

Annotation Protocol:

For each tweet-study pair, where the tweet contains an implicit reference to a scientific study, assess whether the implicit reference corresponds to the specific study in the pair.

Specifically, answer the following questions:

- **(Q1)** Does the tweet mention a SINGLE scientific study? If not, move on to the next tweet.
- **(Q2)** Does the tweet text mention the study? Answer with either **"Yes"**, **"No"**, or **"I don't know"**.
- **(Q3)** What elements from the tweet helped you answer Q2? Detail your reasoning.

For each candidate tweet-study pairs, the following metadata is made available to help answer the protocol's questions:

- The study's title, abstract, publication date, author affiliations and publication venue are included.
- The tweet's text and publication date.

The following 4 sample tweet-study pairs below are shown as examples which have already been answered.

Study title	Study date	Study affiliation/venue	Study abstract	Tweet text	Tweet date	(Q1) Does the tweet mention a SINGLE scientific study? If not, move on to the next tweet	(Q2) Does the tweet text mention the study?	(Q3) What elements from the tweet helped you answer Q2? Detail your reasoning
Effectiveness of Covid-19 Vaccines against the B.1.617.2 (Delta) Variant	21-07-2021	New England Journal of Medicine Jamie Lopez Bernal, Nick Andrews, Charlotte Gower, Eileen Gallagher, Ruth Simmons, Simon Theilwall, Julia Stowe, Elise Tessier, Natalie Groves, Gavin Dabrera, Richard Myers, Colin N J Campbell, Gayatri Amirhalingam, Matt Edmunds, Maria Zambon, Kevin E Brown, Susan Hopkins, Meera Chand, Mary Ramsay From Public Health England (J.L.B., N.A., C.G., E.G., R.S., S.T., J.S., E.T., N.G., G.D., R.M., C.N.J.C., G.A., M.E., M.Z., K.E.B., S.H., M.C., M.R.), the National Institute of Health Research (NIHR) Health Protection Research Unit in Vaccines and Immunisation, London School of Hygiene and Tropical Medicine (J.L.B., N.A., C.N.J.C., G.A., K.E.B., M.R.), the NIHR Health Protection Research Unit in Respiratory Infections, Imperial College London (J.L.B., M.Z.), and Guy's and St Thomas' Hospital NHS Trust (M.C.), London, and Healthcare Associated Infections and Antimicrobial Resistance, University of Oxford, Oxford (S.H.) - all in the United Kingdom.	Background: The B.1.617.2 (delta) variant of the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the virus that causes coronavirus disease 2019 (Covid-19), has contributed to a surge in cases in India and has now been detected across the globe, including a notable increase in cases in the United Kingdom. The effectiveness of the BNT162b2 and ChAdOx1 nCoV-19 vaccines against this variant has been unclear. Methods: We used a test-negative case-control design to estimate the effectiveness of vaccination against symptomatic disease caused by the delta variant or the predominant strain (B.1.1.7, or alpha variant) over the period that the delta variant began circulating. Variants were identified with the use of sequencing and on the basis of the spike (S) gene status. Data on all symptomatic sequenced cases of Covid-19 in England were used to estimate the proportion of cases with either variant according to the patients' vaccination status. Results: Effectiveness after one dose of vaccine (BNT162b2 or ChAdOx1 nCoV-19) was notably lower among persons with the delta variant (30.7%; 95% confidence interval [CI], 25.2 to 35.7) than among those with the alpha variant (48.7%; 95% CI, 45.5 to 51.7); the results were similar for both vaccines. With the BNT162b2 vaccine, the effectiveness of two doses was 93.7% (95% CI, 91.6 to 95.3) among persons with the alpha variant and 98.0% (95% CI, 85.3 to 99.1) among those with the delta variant. With the ChAdOx1 nCoV-19 vaccine, the effectiveness of two doses was 74.5% (95% CI, 68.4 to 79.4) among persons with the alpha variant and 67.0% (95% CI, 61.3 to 71.8) among those with the delta variant. Conclusions: Only modest differences in vaccine effectiveness were noted with the delta variant as compared with the alpha variant after the receipt of two vaccine doses. Absolute differences in vaccine effectiveness were more marked after the receipt of the first dose. This finding would support efforts to maximize vaccine uptake with two doses among vulnerable populations. (Funded by Public Health England.)	Peer-reviewed in the New England Journal of Medicine regarding Delta (B.1.617.2): •Pfizer is ~90% effective •AstraZeneca is ~70% effective. This falls in line with vaccine efficacy of other variants. Yes, the vaccines ARE indeed effective against Delta.	22-07-2021	Yes	Yes	1) - 2) The venue referred by the tweet (New England Journal of Medicine) matches the study's venue 3) The scientific claim contained in the tweet matches the study's main claim
The FDA-approved drug ivermectin inhibits the replication of SARS-CoV-2 in vitro	03-04-2020	Antiviral Research Leon Caly, Julian D Druce, Mike G Catton, David A Jans, Kylie M Wagstaff Victorian Infectious Diseases Reference Laboratory, Royal Melbourne Hospital, At the Peter Doherty Institute for Infection and Immunity, Victoria, 3000, Australia. Biomedicine Discovery Institute, Monash University , Clayton, Vic, 3800, Australia. Biomedicine Discovery Institute, Monash University , Clayton, Vic, 3800, Australia. Electronic address: kylie.wagstaff@monash.edu.	Although several clinical trials are now underway to test possible therapies, the worldwide response to the COVID-19 outbreak has been largely limited to monitoring/containment. We report here that ivermectin, an FDA-approved anti-parasitic previously shown to have broad-spectrum anti-viral activity in vitro, is an inhibitor of the causative virus (SARS-CoV-2), with a single addition to Vero-hSLAM cells 2 h post infection with SARS-CoV-2 able to effect ~5000-fold reduction in viral RNA at 48 h. Ivermectin therefore warrants further investigation for possible benefits in humans.	Published in the journal Antiviral Research , the study from Monash University showed that a single dose of ivermectin could stop the coronavirus growing in cell culture -- effectively eradicating all genetic material of the virus within two days.	05-04-2020	Yes	Yes	1) The affiliation referred by the tweet (Monash University) matches the study's affiliation 2) The venue (Antiviral Research journal) referred by the tweet matches the study's venue 3) The scientific claim contained in the tweet matches the study's main claim
SARS-CoV-2 Receptor ACE2 Is an Interferon-Stimulated Gene in Human Airway Epithelial Cells and Is Detected in Specific Cell Subsets across Tissues	27-04-2020	Cell journal Carly G K Ziegler, Samuel J Allon, Sarah K Nyquist, Ian M Mbano, Vincent N Miao, Constantine N Tzouanas, Yuming Cao, Ashraf S Yousif, Julia Bals, Blake M Hauser, Jared Feldman, Christoph Muus Program in Health Sciences & Technology, Harvard Medical School & Massachusetts Institute of Technology , Boston, MA 02115, USA; Institute for Medical Engineering & Science, Massachusetts Institute of Technology, Cambridge, MA 02139, USA; Koch Institute for Integrative Cancer Research, Massachusetts Institute of Technology, Cambridge, MA 02139, USA; Ragon Institute of MGH, MIT, and Harvard, Cambridge, MA 02139, USA; Broad Institute of MIT and Harvard, Cambridge, MA 02142, USA; Harvard Graduate Program in Biophysics, Harvard University, Cambridge, MA 02138, USA; Institute for Medical Engineering & Science, Massachusetts Institute of Technology, Cambridge, MA ...	There is pressing urgency to understand the pathogenesis of the severe acute respiratory syndrome coronavirus clade 2 (SARS-CoV-2), which causes the disease COVID-19. SARS-CoV-2 spike (S) protein binds angiotensin-converting enzyme 2 (ACE2), and in concert with host proteases, principally transmembrane serine protease 2 (TMPRSS2), promotes cellular entry. The cell subsets targeted by SARS-CoV-2 in host tissues and the factors that regulate ACE2 expression remain unknown. Here, we leverage human, non-human primate, and mouse single-cell RNA-sequencing (scRNA-seq) datasets across health and disease to uncover putative targets of SARS-CoV-2 among tissue-resident cell subsets. We identify ACE2 and TMPRSS2 co-expressing cells within lung type II pneumocytes, ileal absorptive enterocytes, and nasal goblet secretory cells. Strikingly, we discovered that ACE2 is a human interferon-stimulated gene (ISG) in vitro using airway epithelial cells and extend our findings to in vivo viral infections. Our data suggest that SARS-CoV-2 could exploit species-specific interferon-driven upregulation of ACE2, a tissue-protective mediator during lung injury, to enhance infection.	Published in the journal Antiviral Research , the study from Monash University showed that a single dose of ivermectin could stop the coronavirus growing in cell culture -- effectively eradicating all genetic material of the virus within two days.	05-04-2020	Yes	No	1) The affiliation referred by the tweet (Monash University) doesn't match the target study's affiliation (Harvard and MIT) 2) The venue referred by the tweet (Antiviral Research Journal) doesn't match the target study's venue (Cell Journal)

The FDA-approved drug ivermectin inhibits the replication of SARS-CoV-2 in vitro	03-04-2020	<p>Antiviral Research</p> <p>Leon Caly, Julian D Druce, Mike G Catton, David A Jans, Kylie M Wagstaff</p> <p>Victorian Infectious Diseases Reference Laboratory, Royal Melbourne Hospital, At the Peter Doherty Institute for Infection and Immunity, Victoria, 3000, Australia. Biomedicine Discovery Institute, Monash University, Clayton, Vic, 3800, Australia.</p> <p>Biomedicine Discovery Institute, Monash University, Clayton, Vic, 3800, Australia. Electronic address: kylie.wagstaff@monash.edu.</p>	<p>Background: Severe acute respiratory syndrome (SARS) is caused by a newly discovered coronavirus (SARS-CoV). No effective prophylactic or post-exposure therapy is currently available.</p> <p>Results: We report, however, that chloroquine has strong antiviral effects on SARS-CoV infection of primate cells. These inhibitory effects are observed when the cells are treated with the drug either before or after exposure to the virus, suggesting both prophylactic and therapeutic advantage. In addition to the well-known functions of chloroquine such as elevations of endosomal pH, the drug appears to interfere with terminal glycosylation of the cellular receptor, angiotensin-converting enzyme 2. This may negatively influence the virus-receptor binding and abrogate the infection, with further ramifications by the elevation of vesicular pH, resulting in the inhibition of infection and spread of SARS CoV at clinically admissible concentrations.</p> <p>Conclusion: Chloroquine is effective in preventing the spread of SARS CoV in cell culture. Favorable inhibition of virus spread was observed when the cells were either treated with chloroquine prior to or after SARS CoV infection. In addition, the indirect immunofluorescence assay described herein represents a simple and rapid method for screening SARS-CoV antiviral compounds.</p>	<p>2015- under Obama, Dr Fauci gave Wuhan Lab a \$3.9M grant for research on bats that's illegal in US.</p> <p>2017 Dr Fauci predicts a 'surprise outbreak' in Trump's first term.</p> <p>Fauci & #DeepState along w/China are responsible for the outbreak of #Covid & the continued rapid spread!</p>	29-07-2020	No		-
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