Re: Fwd: Your JID Brief Report re the Australian pertussis epidemic in 2008-2010

From: Elizabeth Hart <eliz.hart25@gmail.com>
Date: Tue, Dec 11, 2012 at 11:01 PM
Subject: Fwd: Your JID Brief Report re the Australian pertussis epidemic in 2008-2010
To: lyn.gilbert@swahs
Cc: r.lan@unsw Beate Mies@

Professor Gilbert

Please see below my email to Professor Lan, including queries in reference to the Australian pertussis epidemic in 2008-2010.

Please note I have no expertise in this area, and I may have misinterpreted the information presented in the report, and Professor Lan's comments. I would appreciate clarification on this matter, but I have not received a response from Professor Lan.

Professor Gilbert, I also note that you have stated:

"We could give vaccines earlier - to newborns, as long as it was done safely, we could try a combination of the old vaccine and the current one at different times, we could administer more boosters of the current vaccine, or we could try a whole new vaccine". (1)

This all sounds rather experimental...

I would appreciate your response to my queries, as outlined in my email to Professor Lan below.

Yours sincerely

Elizabeth Hart


---------- Forwarded message ----------
From: Elizabeth Hart <eliz.hart25@gmail.com>
Date: Tue, Dec 4, 2012 at 8:50 PM
Subject: Your JID Brief Report re the Australian pertussis epidemic in 2008-2010
To: r.lan@unsw

Professor Lan

I have recently read your JID Brief Report regarding the Australian pertussis epidemic in 2008-2010 (1) which states

"B. pertussis isolates collected from 4 Australian states during an ongoing pertussis epidemic that began in 2008 were classified using SNPs, MLVA, fim3, prn, and ptxP typing. SNP cluster 1 strains,
primarily SP13 and SP14, accounted for 86% of isolates...This is a significant increase from our previous study of isolates collected between 2000 and 2007, in which SNP cluster 1 represented only 31% of isolates. This suggests increasing selection among the *B. pertussis* population in Australia in favor of strains carrying antigens that differ from those represented in ACVs." (My emphasis.)

and

"The prn2-pxtP3 isolates have the potential not only to evade the protective effects of ACV but also to increase disease severity as a double act of *B. pertussis* adaptation. Therefore, vaccine-induced selection could contribute to the expansion of cluster I, specifically SP13 and SP14, because of the presence of both prn2 and pxtp3. These 2 SPs have swept across Australia during the epidemic period. (My emphasis.)

Given these statements, I was confused by your comments in the UNSW website article (2) on this subject, i.e. "The prolonged whooping cough epidemic in Australia that began during 2008 has been predominantly caused by the new genotype of *B. pertussis*...The genotype was responsible for 31 percent of cases in the 10 years before the epidemic, and that's now jumped to 84 per cent - a nearly three-fold increase, indicating it has gained a selective advantage under the current vaccination regime...The vaccine is still the best way to reduce transmission of the disease and reduce cases, but it appears to be less effective against the new strain and immunity wanes more rapidly. We need to look at changes to the vaccine itself or increase the number of boosters." (My emphasis.)

Professor Lan can you please clarify for me how increasing the number of 'boosters' of the existing vaccine protects against the new strain?

Also, how is vaccination "still the best way to reduce transmission of the disease and reduce cases" particularly if "vaccine-induced selection could contribute to the expansion of cluster I"?

I would appreciate your response on this matter.

Yours sincerely

Elizabeth Hart

References: