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Could Your Body Render Future COVID Vaccines Useless?

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STORY AT-A-GLANCE -

- The term "original antigenic sin" was first used by Thomas Francis in 1960, who determined that antibody response to a viral infection was highest against strains of seasonal influenza to which different age cohorts had first been exposed
- While imprinting can enhance your protection against future infections if you're exposed to antigenically related strains, if you're exposed to a distantly related strain, it may increase susceptibility to infection
- If the immune system reaction triggered by the COVID-19 vaccine acts as the original imprint, subsequent COVID-19 vaccines — updated to target emerging variants of SARS-CoV-2 — could be rendered ineffective
- A related phenomenon is pathogenic priming, in which, rather than enhancing your immunity against the infection, exposure to a virus or vaccine enhances the virus' ability to enter and infect your cells, resulting in more severe disease
- Significant concerns have also been raised surrounding antibody-dependent enhancement (ADE), and the possibility that COVID-19 vaccines could worsen COVID-19 disease via ADE

Get vaccinated. It's the latest COVID-19 propaganda message appearing everywhere from TV commercials to social media feeds, and it's being pushed by celebrities and government officials alike. Yet, a sizeable population of Americans aren't ready to roll up their sleeve just yet.

A January 2021 poll found 31% were taking a "wait and see" approach to see how the vaccine — or more aptly, **gene therapy** — is working while 7% said they would get the COVID-19 vaccine only if it became required for work, school or other activities, and 13% said they would "definitely not get it."[1]

A cautionary approach is warranted, as none of the COVID-19 vaccines currently on the market are actually licensed. They only have emergency use authorization — which, incidentally, also forbids them from being mandated, although this is being widely and conveniently ignored — as trials are still ongoing.

The fact is, there's a lot that's unknown about these products, including their ultimate effects on your immune response. Increasingly, scientists are asking whether a phenomenon known as original antigenic sin (OAS), or imprinting, may render next-generation COVID vaccines useless.[2]

What Is Original Antigenic Sin, or Imprinting?

The term "original antigenic sin" was first used by Thomas Francis in 1960, who determined that hemagglutination inhibition assay titers — which are used to determine the antibody response to a viral infection — were highest against strains of seasonal influenza to which different age cohorts had first been exposed.[3]

In other words, the first influenza virus that you're exposed to affects the way your lifelong immunity to that virus plays out.[4] Later infections with virus strains similar to the first one will boost your antibody response against the original strain, and it's not only influenza that this applies to. Imprinting is also known to occur in children with multiple dengue virus infections, for instance.[5]

In some cases, imprinting can be beneficial, but it can also be problematic. One study found that birth-year cohorts that had a first influenza exposure to seasonal H3 subtype viruses were less susceptible to avian influenza H7N9 virus later in life, while those exposed to H1 or H2 subtype viruses in childhood were less susceptible to avian H5N1-bearing viruses when they were older.[6]

"Using data from all known human cases of these viruses, we show that an individual's first IAV [influenza A virus] infection confers lifelong protection against severe disease from novel hemagglutinin (HA) subtypes in the same phylogenetic group," the researchers explained.[7] Imprinting has been suggested as one reason why flu vaccines are often ineffective.

Scott Hensley, an associate professor of microbiology at the University of Pennsylvania, explained to STAT News, "We've all been trained on different influenza viruses. If you vaccinate 100 people, guess what? They're all going to respond differently. We think a large part of that is that we all have a different immunological imprint."[8] He referred to a flu vaccine from 2017, when experts suggested a new H1N1 strain should be added. STAT News reported:[9]

> *"The one they had been using seemed to work fine for most people. But it wasn't working well for a slice of the population — adults between the ages of about 30 and late middle age.*
>
> *Hensley and his lab discovered that the vaccine target was making people who had their first flu exposures between 1977 and 1985 create antibodies to a version of H1N1 that was circulating back then — their imprinting virus. The decades-old H1N1 strains were too different from the 2009 version for the vaccine to work well in these people."*

The same thing could be happening with COVID-19.

Imprinting Could Mean Next COVID-19 Vaccines Won't Work

While imprinting can enhance your protection against future infections if you're exposed to antigenically related strains, if you're exposed to a distantly related strain, it may increase susceptibility to infection. According to researchers in The Journal of Immunology:[10]

> *"OAS-like responses were also problematic during the 2013–2014 influenza season, when H1N1 viruses acquired a mutation in an HA [hemagglutinin]*

epitope that was the primary target of the Ab [antibody] response mounted by middle-aged individuals.

The cohort generated a focused Ab response against this epitope during early life exposure to seasonal H1N1 viruses that circulated in the 1970s. As reported by the Hensley laboratory, this epitope was conserved in the original 2009 H1N1 pandemic strain.

However, the drifted H1N1 strain that emerged in 2013-2014 contained a mutation in this region of HA that resulted in poor Ab binding and subsequently unusually high mortality for middle-aged individuals."

In the case of COVID-19, it's possible that the immune system reaction triggered by the vaccine will act as the original imprint, leaving subsequent COVID-19 vaccines — updated to target **emerging variants of SARS-CoV-2** — ineffective.[11]

Michael Worobey, a professor of evolutionary biology at the University of Arizona, who conducted research on imprinting with influenza,[12] told STAT News, "I do think it's something that we need to be thinking about. We might actually see lower efficacy five years from now, if people are still locked into recalling the response to the first [SARS-2] antigen that they saw."[13]

Evidence of Coronavirus Imprinting

Some have argued that SARS-2 viruses don't appear to mutate as rapidly as influenza viruses, making imprinting less of a concern, but Hensley has already seen evidence of coronavirus imprinting while working to develop COVID-19 antibody tests. Blood samples of people with COVID-19 had "dramatic" rises in antibodies to OC43, a coronavirus that causes the common cold and is related to SARS-2, and the viruses that cause SARS and MERS.[14]

"These antibodies were not associated with protection against SARS-CoV-2 infections or hospitalizations, but they were boosted upon SARS-CoV-2 infection," Hensley and colleagues wrote in the journal Cell.[15] Hensley suggested that the immune response

from COVID-19 gene therapies may be so strong that it overrides imprinting impacts, while immunity from the natural infection could lead to imprinting that makes variants harder for the immune system to handle.

But the reality is, no one really knows what's going to happen. As immunologist David Topham, director of the New York Influenza Center of Excellence, told STAT News, one of three scenarios could occur, ranging from problematic to beneficial for those who have immunity from prior COVID-19 infection:[16]

> *"It can be a problem, because the immune cells specific for S2 [a spike protein] outcompete immune cells against other components of the spike protein that you really need in order to get protection. It can be inconsequential in that eventually the responses to the other parts of the protein catch up and it doesn't matter. Or it could actually be a benefit because it gets the immune system revved up more quickly."*

Problems With Pathogenic Priming

A related phenomenon is pathogenic priming, in which, rather than enhancing your immunity against the infection, exposure to a virus or vaccine enhances the virus' ability to enter and infect your cells, resulting in more severe disease.[17]

Research published in the Journal of Translational Autoimmunity confirmed that treatment with a vaccine may increase the risks associated with a wild type virus rather than protect against it, and concluded, as its title suggests, "Pathogenic priming likely contributes to serious and critical illness and mortality in COVID-19 via autoimmunity."[18] According to the study:[19]

> *"Pathogenic priming may be more or less severe in vaccine or infection induced immune responses to some proteins than for others due to original antigenic sin; the immunologic reaction against self-antigens may be made less severe as fast-evolving viruses evolve away from the original vaccine type."*

The Journal of Translational Autoimmunity article, written by James Lyons-Weiler with The Institute for Pure and Applied Knowledge, a nonprofit organization that performs scientific research in the public interest, explains how pathogenic priming occurred during previous trials of a SARS coronavirus vaccine:[20]

> *"In SARS, a type of 'priming' of the immune system was observed during animal studies of SARS spike protein-based vaccines leading to increased morbidity and mortality in vaccinated animals who were subsequently exposed to wild SARS virus.*
>
> *The problem, highlighted in two studies, became obvious following post-vaccination challenge with the SARS virus … recombinant SARS spike-protein-based vaccines not only failed to provide protection from SARS-CoV infection, but also that the mice experienced increased immunopathology with eosinophilic infiltrates in their lungs.*
>
> *Similarly … ferrets previously vaccinated against SARS-CoV also developed a strong inflammatory response in liver tissue (hepatitis). Both studies suspected a 'cellular immune response.'*
>
> *These types of unfortunate outcomes are sometimes referred to as 'immune enhancement'; however, this nearly euphemistic phrase fails to convey the increased risk of illness and death due to prior exposure to the SARS spike protein. For this reason, I refer to the concept as 'pathogen priming'."*

Strong Evidence of ADE Risk From COVID-19 Vaccines

Significant concerns have also been raised surrounding **antibody-dependent enhancement** (ADE), and the possibility that COVID-19 vaccines could worsen COVID-19 disease via ADE.[21] Timothy Cardozo of NYU Langone Health and Ronald Veazey with the Tulane University School of Medicine set out to determine if enough research existed to require clinicians to disclose the specific risk that COVID-19 vaccines could worsen disease if the recipient is exposed to circulating virus.

They reviewed preclinical and clinical evidence, which revealed that ADE is a significant concern. They noted:[22]

> *"COVID-19 vaccines designed to elicit neutralizing antibodies may sensitize vaccine recipients to more severe disease than if they were not vaccinated. Vaccines for SARS, MERS and RSV have never been approved, and the data generated in the development and testing of these vaccines suggest a serious mechanistic concern:*
>
> *that vaccines designed empirically using the traditional approach (consisting of the unmodified or minimally modified coronavirus viral spike to elicit neutralizing antibodies), be they composed of protein, viral vector, DNA or RNA and irrespective of delivery method, may worsen COVID-19 disease via antibody-dependent enhancement (ADE)."*

They concluded that, in order to meet medical ethics standards of **informed consent**, people taking part in COVID-19 vaccine trials, as well as those who have received it after approval, should be clearly warned of the "specific and significant COVID-19 risk of ADE."[23] This, however, has not occurred, and most receiving it have likely not even heard of ADE, much less its association with the experimental COVID-19 vaccine.

Already, vaccinated individuals do appear to be more susceptible to infection by certain variants of SARS-CoV-2, although it remains to be seen whether they are more prone to serious illness.

A study by researchers at Tel Aviv University and Clalit Health Services in Israel found the South African variant of SARS-CoV-2, dubbed B.1. 351 — which presently accounts for about 1% of COVID-19 cases in Israel — affects people vaccinated with **Pfizer's mRNA vaccine** to a greater extent than unvaccinated people.[24]

There continue to be many unanswered questions surrounding COVID-19 vaccines, many of which most of the public has never heard of — Th2 immunopathology, for another example. If you choose to get a COVID-19 vaccine, you're participating in this giant experiment, acting as a guinea pig to see what will ultimately bear out.

That being said, if you or someone you love have received a COVID-19 vaccine and are experiencing **side effects**, be sure to report it. The Children's Health Defense (CHD) is calling on all who have suffered a side effect from a COVID-19 vaccine to do three things:[25]

1. If you live in the U.S., **file a report on VAERS**
2. Report the injury on **VaxxTracker.com**, which is a nongovernmental adverse event tracker (you can file anonymously if you like)
3. **Report the injury on the CHD website**

Sources and Reference

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