which we could put an error bar on, then there must be a systematic error, statistical bias (spectrum, estimator, omitted variable etc.) or our experiment is disqualified by a different factor.

5. The basic Requirements

To be certain, that we are actually making a valid measurement, there are at least three requirements:

A. The First Requirement

If the risk we are exposed to is the result of a pathogen killing a percentage of the hosts it infects, this effect must be clearly visible every time we measure. If we do not detect a signal, although our measurement-method is confirmably functional, then we must assume that there actually is no signal. Or in familiar scientific terms;

Requirement I: *There must be **reproducibility** of results (between measurements/experiments)!*

B. The Second Requirement

In sum of all single measurement results we expect an accumulation around the measured value, forming a bell curve and confirming a plausible and correct measurement and thus, qualifying it as a tool suitable for the purpose.

Requirement II: *We need to see a **bell curve**!*

This is critical to understand:

The sub-measurement results need to accumulate around the measured value! The measured percentage value must be a characteristic property of the measured object: the virus' interaction with its host organism - and it must be measurable *independent of the location* of measurement. If there is no signal, there can be no bell curve - and vice versa.
Sharp peak or bell curve, whichever; We must see *any kind* of indicator hinting at the result!

The relevant Factors

Many factors can and will play a role and cause some distortion, such as a shifted/flattened/widened or asymmetric bell curve. Genetic variety as well as demographic parameters, like age distribution, avg BMI, quality and availability of nutrition, density/availability of local healthcare infrastructure facilities, cultural habits, hygienic standards etc. etc. asf. All of these will have an influence on the measurement of the value we try to measure. Hence, variability will inevitably affect the precision of

the measurement to some extent and we are very aware of that. And that is why we expect to see a bell curve and not a sharp peak signal.

C. The Third Requirement

The measured parameter must be reproducible within a certain range, that delivers and determines the confidence that we can put in the measured value. The coefficient of determination is directly related to the significance, which can be calculated and noted next to the result we measured.

Requirement III: We have to be able to determine the **significance** of the measurement result.

6. The Data

The absolute numbers of total confirmed cases (number of confirmed infections) and the number of casualties as reported officially by the countries and available from various sources (ECDC in our case) were used to calculate the case-lethality rates as described above. For every day of reporting, a number for confirmed infections (cases) was reported together with an updated absolute number of lethalities, respectively. As we have to expect due to the fact that a significant share of infections remains asymptomatic, there should be a substantial number of infections unregistered and unnoticed. The calculated rate can and probably will be higher than the actual rate, but not lower. Therefore, the calculated values will each provide us with a reliable data point (measurement value) for every country per reporting day during determination of the highest possible lethality rate. A country's high-level results will represent the national average over all age groups, BMIs, income levels and many other factors that in smaller population sizes could cause and explain distortions. On a national level however, enormous variances between the entire populations of two countries are not expected.

Most discussions about the results ended up in the data quality being questioned.

Therefore, again:

Yes, there might be variances in the protocols they used to test the patients. Yes, there could be countries who, for whatever reason, reported incorrect numbers. Yes, there are differences within each population. Yes, there are differences between separate populations. We expect variance and consider a tolerance range, wide enough to include for all these values. But claiming a majority of the reported numbers to be heavily distorted when otherwise having to acknowledge something is a very weak argument.
We must assume that the tests were qualified to reliably detect an infection and performed by routine professional personnel. The confirmed infections are reflected in the Total Case numbers. About the detection and absolute quantification of casualties in their result we do not have to be in doubt, presumably.

The Missing Peace

There is obviously a high variance in the reported Lethality numbers, while this should be the characteristic biological interaction that must me measurable within an acceptable range of precision.

The basic principles of scientific research do not allow us to put an **error bar from some value X+(Something) down to 0** and claim to be generating any scientifically useful results!
Correctly interpreted, this actually translates to saying ***"We are not able to demonstrate a direct relation between two factors."***

Taking comfort in accepting an average is not exactly what we call *Good Scientific Practice*. No journal would ever accept a paper with results like these or any conclusion based thereon.

*There **must** be another piece of the puzzle* that we have missed so far!

7. The *Meta*-Analysis

The CLR values were calculated *per country*, each one of which represents an individual contributor to our study, providing measurement values (probits) for the deadliness potential Λ of COVID-19.

Countries participating in the study must be able to provide a sample size of N ≥ 1.000 (or more where indicated) to ensure their contributions significance and to optimize the data quality for our analysis.

The obtained results were reported by the countries on a daily basis. The summarized reports were compared and analysed for obvious or hidden reasons that could explain the huge variance of symptomatics themselves and of their intensities, as well as of the mortality rates specifically, which we can observe.

According to H_0 and H_a:

From equation (viii): $\boldsymbol{\Lambda} \overset{\text{def}}{=} |\boldsymbol{X}| \equiv \boldsymbol{CLR}$ (?),

knowing we expect a linear regression line for Λ and therefore also for |X|, which (theoretically/ideally) is expected to be a line parallel to the x-axis in *quadrant* $\mathbb{I}$ on a plot against the CLR values on the y-axis. When considering the actually measured (recorded) CLR data the result of both origins, virus infection as well as the deaths caused by the nocebo effect, we can proclaim, that above the required baseline, we expect to see the casualties additional to the victims of a virus. For the number of potential deaths caused by the nocebo effect, we introduce a new variable Ψ:

ix. $CLR = \Lambda + \Psi$ which equivalently converts to

x. $\Lambda = CLR - \Psi$ and

as the identified baseline (minimal CLR observed in every (or at least in the majority of) location(s) will represent Λ.

Every number above that baseline must be in result of Ψ:

xi. $\Psi = CLR - \Lambda$

We will dive into differences from country to country, but also observe the development of reported deathrates within a set of countries over a period of time.

Friendly reminder for full clarity:

We are looking **only** at cases of **infections** registered and **confirmed**.

The way of infection and any epidemiological spread related factors are completely irrelevant here. We are looking at total numbers (except for the time series), from beginning of the pandemic: When how many tests were performed where and at what time are all irrelevant.

#Detected Infections ⇒ #deaths in result.

We are looking *only at country level data of confirmed infections* and compare the relative deadlinesses each country found after 1000+ infected samples.

IV. RESULTS

Table 1: CLR total per country Comparative Overview

France	30.696	267.077	11,46
Hungary	614	5.511	11,14
Mexico	63.146	585.738	10,78
Netherlands	6.211	69.063	8,99
Chad	77	1.008	7,64
Canada	9.108	127.358	7,15
Sweden	5.821	83.958	6,93
Liberia	82	1.298	6,32
Sudan	823	13.045	6,31
Ireland	1.777	28.579	6,22
Niger	69	1.175	5,87
Ecuador	6.504	112.141	5,80
Iran	21.249	369.911	5,74
Egypt	5.362	98.285	5,46
China	4.718	89.836	5,25
Andorra	53	1.124	4,72
Mali	126	2.736	4,61
Slovenia	128	2.797	4,58
Peru	28.471	629.961	4,52
Indonesia	7.169	165.887	4,32
Angola	106	2.471	4,29
Bolivia	4.846	114.409	4,24
Macedonia	504	13.927	4,19
Switzerland	1.724	41.240	4,18
Finland	335	8.042	4,17
Romania	3.507	84.468	4,15
Burkina Faso	55	1.358	4,05
Syria	103	2.563	4,02
Germany	9.289	240.986	3,85
Kosovo	488	12.938	3,77
Bulgaria	603	16.065	3,75
Denmark	624	16.700	3,74
Guatemala	2.709	72.921	3,71
Afghanistan	1.402	38.140	3,68
Sierra Leone	70	2.013	3,48
Gambia	96	2.797	3,43
Algeria	1.483	43.403	3,42
AVERAGE	837.579	######	3,38
Portugal	1.915	57.074	3,36
Colombia	10.767	590.520	3,18
Malawi	174	5.523	3,15
Brazil	119.504	######	3,14
Honduras	1.027	58.810	3,11
Poland	2.618	85.480	3,06
Bosnia and Herzego	591	19.214	3,08
United States	161.773	######	3,07
Lithuania	86	2.810	3,06
Zimbabwe	195	6.388	3,05
Nicaragua	137	4.494	3,05
Iraq	6.814	223.612	3,05
Vietnam	31	1.038	2,99
Guyana	35	1.180	2,97
Lesotho	31	1.051	2,95
Albania	271	9.195	2,95
Somalia	95	3.275	2,90
Uruguay	43	1.556	2,76
El Salvador	702	25.415	2,76
Chile	11.132	405.972	2,74
Austria	733	26.783	2,74
Moldova	981	35.904	2,73
Estonia	64	2.343	2,73
Greece	259	9.900	2,64
Democratic Republi	254	9.993	2,54
Norway	264	10.543	2,50
Latvia	34	1.375	2,47
Haiti	201	8.151	2,46
Bahamas	50	2.057	2,43
Kyrgyzstan	1.057	43.712	2,42
Zambia	283	11.779	2,40
Cuba	92	3.886	2,38
Turkey	6.245	265.515	2,35
Australia	583	25.448	2,29
Serbia	709	31.207	2,27
Mauritania	158	6.977	2,26
South Africa	13.743	620.123	2,22
Panama	1.966	90.624	2,17
Cameroon	411	19.142	2,15
Tunisia	74	3.461	2,14
Argentina	8.129	380.279	2,14
Ukraine	2.438	114.497	2,13
Pakistan	6.284	295.372	2,13
Senegal	279	13.384	2,08
Swaziland	91	4.461	2,04
Armenia	869	43.451	2,00
Togo	27	1.365	1,98
Montenegro	92	4.663	1,97
Congo	78	3.979	1,96
Nigeria	1.611	83.477	1,93
Japan	1.255	66.423	1,89
Croatia	180	9.549	1,89
Luxembourg	124	6.580	1,89
South Sudan	47	2.518	1,87
Benin	40	2.145	1,86
India	62.660	######	1,81
Morocco	1.052	58.489	1,80
Libya	226	12.629	1,79
Dominican Republi	1.648	93.390	1,76
Paraguay	280	15.874	1,76
Czech Republic	419	23.777	1,76
Russia	16.914	980.405	1,73
Suriname	66	3.848	1,72
Thailand	58	3.411	1,70
Kenya	567	33.630	1,69
Equatorial Guinea	83	4.928	1,68
South Korea	321	19.400	1,65
New Zealand	22	1.376	1,60
Philippines	3.325	209.544	1,59
Ethiopia	758	48.140	1,57
Guinea Bissau	33	2.149	1,54
Azerbaijan	527	35.986	1,46
Cyprus	21	1.481	1,42
Kazakhstan	1.781	130.040	1,37
Bangladesh	4.174	306.794	1,36
Malaysia	125	9.306	1,34
Puerto Rico	424	31.988	1,33
United States Virgir	14	1.075	1,30
Georgia	19	1.462	1,30
Central African Rep	61	4.700	1,30
Madagascar	187	14.696	1,27
Trinidad and Tobag	19	1.554	1,22
Saudi Arabia	3.813	312.924	1,22
Djibouti	66	5.439	1,21
Costa Rica	407	38.485	1,06
Uganda	28	2.756	1,02
Cape Verde	38	3.745	1,01
Lebanon	148	15.613	0,95
Jamaica	19	2.011	0,94
Namibia	65	6.906	0,94
Belarus	667	71.346	0,93
Slovakia	33	3.728	0,89
Venezuela	358	43.879	0,82
Jordan	15	1.889	0,80
Tajikistan	68	8.481	0,80
Israel	894	112.000	0,80
Guam	10	1.287	0,78
Oman	650	85.005	0,76
Uzbekistan	305	40.953	0,74
Cote d'Ivoire	115	17.797	0,65
Guinea	59	9.251	0,64
Kuwait	525	83.578	0,63
Gabon	53	8.505	0,62
Ghana	270	43.949	0,61
Palestine	160	28.110	0,57
Mozambique	21	3.697	0,57
United Arab Emirate	379	68.901	0,55
Malta	10	1.820	0,55
Nepal	195	36.456	0,53
Iceland	10	2.092	0,48
Aruba	9	1.906	0,47
Rwanda	16	3.747	0,43
Sri Lanka	12	2.989	0,40
Maldives	28	7.469	0,37
Bahrain	189	51.113	0,37
Botswana	6	1.633	0,37
Qatar	196	118.196	0,17
Singapore	27	56.666	0,05

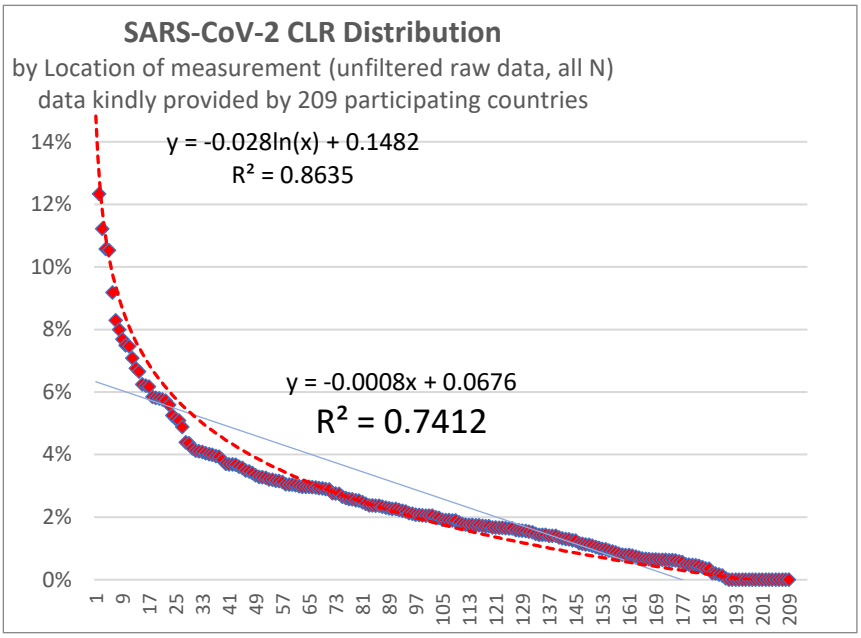


Figure 1: Relative CLR Value Distribution Plot

Raw data: No linear correlation visible. Exponential regression shows higher certainty.

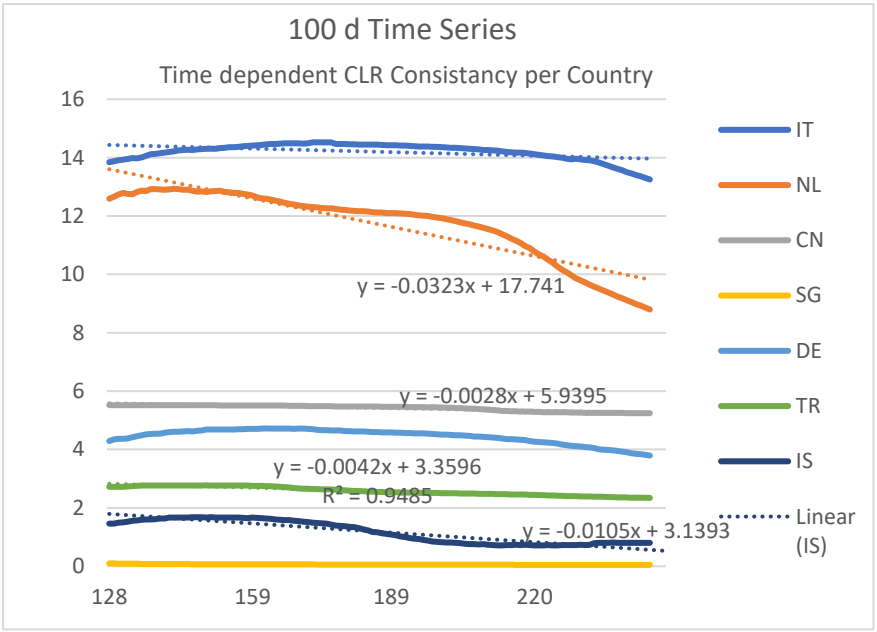


Figure 2: Time Series over a period of 100 days.

The measured values differ between countries. Within a country, they remain quite stable over longer periods of time

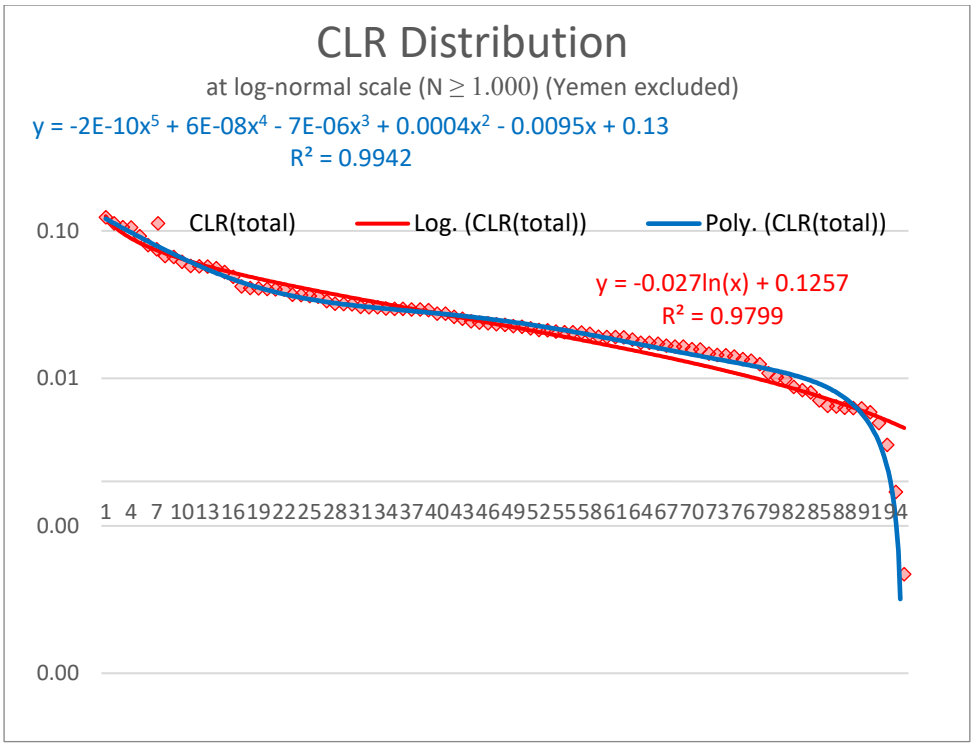


Figure 3: Lethality rates, Log-normal Distribution analysis.

Yemen was removed due to ongoing crisis. Values “too close to 0” were kept. Plotting on log scale confirms value distribution according to a power law with a certainty of r^2 = 98%, corresponding to a significance of p = 0.02! We have Pareto distribution. A 5th order polynomial regression gives p = 0.006.(!!)

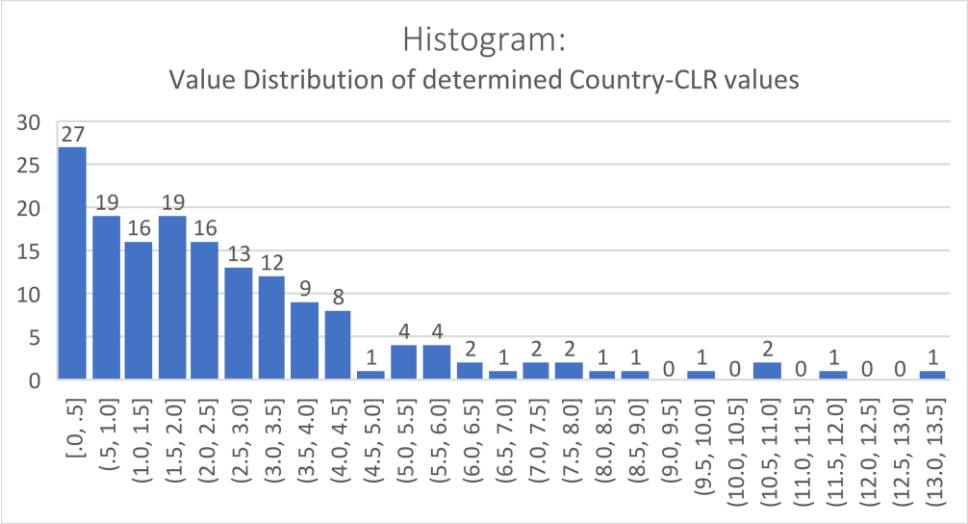


Figure 4: Histogram

The measured value distribution peaks between 0 – 0.5 %. While the majority of countries reported/calculated numbers are closer to zero, big lethality numbers are rather exceptional.

V. DISCUSSION

1. The Evidence

Table 1: CLR total per country Comparative Overview and Figure 2: Time Series over a period of 100 days. show that there is no consistent value we can identify and interpret as a distinctive signal baseline. The linear regression gives p = 0.26 and intersects the x-axis. The expected baseline cannot be identified.

While we expected a linear correlation between infections and deaths and hence, an approximately constant minimal rate, we see several countries, where even in 50000+ patients confirmed to be COVID positive (sample size N ≥ 50000 !!) the case lethalities are very low, more than a few are close to **0**:

Georgia	5552	28	0.50%
Slovakia	9078	44	0.48%
Cayman Islands	210	1	0.48%
Monaco	210	1	0.48%
UAE	91469	412	0.45%
Sri Lanka	3360	13	0.39%
Iceland	2623	10	0.38%
French Polynesia	1332	5	0.38%
Bahrain	68775	242	0.35%
Maldives	10098	34	0.34%
Curacao	360	1	0.28%
Burundi	485	1	0.21%
Qatar	125084	214	0.17%
Western Sahara	766	1	0.13%
Singapore	57700	27	0.05%
Anguilla	3	0	0.00%
Vatican	12	0	0.00%
Falkland Islands	13	0	0.00%
Greenland	14	0	0.00%
Saint Kitts and Nevis	19	0	0.00%
Laos	23	0	0.00%
Dominica	24	0	0.00%
Grenada	24	0	0.00%
Saint Lucia	27	0	0.00%
Timor	27	0	0.00%
New Caledonia	27	0	0.00%
Saint Vincent and the Grenadines	64	0	0.00%
Seychelles	143	0	0.00%
Bhutan	273	0	0.00%
Cambodia	276	0	0.00%
Mongolia	313	0	0.00%
Eritrea	375	0	0.00%
Gibraltar	379	0	0.00%
Faeroe Islands	460	0	0.00%

If a single "measured 0" (false negative?) were due to an error that causes a coincidental wrong result we would expect that measurement to be an exception. Instead, we see that values close to zero seem not to be exceptional at all.

If we allow for an error of a single probit, then 23 out of 209 countries, 11 % have apparently no problem at all, although the virus evidently was there.

Exhibit A: To emphasize what this fact alone actually means: Assuming each value we get reported gives us a significance of $p < 0.05$ (as they all align on the trend line, confirmed to be significant) the p-values of these countries need to be multiplied with one another. This leaves very limited options of interpretation…

But we see patients filling up hospitals and actually dying…!?!

If we consider every country a study with N = "# Cases" ≥ 1000, and a related number for the lethalities in result of these, we can calculate a death ratio which with N ≥ 1000 already should by itself be a representative and significant value for the measured dimension. Most regular "lab-scale" studies and their results we trust in are based on much smaller sample sizes.

Filtering by a sample size of N = 10.000+ samples does not change the characteristic, but increases the significance, which is another indicator confirming that our results are conclusive and plausible.

If the death cases were in result of the virus, we should see the same or at least an approximate value towards which most values tend. Reminder: In sum of all approximations (sub-measurements) we expect a gaussian normal distribution with a peak at the correct value. What we find is that **the majority** of results is closer to 0, while a smaller number of countries shows extremely high lethality rates.

Naturally, everyone is focused on the people dying to try and find the reason. Instead, even if it is hard to ignore the many casualties, when focusing purely on the numbers, considering them data points of measurements and applying the knowledge we already have and applied all the time, the facts are unambiguous.

Looking at Figure 4: Histogram, we realize, that in the *frequency distribution* of all measured values, we find the peak between 0 – 0.5 %. Correctly interpreted, this indicates, that the death potential of the virus is close to or potentially even precisely 0.

Exhibit B: Any value higher than that would else had been **necessari**ly visible in **every** country! The "smear" we see instead appear to be the statistical deviations – distortion - from the value we try to measure! They are the measurements that must be distorted by the effects of a nocebo mechanism that we will further investigate.

The Missing Bell Curve

This also explains the missing bell curve. We cannot see a bell curve because **we are trying to measure a 0**. We cannot have less than 0 deaths and therefore cannot measure negative values. The sub-measurements cannot deviate to both sides of the correct value. The measurements deviate only to the positive side where they accumulate. In sum of all measured sub-values, they clearly converge towards 0:

$$\lim_{N\to\infty} \Lambda_N = 0$$

The High-Level View

From single probits, adding up to measurable numbers and values, we now look at the quantities of actually measured results: how often do we observe which values? In the according histogram we see another peaking signal, but this time we are measuring a higher dimension of signal.

Exhibit C: The resulting peak in Figure 4: Histogram corresponds **to** a single pan-global measurement result and is the final confirmation that our conclusions are confluent and consistent.

We see it has its maximum somewhere very close to 0. Taking into consideration that we would have to allow for a tolerance for the (post mortem) detection of false positive infections and possibly cases of deaths unconfirmed and generalized to be reported as victims of the virus, it appears counter-indicated to assume any value for Λ greater than 0.

And therefore, the correct value and the result we are looking for must be:

$\underline{\Lambda = 0}$

The deadliness potential of the novel Corona virus itself cannot be confirmed to be any higher than 0! Instead of a linear and consistent distribution of measured case lethality numbers, we see that the distribution follows a power law. At log-normal scale we can see that the trend line and the datapoints of our measurement correlate with a certainty of 98%:

$y = -0.027\ln(x) + 0.1257$

$R^2 = 0{,}9799 \Rightarrow p = 0.02$

Exhibit D: The values we measure unmistakably demonstrate exponential behaviour. This means that we cannot be measuring a constant parameter, but the statistical distribution of values according to a power law. The coefficient of determination does not allow for any speculations. But what does this mean?

A. The Pareto distribution

Exponential probability distribution usually indicates randomness in the assignment of included data values. We find patterns of values with Pareto distribution and use the knowledge we have in different areas. The following list is from Wikipedia and links to the according Wikipedia pages:

7) **Occurrence and applications Wikipedia)**

Regression with a 5th order polynomial trend line finds

$y = -2E\text{-}10x^5 + 6E\text{-}08x^4 - 7E\text{-}06x^3 + 0.0004x^2 - 0.0095x + 0.13$

and

$R^2 = \underline{0.9942}$.

As we can see, the coefficient of determination rises to 99.42 %, which corresponds to a significance of **p = 0.0058**.

Considering the sample size we used, this would also be the level of significance we expect to see and is therefore also conclusive.

B. The Country-specific Parameter

Looking at the Case-Lethality Rates *per country* we can see that in Figure 2: Time Series over a period of 100 days., an approximately linear correlation in dependence of the country (= location of experiment) is visible. Data points demonstrate a suspiciously high value-consistency within many countries. The numbers on the x-axis represent the according day of the year. The locally measured values actually appear to be quite constant over a period of time contradicting a randomness based explanation.

The high consistency over time suggests that this value must represent a causality that varies between countries. Even neighbouring countries with high genetic and cultural identity demonstrate significantly different lethality ratios that are self- and time consistent, ruling out coincidental effect as an explanation.

Exhibit E: This confirms that these results cannot be related to any characteristic biological interaction with a pathogen.

2. The Ψ-co-Logical Factor

Obviously**,** Ψ cannot be a biological factor and a supposed relation to the wealth in a country is also not supported by the numbers. There is another country on the list which was removed from the statistics due to an ongoing crisis that could have distorted the significance of their data: Yemen, with almost 29% case lethality. It may be a coincidence but if the high number is credible to be a characteristic indicator and the increased rate was influenced by the fact of being in the middle of a war, this could be a factor of fear, pain, despair, suffering... maybe hope...?

It is not easy to bring this into a positive context.

From xi. (4.6) we noted

xi. $\Psi = CLR - \Lambda$.

Knowing that $\underline{\Lambda = 0,}$ we now <u>know</u>:

xii. **$\underline{\Psi = CLR}$**

Exhibit F:

This is the proof that the suspect is (almost) completely innocent. The nocebo effect alone must be the villain!

So, what is Ψ then? One idea was, that there could be an influence of average "media consumption", which would relate to repeated induction of the prophecy. However, that could not explain the huge variances and relatively low numbers in countries with comparatively high media consumption.

Media consumption as a function for repetitive induction could not be confirmed. This indicates that the influence of repetitiveness appears to be rather low in comparison.

Since the element we search for appears to be a major factor, capable of inducing a psychogenic death, it must be a very psychologically relevant phenomenon. As faith/susceptibility is apparently a factor that plays the main role with both creabo effects and repetitiveness does not seem to have a strong impact, there must be another factor that affects if a trigger will initiate a nocebo effect. The patient has to accept the information as a truth. Since believing a new information is related to not knowing better, education could have been be a relevant factor but that too is not reflected in the numbers at the national level pooled data we use.

On top of the list in Table 1, we see many European countries, basically all of them, except Mexico. With Italy on top of the list, one striking observance is noteworthy:

It appears that what could be commonly associated with *almost all* the countries showing the highest lethality rates, seems to be a high share of Roman Catholic population. In context with COVID-19 and the nocebo effect this could hint to something related to faith.

We know both creabo effects are directly related to believing something. However, this would lead to the assumption that members of other religions were less faithful, which appears to be an erroneous conclusion.

The Inverse Happiness Hypothesis

As believing is not simply the acquisition of information that does not contradict with the knowledge we have, there must be another factor that serves as a kind of gate keeper. This could be a factor of willingness. The readiness to accept the information as part of their truth. This is a very vague hypothesis and it would need to be investigated by psychologists:

A depressive person could accept a prophecy predicting him or her to die more readily than someone who enjoys life. Therefore the 'gate keeper' factor could be affecting what information we accept. If a depressive person is tired of life, (s)he is more likely to accept a negative prophecy as (s)he might perceive the prophecy about his or her death as a liberation from suffering, rendering the bad news to something worth wishing for.

From that sad view, the numbers could be interpreted as an indicator of the general depression level in a country. Or with other words: it could be *an inverse indicator for the average happiness level*. In that case, the news about the lethality of a virus would be readily accepted by people with a subconscious, latent or even discrete death wish.

I am not a psychologist and not educated or qualified to be able to make assumptions on the quantification of subjective psychological quality aspects and if measurements thereof would be able to confirm Pareto distribution.

Further investigation however, focused on countries on top of the list, led to results that are hard to ignore:

Chad, Niger, Sudan – also high rate countries, but without a pre-dominantly catholic population – are currently, or were in a crisis, recently.

If we include it into evaluations, Yemen leads the list with 28 %.

There is an active war in Yemen, fresh scars from a civil war in Sudan and Chad, and insurgencies in Niger. This strongly suggests general average anxiety could very likely be a factor we can expect to find at elevated levels. Reviewing background information with these factors in mind resulted in a row of further insights and conclusions.

VI. CONCLUSIONS

Looking at the results and the evidence they force us to acknowledge, we have to accept, that many of the mysterious observations we make in context with COVID-19 are actually not very mysterious at all. There is nothing about the 'pandemic', that cannot be explained by knowledge we already had. All those facts are very well known to science and modern science itself is even based thereupon.

It appears to be a fact that we are able to successfully ignore all the facts we know, simply to replace our most fundamental scientific knowledge by unvalidated common faith!
While certainly not helpful today, this will hopefully be the only occasion in human history, when we look back and think that 'a global collective head-palm' could be considered "probably appropriate".

1. Virological

The results provide evidence that an infection with the SARS-CoV-2 virus cannot be the main reason for the death of all these people. The intrinsic influence of the virus itself appears to be responsible for not more than an infinitesimal ratio of the lethal consequences. The exponential increase of lethality cannot be explained biologically.

A. The unviro-Logical Analogy

If we were to determine the case lethality rate of **blindfold crossing random streets**, trying long enough and generating enough data, the results we expect would look pretty much like what we see in Figure 1. This rate will not be constant either;

It depends on the street, which we want to cross. The street could be a small side-street which we can find in high abundancies, or a major highway which, as a share of the total number of all streets, is a much smaller chance to hit. This would be the rare deviation, the value that we measure would be one of the rare far-off measurements, while the many small streets are statistically much more abundant and the survival rate is generally high.

It's not the crossing of a street that kills us.

It's the cars, that we have to watch out for!

B. The *Metro*-Logical Explanation

In context with our nocebo hypothesis, this explains the huge variance in lethality rates which we expected to be a quantifiable parameter at a roughly constant level.
But what else are we measuring then instead? What are "the cars"? What causes the distortion that falsified our measurement of SARS-CoV-2 intrinsic Potential of Lethality?

Not being distracted by fellow humans dying in such abundance is clearly not easy. Considered as a simple numerical problem however, has revealed that the high death rates are actually nothing more than an anomaly, a distortion seen during the measurement of COVID-19 specific intrinsic potential of lethality.

We come to the logical and mathematically evident conclusion that COVID-19 cannot be the primary reason whatsoever. But it is a co-factor responsible for the many dead. Therefore, death appears where the virus is. But the virus appears also elsewhere where it does not kill many people.

2. Thanatological/Cardiological

Once a patient has believed us, that an infection will potentially lead to his death, this will cause them to be worried. Being confronted with our own end is a moment in life we all will have to face at some point. Even if our time has not come yet, the perspective of death and also what we possibly expect afterwards *"on the other side"* can and therefore will cause fears. The elevated possibility causes worries, inevitably. And as a psychosomatic consequence, worries lead to physical corticosteroid induced decrease in probability of survival.
Being told to be in one of the high-risk categories will lead to even more worries and higher levels of anxiety and accordingly, a more intense related physical stress response.

C. The Imagination of Death

Einstein said: *"Imagination is more important than knowledge".*

Therefore, let us imagine, **we are an average COVID-19 victim to be:**

As the nomenclature of our species indicates, we are all same. Hence, we should feel and think the same way. What would *we* feel?

We are a man, senior age, slightly over-weighted, (ironically) had a **corona**ry heart disease diagnosed already, and we are told (**induction**) that we will die (**prophecy**), once we get infected by the virus (condition and **trigger**-signal). The source of the prophecy could not be more trustworthy (**credibility**) and we see that people around us actually die (confirmation and **more credibility**, as *seeing is believing*):

The awareness of being in a high-risk group ourselves has been worrying and caused increased nervousness throughout the year. Being confronted with a ubiquitarian reminder of oneselfs transitoriness leads to the perception of a permanent subtle life-in-threat state, leading to activation of the sympathetic nervous system and increased levels of stress hormone release:

Catecholamine secretion leads to hypercardia while the awareness of its negative impact on our CHD create a positive feedback loop. The sustained perception of a threat of our life, over months and over the entire population– **will** result in an elevated total stress level **in average over the population**, while we are the ones (told to be) most vulnerable and hence, are the most affected and have the highest average stress level of all!

Once we notice, we have an infection, we anxiously go to a hospital, like we were asked to and where we hope to find healing and salvation. The setting (in terms of the environmental factors influencing our emotional perception) adds up to the positive feedback loop, which we will hereafter refer to as "*E**x**ponential **E**scalation of A**n**xiety **O**vershoot-**S**piral*" or simply ***XENOS:***

8) **The XENOS Mechanism**

The test confirms: We have COVID-19!!

The (perceived) worst-case scenario! What now?

Emotional excitation, epinephrine secretion, tachycardia, more fear!

The XENOS accelerates. This could potentially be accompanied by hallucinations or visions of what to expect ***"on the other side"*** - depending on which, perchance, this could have an extra amplificative effect on our acute episode of anxiety and the traumatic situation we are going through.

Depending on religious beliefs and pre-induced imagery of a possible hell, combined with self-doubts or self-awareness and self-confirmation of not being free of sins, are likely to be possible additional factors feeding the XENOS further up.

As the threat is intangible, muscular defense mechanisms to repell the threat are meaningless. *"You can't run, you can't fight!"* Two of the three options in a *fight-flight-freeze response* are deemed to fail, leaving a helpless freeze of fear. Linguastically, commonly referred to as "being petrified".

Vigorous reactions might therefore not be observed or recognized externally!

> ***"People of all ages who experience fever and/or cough associated with difficulty breathing/shortness of breath, chest pain/pressure, or loss of speech or movement should seek medical attention immediately."***
>
> ***– WHO Q&A, Symptoms of COVID-19***

Fever and cough can be assigned to the virus infection. All others **could be symptoms of an acute anxiety attack!** And those will be amplified in the hospital/emergency setting, where the people are asked to come to.

Once arrived in the – probably very familiar - hospital setting, where we see and hear about other patients dying, XENOS would accelerate building up momentum. A reaction might not necessarily be visible immediately, since phychological factors as trauma, shock, mental processing etc. can cause a delay, where the patient has time to realize his situation and tries to prepare himself for his own end. Possibly, after a while of helplessness and total submission to external factors – and seeing other people die around us, we would at some point start panicking while our CHD prevalent and additionally corticosteroid weakened heart would reach the limits of its durability:

…tachycardia, tachyarrythmia, myocardial infarction, cardial arrest, asystole and Exitus!

9) **The End**

C = 1

*Another one of the "**non-SARS**-like" symptomatic cases of **SARS**-CoV-2 induced **death**.*

In Figure 6 we see that the age distribution ***peaks between 80 and 90 years of age***. The drop at 90+ would be an expected effect in result of neurodegenerative symptoms of aging. Senile dementia, caused by Alzheimer's disease or as a complication of Morbus Parkinson, will keep the patients from receiving the induced message. As they are not aware of the prophecy, they cannot perceive the induced life-in-threat situation, causing less fear, less anxiety induced stress and thus, demonstrating a lower CLR.

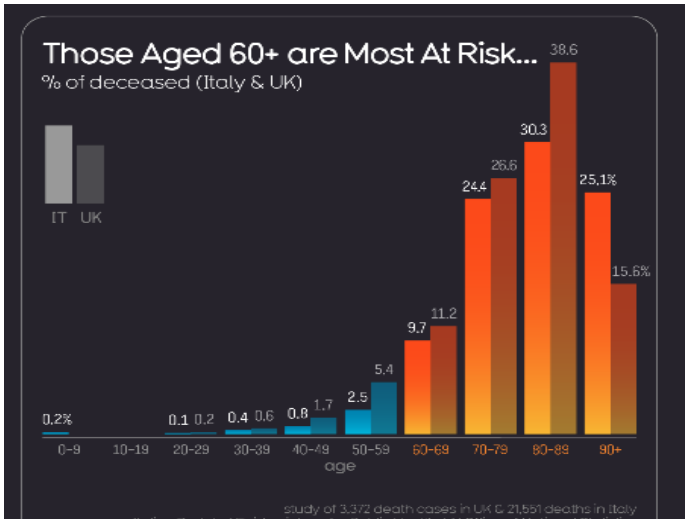


Figure 5: Death cases Age Distribution

Patients with Alzheimer's might simply forget there was a pandemic at all. However, me make sure that they are reminded day after day and maximize efficiency.

1) The Five Stages of Grief

According to the **Kübler-Ross** model of the five stages of grief, the confirmation of an infection itself could represent the **shock event**. In case of infernophobial condition(-ing), this might lead to an anomaly during processing, that **avoids surpassing stage 2** of resistance and reaching catharsis, which would lower the anxiety induced stress. Instead, previously conditioned Infernophobia could **lead directly into the XENOS** wheel instead

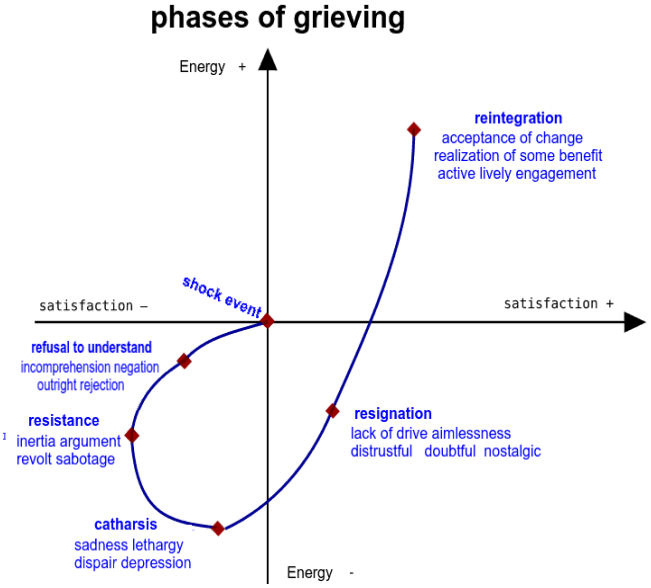


Figure 6: Phases of Grieving acc. to the Kübler-Ross-model

While we are not able to say, if this particular "sub-nocebo" would lead to the fulfilment of a self-fulfilling prophecy, for the regular case of acute thanatophobia, we must assume it could.

Further details and correlation with measured CLR values can be found in the Appendix VIII.11) The highest High-Risk Group

D. The Efficient Homicide

As the investigation has unvailed, the virus seems to be rather harmless and innocent. We ignited a Tsar bomb to kill a mockingbird. The villain is the anxiety itself that builds up due to the fatal predictions of the prophecy, while corticosteroids appear to be the most likely candidate for the main lever of the psychosomatic response that distorts our measurement by killing primarily CHD patients! They are what we detect as probits – **dead human samples** - instead of the lethality probits in result of COVID-19.

But it does not only falsify our intended measurement. We can quantify the distortion as its effect will be proportional to the distortion it causes locally. Per sample population we measure on, this can be interpreted as a basic anxiety level or a common pre-existing trauma. These resemble prestressed samples, which, once the XENOS spins, ends up deadly with a higher probability. With regard to the logical analogy this relates to "the cars" or the traffic on the street we cross. The survival rate will sink with increasing traffic, and as we can conclude about the expected death rate, the CLR will increase as well. As we can confirm, it does. And **it grows exponentially,** so we can thus confidently conclude by extrapolation:

> ***Inducing enough fear*** (increasing the number or speed of the cars), ***it is possible to effectively and efficiently reach <u>100 % death rate.</u>***
> Congratulations, gentlemen. Good job!

⇒ Analog to the ***bacter***icide we use against it, the fear from Corona we induce would relate to an effective ***hom***icide of highest effectivity.

3. Psychological

It may seem hard to understand that people can actually die, apparently, only because someone told them they would. While a single random person would probably not, as a mass phenomenon, <u>this is an inevitable consequence</u>. To connect the dots, we had to zoom out instead of looking closer at the victims. From the perspective of the experimenter, the analysis is easier because more familiar. And things we are familiar with, induce less fear than being confronted with death ourselves, hence allowing the correct part of the neural pile of biomass

between our ears to make the analysis and come to correct results. Also it keeps the conclusions from being distorted by the own fears of the experimenter.

Recommendation: It is advised that the patients focus their cerebral activity to their prefrontal cortex, and try to keep it isolated from excitational signals of their amygdala. These cause distortion of the decision making process and hence, lead to errors in evidently 100 % (1 false(?) probit out of 8 billion samples) of the patients!

E. The Gatekeeper Mechanism

Many scientist friends confirmed and agreed, there could be cases of death due to a nocebo "side" effect. Everyone accepted it to be a realistic possibility, but no one even wanted to imagine it could make a significant part of what we experience. Let alone, that most deaths might *not* be caused by a virus. There was no data at all, based on which we could have assigned a relative ratio. They simply could not believe it. We accept placebos for a fact and we know the significance of its effects. Talking about the nocebo and that this would resemble a global nocebo mass experiment, which would very well be a viable explanation for many of the observances in context with the pandemic was rejected vehemently by most of them.

They simply could not believe it. And it seemed they did not want to.

Even an objective scientific discussion about **what everybody should be talking about: 'possible reasons for the irregularities we observe'**, became impossible once even hypothetically considering that a nocebo could be responsible for a bigger portion than "a few percent". The imagination alone seemed to activate a defence mechanism that had built up against deniers. ***"Pagans!!"***

Some of the most intelligent people I know started saying things at some point that made no sense. It was reminiscent of a displacement activity. It seemed like their minds were repulsing what they were confronted with and started bending to avoid having to integrate things that collided with what they were believing in.. Or simply because they wanted to believe something else. It was nothing but a matter of faith. An ultimate creabo effect.

F. The Polarizational Blockade

The strong polarization among citizens and general pre-judgement of deniers ***"Heretics!"*** must have played a huge role here. There is no other way the entire world of scientists did not see the simple solution and actually started ignoring facts that did not fit into their models.

The world is not always what we believe it to be. And what we believe is often not based on confirmed knowledge but pre-judgements and generalization which is influenced by fears.

It is all in our heads. We saw people dying, we panicked and overreacted. Seeing is believing. **But believing is not an option in science**. Only understanding leads to knowledge.

G. The Characterization

Thanatophobia and - as an extreme form of it - infernophobia(!), can and therefore will lead to elevated states of anxiety, which can further increase up to acute thanatophobic episodes, initiating a deadly self-fulfilling-prophecy mechanism by leveraging an epinephral stress response proportional to the psychological subjective perception of fear, that predominantly endangers patients with pre-existing CHD conditions.

A thanatophobic death obviously is the most extreme symptom of anxiety induced symptomatics. Psychogenic self-necrosis cannot be the result of a generalized disorder.

Obviously, Infernophobia has an amplificative effect here and is a real thing, making it an extraordinary form of acute thanatophobic episodes. Both can lead to stress levels, able to induce a psychogenic death. Therefore, both must be considered separate from generalized anxiety disorders and cannot be generalized as such, as they are currently according to DSM-5!

4. Metaphysical

H. The Miracle

While we would not be able to come up with any possible mechanism for malign tumors to vanish unexpectedly and contrary to best scientific knowledge, this could be considered a "***miracle***":

> *A miracle is an event not explicable by natural or scientific laws.[2] Such an event may be attributed to a supernatural being (especially a deity), magic, a miracle worker, a saint, or a religious leader. - Wikipedia*

Fear respectively, is something very familiar, insomuch as being “scared to death” is even a common saying in many different languages. Death in result of fear can be explained by a very imaginable and even likely mechanism for the worst form of a nocebo effect.
The nocebo effect is not just a voodoo myth. What we see here is a mass effect, caused by false information.

Further investigation into high-Lethality countries revealed and confirmed high percentages of Roman Catholic population:

Country	Religious Information [according to CIA - The World Fact Book] (state of August, 31st)
Belgium	**Roman Catholic 75%**
Brazil	**Roman Catholic 64.6%**
Canada	Catholic 40.6**%** (includes **Roman Catholic 38.8**%, other Catholic .2%)
Chad	Muslim 53.1%, Catholic 20.1%,
Ecuador	**Roman Catholic 74%,**
France	Christian (overwhelmingly **Roman Catholic) 63-66%**
Germany	Protestant 34%, **Roman Catholic 34%**
Hungary	**Roman Catholic 37.2%**
Ireland	**Roman Catholic 84.7%**
Italy	**Christian 80% (overwhelmingly Roman Catholic)**
Liberia	*Christian* 85.6%, Muslim 12.2%
Mexico	**Roman Catholic 82.7%**
Netherlands	**Roman Catholic 28%,** Protestant 19%
Niger	Muslim 80%, other (includes indigenous beliefs and Christian) 20%
Sudan	Sunni Muslim, small Christian minority
Sweden	Lutheran 87%, other (**includes Roman Catholic**, …
UK	Christian (includes Anglican, **Roman Catholic**, Presbyterian, Methodist) 59.5%

Countries that do not have a dominantly Roman Catholic population show a different aspect they have in common. They are predominantly Islamic countries with a recent or ongoing conflict:

Country	Most recent Conflict
Chad	Chadian Civil War (2005–10)
Niger	Boko Haram insurgency (2009–present)
Sudan	South Sudanese Civil War (15 December 2013 – 22 February 2020)
Yemen	Second Yemeni Civil War (2015 – present)

The very last thing I would want to do is to point a finger at a community of faith, no matter which one. My apologies, should I cause someone to be hurt in his religious feelings. That is in no way intended by me.

What the data indicates, is that in countries with predominantly Roman Catholic populations, the lethality rates are higher, which in turn would indicate, that the general anxiety induced stress level – in average - is elevated, as compared to all other religions and also other communities of Christian Religion.

About Italy, we know(?) the high numbers are also in result of a high number of older people in the population. Specifically, being in acute threat-of-life situations, seems to have an accelerative effect on the increase of anxiety, where old people could feel less resistant, especially when a life-threatening virus is about to kill us from within our bodies. But how does that relate to religion?

This could be explained by the expectation of what may come after death.

The worst-case scenarios vary. ***The prognosis of dying is one thing. The prognosis of hellfire is quite another.***

The most rational explanation seems to be the imagination of God to be less merciful leading to a perceived increase in the probability of being sanctioned, leading to “hellfire expectation” and related fears.

I. The Un-natural Selection

Once the national anxiety level reaches a critical level, all samples who fall under the left egde of the bell curve will die! Dead samples are not remeasuarable. We have filtered them out already and hence, we cannot see their signal under the statistics any longer and so the remaining bell curve of the total measured signal gets sharper.

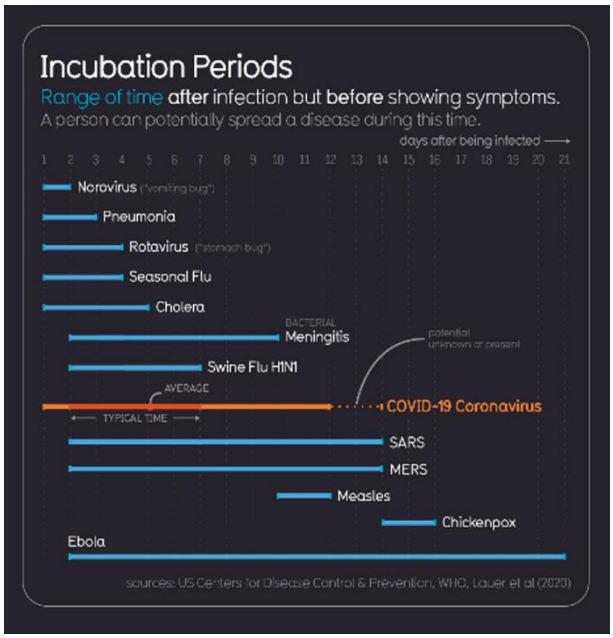
5. Epidemiological

As we must assume, the induced information can spread in every way a rumor can. Every pathogen could then represent a trigger for another collective nocebo response.

As it appears that the induction frequency of the information is not a significant factor, it seems not preclusive that even the Spanish flu could have been a regular flu fired by the nocebo effect. It was reported to be an *above average* regular flu in the beginning and later "caused by a mutation" (?) suddenly became much more deadly. In 1918 there were newspapers. But there was no PCR or DNA sequencers nor immunological methods. Therefore, the mutation can only be one of speculative nature.

Looking at the situation caused by a virus that apparently is much less harmless than influenza, a regular seasonal flu would represent a much better trigger, since it does usually not remain asymptomatic, as SARS-CoV-2 does in the vast majority of infections. Accordingly, this is a hypothetic case that at this point in time is <u>not preclusive at all</u>.

The Recommendation



Under these aspects, it is strongly indicated, that many of the epidemiologically relevant diseases have to be revised. Similar to COVID-19, they also show a way too wide range of uncertainty. If there is no bell curve visible in the statistical data, then there must be something wrong with the measuring method. The only acceptable ranges in the table aside seem to be Noro, Measles and Chickenpox. While incubation periods may be complicated due to uncertainties regarding the point in time of infection, the CLR **<u>must</u>** be within a reasonable **range that <u>does not</u> include values down to zero**, or at least 95 % of the integral under the bell curve of which not, according to the demanded significance of p = 0.05!! Everything else means we are not able to determine a number, therefore we must be missing something important!

J. The Domino Effect

Another recurring argument against the nocebo hypothesis is that people have become sick and died, who were deniers themselves. The issue is not easy as that, as people are always part

of a social environment. They can claim something under social influences like peer pressure, avoiding potential conflicts -which are likely- in result of the polarization we have created. If and when someone under such circumstances dies, the next denier will think his fellow had been a denier too, and died – inducing credibility of the prophecy. The denier is in doubt of denial and therefore not safe from fear, and there falls the next domino piece. And so might have started the global nocebo wave.

K. The worstmost Case Scenario

Technically, this domino effect could even lead to a chain reaction. Here, we have the virus acting as a limiting factor, required to trigger and ignite the nocebo and make the prophecy come true. And this one causes asymptomatic or only slightly symptomatic infections in most of the cases. An infection like a regular seasonal flu hardly goes unnoticed, as most of us will have experienced. The outcome in such a case would be devastating! But even then, we would still have a virus-infection as a limiting factor.

If a prophecy is successfully induced accordingly, and if there were no limiting factor, for example if the prophecy were triggered by an event, astronomical - a solar eclipse or maybe a minor impact, or something else, that convinces a critical number of people to be the trigger signal for an unavoidable prophecy, the nocebo effect could technically lead to *an unconstrained chain reaction*. And from what we know about chain reactions……!

We would not even want to imagine. While this ***nocebo ultimo event*** is hopefully an unlikely **very**-*worstmost* case scenario, being aware of the possibility of this remarkable phenomenon seems advisable.

VII. SOURCES

To save time spent for formalities, the sources are linked directly.

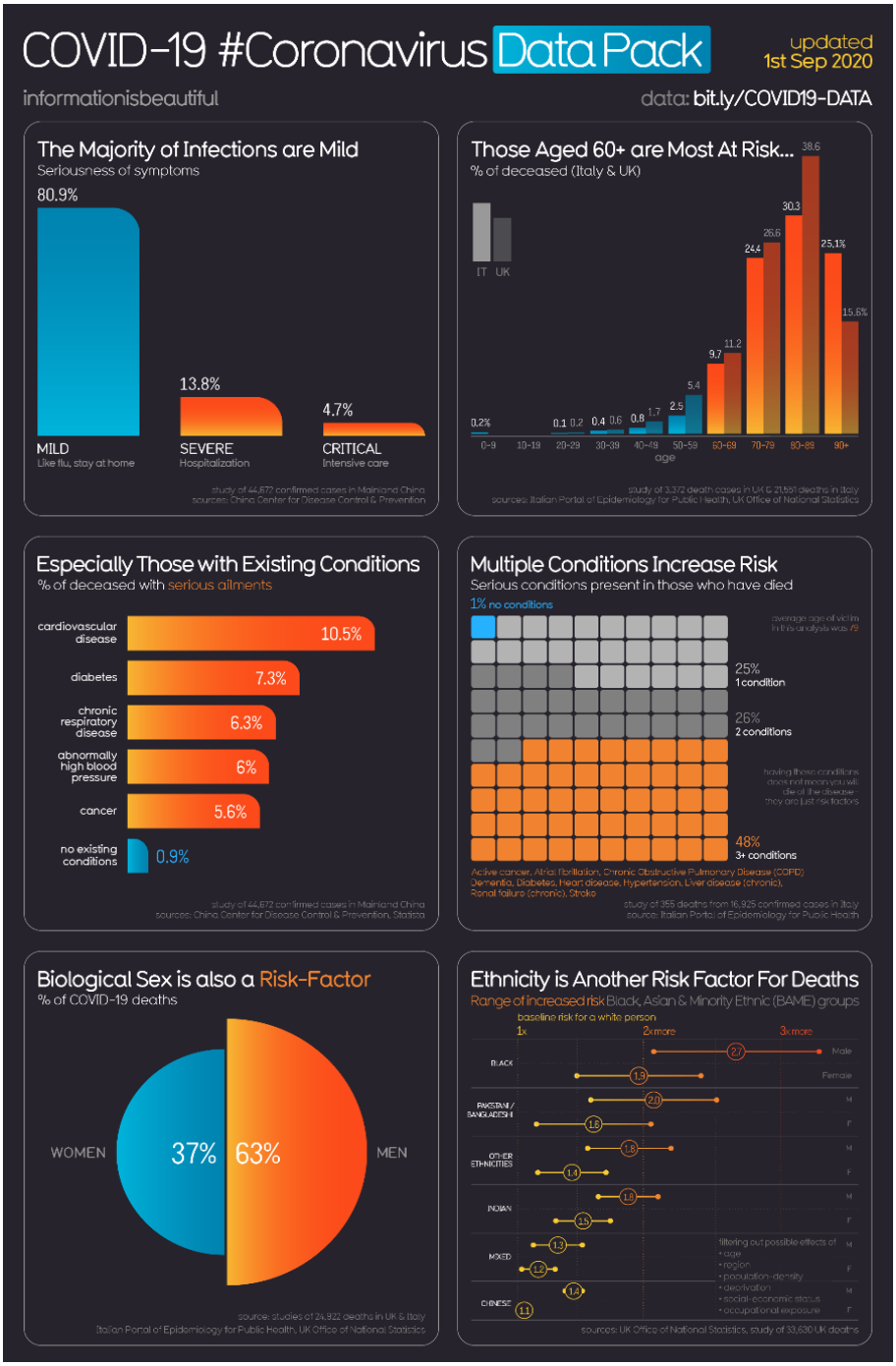
Further Information for comparison with CHD, among other sources, can be found here:

https://www.revespcardiol.org/es-the-epidemiology-of-coronary-heart-articulo-S1885585713003381

VIII. APPENDIX

1. The Reality Check

1) The highest High-Risk Group



The age distribution is identical to that of Coronary Heart Diseases (CHD) and lethalities thereof. It is also overlapping with the demographic distribution of Parkinson's or Alzheimer's disease; they are our **old people, who are dying**!

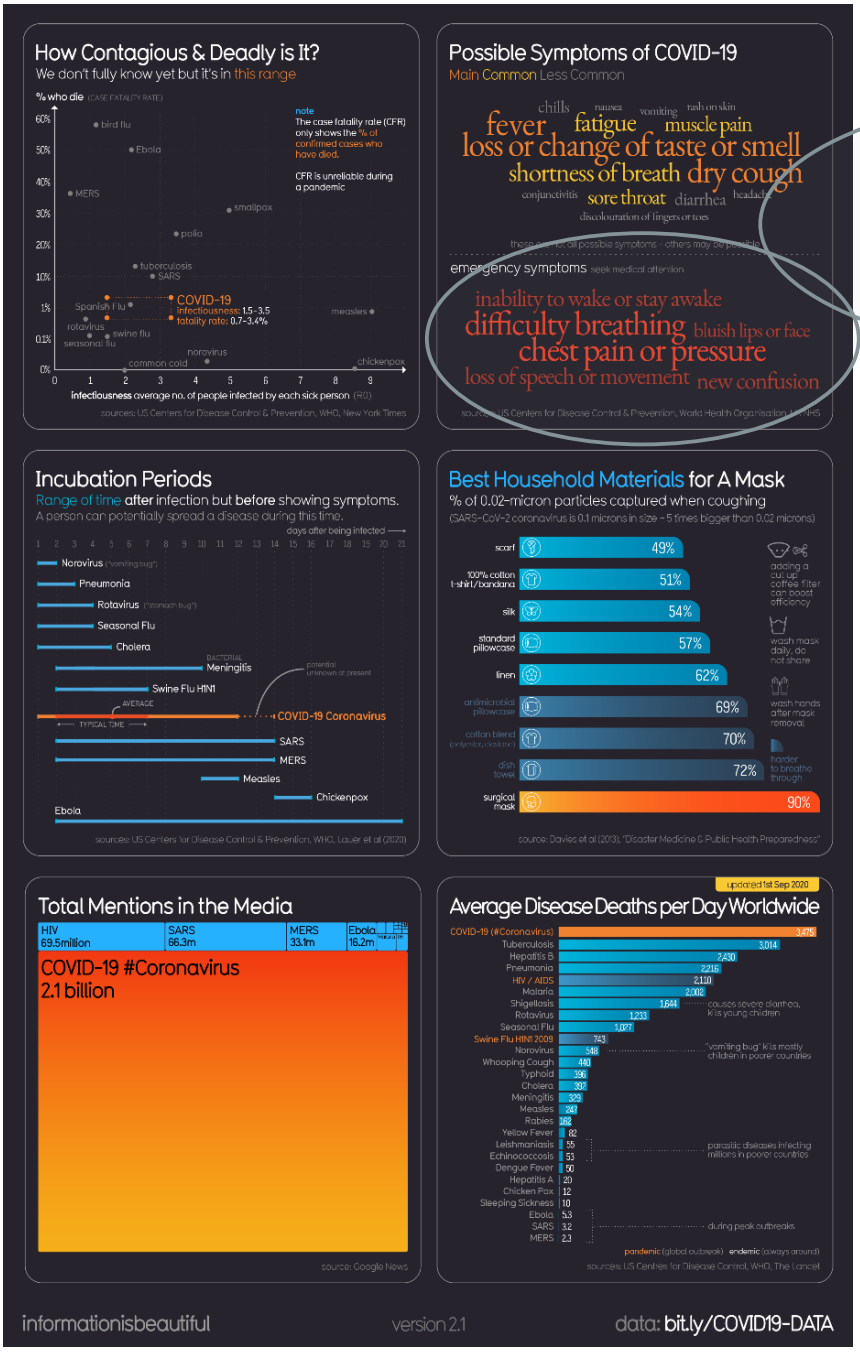
The decrease at higher ages is expected in result of senile dementia, leading to disconnection from the perceived environment, which makes them less vulnerable to external anxiety induction. This is in line and confirms our conclusion. If this effect did not distort our measurement of age distribution, the values would go further up. In this case we would see once again: Exponential behaviour.

2) The 'Biological Sex'

is self-explanatory, once the demographic character of CHD is understood.

3) The possible Symptoms

Apart from usual light symptoms of an immune reaction towards the infection with the virus (fever, coughing, sore throat, and general activation of the lymphatic system); the more severe cases listed here demonstrate suspiciously many symptoms in common with panic attacks:



The Symptoms of a Panic Attack:

feeling faint (inability to wake or stay awake)
a choking sensation (difficulty breathing)
chest pain
dizziness (new? confusion)
feeling like you're not connected to your body
(Loss of speech or movement: motoneural dysfunction!)"
a feeling of dread or a fear of dying *numbness or pins and needles* (sensory neural dysfunction)
shortness of breath
nausea
hot flushes (*rush on skin?*)
chills
sweating
a racing heartbeat
a need to go to the toilet
dry mouth
trembling
shaky limbs
ringing in your ears
a churning stomach
a tingling in your fingers

Source:

https://www.nhs.uk/conditions/panic-disorder/

4) The Ethnicity

is definitely **not** a risk factor here! People of African origin appear to be 3 times more likely to die in result of the infection, which is likely the source of this **belief!** Looking at Africa, the rates are **actually very low**. This contradicts heavily with a possible genetic origin of causality. However, considering the numbers here are even naming another common characteristic: BAME is correctly identified, and must be considered a possible common factor. **This can be observed in the US as well as in the UK and most likely also in France.**

It is not the race that plays a role, it is their status within a population. BAME, as this abbreviation indicates are generally immigrant minorities of origin from different continents.

Minorities are more likely to be on the poorer side of a population. Therefore, in general, having less of a financial fundament and more existential pressure to worry about, leading to increased levels of stress. While not confirmable on national level, another explanation would be education, but specifically within this sub-population. He who knows less has less capacity of critical thinking and hence, has to believe more of what others say. A third factor that should not remain unmentioned is the reciprocal correlation of education and experienced violence on a core-family level. People growing up in lower educated families are subject to an elevated risk of exaggerated patriarchy and physical or psychologic violence within the families.
Each of these factors, over longer durations will render the individuals subject to higher levels of anxiety induced stress and thus, these will inevitably demonstrate higher CLR's.

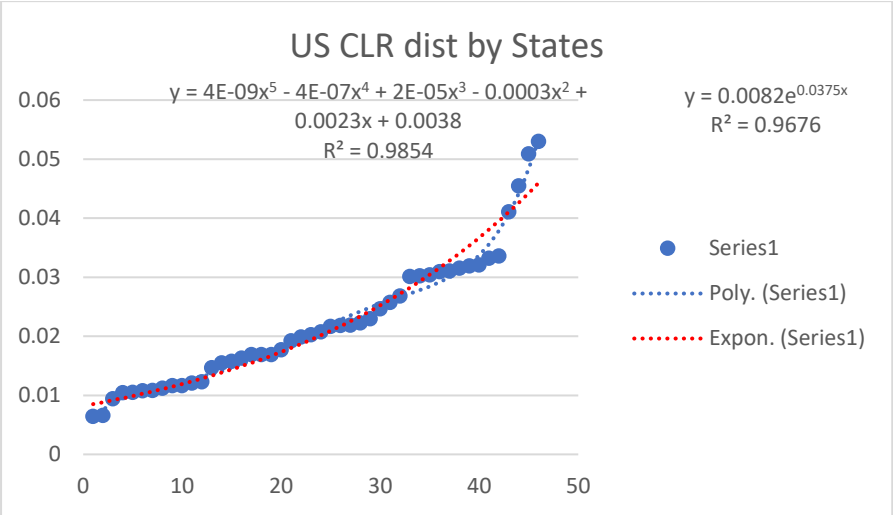
2. The Closer Look

Analysis of CLR (or Ψ) values confirm Pareto distribution on every level. Countries, states or regions within countries or in 'town by town' comparison.
And within each town it will probably reflect the share of population, that is subject to the highest stress levels in general, plus the additional stress load due to induced anxiety, facilitating development of acute thanatophobic episodes.
The local geographic peaks in signal (most deaths) will be in the hospitals of course, where we also expect them. But we would not have expected, that the hospital itself would have a risk increasing effect. However, this would be your explanation why in many African and other poor countries with less dense medical infrastructure the death numbers are so inexplicably low, although the worst scenarios were expected to take place there:
The population has less access to a hospital and therefore, the setting in which the deaths are concentrated is not given. Our hospitals attract the afraid population. Typically, the ones most afraid will be the first in a hospital – and unfortunately, the first to die - (and up goes the credibility of the prophecy in result of the first cases of death).
If this can be confirmed, this will prove that the hospitals themselves resemble concentration camps of XENOS.

“Class” dependency must be clearly visible in the statistics. BAME, minorities, people on the lower end of our communities and whoever else spend their lives (and esp. the recent months) with elevated stress levels due to the perceived feeling of an existential threat.

And within these “classes”, we are killing primarily our elderly men and women.

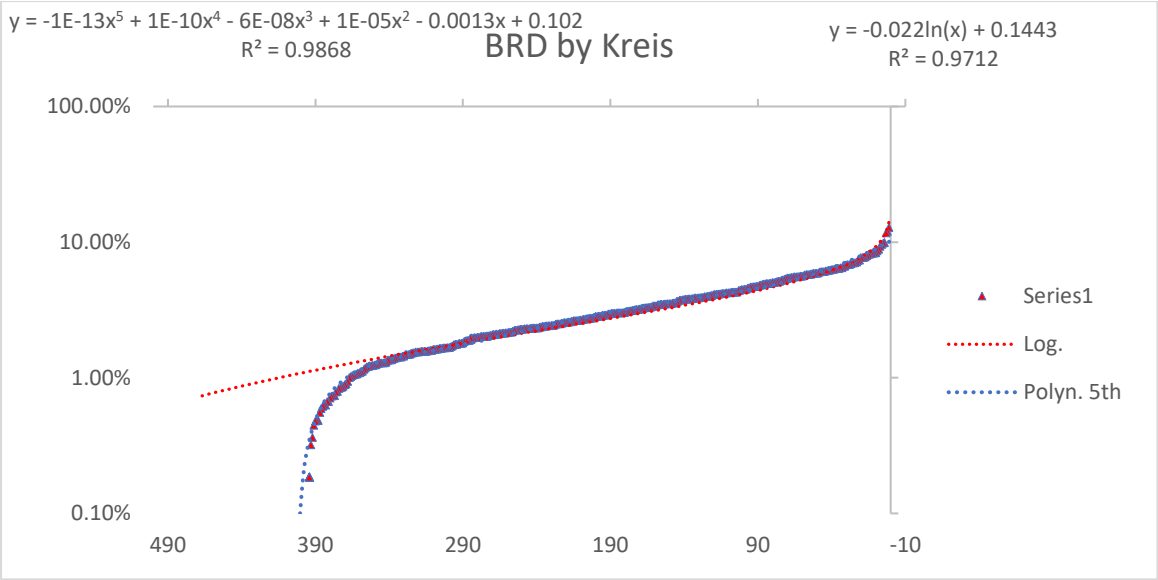
The United States



Pareto distribution: *true*.

Plausibility of biological interaction: *false*

The German Circles



Pareto distribution: *true*.

Plausibility of biological interaction: *false*