Did the failed acellular pertussis vaccine reduce rates of meningococcal disease in the US?

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The acellular pertussis vaccine (APV) does not produce mucosal immunity. It therefore makes recipients susceptible to colonization by the *Bordetella pertussis* bacteria. Such colonized individuals transmit the bacteria (1–3).

Meningococcal disease rates in the US have been declining since the late 1990s (4). Meningococcal vaccines introduced in the 1970s or 2005 obviously cannot explain this trend.

One possible explanation is the introduction of APV in 1991. Widespread *B. pertussis* colonization in the population occurred following APV introduction. With *N. meningitidis* and *B. pertussis* competing for the same nasopharyngeal (NP) real estate, a reduction in *N. meningitidis* colonization can be expected. The APV also resulted in IgE mediated sensitization against *B. pertussis* proteins (5). The result can be local allergic reactions that perturb NP colonization (by any bacteria) upon exposure to *B. pertussis*. One can expect IgE mediated mast cell degranulation, histamine release, mucus production that result in perturbation of colonies. So *N. meningitidis* colonization of NP may have been impacted by competition with *B. pertussis* or perturbation due to IgE mediated immune reactions.

This may be one more unintended consequence (a rare positive one) of poorly understood vaccines, developed using trial and error (6). Of course, the APV more than made up for this benefit by contributing to life-threatening milk allergy, autism, asthma and numerous autoimmune disorders (7–9).

References

1. Rubin K, Glazer S. The potential role of subclinical Bordetella Pertussis colonization in the etiology of multiple sclerosis. Immunobiology. 2015 Dec;
2. Zhang Q, Yin Z, Li Y, Luo H, Shao Z, Gao Y, et al. Prevalence of asymptomatic bordetella pertussis and bordetella parapertussis infections among school children in China as determined by pooled real-time PCR: A cross-sectional study. Scand J Infect Dis. 2014;46(4):280–7.
3. Arumugham V. Influenza and acellular pertussis vaccines not only fail to protect, they increase susceptibility and severity of disease upon infection – benefits are overrated and the risks are being ignored [Internet]. 2019. Available from: https://doi.org/10.5281/zenodo.2532166
4. Meningococcal | Surveillance | CDC [Internet]. [cited 2019 Jan 22]. Available from: https://www.cdc.gov/meningococcal/surveillance/index.html
5. Edelman K, Malmstrom K, He Q, Savolainen J, Terho EO, Mertsola J. Local reactions and IgE antibodies to pertussis toxin after acellular diphtheria-tetanus-pertussis immunization. Eur J Pediatr. 1999 Dec;158(12):989–94.

6. Pulendran B, Ahmed R. Immunological mechanisms of vaccination. Nat Immunol. 2011 Jun;12(6):509–17.

7. Vanood A, Wingerchuk D. Systematic Review Investigating Relationship Between Neuromyelitis Optica Spectrum Disorder (NMOSD) and Vaccination (P1.2-003). Neurology [Internet]. 2019;92(15 Supplement). Available from: https://n.neurology.org/content/92/15_Supplement/P1.2-003

8. Arumugham V. Vaccines cause autoimmune diseases: The latest evidence implicates influenza, tetanus, diphtheria, and pertussis (Tdap), human papilloma virus, pneumococcal, hepatitis A, hepatitis B, typhoid, yellow fever, and Japanese encephalitis vaccines in Neuromyelitis Optica Spectrum Disorders (NMOSD) [Internet]. 2019 [cited 2019 Jun 12]. Available from: https://doi.org/10.5281/zenodo.3244630

9. Arumugham V. Vaccines and Biologics injury table based on mechanistic evidence – Mar 2019 [Internet]. 2019 [cited 2019 Apr 20]. Available from: https://doi.org/10.5281/zenodo.2582634