Pre-existing immunity to Covid-19 – Marc Girardot of PANDA unpacks its evolution

16th August 2021 by Nadya Swart

In June, BizNews published an article by Marc Girardot – a strategy consultant and a member of PANDA – in which he thoroughly addressed the question of whether those who have recovered from Covid-19 should get the vaccine. In order to answer this question, Girardot focused on the robustness of natural immunity. In this article,

an equally important and powerful follow up, Girardot explores the extent and evolution of our pre-existing immunity to Covid-19. Girardot eloquently shows that before it even existed – human beings were largely immunised against Covid-19 due to cross-immunity. He underpins this assertion with numerous references to studies and research papers, one of which found that 80-98% of blood samples taken throughout the globe had cross-reactive T-cells. In simple terms, this means that 80-98% of the world population likely has a significant degree of pre-existing immunity against SARS-CoV-2, which Girardot argues is an explanation for the overall low fatality rates of Covid-19. Using simple analogies, Girardot addresses the various concerns that surround Covid-19. He compares the pandemic to a wildfire without "dry wood", stating that 'this pandemic had no fuel. In other words, people were somehow "immune", and indeed many were asymptomatic.' As with his first article, this article is loaded with hyperlinks – each of which provide the source of those facts asserted in the article. Girardot thoroughly looks at Covid-19 infection in children, concluding that "Beyond the psychological harm, it is very plausible that the isolation measures have deprived elderly of their best weapon to fight: the gift of super immunity from their grandchildren." – Nadya Swart

A novel perspective on a not so novel virus

By Marc Girardot*

Within a few years of Columbus' landing in America, 95% of native Americans were decimated by viruses imported from Europe: 19 out of 20 American Indians died. Not cohabitating with livestock as intensely as Eurasians had for millenia, Aztecs had not built layers of immune defences against influenza, smallpox or measles. Surprisingly, Spanish conquistadores resisted better in the face of the Indians' own viruses. Likely protected by the Aztecs' own herd immunity, the Spaniards were not overwhelmed by massive viral loads contingent with an epidemic in a defenceless community.

If these "Noble Savages" weren't without sin, the most gruesome of which was

child sacrifice – they were without virus, at least European ones: Native Americans were "naive" immunologically. Confronted with European viruses, their immune system was like a new-born child seeing for the first time: it had the hardware, but not yet the appropriate software to fight back. No one being immune, the epidemic "avalanche" was uncontained and unstoppable: everyone cross-contaminating one another repeatedly with higher viral doses. The death toll of the invasion of the Americas ended up being cataclysmic: **2,000 times deadlier than Covid-19**!

Image 1 - Native American dying of smallpox introduced by the Spaniards

Credit: Bridgeman Images

Since January 2020, WHO, health authorities, academia and mainstream media have been contending that SARS-CoV-2 is a completely novel virus, and that it is particularly lethal. There is no doubt, SARS-CoV-2 can be a nasty virus at the individual level. But, why hasn't the same cataclysm hit our highly connected "naive" world? Why hasn't the same vicious circle of repeated high viral load contaminations occurred? Why hasn't "the fire caught on and consumed this immense quantity of dry wood?"

The only logical answer is that **SARS-CoV-2 was absolutely not novel.** And therefore everything has been blown out of proportion with some very dire consequences for public health and for public liberties.

Having killed allegedly 0.05% of the world population in 18 months, SARS-CoV-2 is evidently not particularly lethal to the community. A simple comparison with the ravages of the Conquistador viruses suggests that SARS-CoV-2 isn't novel after all, and that populations were originally largely immune to it from the beginning.

In the following lines, I will debunk with facts, data and reason further fallacies and explain why SARS-CoV-2 was never the monstrous threat pictured by the media, by academia and by health authorities.

Like a Running Wildfire?

Viral epidemics are as hard to model as wildfires. Too many variables can influence the outcomes and the unfolding. What we do know from experience is that when all the conditions are right – plenty of dry wood, scorching sun, strong wind, dry bushes… everything will burn with intensity and there is no stopping it: protected cork-oak forest, green meadows… everything will burn.

> According to the media, all the conditions were right for the SARS-CoV-2 "wildfire": an overcrowded planet, ever growing urbanisation, traveling and interacting like never before, a large ageing population and a 'novel' virus.

There is no question that, if indeed we had absolutely no immunity against this virus, all the conditions would have been combined for a catastrophe of epic proportions. An epidemic that would have made the Aztec extinction look anecdotal.

But, as a wildfire without "dry wood", this pandemic had no fuel. In other words, people were somehow "immune", and indeed many were asymptomatic.

Supersized or fantasised?

Prior to March 2020, doctors treated patients with COVID-19 so well that most didn't notice it had become prevalent. With the incomprehensible ban on family doctor treatments, the systematic use of intubation, the panic in hospitals and care homes, the inordinate use of qPCR, along with financial incentives to diagnose COVID, the death numbers were likely artificially inflated.

We now know that – even untreated before hospitalisation – the COVID-19 mortality was much lower than initially stated. We also know it could have been much lower had COVID-19 been treated as it was prior to March 2020, or as per the recommendations of many doctors on the frontline.

In the first few days of the crisis, dramatic images of deaths were broadcast across the globe, reminiscent of the Great Plague. Experts were spewing out scary fatality rates as high as 6% which would have equated to each and every one of us losing 9 people we love! Thankfully, this apocalyptic scenario never materialised!

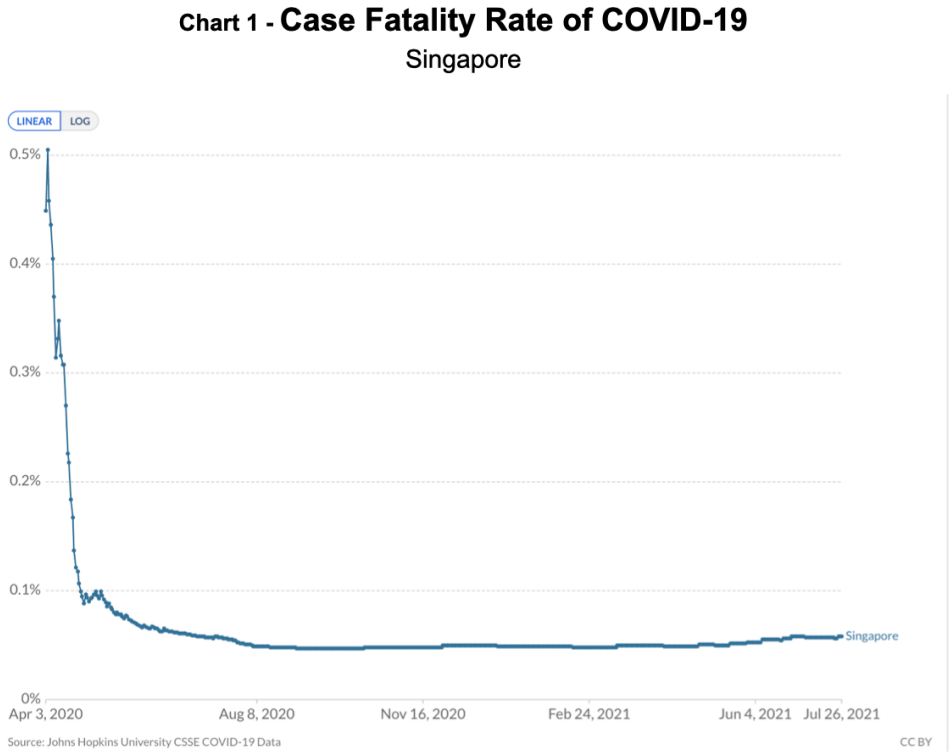
From the start, fatality estimates had been on a roller-coaster ride: From a panic-seeking 6% to a steep fall to the Diamond Princess's 0.6%, down again at 0.36% with Prof. Streeck, to a more realistic low plateau at 0.1% with Prof. Giesecke, up again to 0.68% in an Australian piece, down again early this year when Prof. Ioannidis stated the average fatality rate was 0.15% for 2020. The good news is roller-coaster rides always end on the low side. A few hard-core fans want another run, but quite frankly the run is over.

After 18 months of scientific ethics stampede, who can still trust scientists? So let's see if we – simple mortals – can find trustworthy data to confirm SARS-COV-2 lethality without complex modelling…

Three proxies to estimate the COVID fatality rate

Here are a few simple ways to estimate the true mortality of COVID using verifiable data:

- Imagine a detective investigating every single COVID death widely. Singapore – by that metric – is unmatched with **423,570 tests per COVID death.** The US, France or Germany respectively have tested 831, 893 and 716 per death. In other words, Singapore has tested 500+ times more than other leading nations, and naturally commands a greater precision in its findings. **The estimated fatality rate in Singapore is 0.06%**, a serious stick in the ground: 100 times less than the original scaremongering prediction…



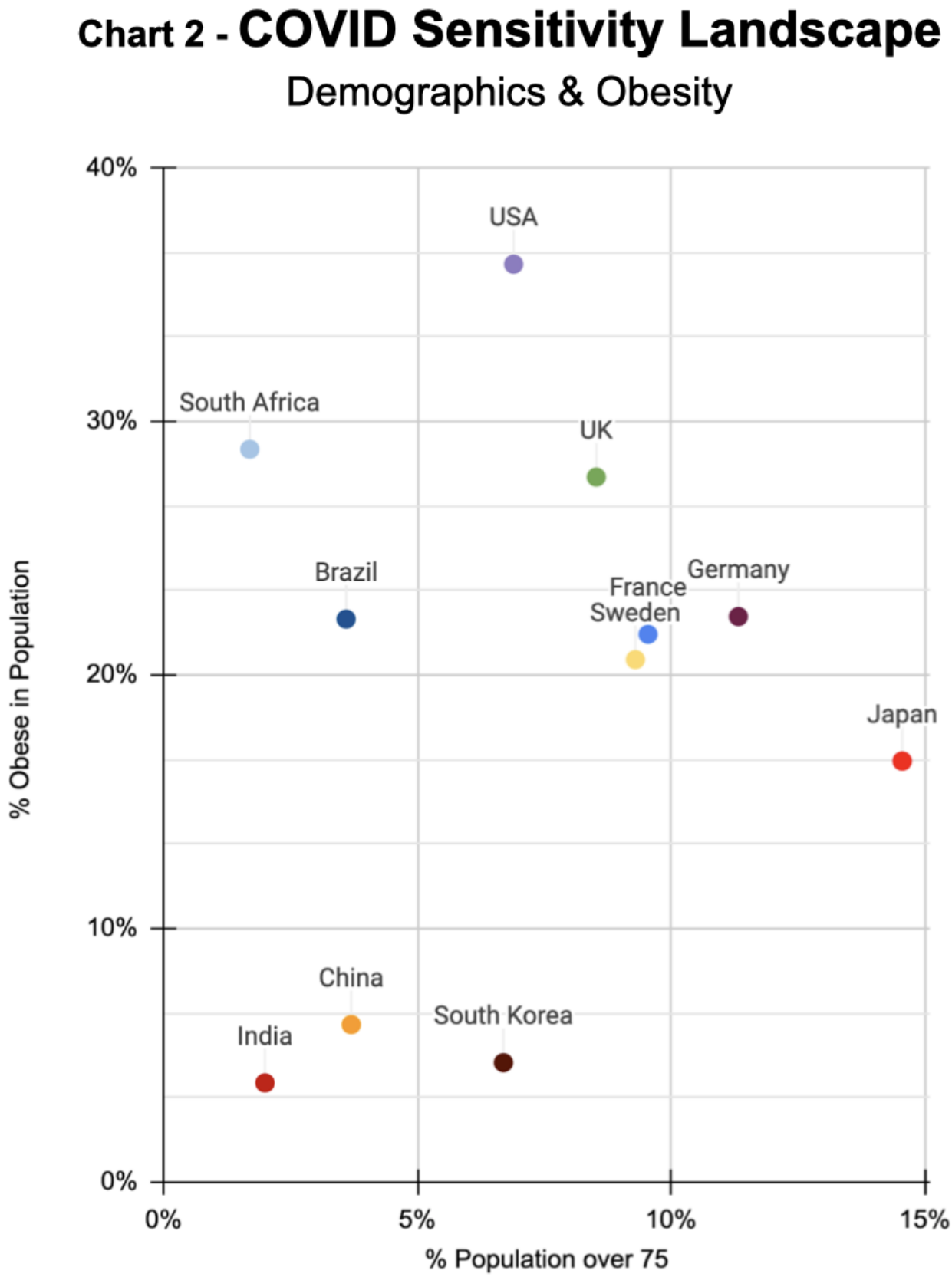
Source: ourworldindata.org

- Another interesting piece of information comes from pharmaceutical company Regeneron. Last March, they announced promising results from their antibody treatment clinical trial. Their placebo arms comprising 2,089 untreated infected patients caught my attention because only 4.1% ended up in the hospital. Knowing that "at risk patients" represent a maximum 25% of a population, that 31% of hospitalised patients typically transfer to the ICU, and that today – with decent treatment in the hospital – 19% in the ICU die. The

"Regeneron" data points to a US IFR of 0.06% today and **0.12% in 2020**, i.e. 15-30 times less than current US case fatality rate.

- In the spring of 2020, I set out to simulate the New York City epidemic. Using only real verifiable and redundant data: temperature reading from Kinsa connected thermometers, asymptomacy levels of caregivers to cancerous children and delivering mothers, NYC mortality growth rates... All these data combined into a number of infected in New York City of 15.5 million (estimate on May 10, 2020). At the time, the official death toll was 15,217! So there is no escaping it: in New York **the COVID IFR was ~0.1%**.

Depending on timing, on demographics [see.Chart 2], on treatments, on general public health and on density levels, the infection fatality rate in a general population seems to range between 0.01% and 0.2%, essentially comparable to a mild to robust flu, but then again family doctors get to treat influenza.



The Tip-of-the-Iceberg

You're probably thinking "*Hold on, Cowboy! You are saying 15.5+million had been contaminated in New York in the spring of 2020?!*" Yes, that's what the data

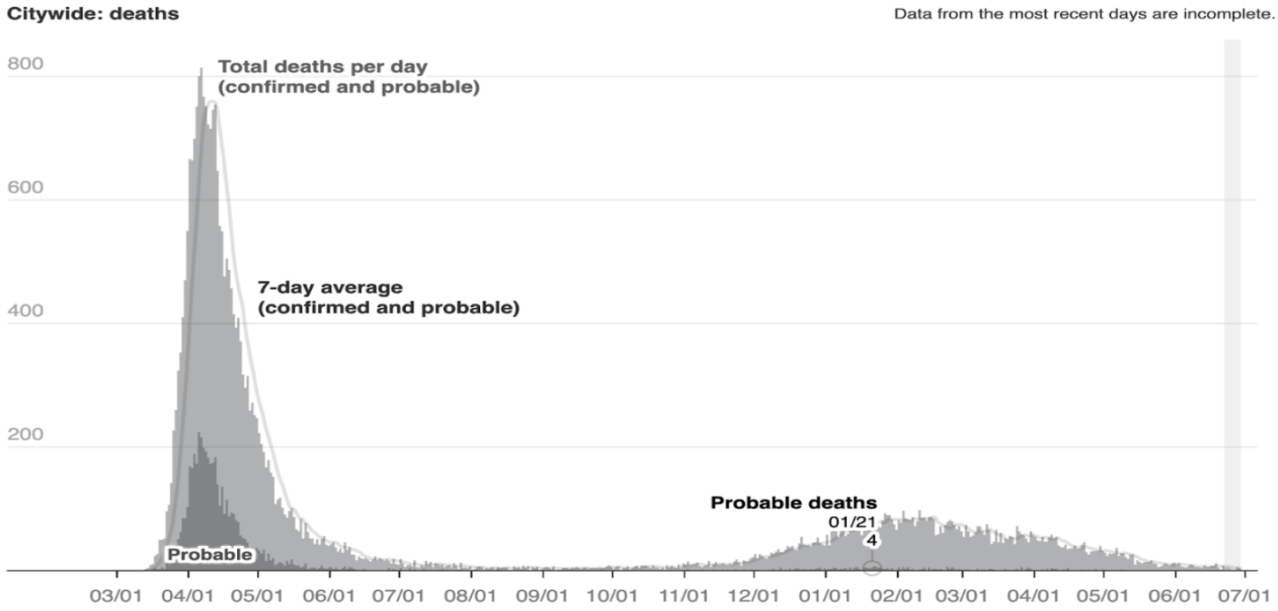
indicates. If Health authorities didn't see the tidal wave of cases, it doesn't mean they didn't exist; it simply means they were invisible. Most probably never even seroconverted.

NYC health authorities weren't alone in not seeing what was right in front of them. At the time, the US Navy's surgeon general said: "*We're learning that stealth in the form of asymptomatic transmission is this adversary's secret power*". Similarly, when the French aircraft carrier *Charles-de-Gaulle* came back to its base, the French Navy was convinced it had a mere 50 infections on board. In reality, they had 1,046. For every 1 spotted, they missed 20. Experts at threat detection managed to identify only 5%; the remaining 95% hidden in plain sight, as **the asymptomatic contaminations weren't actually making sailors sick**.

Therefore, it is reasonable to think that, on land with a proportion of elderly and sick in the population, health authorities would spot five times more, and still miss 3 out of 4. If only 1 in 5 New Yorkers had been contaminated last year, another 80% would have been susceptible. Another sizable seasonal peak would have naturally occurred. But the second tsunami never materialised as the chart below clearly demonstrates [see.Chart 3].

Chart 3 - New York City Covid-19 Death Toll

March 2020-July 2021



credit: New York Health - source: NYC.gouv

A NOT SO NEW VIRUS

So, why is this virus not making everyone sick? Why haven't we seen the catastrophic predictions, materialise?

> Ironically, most researchers are focused on the many viral and immune intricacies of SARS-CoV-2, but essentially, they miss out on taking a more systemic perspective that explains quite simply what has occurred: almost nothing is new with SARS-CoV-2.

Like a water leakage, the sooner stopped the better

Let me try an analogy close to home to describe virus propagation through the body. A viral infection is like a water leak in your home: the bigger the leak (aka *viral dose*), the larger the damage. If you are lucky enough to have an automated detection system (aka *Resident memory immunity*), the water supply will be

stopped instantly with no damage (aka *asymptomatic*). If not, the water will continue flowing, adding up, becoming visible enough (aka *fever*…) for you to realise it and run to cut the water supply. You'll have a lot of mopping and drying up to do, occasionally leaving stains on the walls and ceilings (aka *long COVID*). But if you are away, and come back after work, with a very delayed response time (aka *immunocompromised*), your house might be entirely in ruins. You will need to run everywhere, actively try to save the house from actually being destroyed.

A Typical Unfolding

In reality, the SARS-CoV-2 propagation in the body unfolds with exactly the same process as the flu penetrating the respiratory gateways:

1. A viral dose enters the body through the airways, either in one discrete event or possibly multiple successive events.
2. The immune system has the capacity to reject it immediately – or not – based on context, on dose level and on immunity status.
3. If it can't reject it, the virus continues to propagate until the immune alert "rings" and triggers a more robust immune arsenal.
4. The arsenal – fever, T-cells, antibodies… – then rids the body of all virions and kills every infected cell in the upper respiratory tract, causing minor cellular damage (loss of taste/smell…).
5. If the immune alert or the reaction is delayed and/or if the immune reaction is weakened, the virus continues to propagate and grows exponentially, penetrating deeper into the body, conquering and damaging critical organs like the brain, the heart, the lungs…
6. When the immune arsenal finally kicks in, the battlefield is no longer in the mouth or the nose, but across the entire body,,.
7. T-cells scuttle precious infected cells, causing major damage to vital organs and creating life-threatening inflammation and thrombosis, throughout the body.

8. Finally, if the patient survives, the body-battlefield needs to heal where it can, and suffer, occasionally irreparable damage (fibrosis…), a Long Covid, with lifelong consequences.

In other words, minimising the clinical impact of COVID-19 is all about early intervention, either therapeutic or immunological, and also about avoiding high dose contamination as much as possible. That is true of all coronaviruses and of the flu, and any decent doctor knows that.

Most Were Already Immune

> From the very beginning, everyone was oblivious of the "elephant in the room": **COVID-19 shares 65-82% genome with other main Coronaviruses.**

Why is this fact so critical?

Roughly 21,000 nucleotides are shared between SARS-CoV-2 and other coronaviruses, that's nearly 15 times more than the Moderna and Pfizer vaccines. Common coronavirus colds permanently circulate the planet, and have been infecting people for centuries: billions of people have had to gain immunity against the most immunogenic parts of this long string of RNA.

Past infections have had to act as yearly "vaccination campaigns". Year after year immunising the population. **Even before it ever existed, people were largely immunised against COVID-19.** This is a well-known concept called cross-immunity that exists also for the flu. **Cross-immunity is a natural immunological safety net, most likely, the principal reason for the relative mildness of this pandemic**.

All brushed under the carpet…

No media has addressed the topic that SARS-CoV-2 is absolutely not new. In fact, many scientists have been aggressively trying to discount it: Interestingly, some contend circulating antibodies and T-cells triggered by the vaccines are a proof of immunity, but that pre-existing cross-reactive antibodies and T-cells aren't… as if the immune system was able to discriminate.

> Cross-immunity and vaccines are based on the same immunological processes. **Natural and vaccine-induced immunity are simply two instances of the same biological platform.**

If one believes in vaccines against COVID, one necessarily needs to believe that past coronaviruses have already robustly immunised a big part of the population. The immune system is indeed agnostic to the virus, and processes the vaccine and the virus in the same way at a biological level. The only difference – for now – is that current intramuscular vaccines do not mimic virus penetration, and miss out on a formidable opportunity to be much more effective…

A recent survey found that 80-98% of blood samples taken throughout the globe had cross-reactive T-cells. Cross-reactive cells are immune cells that can target shared elements between distinct viruses, notably all the commonalities within the coronavirus family.

> You can go back and read that again: **80-98% of the world population likely has a significant degree of pre-existing immunity against SARS-CoV-2**, and that surely explains the overall low fatality rates.

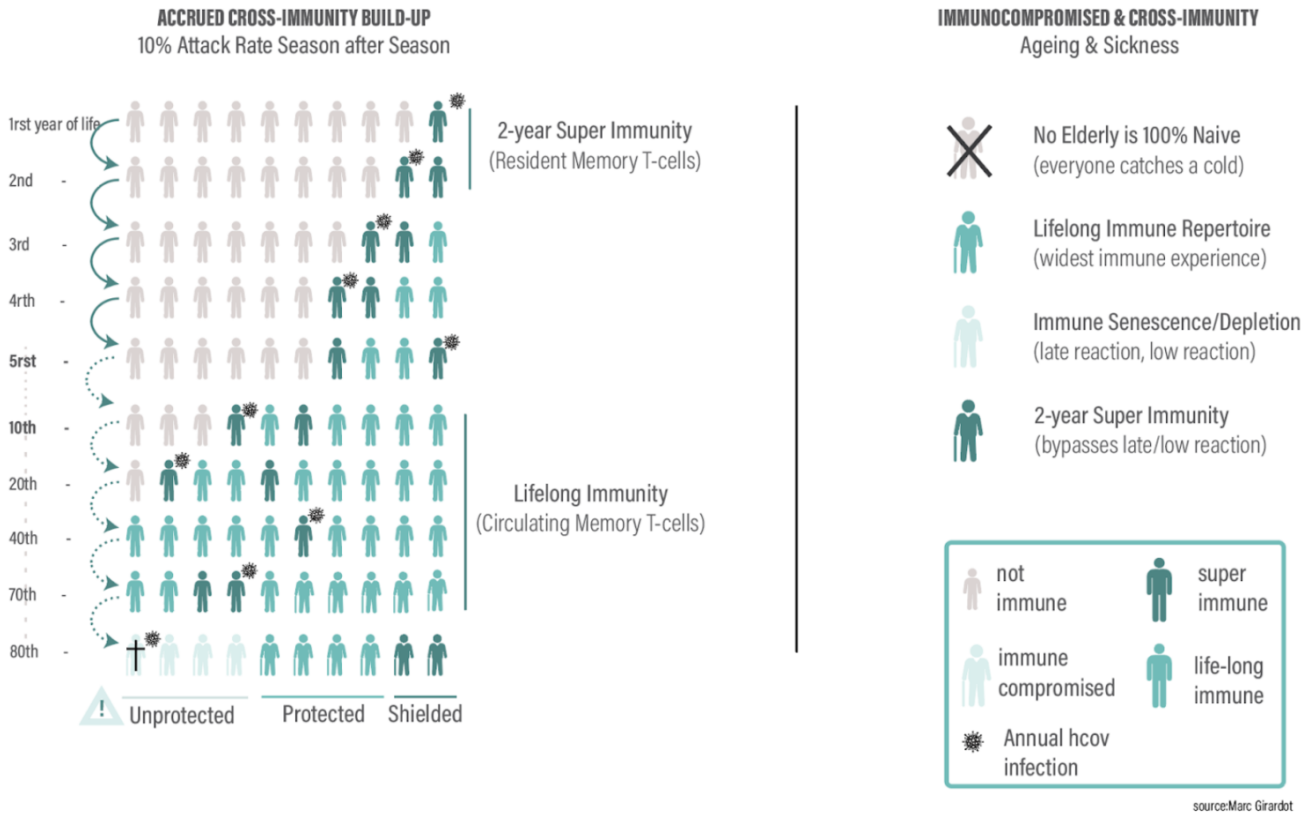
This layered immunisation acquired throughout the years by communities across the world is **confirmed by more than 20 different papers** in all continents [Exhibit 1]. This confirms that this virus is not at all novel to our immune systems, and why this pandemic is in fact relatively mild, contrary to what some epidemiologists predicted.

The Protective Umbrella of Cross-Immunity

Let me articulate how cross-immunity likely translates in the epidemic dynamic. Every year, coronavirus induced colds hit the population essentially based on the duration of the winter weather – how long we lock ourselves inside – and on the density of the population. The more people in a square kilometer, the higher the virus density, the more people are contaminated. The denser the city, the more prominent the "super spreader" conditions.

Imagine coronaviruses hit roughly 10% of the population every winter season. A 1-year old will have a 10% chance of being immune. A 2-year old will have a 19%, and a 10-year old 65%. A 25-year old would be immunised in 93% of the cases. And this is consistent with the data collected on the field; for example in Kansas where 67% of children had pre-existing antibodies to common colds whereas adults had 93%. [Exhibit 2]

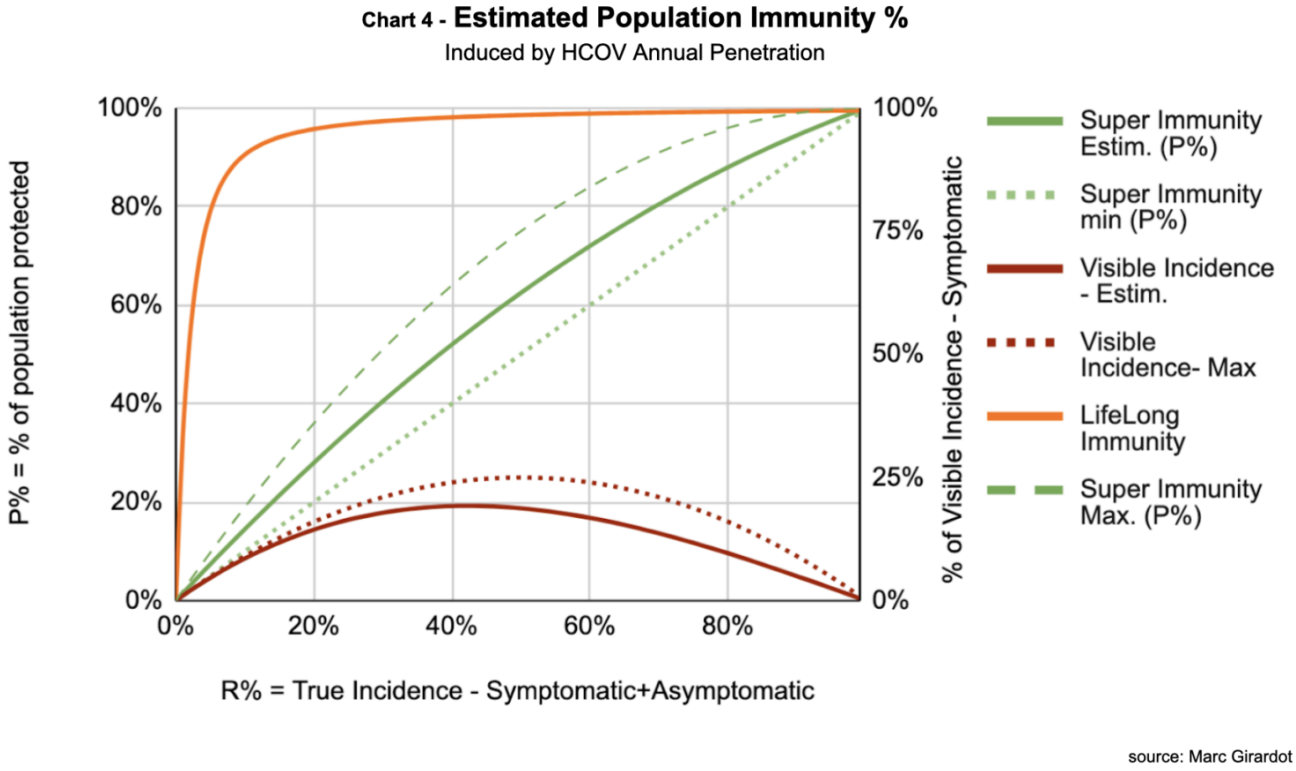
So, if adults had pre-existing immunity, via cross-immunity, why are people still falling sick?



To understand this, one must understand four distinct immune populations and how they play out in different contexts:

1. **Immunologically Naive** – those are essentially children and some teens untouched so far by coronaviruses. They benefit from a very strong innate immunity, but their immune system database is limited.
2. **Super Immune** – those infected by a coronavirus in the 2 years before SARS-CoV-2 benefit from a form of temporary "super immunity". After an infection, a very strong preemptive immune force develops in the portal of entry of the virus – called tissue resident memory cells. This force impedes renewed infection for roughly 2 years.
3. **Immune for Life** – those infected more than 2 years ago still benefit from a vigorous reactive immunity – but not instantaneous – response. They would generally not be in danger, but the infection can be symptomatic.
4. **Immune Compromised** – either temporarily or permanently, despite a life-long immunity, these individuals have the immune information on how to fight, but either the alert message gets delayed or they end up having a weak immune

response. These are the people principally falling seriously ill and dying of COVID-19.



source: Marc Girardot

The percentage of each of these four populations will vary considerably depending on the infection rate of the region they live in [see.Chart 3]. The denser the region, the higher the rate of infection and the corresponding protection, notably this 2-year super immunity. If common colds hit 25% of a particular region every year, up to 44% of the population would have super immunity [25%+25%.(1-25%) = 44%] and 97% would have a life long immunity.

This likely explains the extraordinary track record of densely populated areas such as Asia and Africa during this pandemic, more so than NPIs that were often started too late to be effective. A recent serology study from Hyderabad in India showed 54.2% had antibodies against SARS-CoV-2 in January 2020. Given the rapidly waning of antibodies, it's probable Hyderabad was hit 80% or more, giving its population in the densest areas 86% super immunity, in line with 9 asymptomatic in 10 infected revealed by the study.

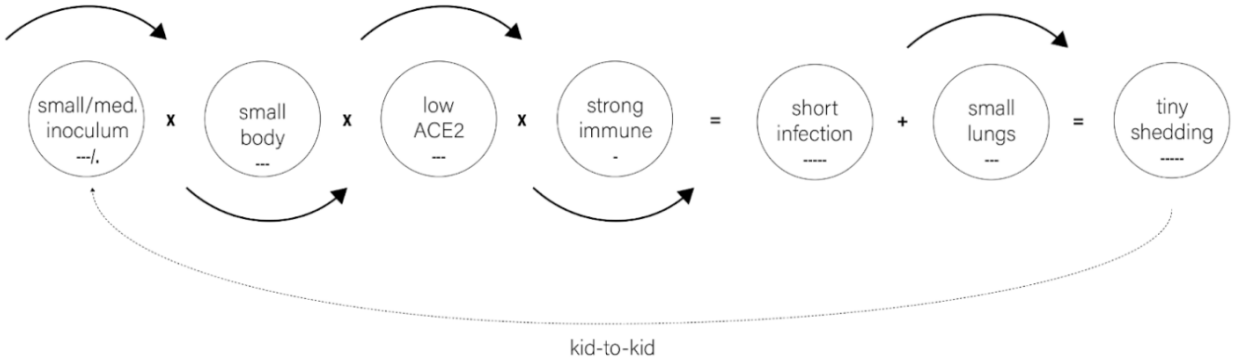
Even less dense regions where only 5% of the population catch common colds each year would have a 10% “super immunity” and a 79% “lifelong immunity”. It is very clear that cross-immunity likely prevented a cataclysmic pandemic like the one Native Americans succumbed to by simply dampening the virion doses produced by each infected person. And highest density countries provided a super immunity shield to their eldest citizens more so than in lower density countries explaining the mortality discrepancy between Asian countries and Europe or the US.

Harmless Children

In my earnest opinion, scientists finger pointing at children couldn’t be more wrong: kids are precious contributors dampening the consequences of epidemics, and likely play a critical epidemiologic role in providing protection to the elderly. They have been shown not to contribute to family disease. Couples with children are more likely to be asymptomatic than couples without. One study found that “Children were five times more likely to have seroconverted without symptoms compared to adults”. Outrageously, and in contradiction with scientific evidence, kids have been accused of endangering the lives of the very grandparents they cherish.

> Recently, a John Hopkins study announced that **ZERO healthy kids had died of COVID-19 in the United States**. So, if kids are so poisonous, why aren’t they poisoning each other? Why aren’t they poisoning themselves?

Scheme 1 - Naive kid Viral Dynamics



source: Marc Girardot

The reality is kids have much smaller bodies, so their virus production capacity is naturally smaller. They have far less ACE-2 receptors per cell than adults which hinders the production of virions. On top, children have very effective and vigorous immune systems, extremely reactive which cut short virus propagations. With smaller breathing volume and mild cases, it is quite evident that – despite being proportionately more naive immunologically – they never constituted a threat to the community and most likely not to the elderly. In any case, children are much less a threat than the vast majority of adults. It is also very likely that a higher percentage of kids are "super immune" (see below). Hence, the narrative that children are dangerous is as ridiculous as saying that the main risk on a high traffic road filled with heavy trucks is a small bicycle…

> Beyond the psychological harm, it is very plausible that the isolation measures have deprived elderly of their best weapon to fight: the gift of super immunity from their grandchildren. It is quite evident that it is preferable for an elderly to get infected by a grandchild occasionally, than by immunocompromised neighbours constantly bombarding them with heavy load inocula in care homes.

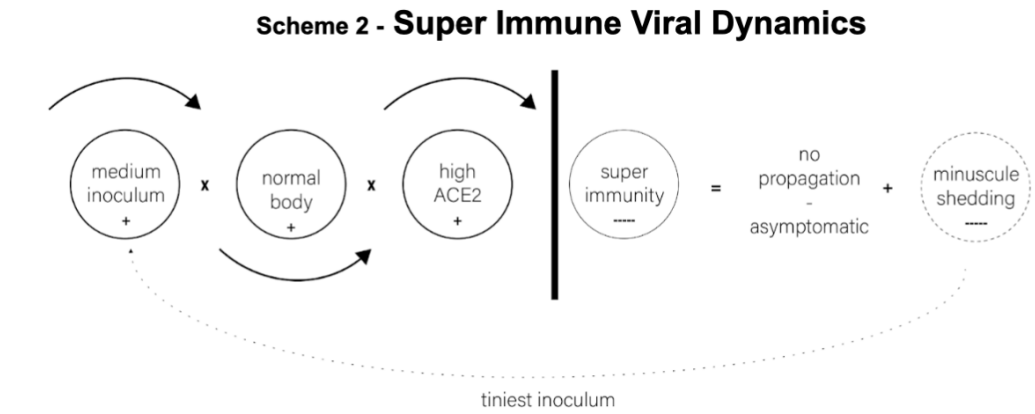
The children were the victims of this pandemic. Not only have our children been grossly mistreated: uselessly gagged for months, deprived of education, of motor development and of indispensable social interactions, but also they've been put in grave danger physically, immunologically, and psychologically. One recent study – rapidly retracted – seemed to show children were breathing for months – at a minimum – 3 times the 0.2% CO2 legal limit. In Germany, another study highlighted 53% children reported headaches, 42% malaise and 38% impaired learning from mask-wearing. Facts are that we have abused our kids all for nothing.

The Super Immune

After an infection, in the portals of entry of the virus – mouth, nose, lungs or digestive system – battalions of sentinels – called resident memory cells, both B and T – build up in the tissues and are ready to kill the next infection "*in the egg*". They are ideally located to quell the infection in the passage of entry where the virus is most vulnerable. If virions pass through, this first line will inevitably have dampened the impact of a high dose inoculum, like soldiers rejecting siege ladders to limit the numbers of invaders into a castle.

This vigorous instantaneous immunity typically lasts up to 2 years – if not stimulated again. This makes a lot of evolutionary sense as most epidemics last two years. When you've just had a war, you keep troops guarding the curtain wall for a while… then after a few years, it's too many resources allocated versus a waning risk, so you just leave one fellow to ring the alarm, and redirect resources to where they are more needed.

The presence of resident memory T-cells is dependent on one meeting a virus with significant genetic similarities in the 2 years prior to the infection. The denser the place one lives in, the higher the chance one benefits from this super-immunity.



source: Marc Girardot

This would be a particularly important mechanism of immunity for anyone immunocompromised – elderly and/or sick – because it is off-the-shelf, there is no delayed reaction. In that respect, in the traditional family model, elderly probably benefited greatly from this permanent protective umbrella acquired through the very low viral shedding of their grandchildren. The low death rate in China points to elderly possibly benefiting in large numbers from that protection. Building resident memory T and B-cells in the respiratory tract is a particularly interesting endpoint for a new class of vaccines: **an aerosolised vaccine that could engender the proper immune response to kill the infection early on and stop transmission,** as opposed to existing vaccines that have failed to provide "sterilising immunity".

The cross-immunity safety net

As shown by many studies [Exhibit 1], a very large proportion of the population, **essentially almost all the adults, had a significant pre-existing cross-immunity against SARS-CoV-2**. As one ages one necessarily has caught a cold, at least once, if not repeatedly.

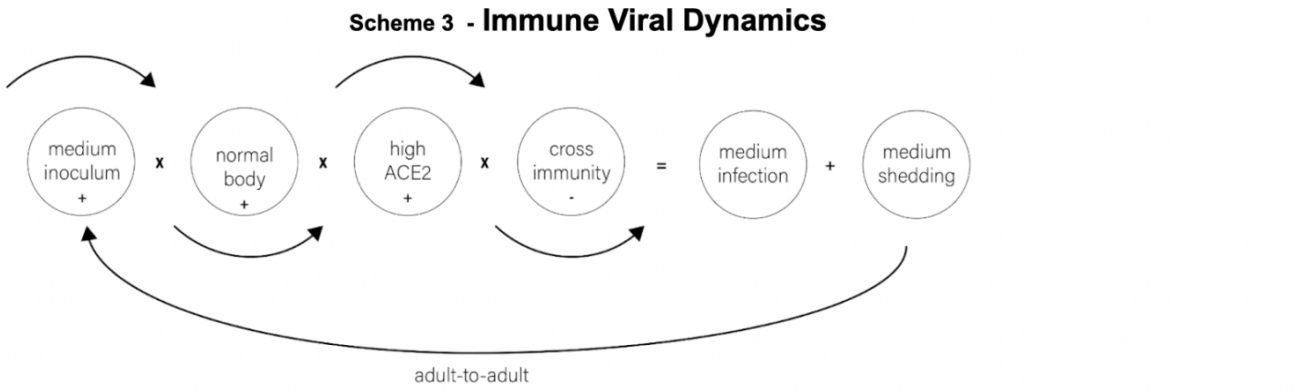
If not stimulated anew, the resident memory cells recede from the respiratory tract, possibly to leave space for new ones triggered by another seasonal epidemic, or simply as a matter of resource reallocation.

However, sentinel T-cells called "circulating memory T-cells" – imagine roaming

guards – are still active. In case of renewed "invasion", they cannot cope with the enemy just yet, but they can sound the alarm and trigger a sort of "immune conscription" to launch a specific counter attack to a new infection.

Given the more diffuse presence in the blood and the lack of *action-ready* T and B-cell cohorts, a short delay to sound the alert and build the immune battalions is required, time during which the virus can continue to expand.

If a parent gets a small dose from a child, he/she will likely remain asymptomatic. But a medium dose inoculum will trigger symptoms despite this lifelong immunity. This immunity protects against serious illness as long as the host is healthy. However, it does not guarantee against transmission, nor against symptoms.



source: Marc Girardot

> Overall, this wide immunity – largely dispersed throughout the community – acts as a safety net, reduces the average viral load transmitted and mitigates the "avalanche" effect experienced by Aztecs.

However, obese individuals might still be at risk because their chronic state of inflammation and higher level of ACE-2 receptors make their bodies more prone to severe COVID.

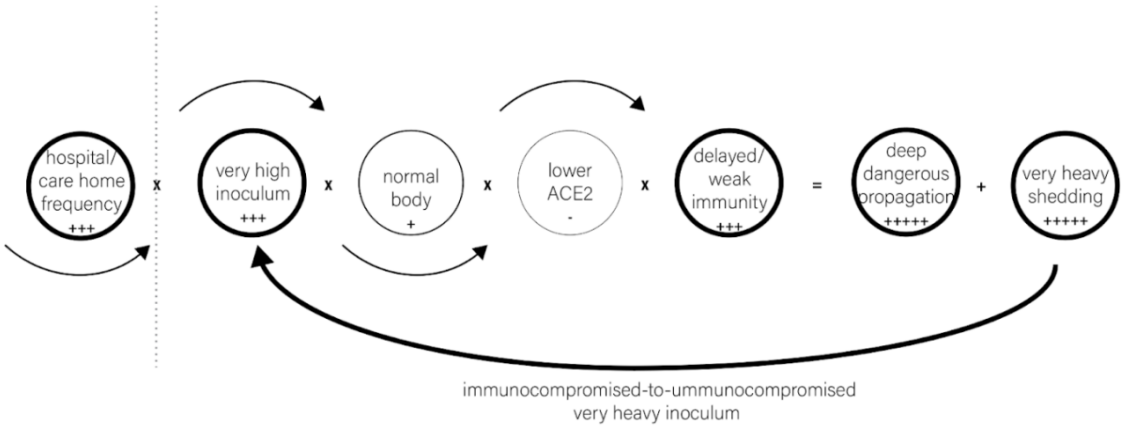
This type of life-long immunity becomes ineffective when the immune system becomes senescent or compromised. This is also the Achilles heel, of current vaccines which will be dependent on the very same delayed immune arsenal.

Compromised immune systems

If you remember the "water leakage" analogy, immunocompromised are those that are away when the *leak* occurs (aka *Infection*), come back late (aka *delayed/weak response*) and have a very bad situation on their hands (aka *severe* COVID).

Very old age – as well as certain medical conditions (diabetes…) – can impact the normal unfolding of immune reactions: the response can be significantly delayed, which can have catastrophic consequences when combined with a large initial dose of virions capable of multiplying exponentially for a long period of time [Scheme 4] .

Scheme 4 - Immunocompromised Viral Dynamics



source: Marc Girardot

These people's immune systems have necessarily met a SARS-CoV-2 cousin and/or another during their long lives. But this is no longer enough to protect them. The severe COVID patients have dysfunctional immune systems. And **this is at the core of the COVID-19 crisis**.

Resident angels

In the water leak analogy, I referred to an "automated detection system" stopping the leak early on. Evolution has integrated that "automated detection" into our immune system with mucosal resident memory immune cells strategically located at the last entry points of the virus: mouth, nose, lungs and digestive system.

Lasting resident memory T and B-cells provide protection – notably with IgA antibodies – against never-ending self-replicating epidemic cycles. Without them, people would get reinfected continuously. Evolution would have weeded out these – now extinct – communities that did not benefit from that protection, either because they died of constantly being weak, or were eliminated by healthier tribes.

The most effective, the safest and the most resource optimised way to stop an invasion is to counter-attack at the most vulnerable point of the enemy's progression path, and with a rapid and robust immune force capable of instantly destroying the virions. The resident memory immune arsenal are our Resident Angels, specially protecting the densest communities, that would be unlivable without it.

Considering SARS-CoV-2 was novel, epidemiologists have disregarded entirely one of the most important factors in this pandemic: pre-existing immunity. Nonetheless, **pre-existing immunity materialised in extremely high levels of asymptomatics**: 9 in 10 infected in Hyderabad, 20 out of 21 on the Charles-de-Gaulle aircraft carrier…

> The Super Immunity impact on an epidemic can be negligible or far-reaching. It is likely dependent on population density that drives infection rates within a population and the corresponding accrued rate of Super Immunity.

In Norway or Finland, where the lived density is 219 and 234 inhabitant per square kilometer, it is quite easy to imagine the Super Immunity protection to be very low, possibly below 5%, but it's okay since it also means the typical viral incidence impact is very low. On the opposite side of the spectrum, residents in cities such as Manhattan, Paris or Hyberadad, amongst the densest cities in the world – respectively with 25,846 , 20,755 and 18,480 inhabitants per square kilometer – would be essentially protected: 6 out of 7 were asymptomatically infected – or re-infected – so as to be protected the following season.

Only the immunocompromised left within the remaining 15% of the population not under the Super Immunity shield would face a significant probability of death. They represent up to 0.3% of these cities' populations. Elderly living separately might stand a chance, but those in care homes and hospitals would unfortunately be the main victims, as shown by the data .

In other words, infection penetration driven each year by population density acts as a protection for the next 2 years or so. Were it not for Super Immunity, very dense cities would likely be unbearable from a health standpoint with the combined negative effect of numerous sick people, of very high average shedding and of longer infections. One can view this mucosal immunity as a foundational tenet of an urbanised civilisation.

The China conundrum

From the start, many have accused China of lying about their numbers. But one needs to put into perspective the driving forces of the COVID-19 pandemic, immunity, age and obesity, and acknowledge that China has a radically different context. With proportionally 1.9 times less elderly and 5.9 times less obesity, China can be 10 times less susceptible. Add on the benefits of Super Immunity, in a country nearly 7 times denser than the United States where traditional families still exist. It is probable that China had a fatality rate 20 times lower than the US, likely bringing it below the 0.01% fatality rate.

The Inoculated Dose is the Poison

One striking reality of COVID-19 – gone unnoticed – is that overwhelmingly patients die without any active SARS-CoV-2 virion in their bodies. In other words, a healthy individual can deal with everyday doses of SARS-CoV-2 quite mundanely, often without realising he/she was ever infected. Even the "at risk" population fundamentally deals with this virus well: when infected and untreated, 96% never need to be hospitalised according to a clinical trial by Regeneron.

So what do people die of?

Severe Covid patients die of the spread of the initial dose and/or their lack of capacity to contain the expansion and the spread. The higher the number of contaminated cells, the higher the cellular damage, the higher the inflammation and the clotting, and the more critical organs are affected. Just like uncontrolled cancerous cells, infected cells become too numerous for the patient to survive.

> The immune system can scale rapidly, nevertheless it has defined limits in its capacity to process, as such the initial dose – or doses – is necessarily a

key driver of severity. The more diluted the dose the safer.

Once it is recognised that the virus is airborne, health authorities should act decisively by focusing on the activities and zones where viral production is the most active so as to reduce viral concentrations in the air. This doesn't require a PhD to understand: if many weak people are grouped together and share high doses with each other, they end up poisoning one another, repeatedly – just like the Aztecs did . **Care homes turn into virus production factories releasing massive viral quantities in the ambient air.**

Mitigating the Viral Network Effect in Community Care

Like firemen attacking a fire at its hottest point, the focus should always have been on care homes and hospitals. These community care institutions are formidable instances of the power of network effects applied to virus contamination and its devastating consequences.

Care homes accounted directly for 41% of the victims of this pandemic, possibly more. Community care facilities – be it retirement homes or hospitals – likely produce the largest viral quantities within a geographic area, therefore producing the most lethal doses. Simple mathematics: the weakest people given the highest viral load repeatedly, sick the longest, all concentrated in one place, will inevitably shed the greatest amount of virus.

Breaking the toxic network effect is imperative, possibly by temporarily scaling down the care facilities by redistributing geographically the residents: some going back to their families, the most independent ones relocated into hotels. Keeping the virus out of care homes and hospitals.

Defusing the Real Viral Bombs

Given the correlation between dose and mortality, a study logically demonstrated that the closer from the source of the epidemic, the more lethal the virus was. Similar to a bomb blast. In other words, if the virus is airborne and dose correlates with lethality, the virus "production centres" should absolutely be curtailed, the most strategic way to limit casualties is to limit virus production at the source, defusing the viral bomb.

One can easily relate to the importance of disarming the central power of virus production in hospitals and care homes :

- **Breaking up the mortal network effect** – Care home and hospital sanitation technologies and processes should be upgraded to make sure contaminated air is not shared within the care community, nor with the outside world. The air would need to be constantly filtered and sanitised – like in airplanes – so that cross-contamination is absolutely avoided. Using remote patient monitoring technologies, such as the one trialed by the NHS in 2020, can possibly limit the fatality rate by spatially distributing patients at home, or within their families, and by avoiding snowball effects.
- **Let fever play its part** – Contrary to what many think, fever is another fantastic legacy of evolution. Early on, at a critical moment when the immune arsenal is unprepared, when T-cells and antibodies are nowhere to be seen, raising the body temperature likely damages virions massively hindering virus propagation and avoiding damage done to healthy cells. Any virion then destroyed is thousands of healthy cells saved and less accrued inflammation later. Studies have shown the epidemiological impact of fever can be critical on the evolution of an epidemic. Early generalised usage of paracetamol probably contributed to much longer infections and consequently a much wider epidemic. In that perspective, paracetamol or other fever-reducing drugs should absolutely not be generalised as it is in certain countries, and should be limited to personal situations where the cost of fever becomes superior to its benefits, at a later stage.
- **Harnessing Super-Immunity –** Unfortunately, current intramuscular vaccines likely don't trigger resident memory T and B cells in the point of entry of the virus. Only resident immune cells can react early independently of the delayed immune response. In 2016, a study had already demonstrated that for other coronaviruses. Aerosolised vaccines producing mucosal resident immune cells likely better mimic a natural infection and trigger "Super Immunity" to provide better protection, notably for the elderly. This type of vaccine should be accelerated as one of the safest and most effective measures in the near future, more so than more invasive vaccines.

- **Prophylactic treatments** – While waiting for novel and safer vaccine technologies to be validated, another way to limit production of virions in the community is through proven prophylactic treatment that limit the access of cells to be infected, such as Ivermectin.

To conclude:

- **SARS-CoOV-2 was never a novel virus**.
- There is no doubt that SARS-CoV-2 is a dangerous virus.
- The threat of SARS-CoV-2 was grossly exaggerated.
- The vast majority of people were partially, or totally immune to it.
- In the densest regions and cities, where epidemics are regular and widespread, many seem to have benefitted from a "super immunity" granted by tissue resident immune cells.
- Healthy children are not at risk from the virus, nor a threat to adults.
- The main potential victims remain people with a dysfunctional immune system
- Mucosal vaccines that trigger "super immunity" are likely one way forward: more effective and safer than intramuscular vaccines.
- Fighting high viral production in care homes and hospitals should have been – and should be – the cornerstone of the fight against COVID-19 instead of useless vaccine passports and ineffective lockdowns.

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