Investigation of Sapovirus Gastroenteritis Outbreaks in British Columbia using Real Time RT-PCR, Oct 2007 to Dec 2011

B Auk, N Prystajecky, P Tang, L Li, A Li, J Fung, J Wong, B Wong, R Chen, V Montoya, A McNabb, JL Isaac-Renton

In April 2012, the Environmental Microbiology Program of the BCCDC PHMRL implemented sapovirus testing using molecular (fast, sensitive) Real Time RT-PCR tools for gastro-enteritis (GE) outbreak investigation. This test will be an important additional tool in providing information for infection control and disease tracking.

Our investigation of sapovirus testing started in December 2008, where it was noted that more than 50% of GE outbreaks during a two week period were caused by unknown etiology where in previous seasons, approximately 90% or more of GE outbreaks were caused by norovirus. A small retrospective study of unknown GE outbreaks was undertaken using a novel virus microarray called the Virochips; the use of Virochips detected sapovirus amongst the unknown GE outbreak samples. At the same time, Alberta’s Public Health Laboratory along with its collaborators published an article outlining the fact that sapovirus was an important cause of GE in their province (Pang et al, 2009).

Sapovirus is part of the same Caliciviridae family as norovirus. According to literature, symptoms and transmission patterns of sapovirus is similar to norovirus, though sapovirus is generally found most often in young children.

As part of ongoing improvement of BCCDC PHMRL’s molecular microbiology capabilities, with Alberta Public Health Laboratory help, our staff in Molecular Microbiology & Genomics and Environmental Microbiology validated and implemented sapovirus Real Time RT-PCR for GE outbreak investigation. This work started by retrospective testing of all unknown GE outbreak samples from Oct 2007 to Feb 2012. This showed that it is indeed an important cause of GE outbreaks in BC, as the second leading source of GE next to norovirus (Figure 1). Sapovirus rates in BC fluctuate significantly season to season, from a high of 7.7% (08-09) to a low of 1.2% (10-11). During the 08-09 season, the majority of reported sapovirus outbreaks occurred in residential care facilities (47.6%), followed by hospitals (28.6%) and day cares (19.0%). In the current season, however, sapovirus is mainly causing outbreaks in day care facilities in BC. Testing of sapovirus in GE outbreaks is currently ongoing for all norovirus negative outbreaks.

Ongoing development of even more enhanced molecular microbiology assays for other causes of GE outbreaks continues.

Immunity to Measles Virus Infection in Prenatal British Columbians

A Mak, V Sahni, M Naus, M Petric, M Krajden

A sero-survey measles antibody study of prenatal women was conducted to inform measles immunization and control policies in BC and to correlate the levels of immunity in relation to an individual’s year of birth.

Two different measles virus serology assays were used for this serological study: VIDAS Measles IgG Assay (Biomerieux); and, Enzygnost Anti-Measles-virus/IgG assay-Quantitative in mIU/mL (Dade-Behring, a division of Siemens).

It was found that younger women demonstrated lower measles IgG antibody reactivity: women born in the 1960s were 95% IgG reactive compared to 88% of women born in the 1970s (Table 1). Further examining IgG results by birth year revealed that the decrease in immunity with younger age may be partly attributable to an increase in the proportion of women with equivocal results (Figure 2).

Overall there were more samples that tested in the equivocal range by the Behring Enzygnost assay and non-reactive by the VIDAS assay. However, this would not have appreciably affected the number of women designated as immune to measles as the two assays compared favourably.

Table 1
Year of birth and IgG positivity.

<table>
<thead>
<tr>
<th>Year of Birth</th>
<th>1960-69</th>
<th>1970-79</th>
</tr>
</thead>
<tbody>
<tr>
<td>% Measles IgG Positive</td>
<td>95% (631/661)</td>
<td>88% (588/665)</td>
</tr>
<tr>
<td>(42-51 years)</td>
<td>(32-41 years)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Year of Birth</th>
<th>1960-64</th>
<th>1965-69</th>
<th>1970-74</th>
<th>1975-79</th>
</tr>
</thead>
<tbody>
<tr>
<td>% Measles IgG Positive</td>
<td>97% (228/234)</td>
<td>94% (403/427)</td>
<td>91% (303/332)</td>
<td>85% (285/333)</td>
</tr>
</tbody>
</table>

Figure 1
Gastroenteritis outbreak etiologies in BC, Oct 2007 - Feb 2012. * Other uncommon etiologies included Diarrhetic Shellfish Poisoning, Cyclospora and scrombroid poisoning. ** Mix refers to more than one etiology detected.

Figure 2
Quantitative IgG (Behring) result by birth year.
Carbapenemase Resistant Enterobacteriaceae (CRE)

The latest counts for cases of carbapenemase resistance in BC can be found in Table 2 (updated from our February 2012 issue). Sixteen cases with the New Delhi Metallo-β-lactamase gene (NDM) endemic to South Asia have been detected since this work began in 2010. Two cases had the *Klebsiella pneumoniae* carbapenem (KPC) β-lactamase gene (one case with KPC as well as a Verona integron-encoded metallo-β-lactamase (VIM) gene) and three cases with only the VIM gene. Two cases with the IMP-type β-lactamase has also been detected.

To date, carbapenem resistance has been isolated in a variety of organisms including *E. coli, Klebsiella pneumoniae, Pseudomonas aeruginosa, Citrobacter freundii, Morganella morganii* and *Enterobacter cloacae* and *Acinetobacter baumannii*.

Table 2. Carbapenem Resistant Enterobacteriaceae Detected, Bacteriology & Mycology Program, PHMRL.

<table>
<thead>
<tr>
<th>Type</th>
<th>No. of Cases</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>NDM</td>
<td>17</td>
<td></td>
</tr>
<tr>
<td>KPC</td>
<td>2</td>
<td>1 case also harboured the VIM gene</td>
</tr>
<tr>
<td>VIM</td>
<td>3</td>
<td>In addition to above KPC/VIM case</td>
</tr>
<tr>
<td>IMP</td>
<td>2</td>
<td></td>
</tr>
</tbody>
</table>
Gastrointestinal Outbreaks

In May, there were 12 gastrointestinal (GI) outbreaks investigated at the PHMRL. The number is consistent with the volumes typically seen at this time in past years (Figure 3). Outbreaks were identified from 9 longterm care facilities, 2 daycares/schools and 1 hospital. Samples for laboratory testing were submitted for 8 (67%) of these outbreaks. Of these, norovirus was confirmed in 5 (62%) outbreaks. Rotavirus was detected in one longterm care facility and sapovirus was detected at a daycare/school.

The data available are from outbreaks in which the PHMRL has been notified. Some acute care microbiology laboratories are also testing for norovirus in the province and these data do not include outbreaks from Vancouver Island Health Authority. Given the nature of GI outbreaks, samples are not always available for testing.

Figure 3
Gastrointestinal outbreaks investigated since January, 2012, Environmental Microbiology, Bacteriology & Mycology, Parasitology and Virology Programs, PHMRL.
Respiratory Outbreaks

In May, samples were submitted to the PHMRL for 4 respiratory outbreak investigations from 3 long term care facilities and 1 hospital. The number of outbreaks investigated was on the higher end of what has been previously observed at this time for week 21 (Figure 4). Using PCR and Luminex methods, influenza A(H3) was detected in 1 long term care facility.

Figure 4
Respiratory outbreaks investigated in the 2011/2012 respiratory season, Virology Program, PHMRL.
Influenza Surveillance

Respiratory testing volumes have been slightly above that of the same weeks from the 2010/11 season and particularly for week 22 (Figure 5). Influenza positivity rates have varied from 12-43% in weeks 18-22 (Table 3). Influenza A (H3N2) was the major virus type detected this period with 63 (12.68%) positive specimens, followed by 47 (9.46%) detections of influenza B and 14 (2.82%) detections of (H1N1)pdm09 during this time. Influenza A rates were higher this period compared to this time last season, rising to a season high of 26% in week 22 (Figure 5). Influenza B rates have been between 6% to a season high of 17% positivity.

Of the other respiratory viruses, rhino/enterovirus continues to be the most detected at 14-18% positivity. There has been a decrease of RSV this month along with human metapneumovirus, adenovirus and parainfluenza virus to rates below 5% positivity.

Table 3

<table>
<thead>
<tr>
<th>Positive influenza A and B detections for weeks 14-17 (April 29- June 2, 2012, Virology Program, PHMRL. (H1N1)pdm09 refers to the 2009 influenza A(H1N1) pandemic virus.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Week 18</strong></td>
</tr>
<tr>
<td>Number of Specimens Tested</td>
</tr>
<tr>
<td>Number of Positive Specimens</td>
</tr>
<tr>
<td>Influenza A</td>
</tr>
<tr>
<td>(H1N1)pdm09</td>
</tr>
<tr>
<td>sH3N2</td>
</tr>
<tr>
<td>Not typeable</td>
</tr>
<tr>
<td>Influenza B</td>
</tr>
</tbody>
</table>
Influenza Surveillance continued

National influenza trends in May demonstrated low to moderate levels of activity with influenza B rates surpassing rates of influenza A in most of the country except in BC and the Atlantic Provinces. Rates of influenza B decreased in the Atlantic Provinces were the highest early on in May (21%) but decreased to 0% by end of the month. Influenza B rates decreased as well in Quebec and Ontario (from 12% to less than 3% positivity) but have remained steady at around 5% in the Prairies. Influenza A rates decreased in Quebec, Ontario and the Prairies with rates from 0.3-1.5% positivity by the end of the month. Unseasonably cool weather in BC is likely causing influenza to persistent in the province.

The World Health Organization (WHO) reports that the 2011-2012 season is ending for most regions in the temperate northern hemisphere. In the 2011-12 season, the United States (US) and Europe, the majority of infections were due to influenza A(H3N2) while Mexico saw mainly influenza A(H1N1)pdm09. Canada generally saw a predominance of influenza B, although there was unequal distribution. Most viruses tested were antigenically closely related to those found in the current trivalent seasonal vaccine at the beginning of the season; by mid-season, there was divergence in the A(H3N2) viruses in the US and Europe. Influenza B virus detections have been both from the Victoria and Yamagata lineages (WHO, 25 May 2012 Update).
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