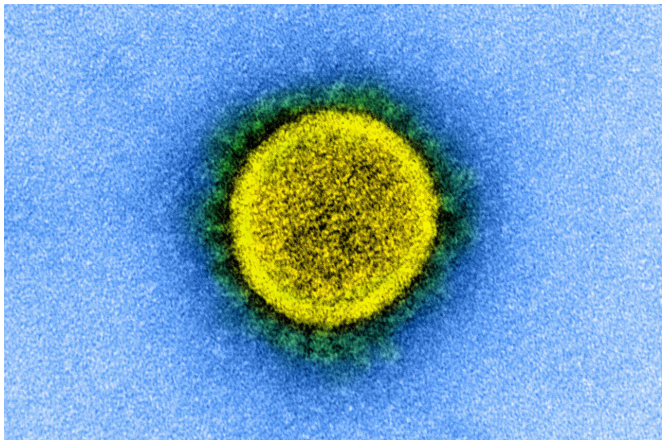
The dirty life and times of SARS-CoV-2

13 January 2021, by John Hewitt



SARS-CoV-2 (shown here in an electron microscopy image). Credit: National Institute of Allergy and Infectious Diseases, NIH

It took the coronavirus for scientists to finally question the standard ethics of the placebo controlled study. Even the top mainstream science journals, like *Nature*, have now at long last finally admitted that no one should be putting their elderly parents, children or essential co-workers in the line for a dummy injection against this virus.

Frankly, this is a stunning departure from the official medical narrative of double-blind placebo controlled trials at all cost. Science has spoken again, it would seem, and it says big pharma can keep their placebos. Instead, those willing to brave new vaccines can enroll in more fitting biomarker studies where everyone gets a shot to be saved. The observables in these studies are not simply whether you succumb or survive, but rather, which antibodies and what protections your own unique defensive systems mounted or failed to mount. One might imagine the rest of medicine is not too far behind.

But what if vaccines can't keep up with all the new variants, for example, the E484K variant first noted in South Africa, which are now thwarting attempts to control the spread? For that matter, how are these new viruses being propagated? Are they one-time founding events that then fly in from distant countries of origin, or are these multiregional mutations, arising de novo in local populations? Even more importantly, where did the first virus come from?

There may still be a lot of uncertainty regarding the origins of SARS, but there's one thing we can be confident of at this time—only weak-minded fools let another person tell them what is real information and what is misinformation. In a healthy mind, the truth about SARS, as in other complex matters, is never something that is imposed from without; it must be something we each arrive at from within via experience. A recent well-publicized paper in *Trends in Ecology and Evolution* titled "Unraveling the Zoonotic Origin and Transmission of SARS-CoV-2," by virtue of its title alone, would appear to have special access to the coronavirus truth.

The authors note that a few bat SARS-related coronaviruses, such as the RaTG13 sequence, contain up to 96.2% sequence identity with SARS-CoV-2. This virus could therefore potentially be our source, or it could have simply evolved in parallel to it. In the case of the SARS outbreak in 2002-2003, the likely zoonotic source was civets (Paguma larvata), which carried a SARS virus with 99.8% identical sequence to that of the outbreak virus. The authors go on to describe how a virus that infects pangolins, a so-called intermediate host, possesses intriguing similarities to SARS-CoV-2 in the crucial ACE2 receptor binding region of its own spike protein. Interesting, although in fairness to the pangolin, at the whole-genome level, these viruses only have 85.5–92.4% similarity to SARS-CoV-2. Therefore, they would require no small measure of natural evolution or laboratory passaging to morph into the virus we are now confronted with.

In the face of ambient uncertainty, the average but inquisitive person must either become an expert themselves, or find experts whom they trust. In other words, they should identify of those sufficient

boldness to push back against suspect official narratives when shortcomings are found. When searching for such learned individuals in the digital aether, new information comes to light regarding this particular anti-pangolin paper. Twitter user @WackyScience points out the *Cell* paper relies on a *Nature* paper for inspiration on these pangolin matters, into which an important addendum has quietly been slipped. Namely, that the pangolin samples were contaminated with human and mice material. For those who might want to dig deeper, @NoWackyScience offers a few neatly compiled motivational links:

1. Time to exonerate the pangolin
2. No virus in wild pangolins
3. Doubts have been cast
4. No PCR/meta-data
5. Broad Institute rebukes pangolins

The last entry above is co-authored by an outstanding post doc member of the Broad Institute, Alina Chan, who has done as much as anyone to elucidate the increasingly mysterious origins of the RaTG13 sequence. It seems there is more to the story than at first meets the eye. Chan is almost apologetic in arriving at her conclusions regarding the shenanigans in the Chinese promulgation of this RaTG13 sequence. She says that just because this "conspiracy theory" regarding the origin of these viral sequences could be true in her opinion, other conspiracy theories in other topics are still baseless. However, she says, saying that it is possible that a virus got out of a lab is actually not a conspiracy theory; it is simply a theory. Escape events are a real phenomenon, and they have happened so many times in recent history that it boggles the imagination to think that our bioweapons lab at home—and those we fund on more permissive foreign soil—still operate in the way they do.

While it is certainly possible, perhaps even likely, that all the unique transformations of the present coronavirus that enabled its unprecedented infectivity evolved naturally in other animals and seamlessly burst through species barriers to us (in other words, were entirely zoonotic), other logical explanations of the observed data do exist. Those other explanations, like escape from a lab that specializes in viral function transformations, are now being looked at more closely on several surprisingly mainstream venues. At this point, it is important to note that the official media narrative—in fact, the existing scientific narrative on virus origins—is that it could not possibly be an escape.

One bombshell story appeared the other day in *New York* magazine that made the hearts of many well-funded infectious disease lab heads skip a beat. The author, Nicholson Baker, is a novelist and essayist rather than an infectious disease virologist. Yet he has somehow managed to document the facts apparent in our present situation more thoroughly and succinctly than any of the experts. Some insight into why this is even possible today can be had by considering the process by which scientific knowledge is now packaged to the public at large. Today, scientists and even science writers are frequently given talking points, either directly or indirectly, about how to present scientific knowledge to the public. In other words, guidance on not just which science should be considered disinformation, but also how to properly respond to certain alleged disinformation.

For example, an esteemed team of academic scientists has now assembled something called the "COVID-19 Communication Handbook" to instruct on how to deal with vaccine conspiracists. Namely, how to respond to someone asking logical questions about which vaccines might successfully fight which viral variants. In a nutshell, the publication strongly condemns what they call "vaccine behaviors" that run counter to its own goals. Curiously, many folks who seem to exhibit these undesirable vaccine behaviours also question the official narrative of SARS origins.

Perhaps the only way to definitively prove that a bat virus in Chinese caves naturally mutated and transported itself 1000 miles to the Wuhan outbreak site, and that the world's most funded and advanced lab for converting uninfectious SARS viruses into human-infectious SARS variants was not involved, is to pool our existing knowledge of how individual DNA and RNA sequences actually mutate and transform.

This is something we already understand

surprisingly well. In other words, which specific bases naturally tend to progress over time into other specific bases, and by what natural processes. We also have a fairly good idea of how sequences are maintained by various DNA repair mechanisms in more advanced organisms, and how mutations are often seemingly directed, or biased in some instances, according to the state of the organism or host. In the literature, these processes are called base transitions, and are frequently couched in terms of dynamic landscapes of maximum likelihood estimations of base substitutions.

They come in two general types: Transitions are interchanges of two-ring purines (A-G) or of one-ring pyrimidines (C-T), while transversions are interchanges of purine for pyrimidine bases. All told, we have 16 possible types of substitution scenarios, each with independent likelihoods. In human mitochondrial DNA, for example, we know that the transition to transversion ratio is very high. Mitochondria compete with nuclear DNA for access to nucleotides for replication, transcription and repair, and the relative abundances of each nucleotide in the cell influences the outcome of seemingly diverse random events. Much the same logic can be applied to evolving viral sequences. The time is ripe to start doing this type of analysis for the evolution of SARS sequences we obtain from animals or patient.

More information: Arinjay Banerjee et al. Unraveling the Zoonotic Origin and Transmission of SARS-CoV-2, *Trends in Ecology & Evolution* (2020). DOI: 10.1016/j.tree.2020.12.002