Is the new Wuhan Chinese coronavirus an avatar of a genetically engineered coronavirus to produce a curative AIDS vaccine?
2. SARS-CoV-2 and COVID-19: What technology is available to built a HIV biomimetic coronavirus?

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Abstract *

Usually, common cold is not a danger, but a new strain of coronavirus is killing man species. Why did this new coronavirus, named at first 2019-nCoV and then re-named SARS-CoV-2, emerge without anybody being able to identify neither the reservoir nor the vector? Why did it trigger a multi-faced pandemic disease COVID-19? Why does it look like other human infecting viruses? Is it the result of an accidental genome similarity, an evolutionary convergence or a technological artefact? What is the origin of the virus? This virus could not have emerged spontaneously by natural mutations and wild strains recombinations, it is a genetic chimaera with artificial insertions of modified genes, an engineered genome of a coronavirus within a capsid of a Human Immunodeficiency Virus (HIV). Why the released coronavirus couldn't be a HIV candidate preventive or curative vaccine of AIDS?

What technology is needed to built a HIV biomimetic coronavirus? Don't we get it?

What could we learn from the study of the scientific and social story of the virus and disease?

Keywords: 2019-nCoV, AIDS, coronavirus, COVID-19, curative vaccine, evolution, genetic chimaera, genome engineering, host cell, target cell, Human Immunodeficiency Virus HIV, pandemic disease, SARS-CoV-2

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Introduction

Usually, coronaviruses are responsible in humans for a minor infection and a common mild disease, a cold. In 2003, a coronavirus from a bat (its wild animal reservoir), SARS-CoV-1, was transmitted to humans (its definitive host) via a civet (a wild animal vector). The viral infection triggered a Severe Acute Respiratory Syndrome or SARS. In 2012, another coronavirus, also of wild origin and also from a bat, was transmitted to humans by another animal vector, a domestic camelid. The viral infection triggered another severe acute respiratory syndrome or MERS, as lethal as SARS. Other coronaviruses are highly studied because they infect domestic animals such as bovids. In December 2019, in Wuhan, Mainland China, a new strain of coronavirus, - named 2019-nCoV at first and then renamed SARS-CoV-2 -, emerged without anybody was able to identify, with certainty, neither the reservoir nor the vector. It triggered a pandemic human disease COVID-19. **Everything is as if the virus was totally humanized right from the beginning of the outbreak**. In the middle of May 2020, more than 3 million victims and more than 250,000 deaths were officially identified in 193 countries or territories. Depending on the affected person, this new human disease is both a-symptomatic and may go unnoticed, or multi-symptomatic and very severe, or even lethal (Baud et al., 2020).

What is the origin of this virus? Why does the disease express a multi-factor changing face?

1. From 2019-nCoV to SARS-CoV-2: the story of the emergence of an uncanny virus.

In February 2020, *a French mathematician, also an inter-disciplinary expert, emeritus engineer in computer science and researcher at the IBM Center for Research in Artificial Intelligence, at the University of Bordeaux*, identifies into the genome of the isolated **2019-nCoV** strain, **patterns that break** the symmetry of the usual genetic organization of a coronavirus (Perez, 2020). New information patterns were inserted into the gene encoding **the envelope spike protein** (protein S) and into the genes encoding the poly-protein which is at the origin of both **the viral protease and polymerase**, enzyme activities that are present in every coronavirus. These insertions were identical to the same information patterns found into the genome sequences of the similar proteins Open Reading Frames (ORF) of another well known RNA virus, the HIV.

Is this a random similarity or an evolutionary curiosity or is the virus a synthetic, artificial one?

Using a software tool to study statistical correspondences, the genome of this new coronavirus was compared with those of previously isolated coronaviruses known since 2003. **Paradoxically**, the result highlighted a phylogeny in which the new virus 2019-nCoV (the most recent one) was older, **in terms of genetic distance**, than less recent ones, SARS 2015 and SARS 2017 that appeared before. Everything seems to point out as if it was less evolved than the oldest known SARS coronaviruses (SARS 2003 and SARS 2004). Just as if, in a family tree, the grandfather was to be born after his grandson!

1.a. What was the origin of 2019-nCoV?

The genetic sequencing of this 2019-nCoV virus indicated that its genome looked like that of another coronavirus that was issued from a bat and isolated in 2013. What would then be the intermediate host vector between bat and man? The 2019-nCoV genome appeared to be located out of all the clades of humanized coronaviruses, whose reservoirs and vectors were known. Everything happens as if the virus had passed directly from bat to man, without an intermediate vector, or even **directly from man to man** (Bricage, March 2020). But how then was this virus, supposedly of wild origin, humanized? Other authors considering that the initial reservoir could only be a bat species (Hu et al., 2015) discussed about various vectors, first a species of reptile (Zhang et al., 2020), then finally a pangolin species. Yet, their new phylogenies showed that the genome of 2019-nCoV was **as close or as far, in terms of genetic distance**, to the known genomes of bat coronaviruses as it was to those of domestic animals. How could the viral genome then be simultaneously very different from the genomes of its previous ancestral viruses, that were at the origin of the SARS and MERS, and coming from the same reservoir of origin, but without a known vector? Why was there such a mess?

With the registration, in 2020, of the viral strain RaGT13, that was isolated in 2013 (as its name suggests), other phylogenies were then proposed (Bedford & Hodcroft, 2020). They replaced the SARS-CoV-2 in an evolutionary position more fitting in with the past known ones. **Why was the alleged ancestral strain, isolated in 2013, and on which research has been carried out, not registered in the genetic data banks before 2020** and only after the 2019-nCoV registration, which strain was then renamed as **SARS-CoV-2**?

1.b. A HIV biomimetic coronavirus?

Meanwhile, *a team of young computer scientists, from the Indian Institute of Technology in New Delhi*, had uploaded a work, in bioRXiv, the open online archive, showing an unknown and totally uncanny similarity between the genomes of 2109-nCoV and HIV (Pradhan et al., 2020). The 2019-nCoV genome contained patterns similar to some found in the HIV genome, in its envelope protein S and in its poly protease-polymerase genes pool; thus validating the previously published work of Perez (2020).

How could identical genetic sequences be present in such different viruses?

Was this an adaptive evolutionary convergence phenomenon? (Mills, 2006)

How, in nature, could a set of similar mutations give the same functional result in genetic systems evolving within completely different hosts, (bats and humanoid primates)? Or, if it was a genetic transfer, how, in nature, were such targeted genetic recombinations possible (Bricage, March, April & May 2020)?

In what host could the simultaneous presence and the invasion of the same host cells, by both viruses, have arisen at once? Afterwards, this unpublished article was withdrawn; cancelled without any specific comment. Simultaneously, the previously published work was *framed as a fake news*, while the 2019-nCoV became the SARS-CoV-2. Why so many troubles?

What did the work of the Indian team show?

If we compare the proteins S of the initial 2019-nCoV strain with those of the other coronaviruses, the amino acid islands observed in the homologue proteins sequences turn out to be thoroughly different. Could they result from point mutations in the corresponding nucleotide coding sequences? As a whole, these inserts, preserved in all the first analyzed genomes, do represent less than 1% of every genome. This is much less than the rightfully expected overall genetic divergence with every coronaviruses, whose sequences are known. There is an astonishing similarity between the sites of cutting by the proteases: the same sites are present in these proteins of both the coronavirus and the HIV (and also some influenza viruses). These sites are different from those of usual coronaviruses; either in wild reservoirs or vectors, wild or domestic vectors, or even in every known humanized coronaviruses. How could a natural change of such magnitude, by viable and specific mutations, have affected only the cut-off sites, which represent less than 3% of the protein sequence? One can only envisage a series of targeted and repeated mutations, whose presence simultaneously created a new structure of the virus binding protein at its receptor site. Isn't it highly unlikely that a wild virus could acquired, in so few years, such unique insertions similar to those present in the HIV-1 gp120 and Gag proteins?

None of these 4 inserts may result from a random single-point mutation. This type of mutation usually affects only 1 amino acid and not a defined protein pattern (**a series of specifically changed amino acids**). A point mutation can also cause a shift in the reading frame, but this usually results in a non-functional protein. Yet 2 of these inserts present a deletion (Su et al., 2020), an interruption, a partial break into their sequence, which not only retains the functionality but also specifically increases the density of the positive charges located at the surface of the active molecule. A deletion is usually lethal and results in a non-functional protein. So how did these new molecules with new properties emerge? And **by what natural process?**

1.c. A story of many troubles and mess...

A North American University research team then confirmed the existence of these extra-coronaviral sequences (Beal et al., 2020). Into the surface glycoprotein they determine the kind of cell receptors that are potentially recognizable by the new virus, for its entry into a target cell. Inserted into the ORF1ab poly-protein, which codes for intra-cellular viral functional enzymes, that can be activated only by cuttings either by viral or cellular (Millet & Whittaker, 2015) proteases, they determine the viral protease properties (Anderson et al., 1993).

RNA viruses, such as coronaviruses, usually have a very high mutation rate: 10 times that of single-strand DNA viruses, 10,000 times that of other viruses and 100,000 times that of their eukaryotic host cells. This paramount rate implies a random accumulation of errors in the genome, which are usually unfavorable for the virus survival. The existence of hot spots, where mutations are the most frequent, is well-known into every genome. Reconstructing the history of their emergence has allowed, in HIV clades, to trace the lineage of genes and to evaluate the evolutionary potential of a gene. Let's consider the HIV proteins that may have been used as a model to naturally reconfigure those of SARS-CoV-2, in order to have a biomimetic coronavirus of HIV. For HIV, the envelope protein (env) is evolutionarily much more stable than the pol and gag proteins. How can we then explain that in the new coronavirus the exact opposite is observed in the evolution trend that would have naturally led to the homologous protein to env compared to those that would have led to the modified proteins homologous to pol and Gag? Are these changes random or intentional?

Adapted from Bricage, May 2020, figure1

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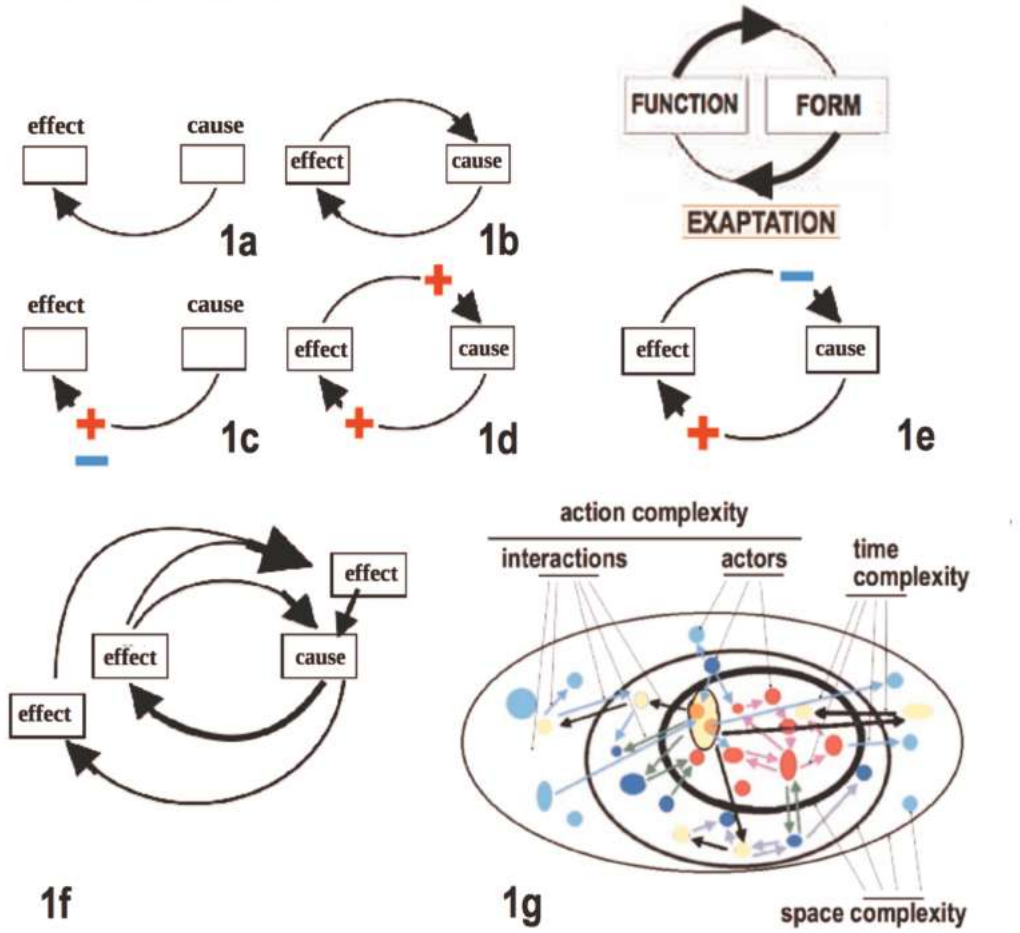
2. Why the SARS-CoV-2 couldn't be a virus of wild origin? Ecological, physiological and genetical aspects.

Figure 1. Living systems *'space-time-action'*: actions, interactions, feedbacks.



-1a- action: from cause to effect. When you can trace an effect back to its cause, *"by removing the cause you remove the effect"*. This is the principle of the action of a drug., -1b- retro-action or feedback: in living systems *the effect can itself be a cause that acts back on its initial cause* (feed back). We no longer know where the cause is, or where the effect is: *"we can delete a local effect without deleting its initial global cause"*. The physiological reciprocal interactions between a form (FORM) and its associate function (FUNCTION) can lead to *the* ***co-construction*** *of new adaptive capabilities* (EXAPTATION) that determine the evolution of a molecular or cellular system: systemic constructal law., **-1c-** the cause may have an action of **stimulation + or inhibition –**., **-1d- cascade amplification**: reciprocal stimulatory, agonist inter-actions, lead to *a runaway phenomenon*., **-1e- reciprocal regulation**: only long-term, ago-antagonistic actions allow the establishment of a stable steady state., **-1f- contingent network of inter-actions**: *"into a set of indissociable effects, we no longer know where the initial cause is"*. Any effect can be a cause, and acts as a new cause of stimulation or inhibition, depending on the context., **-1g-** How to measure the complexity of a system?: evaluation of spatial and temporal modularities. Any system of inter-active actors might be represented schematically by *balls (actors)* and *colored arrows (actions)*. The colors (qualitative aspect) indicate a **type** of actor or action (reciprocal or not) and the sizes indicate the **intensity** (quantitative aspect). The action complexity is measurable by the qualitative aspects (diversity of actors and interactions) or/and quantitative aspects (number and intensity of the inter-actions and number of actors and the parts in every action taken by every actor). Just as there is a **spatial compartmentation**, there is a **temporal compartmentation:** the temporal complexity is measurable by the latency time, the time point and the lifetime of each action or each inter-actions network, the spatial complexity is measurable by the distribution of actors, taking into account the boundaries of the subsystems that make up the system, and the lifespans of actors and subsystems. *Complexity is complex!*

Subsequently a team of Chinese researchers (Zhang et al., 2020) disqualified the role of these insertions through 3-D modeling of the interaction between the S protein and its target receptor ACE2 (Ge et al., 2013). The inserts are located outside the recognition site that binds to the receptor, so they would not have a functional effect. Isn't writing this ignoring the fact that the functional properties of a protein emerge from the remote interactions between its different peptide modules, the different parts that make up the protein, the whole?

2. Don't we get a technology to built a HIV biomimetic coronavirus?

The genome of any coronavirus encodes for a first poly-protein that contains the viral proteases, though inactivated since linked together within a pre-protein complex (ORF1a). Other segments of the genome code for 2 other poly-proteins that respectively contain the enzyme activities for the viral genome manipulation (ORF1b) and the structural envelope protein.

2.a. How does a coronavirus work, compared to another ribovirus like HIV?

Protein cutting of these poly-proteins is critical (Bricage, April 2020).

It can first be achieved by a pre-existing cellular protease if any. Once the viral genome is expressed in its functional proteins, the proteases of viral origin, the Papaïne-Like-Protease (PLP) and Chymotrypsine-Like-Protease (CLP), can work. The genome structure and reading sites of a coronavirus are the same as those of a retrovirus such as HIV. The functioning is the same if not for the fact that there is no need for a retro-transcription activity in the development cycle of a coronavirus, and that the recognized and infected cell types are supposed to be different…,

with the exception of a coronavirus biomimetic of HIV?

In addition, depending the protease that cuts and the site where it cuts, the cutting of the poly-proteins can give birth to a family of homologous proteins: the viral proteases and the envelope protein synthesized by a same cell are not unique but multiple in terms of functional structure (figure 1).

Haven't high-resolution 3-D models shown that in SARS-CoV-2 **the S-protein resembles both those of a coronavirus and HIV**? The topology of the HIV protein is very specific and this specificity is at the origin of the inhibitor structures (conceived or not with the help of an artificial intelligence) that prevent its activity (like mozenavir). Isn't **the topology of the coronavirus protease strangely similar to that of HIV**? The same type of inhibitor can be effectively used because it will locate at both the coronavirus and HIV sites.

2.b. Isn't it possible to make a coronavirus biomimetic of HIV?

Isn't it technologically possible to modify an ancestor of SARS-CoV-2 to produce a biomimetic coronavirus of HIV so as to develop a potential AIDS vaccine (Du et al., 2009)? The 2003 SARS-CoV-1 and the 2013 MERS were the subject of extensive research. The genetic and molecular engineering of coronaviruses has been the subject of numerous books (Canavagh, 2008) and publications with detailed laboratory protocols (Becker et al., 2008). Many works about the construction of genetic chimeras have been published and new patents registered (Baric et al., 2018). For over 20 years, North American researchers, but not only (Amer et al., 2012), have been working on coronaviruses in domestic animals, such as cattle or pets. Under the leadership of Professor Dr Frank Plummer, the National Microbiology Laboratory of Canada (NMLC) acquired a sample of the MERS virus from the Erasmus Medical Center in Rotterdam, in 2013.

Is there one laboratory able of such **an intellectual and technological feat**? The NMLC, in Winnipeg, was led by Dr. Frank Plummer, a member of the National Agency of Public Health of Canada (NAPHC), of which the NMCL is a department. As **a world-renowned expert on AIDS**, with over 300 publications and over 16,000 citations, Professor Plummer also worked at the University of Sheffield and the University of Manitoba, where he headed the **Department of Medical Microbiology and Infectious Diseases**. Plummer was the first to highlight (Fowke et al., 1996) the existence of individuals resistant to HIV infection, among a population of African women sex workers, in Kenya, Nairobi. Since 2004, with teams of Chinese researchers hosted within

the LNMC he has been working on both AIDS (Luo et al., 2011) and SARS (Berry et al., 2004) viruses. He was highly interested on the structural proteins of these viruses; especially the envelope proteins (Ao et al., 2019). One of the leaders of the research teams of the NMLC, also the director of **the antiviral vaccine therapy program**, Ms Xiangguo Qiu, is **an exceptional researcher**. Her works have been referred with more than 4,000 citations. The teams she worked with have developed skills in vaccines development for the Lassa fever virus, the Ebola virus and the Marburg virus. To do so they used one of the known vectors of bat coronaviruses as animal model, the ferret (Wong et al., 2018), the vector of the previous SARS-CoV-1.

Together with other teams of Chinese researchers, she has carried out works on the production of Ebola virus mimetic HIV clones (Ao et al., 2019). She has published results related to the structural proteins of these viruses and their genetic modifications in relation to the phenomena of virus entry and replication in its host cells. These teams have demonstrated skills in using artificial intelligence software tools (Capuzzi et al., 2018). Under the leadership of Frank Plummer and the guidance (or participation) of Xiangguo Qiu, other teams of Chinese researchers have **built biomimetic HIV viruses** (Tang et al., 2012) **expressing modified proteases** (Luo et al., 2012) or modified surface proteins. Isn't it the case for the ones of 2019n-CoV (Coutard et al., 2020)? What more was needed to build a candidate HIV vaccine? Only a humanized supposed harmless coronavirus was! Could we not protect ourselves from AIDS while only risking a simple cold? What could be more dull and benign than a cold? Everything was available in 2012, before the MERS strain acquisition.

2.c. Why couldn't the new coronavirus be a HIV candidate vaccine?

Couldn't the manufacture of a biomimetic coronavirus of HIV then be considered? This **exceptional intellectual and technological feat** was unfortunately not completed, as the vaccine candidate had not been finalized. How did it escape too soon?

In early July 2019, an administrative investigation led to the sudden expulsion, from the P4 Laboratory in Winnipeg, of Xiangguo Qiu, her husband (also a biologist), and all her research students. They were all escorted out of the laboratory. The University of Manitoba ended all their accreditations as well as all their activities, and reassigned all the students to other structures, while formally prohibiting all students and faculty staff from traveling to China. Why? Why did the NAPHC launch an administrative investigation in May 2019, the subject of which was never specified? Weren't the facts reported by Canadian and even Chinese daily newspapers and seen on Canadian television before July 2019?

Why were these reports taken over? Where and by whom were they distorted and reclassified as fake news after the outbreak was declared in Wuhan? Is deliberately adding the fake news label enough to discredit a truth? Isn't it easy to take a real information, to modify it, to give it the appearance of reality, to then better disqualify it, so as to thus be able to decline any information referring to it?

3. Isn't a whole always both more and less than the sum of its parts?

Wouldn't the acceptance of the fact that the SARS-CoV-2 is related to a vaccine candidate, initially designed to fight HIV, would allow to explain the particularities of the COVID-19 disease? How could the fact that the disease, as **an expression of the physiological interactions between the viral genome and the one of the infected cells** in the body, seems to change its face continuously, depending on the infected patient, be explained, if not by accepting that the SARS-CoV-2 is a biomimetic virus of HIV, expressing variable properties depending on how it interacts with its host cells (figure 2)?

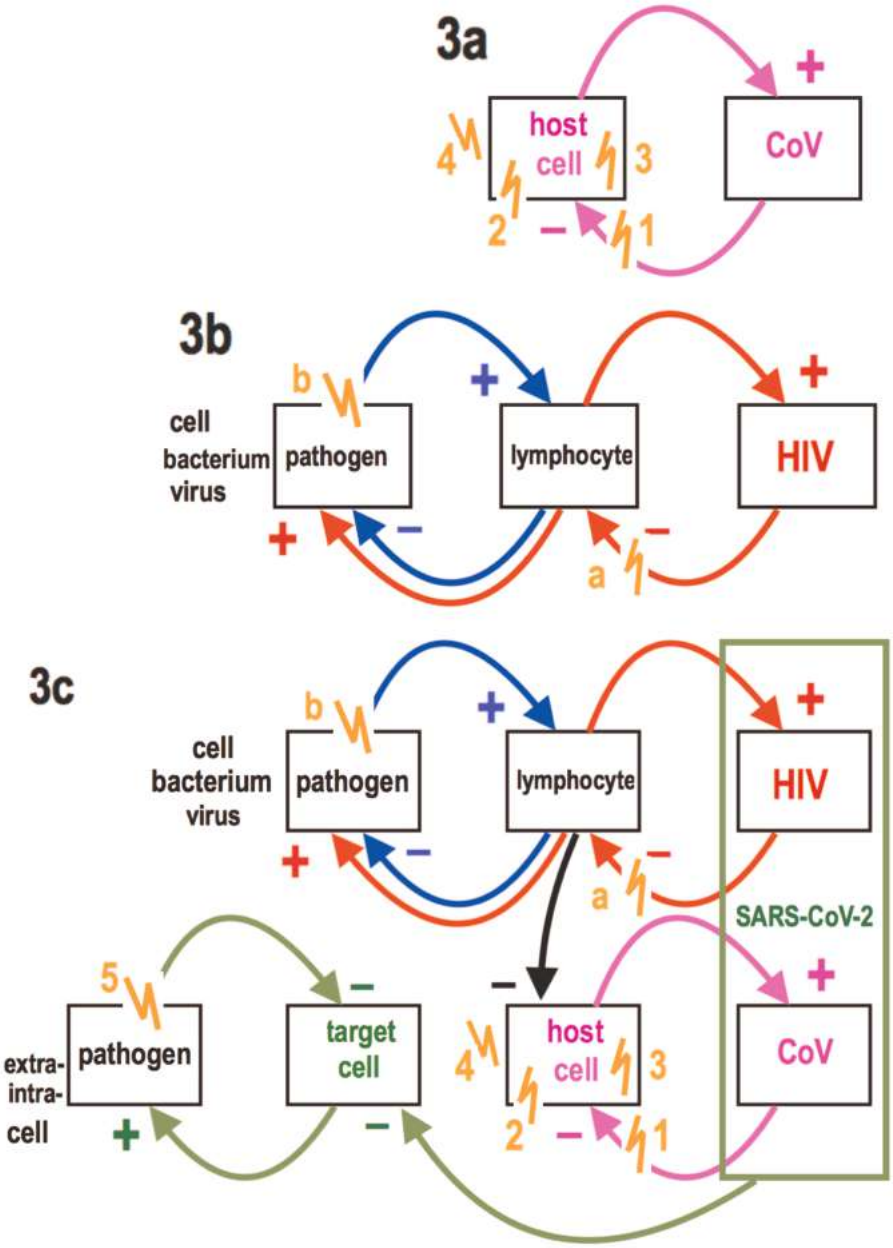
It can either go unnoticed as a common cold coronavirus, or it can trigger symptoms usually associated with AIDS such as lymphopenia and express HIV properties such as neurotropism. Could it not also have **emergent properties associated with new target cells and new symptoms?** Doesn't the fact that it is potentially more or less an HIV, more or less a coronavirus, and more or less something else… account for the biodiversity of the symptoms and thus the diversity of the treatments involved, some of which have nothing to do with anti-viral medication? Is this banal scenario of dependent interactions between the parts of a system anything impossible (Bricage, May 2020)?

Adapted from Bricage, May 2020, figure 3

Is the new Wuhan coronavirus an avatar of a genetically engineered coronavirus to produce a curative AIDS vaccine?

2. Why the SARS-CoV-2 couldn't be a virus of wild origin? Ecological, physiological and genetical aspects.

Figure 2. Why couldn't the SARS-CoV-2 be an avatar of a HIV biomimetic coronavirus?



-3a- What are the different strategies for curing COVID-19 and struggle against SARS-CoV-2?

A coronavirus, CoV, as every virus, survives within a target cell, a host cell, which provides it with the necessary and sufficient functional capabilities for the viral reproduction. The host is the survival **ecoexotope*** of the **endophysiotope*** of the virus. But the virus can also survive in a dormant state of life outside the cells (cell level = i) of an organism (organism level = i+1), and outside the organism. The ecoexotope of survival of the virus is both the endophysiotope of the cells, the endophysiotope of the organism and the ecoexotope of survival of the organism. As every predator it survives only as long as it has live preys to eat! **For the one to survive** (the predator) **the other one** (the prey) **must survive first!** >>>>

Adapted from Bricage, May 2020, figure 3
Is the new Wuhan coronavirus an avatar of a genetically engineered coronavirus to produce a curative AIDS vaccine?
2. **Why the SARS-CoV-2 couldn't be a virus of wild origin? Ecological, physiological and genetical aspects.**

Figure 2. Why couldn't the SARS-CoV-2 be an avatar of a HIV biomimetic coronavirus?

>>>> * ecoexotope: exo = **external**, tope **space-time** of eco = **inhabitation**, endo-physio-tope: endo = **internal**, tope = **space-time** of physio = **of functioning** (Bricage, May 2020, figure 2) (new words and concepts for a new paradigm http://**armsada**.eu)

As for every parasite, the development cycle completion of the virus is aleatory and all the more risky for its fate when its hosts are going rarer or too dispersed. This is why, as every parasite, it produces an extremely large progeny. How to prevent the viral cycle completion (purple arrows) which results in the multiplication of the virus **+** and the destruction of cells **-** ?

Once the virus has entered the body, what strategies are available to protect cells?

- [**strategy 1**] - trapping of the free virus: naturally by antibodies of the organism, with virus destruction by lymphocytes of the immune system, or, when failing in that way, the artificial trapping by lures mimicking the natural receptors of the virus, if they are (all) known..., *otherwise* - [**strategy 2**] locking the entry of the virus into the host cell: either by blocking access to the input receptor(s) or by blocking the internalization of the virus-receptor complex if it is impossible to lock the receptors, *otherwise* - [**strategy 3**] early blocking of the expression of the functions of the virus: either the alteration of the synthesis of its proteins, or the alteration of the synthesis of the copies of its genome, *otherwise* - [**strategy 4**] lysis of the host cell before the complete cycle of production of functional virions; in any case the release of the virus population destroys the cell… !

-3b- The characteristics of an HIV infection. The Human Immunodeficiency Virus **HIV** targets cells of the immune system whose destruction **–** results (red arrows) in both the multiplication of the virus **+** and the multiplication **+** of usually recognized **pathogens** (viruses, bacteria, cancer cells) that are not destroyed **-** by killer lymphocytes or/and macrophages (blue arrows).
To stop **a sooner or later fatal immunodeficiency**, 2 strategies are simultaneously needed: - [**strategy a**] as for **CoV** (strategy a = strategies 1, 2, 3, 4), to stop HIV and AIDS, usually by **a multi-therapy**, and - [**strategy b**] (strategy b = strategy 5) to stop opportunistic pathogens, by antibiotics, anti-viral or cell "toxic" drugs, **depending on the type of invaders**.

-3c- **The peculiarities of an invasion by a coronavirus biomimetic of HIV.** Doesn't **SARS-CoV-2-** get both the functional properties of **CoV** and **HIV** and **emerging properties**? Aren't its target cells both the usual host cells of a **SARS** type coronavirus, whose invasion is accentuated (black arrow) by a partial (?) depression of immunity, some of the target cells of a (more or less attenuated?) HIV, and new unusual cells for a coronavirus (**emergent property**) which invasion may lead to additional secondary infection by either extra- or intra-cellular pathogens, and the invasion of which must be contained by drugs appropriate to their types [**strategy 5**].

Based on the known symptoms and available medications, what facts are validated by the really used treatments #?
- [**strategy 1**] **soluble receptors** (soluble ACE2, soluble CD147), **plasma neutralizing antibodies** of cured patients,
- [**strategy 2**] **inhibitors of receptors binding**, anti-ACE2, anti-CD147 (meperizumab), **protease inhibitors** (lindinavir, saquinavir, lopinavir and ritonavir), other **inhibitors of entry** (camostat mesylate), chloroquine **?
- [**strategy 3**] **viral protease inhibitors** (lindinavir, saquinavir, lopinavir and ritonavir), polymerase inhibitors (sofosbuvir, favipiravir), **nucleoside analogues** (remdesivir, ribavirin), **fever mitigation*?** (non-steroidal antipyretics), hydroxy-chloroquine,
- [**strategy 4**] hydroxy-chloroquine, azithromycin, and other **cell toxic drugs**, **autoimmune antibodies**,
- [**strategy 5**] **fever***, **anti-influenza** (oseltamivir, umifenovir) and other **anti-viral drugs**, chloroquine and derivatives**, **anti-bacterial antibiotics**** (amino-glycosides, chloramphenicol, erythromycin and azithromycin, ceftiaxone), anti-diarrheic drugs (nifuroxazide = ercefuryl), other **toxicants** **depending on the over-infecting pathogens**, colchicine,…
- [**strategy a**] **anti-HIV** (lopinavir et ritonavir), **fever** *,
- [**strategy b**] **anti-influenza** (oseltamivir, umifenovir), **anti-viral drugs,** chloroquine and derivatives, azithromycin, **antibiotics** (anti-tuberculosis), highly toxic **anti-cancer drugs** (colchicine, fluorouracil), **other toxics** depending on the infecting pathogen.
This is a non-exhaustive list of products (usual French names, data for information). The strategy for treating inflammatory**, hyper-allergic or autoimmune reactions [for example with monoclonal antibodies (tocilizumab)] is not specified here.
paradoxical strategies: * should fever, the first anti-viral defense, be reduced or not?, ** Drugs, hydroxychloroquine and/or azithromycin (an antibiotics, such as erythromycin or chloramphenicol), are not antiviral ones, they are **cell toxicants** and used to fight inflammation! They have "plural" effects. What are they really? Couldn't their effects vary from one patient to another?

Isn't **the variable expression of the of SARS-CoV-2 phenotype** the consequence of the variable expression of properties relative to both the phenotype of a weak, strong or intermediate HIV, or/and of properties relative to the phenotype of a weak, strong or intermediate SARS-CoV? Doesn't that explain **the variety of symptoms observed during COVID-19**?

Conclusions

Isn't the environmental and societal story of the virus being rewritten?

The analysis of the evolution of the humanized viral populations showed the existence of at least 2 subpopulations of SARS-CoV-2. The most frequent type, type L, both is the most recent and the most aggressive one. The oldest and least frequent type is also the least aggressive one. **This is totally the opposite of the known natural genetic evolution of wild viruses.** Or would it be possible there were tight from the beginning several types of coronavirus?

Why did a group of 27 prominent scientists insist on discrediting the artificial origin of the new coronavirus? **Are we rewriting the virus history?** Wouldn't transparency prevent this kind of scientific, political and media imbroglio? How can we prevent economics from mixing into issues and thus undermines the social and environmental responsibility (Bricage, 2011) of both research laboratories, health companies and individuals? Does mathematical economics justify the liberal management of science and health services? How to avoid resentment while fostering emulation? Some models exist: - **open access publishing** under the **"Creative Commons"** license, without paying copyrights but still mentioning all the authors, - freely contributing to information archive with both an open access and open discussion, in a structure accessible for everybody, such as **Wikipedia** for teaching and **Research Gate** for research, - encouraging original, individual or collective innovations rather than repressing them by either deterring, refusing or drowning them under constraints or the accumulation of other consensual information.

What can we learn from the emergence of this new pandemic?

A proposal not having been proved, does not mean that it is automatically false; many are those who commit this error of logic. That does not mean it is true either. But, if the source has some legitimacy, as for a simple honest testimony, it is worth testing it! A new article about disinformation appeared in the English version of Wikipedia. Why? Why does it **only deal with "conspiracies" against the consensual version** of the accidental appearance of SARS-CoV-2 from a wild animal (Bricage, 2011)? Why are the majority of the arguments in favor of the consensus track expressed as quickly as possible, in the form of letters, correspondence (Andersen et al., 2020), reports or accelerated papers (Zhou et al., 2020) and not as usual scientific publications? Why are the aspects validating this track the only accepted and published ones? Why are papers dealing with non-consensual versions systematically rejected? Is there a global attempt to hide part of the reality and re-write the history of both the origin of the virus and the pandemic?

Can we be a judge and a part of such an important investigation for humanity?

Finally we should be able to systematically check when the "fake news" label is attributed to a news that the news is not in fact a fake news but that *"the fake news labelling" is itself "a fake news"!*

Unfortunately, this is probably not the latest pandemic due to reckless human actions on the wild environment (Bricage, 2011). The problem is that they are getting closer and closer and the associated viruses are getting more and more aggressive. How to stop this escalation of the violence of interactions between the human species and its host ecosystem? Should environmental faults be used as an alibi to hide technological faults, whatever the reasons? **Primarily all aren't societal, economic or/and political faults?**

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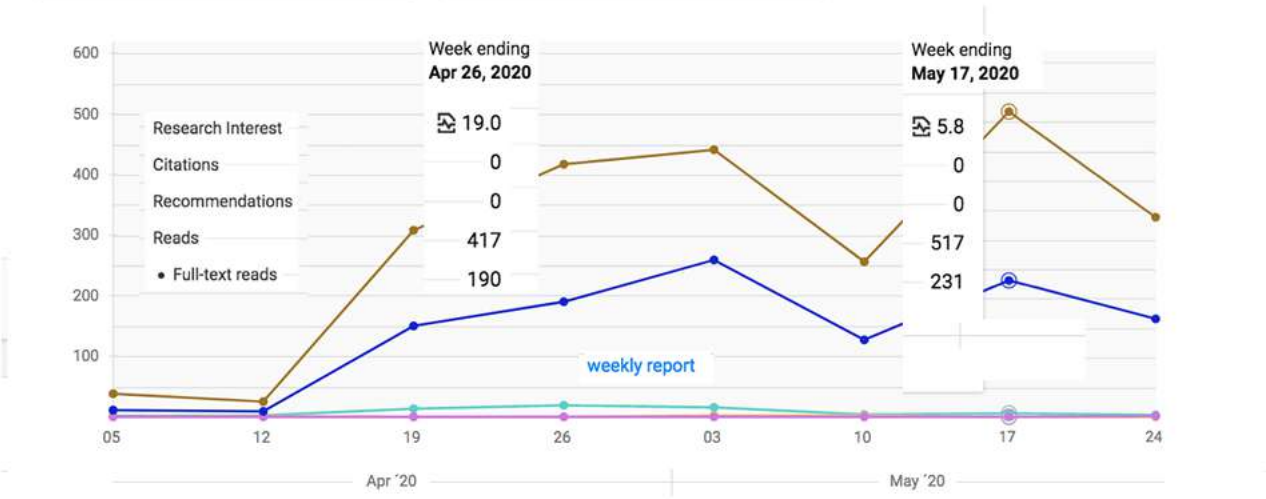

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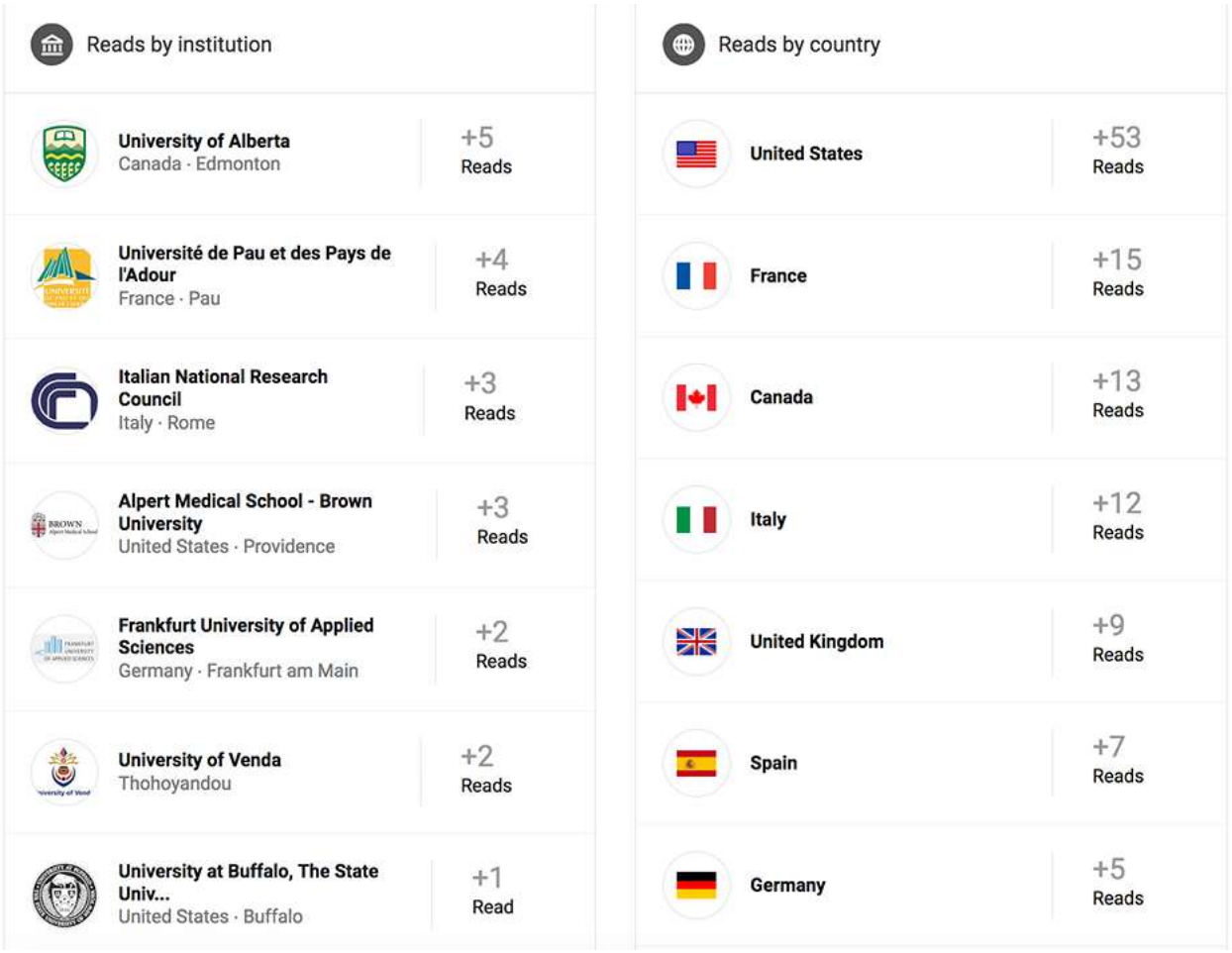

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