

Ron Johnson Hospital Protocol Statement-Christina Parks
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I submit this statement to the Committee as a scientist with a Ph.D. in Cellular and Molecular Biology, not as a physician or nurse, and therefore do not offer testimony concerning bedside medical practice. I ask instead that the Committee examine an important scientific question: **What was known about potential COVID-19 therapeutics before and during the development of federal treatment recommendations, and how was that evidence weighed?**

Dr. Anthony Fauci was not simply a public-health spokesman during COVID-19. From 1984 through 2022, he directed the National Institute of Allergy and Infectious Diseases (NIAID), overseeing an extensive research portfolio encompassing HIV/AIDS, antiviral therapeutics, Ebola, emerging infectious diseases, and ultimately COVID-19.[1] His scientific career was particularly intertwined with HIV/AIDS research and the development and evaluation of antiviral therapies.

Against that background, the preexisting literature concerning chloroquine and hydroxychloroquine deserves examination.

Long before COVID-19, chloroquine and hydroxychloroquine had been investigated for antiviral activity against HIV. Laboratory studies demonstrated inhibition of HIV replication, and small clinical trials in the 1990s reported reductions in HIV-1 RNA with hydroxychloroquine, although subsequent clinical studies produced conflicting results.[2,3] Thus, the potential antiviral properties of these medications were well known within the infectious-disease literature.

More directly relevant, following the original SARS outbreak, researchers reported that chloroquine inhibited SARS-CoV—the coronavirus responsible for SARS—in cell culture. A 2004 study reported antiviral activity at concentrations below those producing cellular toxicity.[4] A subsequent 2005 study found strong inhibition of SARS-CoV infection and spread when chloroquine was administered either before or after viral exposure, with proposed mechanisms involving endosomal pH and altered glycosylation of ACE2.[5]

These findings did not prove that hydroxychloroquine would successfully treat COVID-19. They did, however, provide a scientifically plausible basis for urgently and objectively evaluating an inexpensive, widely available medication with established safety profiles that could potentially be repurposed as an antiviral therapies early in SARS-CoV-2 infection.

The use of Remdesivir in hospital protocols was extremely questionable because preclinical toxicology had already identified the kidney as a target organ, and FDA's

subsequent pharmacology review describes renal tubular injury in rhesus monkeys receiving intravenous remdesivir, including tubular atrophy and casts; in one seven-day study, a no-observed-adverse-effect level was not identified.[6] Its anti-viral efficacy was also in question as it had been investigated during the Ebola epidemic, and in the randomized PALM trial it was inferior in survival outcomes to the monoclonal-antibody therapies MAb114 and REGN-EB3.[7]

This does not establish that remdesivir caused renal failure in individual COVID-19 patients. COVID-19 itself can severely damage the kidneys, making individual causation difficult to determine. But it establishes that renal toxicity was a recognized safety signal warranting careful attention especially in the face of its questionable efficacy as an anti-viral.

I respectfully ask the Committee to investigate why one class of drugs with longstanding antiviral research and specific preclinical evidence against SARS-CoV was rapidly marginalized, while an investigational antiviral with known renal toxicology signals became a cornerstone of hospital treatment.

The issue is not whether hydroxychloroquine ultimately proved to be a “miracle drug.” It is whether competing therapeutic options were evaluated with the same scientific openness, evidentiary standards, and concern for patient safety.

References

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