Resources Supporting Testimony of Dr. Christina Parks

for Michigan Bill HB4471

Do the Covid vaccines prevent transmission??..the words of leading public health officials (CDC, WHO) including Tony Fauci.

https://ipfs.io/ipns/k2k4r8pkk8wevtty3rpqw8mh2njz0snop5xpha1ybafquvb7dcgayh4j/index.html@p=9134.html

Vaccination predisposes those exposed to Antibody Dependent Enhancement (ADE), which results in more severe disease; shown specifically for the Delta variant.

1. Tseng, Chien-Te et al. **"Immunization with SARS coronavirus vaccines leads to pulmonary immunopathology on challenge with the SARS virus."** *PloS one* vol. 7,4 (2012): e35421. https://pubmed.ncbi.nlm.nih.gov/22536382/
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Covid-19: Fully vaccinated people can carry as much delta virus as unvaccinated people, data indicate
https://www.bmj.com/content/374/bmj.n2074?fbclid=IwAR06JSkgiTcx23PlhsSFIkiLua3NAjar93zH133Wm5y8c3sLwDU-rJBEGmA

Fully vaccinated persons made up 74% of people testing positive for Covid in the Barnstable Massachusetts outbreak and 4 out of 5 of those hospitalized.
https://www.cdc.gov/mmwr/volumes/70/wr/mm7031e2.htm

DTaP (containing acellular pertussis vaccine) does not prevent colonization by and infection with the Pertussis bacteria and allows/selects for the formation of vaccine resistant strains.

3. Gill, Christopher et al. **"The relationship between mucosal immunity, nasopharyngeal carriage, asymptomatic transmission and the resurgence of *Bordetella***

***pertussis*."** *F1000Research* vol. 6 1568. 25 Aug. 2017, doi:10.12688/f1000research.11654.1 https://pubmed.ncbi.nlm.nih.gov/28928960/

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Yearly Flu vaccines do not produce immunity to lethal variants, while natural immunity does.

7. Bodewes R, Kreijtz JH, et al. **Vaccination against human influenza A/H3N2 virus prevents the induction of heterosubtypic immunity against lethal infection with Avian influenza A/H5N1 virus.** *PloS One* 2009; 4(5): e5538 https://pubmed.ncbi.nlm.nih.gov/19440239/
8. Skowronski DM, De Serres G, Crowcroft NS, Janjua NZ, Boulianne N, et al. (2010) **Association between the 2008–09 Seasonal Influenza Vaccine and Pandemic H1N1 Illness during Spring–Summer 2009: Four Observational Studies from Canada.** *PLOS Medicine* 7(4): e1000258. https://journals.plos.org/plosmedicine/article/citation?id=10.1371/journal.pmed.1000258
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Yearly Flu vaccines, while demonstrating some efficacy the first year, result in increased susceptibility to both influenza and non-influenza respiratory illness (including coronaviruses), as well as increased influenza-related hospitalization in both adults and children in subsequent years.

10. Ohmit, Suzanne E et al. **"Influenza vaccine effectiveness in the community and the household."** *Clinical infectious diseases : an official publication of the Infectious Diseases Society of America* vol. 56,10 (2013): 1363-9. doi:10.1093/cid/cit060 https://pubmed.ncbi.nlm.nih.gov/23413420/
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Ph.Ds, those with less than a high school education and Africans Americans are the most "vaccine-hesitant" Americans.

https://www.medrxiv.org/content/10.1101/2021.07.20.21260795v1.full.pdf

Tuskegee Experiment

https://www.cdc.gov/tuskegee/timeline.htm

CDC Senior Scientist comes forward as a whistleblower, explaining that key data showing increased risk of autism in African American boys who were vaccinated on time was omitted from their MMR autism study.

https://sharylattkisson.com/2021/01/cdc-scientist-we-scheduled-meeting-to-destroy-vaccine-autism-study-documents/

Questions

Promotion of Cancer by hypercapnia (increased partial pressure of carbon dioxide) and resulting blood acidosis.

Effect of Hypercapnia, an Element of Obstructive Respiratory Disorder, on Pancreatic Cancer Chemoresistance and Progression
https://www.sciencedirect.com/science/article/abs/pii/S107275152030123X

Ibrahim-Hashim, Arig, and Veronica Estrella. **"Acidosis and cancer: from mechanism to neutralization."** *Cancer metastasis reviews* vol. 38,1-2 (2019): 149-155. https://pubmed.ncbi.nlm.nih.gov/30806853/

Mask-wearing and the respiratory acidosis it induces as well as cardiorespiratory stress are a metabolic disaster.

https://www.verywellhealth.com/respiratory-acidosis-4691758

Lee, Sharen et al. **"COVID-19: Electrophysiological mechanisms underlying sudden cardiac death during exercise with facemasks."** *Medical hypotheses* vol. 144 (2020): 110177. https://pubmed.ncbi.nlm.nih.gov/33254499/

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Letter to the Editor

Infection-enhancing anti-SARS-CoV-2 antibodies recognize both the original Wuhan/D614G strain and Delta variants. A potential risk for mass vaccination?

The aim of the present study was to evaluate the recognition of SARS-CoV-2 Delta variants by infection enhancing antibodies directed against the NTD. The antibody studied is 1052 (pdb file #7LAB) which has been isolated from a symptomatic Covid-19 patient[1]. Molecular modeling simulations were performed as previously described[2]. Two currently circulating Delta variants were investigated, with the following mutational patterns in the NTD :

- G142D/E154K (B.1.617.1)
- T19R/E156G/del157/del158/A222V (B.1.617.2)

Each mutational pattern was introduced in the original Wuhan/D614G strain, submitted to energy minimization, and then tested for antibody binding. The energy of interaction (ΔG) of the reference pdb file #7LAB (Wuhan/D614G strain) in the NTD region was estimated to −229 kJ/mol^{-1}. In the case of Delta variants, the energy of interaction was raised to −272 kJ.mol^{-1} (B.1.617.1) and −246 kJ.mol^{-1} (B.1.617.2). Thus, these infection enhancing antibodies not only still recognize Delta variants but even display a higher affinity for those variants than for the original SARS-CoV-2 strain.

The global structure of the trimeric spike of the B.1.617.1 variant in the cell-facing view is shown in Fig. 1A. As expected, the facilitating antibody bound to the NTD (in green) is located behind the contact surface so that it does not interfere with virus-cell attachment. Indeed, a preformed antibody-NTD complex could perfectly bind to the host cell membrane. The interaction between the NTD and a lipid raft is shown in Fig. 1B, and a whole raft-spike-antibody complex in Fig. 1C. Interestingly, a small part of the antibody was found to interact with the lipid raft, as further illustrated in Figs. 1D-E. More precisely, two distinct loops of the heavy chain of the antibody encompassing amino acid residues 28–31 and 72–74, stabilize the complex through a direct interaction with the edge of the raft (Fig. 1F). Overall, the energy of interaction of the NTD-raft complex was raised from −399 kJ.mol^{-1} in absence of the antibody to −457 kJ.mol^{-1} with the antibody. By clamping the NTD and the lipid raft, the antibody reinforces the attachment of the spike protein to the cell surface and thus facilitates the conformational change of the RBD which is the next step of the virus infection process[2].

This notion of a dual NTD-raft recognition by an infection enhancing antibody may represent a new type of ADE that could be operative with other viruses. Incidentally, our data provide a mechanistic explanation of the FcR-independent enhancement of infection induced by anti-NTD antibodies[1]. The model we propose, which links for the first time lipid rafts to ADE of SARS-CoV-2, is in line with previous data showing that intact lipid rafts are required for ADE of dengue virus infection[3].

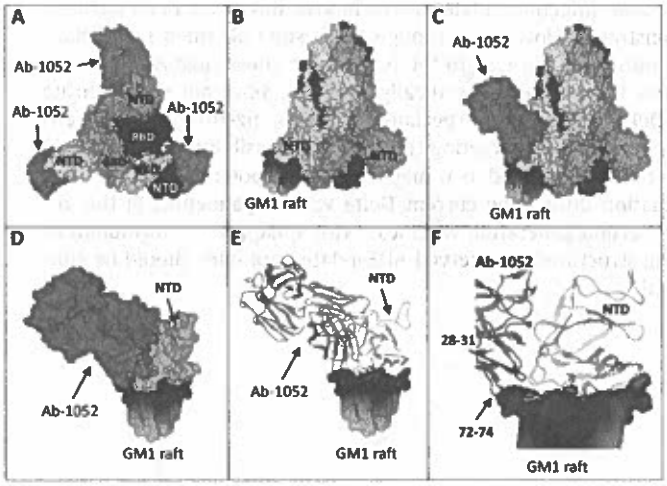

Fig. 1. Infection enhancing antibodies recognize the NTD of Delta variants. A. Molecular model of the Delta B.1.617.1 spike trimer as viewed from the host cell surface (chains A, B and C in cyan, yellow and purple, respectively), with the NTD and RBD of each chain indicated. The 1052 antibody is in green. **B.** Spike trimer with the B subunit bound to a lipid raft (with 6 ganglioside GM1 molecules). **C.** Trimolecular [spike-antibody-raft] complex. **D.** Focus on the NTD-antibody complex bound to the lipid raft. **E.** Secondary structures of the NTD (yellow) and the antibody (green) bound to lipid raft gangliosides. **F.** The 1052 antibody clamps the NTD and the edge of the lipid raft.

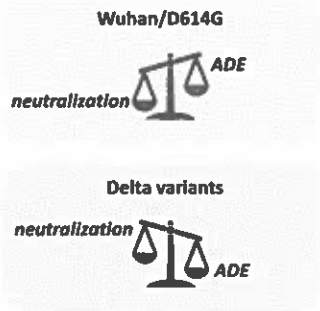

Fig. 2. Neutralization vs ADE balance according to SARS-CoV-2 strains.

Neutralizing antibodies directed against the NTD have also been detected in Covid-19 patients[4-5]. The 4A8 antibody is a major representant of such antibodies[5]. The epitope recognized by this antibody on the flat NTD surface is dramatically affected in the NTD of Delta variants[2], suggesting a significant loss of activity in vaccinated people exposed to Delta variants. More generally, it can be reasonably assumed that the balance between neutralizing and facilitating antibodies may greatly differ according to the virus strain (Fig. 2).

https://doi.org/10.1016/j.jinf.2021.08.010

2018 Final Pertussis Surveillance Report

Reported Pertussis Incidence and Cases

STATES	Incidence (per 100,000)	No. of Cases
ALABAMA	4.48	219
ALASKA	12.48	92
ARIZONA	3.33	239
ARKANSAS	3.28	99
CALIFORNIA	5.85	2315
COLORADO	10.78	614
CONNECTICUT	1.20	43
DELAWARE	19.64	190
D.C.	1.99	14
FLORIDA	1.53	326
GEORGIA	1.85	195
HAWAII	2.32	33
IDAHO	22.57	396
ILLINOIS	2.90	370
INDIANA	2.75	184
IOWA	5.13	162
KANSAS	5.26	153
KENTUCKY	4.43	198
LOUISIANA	2.68	125
MAINE	33.32	446
MARYLAND	1.97	119
MASSACHUSETTS	3.85	266
MICHIGAN	6.46	646
MINNESOTA	7.08	397
MISSISSIPPI	1.44	43
MISSOURI	2.77	170
MONTANA	13.46	143
NEBRASKA	8.35	161
NEVADA	2.83	86
NEW HAMPSHIRE	10.10	137
NEW JERSEY	3.09	275
NEW MEXICO	11.55	242
NEW YORK	3.35	373
NEW YORK CITY	2.20	185
NORTH CAROLINA	3.75	389
NORTH DAKOTA	6.71	51
OHIO	5.45	637
OKLAHOMA	2.61	103
OREGON	11.91	499
PENNSYLVANIA	3.61	462
RHODE ISLAND	3.22	34
SOUTH CAROLINA	3.89	198
SOUTH DAKOTA	18.48	163
TENNESSEE	1.82	123
TEXAS	4.07	1167
UTAH	13.67	432
VERMONT	5.43	34
VIRGINIA	2.88	245
WASHINGTON	8.32	627
WEST VIRGINIA	1.50	27
WISCONSIN	12.04	700
WYOMING	10.73	62
TOTAL	**4.77**	**15,609**

Source: NCHS Bridged Race Intercensal Population Estimate for 2018.

Weeks 1-52, 2018 CDC/NCIRD/DBD/MVPDB

Notice to Readers: Final 2018 Reports of Notifiable Diseases

https://wonder.cdc.gov/nndss/nndss_annual_tables_menu.asp

Reported Pertussis Cases

2017: **18,975** 2018: **15,609**

Reported Pertussis Cases and Percent Hospitalization by Age Group

Age	No. of Cases (% of total)	Age Inc /100,000	% Hospitalized by age**
< 6 mos	1401 (9.0)	72.8	42.3
6-11 mos	630 (4.0)	32.7	11.9
1-6 yrs	3232 (20.7)	13.5	2.6
7-10 yrs	1897 (12.2)	11.6	1.3
11-19 yrs	4922 (31.5)	13.0	0.9
20+ yrs	3520 (22.6)	1.4	7.7
Unknown Age	7 (0.0)	N/A	N/A
Total	**15,609 (100)**	**4.8***	**7.0**

*Total age incidence per 100,000 calculated from 15,602 cases with age reported.
**Age-specific proportion of cases that were hospitalized, calculated from those with a known hospitalization status.

Reported Pertussis Deaths

Age	Deaths
Cases, aged < 1 yr	3
Cases, aged ≥ 1 yr	2
Total	**5†**

*Deaths reported through NNDSS to CDC.
†3 of the 5 deaths were female.

Reported DTaP Vaccine Status of Children with Pertussis, Ages 6 months through 6 years

Age	Vaccine History Unknown	Unvaccinated	Undervaccinated (1-2 doses)	Completed Primary DTaP Series (3+ doses)	Total
	No. (%)	No. (%)	No. (%)	No. (%)	No.
6-11 mo	286 (45.4)	61 (9.7)	92 (14.6)	191 (30.3)	630
1-4 yrs	1194 (49.3)	228 (9.4)	83 (3.4)	916 (37.8)	2421
5-6 yrs	360 (44.4)	75 (9.3)	20 (2.5)	356 (43.9)	811
Total*	**1840 (47.6)**	**364 (9.4)**	**195 (5.0)**	**1463 (37.9)**	**3862**

*Percent calculated from total cases aged 6 months to 6 years, n=3,862.

Footnote: This table reflects reported vaccination history of pertussis cases aged 6 months through 6 years. CDC recommends all children receive at least 3 doses of DTaP by age 6 months. DTaP coverage in the United States is very high. Over 95% of all children 19-35 months of age have received at least 3 doses of DTaP. This table illustrates a similar trend among the pertussis cases reported during 2018—the majority have received at least 3 doses of DTaP. Because protection from DTaP wanes over time, even children who are up to date with their pertussis vaccines may contract pertussis. Unvaccinated children are more likely to contract pertussis and have more severe disease than those who are fully vaccinated. Note: surveillance data have limitations and are often incomplete; more than a third of pertussis cases in this table have unknown pertussis vaccination history. You cannot use these data to interpret vaccine effectiveness or to assess risk, as the data are incomplete and there is no healthy comparison group.

National Center for Immunization and Respiratory Diseases
Division of Bacterial Diseases

CS292279 December, 2019

Acellular pertussis vaccines protect against disease but fail to prevent infection and transmission in a nonhuman primate model

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Edited by Rino Rappuoli, Novartis Vaccines and Diagnostics Srl, Siena, Italy, and approved October 22, 2013 (received for review August 5, 2013)

Pertussis is a highly contagious respiratory illness caused by the bacterial pathogen *Bordetella pertussis*. Pertussis rates in the United States have been rising and reached a 50-y high of 42,000 cases in 2012. Although pertussis resurgence is not completely understood, we hypothesize that current acellular pertussis (aP) vaccines fail to prevent colonization and transmission. To test our hypothesis, infant baboons were vaccinated at 2, 4, and 6 mo of age with aP or whole-cell pertussis (wP) vaccines and challenged with *B. pertussis* at 7 mo. Infection was followed by quantifying colonization in nasopharyngeal washes and monitoring leukocytosis and symptoms. Baboons vaccinated with aP were protected from severe pertussis-associated symptoms but not from colonization, did not clear the infection faster than naïve animals, and readily transmitted *B. pertussis* to unvaccinated contacts. Vaccination with wP induced a more rapid clearance compared with naïve and aP-vaccinated animals. By comparison, previously infected animals were not colonized upon secondary infection. Although all vaccinated and previously infected animals had robust serum antibody responses, we found key differences in T-cell immunity. Previously infected animals and wP-vaccinated animals possess strong *B. pertussis*-specific T helper 17 (Th17) memory and Th1 memory, whereas aP vaccination induced a Th1/Th2 response instead. The observation that aP, which induces an immune response mismatched to that induced by natural infection, fails to prevent colonization or transmission provides a plausible explanation for the resurgence of pertussis and suggests that optimal control of pertussis will require the development of improved vaccines.

whooping cough | T-cell memory | animal models | adaptive immunity | IL-17

Pertussis is a highly contagious, acute respiratory illness caused by the bacterial pathogen *Bordetella pertussis* (1, 2). Infection results in a wide spectrum of clinical manifestations ranging from mild respiratory symptoms to a severe cough illness accompanied by marked leukocytosis and the hallmark inspiratory whoop and posttussive emesis (3). Because acellular pertussis vaccines replaced whole-cell vaccines in the 1990s, pertussis has reemerged at a startling rate in the United States despite nationwide vaccine coverage in excess of 95% (4). With a 50-y high of 42,000 reported cases in the United States in 2012, pertussis is the most common of the vaccine-preventable diseases (5). This resurgence is mirrored throughout the industrial world despite similar high rates of vaccination (6–9). Two common hypotheses for the resurgence have been proposed: *i*) current acellular pertussis vaccines (aP) vaccines are less effective than the whole-cell pertussis (wP) vaccines they replaced and *ii*) aP-induced immunity wanes more quickly than anticipated (10–13). However, pertussis resurgence is not completely understood (14, 15).

Hampering our ability to counteract this resurgence is the fact that pertussis pathogenesis and immunity to natural infection have not been well studied in humans because typical pertussis is sporadic given high rates of vaccination in developed countries. Human challenge studies have been proposed but never conducted due to a variety of logistical and ethical problems including the potential for severe disease, the lack of an effective therapeutic for established disease, and the highly contagious nature of pertussis. Although a variety of small-animal models have been used to study pertussis, none of them adequately reproduce the human disease (16). To address this gap, we recently developed a nonhuman primate model of pertussis using baboons (*Papio anubis*) and found the disease is very similar to severe clinical pertussis. Upon challenge, baboons experience 2 wk of heavy respiratory colonization and leukocytosis peaking between 30,000–80,000 cells/mL, similar to the range in pertussis-infected infants (1, 17). In addition, baboons experience a paroxysmal cough illness characterized by repeated fits of 5–10 coughs. The coughing fits last on average >2 wk in the baboon, although this is less than some severely infected children, where the cough can last up to 12 wk (1, 17). We also characterized airborne transmission of *B. pertussis* from infected to naïve animals, which is the route of transmission postulated to occur between humans (18). Because this is the only model of pertussis to reproduce the cough illness and transmission of the human disease, we believe it provides the unique opportunity to test our hypothesis that aP vaccines fail to prevent *B. pertussis* colonization, thus enabling transmission among vaccinated individuals.

Using this model we have confirmed that, as in humans, aP vaccines provide excellent protection against severe disease in baboons. However, aP vaccines do not prevent colonization following direct challenge or infection by transmission. In addition, aP-vaccinated animals are capable of transmitting disease to naïve contacts. By comparison, wP-vaccinated animals cleared infection significantly more quickly than aP-vaccinated or naïve

Significance

Pertussis has reemerged as an important public health concern since current acellular pertussis vaccines (aP) replaced older whole-cell vaccines (wP). In this study, we show nonhuman primates vaccinated with aP were protected from severe symptoms but not infection and readily transmitted *Bordetella pertussis* to contacts. Vaccination with wP and previous infection induced a more rapid clearance compared with naïve and aP-vaccinated animals. While all groups possessed robust antibody responses, key differences in T-cell memory suggest that aP vaccination induces a suboptimal immune response that is unable to prevent infection. These data provide a plausible explanation for pertussis resurgence and suggest that attaining herd immunity will require the development of improved vaccination strategies that prevent *B. pertussis* colonization and transmission.

Author contributions: J.M.W. and T.J.M. designed research; J.M.W., L.I.Z., and T.J.M. performed research; J.M.W. and T.J.M. analyzed data; and J.M.W. and T.J.M. wrote the paper.

The authors declare no conflict of interest.

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See Commentary on page 575.

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