Epidemiology of foodborne Norovirus outbreaks in Catalonia, Spain

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Abstract

Background:
Noroviruses are one of the foremost biological agents associated with the consumption of contaminated food. The objective of this work was to study the size and epidemiological characteristics of foodborne outbreaks of gastroenteritis in Catalonia, a region in the northeast of Spain. In all reported outbreaks of gastroenteritis associated with food consumption, fecal samples of persons affected were analysed for bacteria, parasites and viruses. Study variables included the setting, the number of people exposed, age, sex, clinical signs and hospital admissions. The study was carried out from October 2004 to October 2005.

Results:
Of the 181 outbreaks reported during the study period, 72 were caused by Salmonella and 30 by norovirus; the incidence rates were 14.5 and 9.9 per 100,000 persons-year, respectively. In 50% of the norovirus outbreaks and 27% of the bacterial outbreaks (p=0.03) the number of persons affected was ≥10; no differences in the attack rates were observed according to the etiology. Hospitalizations were more common (p=0.03) in bacterial outbreaks (8.6%) than in norovirus outbreaks (0.15%). Secondary cases accounted for 4% of cases in norovirus outbreaks compared with 0.3% of cases in bacterial outbreaks (p<0.001)

Conclusions:
Norovirus outbreaks were smaller than bacterial outbreaks, suggesting that underreporting is greater for norovirus outbreaks. Food handlers should receive training on the transmission of infections in diverse situations. Very strict control
measures on handwashing and environmental disinfection should be adopted in closed or partially-closed institutions.

Keywords: Foodborne outbreaks, Norovirus, Public Health
**Background**

Diseases resulting from the consumption of contaminated food cause a considerable disease burden in developed countries [1], and thus it is important to determine their etiology and food vehicles. Although there are difficulties in associating a specific food with the appearance of cases or outbreaks of gastroenteritis [2], reports agree that noroviruses (formerly Norwalk-like viruses) are one of the foremost biological agents involved in cases of gastroenteritis associated with food consumption[3].

The stability of the norovirus in various environmental conditions means that they can remain infectious in frozen and refrigerated food and even food heated to 60ºC for 30 minutes [4], which explains why they can be easily transmitted by foods contaminated by contact with human fecal matter or by unhygienic foodhandling [5].

The infective dose of norovirus is very low: new infections may be produced by person-to-person transmission of very small amounts of virus. Therefore, secondary cases usually appear in foodborne outbreaks vehicled by a single exposure [6].

The available evidence on foodborne gastroenteritis outbreaks due to norovirus is based on national and international public health activities [6,7]. Although most laboratories are equipped to analyse bacterial processes, few are able to make a diagnosis of viral causes of gastroenteritis and therefore confirmation of a possible viral cause of gastroenteritis is not always sought [8].

Analysis of the official statistics provided by different health authorities is frequently
partial and their interpretation is complex[9].

Epidemiological study of foodborne norovirus outbreaks in each community, even knowing that they represent only a part of the real situation due to clinical underreporting and epidemiological and laboratory difficulties, is a necessary step in carrying out interventions at a local level that allow the prevention of the appearance of new outbreaks and improve knowledge on outbreaks and the distribution of specific strains [6,10].

The objective of this study was to determine the size and epidemiological characteristics of foodborne outbreaks due to norovirus in Catalonia between October 2004 and October 2005 and compare them with bacterial outbreaks.

**Material and methods**

We carried out a prospective study of foodborne outbreaks occurring between 15 October 2004 and 30 October 2005 in Catalonia, an autonomous community in the northeast of Spain, with a population of 6.9 million.

A foodborne outbreak was defined as two or more cases with similar symptoms that appeared after consuming the same food which was epidemiologically-confirmed as the disease source.

When an outbreak was reported, an investigation was made to determine the characteristics of the cases and the possible food involved. Likewise, clinical and food samples were collected for laboratory analysis to identify the causal agent. In addition to standard microbiological tests to rule out bacterial and parasitic cause, enzyme linked immunosorbent assay and RT-PCR techniques were carried out on fecal samples of cases and of food handlers when the outbreak was not limited to the family area, to detect the virus.
Stool samples were plated on selective and differential media to study *Salmonella* (MacConkey agar, Salmonella-Shigella agar, Xylose-Lysine-Desoxycholate agar and Selenite enrichment broth), *Shigella* (MacConkey agar and Salmonella-Shigella agar), Shiga toxin-producing strains of O157:H7 *Escherichia coli* (MacConkey agar with sorbitol), *Yersinia* (Cefsulodin-Irgasan-Novobiocin, CIN agar), *Campylobacter* (Charcoal agar), *Vibrio* (Thiosulfate Citrate Bile salt Sucrose, TCBS agar) and *Aeromonas* spp (Pseudomonas-Aeromonas agar with 100,000 IU per litre of Penicillin G, GSP agar).

In outbreaks where a parasitic infection was suspected, the diagnosis was established by direct microscopic examination or after concentration of preserved stool (Merthiolate-iodine-formalin and 10% formalin) to determine the presence of ova, trophozoites or cysts. *Cryptosporidium* oocysts were examined by stained fecal materials (Auramine and Ziehl-Neelsen stains).

Enzyme immunoassay for norovirus genogroups I and II (IDEIA™ norovirus, DakoCytomation), rotavirus group A (IDV Rotavirus-96.Izasa), astrovirus (IDEIA™ Astrovirus, DakoCytomation) and adenovirus serotypes 40 and 41 (IDV Adenovirus-96.Izasa) and RT-PCR were performed. For norovirus, RT-PCR primers designed for partial RNA polymerase region (ORF1) were used: NVp110 (5’- ACD ATY TCA TCA TCA CCA TA – 3’) for RT and JV12 (5’- ATA CCA CTA TGA TGC AGA TTA – 3’) and JV13 (5’- TCA TCA TCA CCA TGA AAA GAC – 3’) for PCR [11]. For rotavirus, the primers used were VP6-3 (5’- GCT TTA AAA CGA AGT CTT CAA C – 3’) and VP6-4 (5’- GGT AAA TTA CCA ATT CCT CCA G – 3’). The primers used for adenovirus were hexAA1885 (5’- GCCGCAGTGGTCTTACATGCACATC-3’) and hexAA1913 (5’-
CAGCACGCGCGGATGTCAAAGT-3’), which amplify a 301 bp fragment within the hexon region of the adenovirus genome [12]. For the genogroup A astrovirus, primer set A1 (5’-CCTGCCCCGAGAAACCAAGC-3’) and A2 (5’-GTAAGATTCAGATTGGTGC-3’) from the hypervariable region of the ORF1a of the genome of astrovirus was used [13] and for the detection of genogroup B astrovirus, primer set A1bis (5’-CCTGCCCCCCGTATAATTAAAC-3’) and A2bis (5’-ATAGGACTCCCATATAGGTGC-3’) [14]. PCR products were detected in a 2% ethidium bromide-stained agarose gel and purified with the QIAquick PCR Purification Kit (QIAGEN, Hilden, Germany).

Norovirus genotyping systems and an automated sequencer (Applied Biosystems) was performed by sequencing the amplimers with the JV12 and JV13 primers using the ABI PRISM Big Dye Terminator Cycle Sequencing kit (Applied Biosystems PRISM 3700) [15]. Likewise, foods suspected of being involved in the outbreak were analysed when some remained.

The variables analysed included the agent, area of the outbreak, date of appearance of the first case, number of people exposed, number of cases, age, sex, symptoms and hospitalization. In non-familial outbreaks, it was determined whether the case was primary or secondary. A secondary case was defined as someone who had not consumed the suspected food [16] and in whom the onset of symptoms occurred after the maximum incubation period of 51 hours [17].

Statistical Analysis:
Differences between medians were compared using the Mann-Whitney U test. Differences between proportions were compared using the χ² test or Fisher’s exact test. The tests were two-tailed. An alpha level = 0.05 was considered statistically
significant. Incidence rates and their 95% confidence intervals (CI) were calculated using the 2005 voter’s list, assuming a Poisson distribution. For the seasonal distribution and calculation of the incidence rates, only outbreaks occurring in a natural year were considered (15 October 2004 - 14 October 2005).

Results

In the study period there were 181 foodborne gastroenteritis outbreaks due to all causes, of which Salmonella (72 outbreaks, 40%) and norovirus (30 outbreaks, 17%) were the most-frequent causes (table 1). Of the 30 foodborne norovirus outbreaks (50% of all norovirus outbreaks), 20 (66.7%) occurred in restaurants, 6 (20%) in families, 2 (6.7%) in residential nursing homes, 1 (3.3%) in a school and 1 in a summer camp (3.3%). Norovirus was identified as the sole agent in one or more samples from cases in 22 outbreaks and there was a mixed etiology in 3 outbreaks (with adenovirus, Salmonella and Vibrio parahaemolyticus, respectively); in the other 5 outbreaks, Kaplan’s criteria were fulfilled. The genotype was determined in 10 outbreaks; 8 were genotype Lordsdale II.4 and 2 were Melksham II.2.

The total number of people affected was 741 in the 30 norovirus outbreaks and 1018 in the 86 bacterial outbreaks. The incidence rates per 100,000 persons-year were 9.9 (95% CI 9.2-10.7) and 14.5 (95% CI 13.6-15.4), respectively.

Although all cases were primary in most of the non-family outbreaks, in 7 norovirus outbreaks a total of 27 secondary cases were recorded (4%); in bacterial outbreaks only 2 secondary cases were detected (0.3%), both in the same outbreak (table 2). The median age of cases was ≤15 years in 10% of the norovirus outbreaks and 6% of the bacterial outbreaks (p=0.4), 16-59 years in 83% and 87 %, respectively.
(p=0.8) and >60 years in 7% in both groups. There were no significant differences between the gender distribution of norovirus outbreaks (51.2% male and 48.8% female) and bacterial outbreaks (52.5% male and 47.5% female), (p=0.6). The most frequent symptoms are shown in table 3.

The size of the outbreaks ranged between 2 and 174 in norovirus outbreaks and between 2 and 123 in bacterial outbreaks. In 50% of the norovirus outbreaks and 27% of the bacterial outbreaks (p=0.03) the number of cases was ≥10. In 70% of the norovirus outbreaks and 74% of the bacterial outbreaks (p=0.81) the attack rate was >50%. Hospitalization occurred in one case in the viral outbreaks (0.15%) and in 87 cases in bacterial outbreaks (8.6%), with the difference being statistically significant (p<0.001) (table 3).

A total of 53% of norovirus outbreaks and 80% of bacterial outbreaks occurred between June and October (p=0.01) (Figure 1).

In the 17 norovirus outbreaks in which the food vehicle was determined by epidemiological analysis, the most common vehicles were fish, and more specifically, bivalve molluscs (8 outbreaks, 26.7%), pastries (3 outbreaks, 10%) and vegetables (2 outbreaks; 6.6%); in the bacterial outbreaks, these figures were 2.3%, 3.5% and 0%, respectively, with only the differences for fish and vegetables being statistically significant (table 4). In 9 of the 30 norovirus outbreaks, laboratory analysis of foods was possible, although the virus was not detected in any outbreak; in 11 of the 86 bacterial outbreaks, the causal agent in the food was confirmed.

Samples from foodhandlers were analysed in 23/30 norovirus outbreaks (76.7%) and one or more samples were positive in 17. The possible involvement of
foodhandlers was detected by investigation in 13 outbreaks (43% ) but was confirmed in only one outbreak by molecular epidemiology. Fecal samples from foodhandlers were analyzed in 30/86 bacterial outbreaks (34.9%) and the epidemiological investigation confirmed the implication of a foodhandler in 7 outbreaks (8.1%)

**Discussion**

The results of this study emphasize the importance of foodborne transmission in gastroenteritis outbreaks due to norovirus. Of the 60 norovirus outbreaks detected in the study period, 30 (50%) were foodborne. This percentage, although high, is lower than that found by Frankauser et al [18] in the United States where, unlike Spain where all outbreaks must be reported, reporting is only obligatory for possible foodborne outbreaks [19].

Swedish [20] and Dutch [21] studies estimate that the proportion of foodborne norovirus outbreaks ranges between 12 and 16 %, while an estimate of 10% has been made for England and Wales [10].

These differences can be explained, at least partly, by the different surveillance systems used. When the basic information comes from samples analyzed in the laboratory [7] it can be more difficult to identify foodborne outbreaks than when information comes from reports received by epidemiological surveillance services, because outbreaks associated with restaurants are more frequently reported to the public health services [2,10].

In this study, norovirus was the second etiologic agent (30 outbreaks) only preceded by Salmonella (72 outbreaks). This is in agreement with other reports [8,22], although some studies have found norovirus to be the first cause of
foodborne outbreaks [3,23-25]. The incidence rate of fooborne norovirus infection was 9.9 per 100,000 persons-year, less than the 15.6 found by Lindqvist et al in Sweden in a study that included all outbreaks detected by a surveillance system enhanced by means of physician visits [25]. In order to evaluate the reporting of norovirus outbreaks in different countries, it should be taken into account that the reasons why people with gastroenteritis consult their doctor and the resulting suspicion of a foodborne outbreak are not homogenous, and therefore it is difficult to determine whether incidence rates really differ or if the apparent differences are due to the ability to detect outbreaks [2,6].

Widdowson et al [22] found that 25% of bacteria-negative outbreaks were not analyzed to detect viral causes, but this type of information is not normally available. In this study, all reported foodborne outbreaks were studied using an identical methodology, searching first for bacterial and parasitic causes and, if these were negative, for viral causes. Therefore, although laboratory analyses were exhaustive for all outbreaks, norovirus outbreaks involved more cases, but less febrile cases and hospitalizations, demonstrating that norovirus outbreaks are less severe than bacterial outbreaks.

Although reported viral outbreaks were larger than bacterial outbreaks, the attack rates were similar, suggesting that smaller viral outbreaks are not reported. Cowden et al [26] in England found that underreporting of norovirus was one hundred times greater than for Salmonella and 30 times greater than for Campylobacter.

In contrast with bacterial outbreaks, no seasonality was observed in norovirus outbreaks. The seasonality of norovirus infections is controversial; some reports
have found no seasonality [18,26], while others have found an increase in winter in outbreaks involving person-to-person transmission [10], but not in foodborne outbreaks [20].

Although not always possible, determining the food implicated in gastroenteritis outbreaks is of great importance for the recommendation of preventive measures. In this study, the food implicated was identified in only 57% of norovirus outbreaks compared with 80% of bacterial outbreaks. In the United States, a 2002 study found levels of 47% and 76%, respectively [23], but in other US studies, the results were similar to ours [22]. With the exception of bivalve molluscs, laboratory techniques to detect norovirus in foods still have a very low sensitivity because the amounts of the virus in foods are very low and because some foods inhibit RT-PCR analysis [24,27].

The low infective dose necessary to produce infection explains why contamination of foods by a foodhandler, with or without clinical symptoms, or even by contact with contaminated surfaces [4,28] easily cause outbreaks. The proportion of norovirus outbreaks in which the involvement of a foodhandler was suspected was 43%, similar to the 48% reported by other authors [22]. However, this involvement was identified by molecular epidemiology in only one case. For this reason, fecal samples should be collected from patients in order to diagnose the outbreak [2] and also from foodhandlers involved in the preparation of the foods consumed in order to demonstrate their involvement. This underlines the need to educate foodhandlers on hygienic practices [3,10,23,29].

It is widely-accepted that foodhandlers should not return to work until 48-72 hours after the disappearance of clinical signs of disease [26,28,29]. However, other
reports suggest extending this period [30,31]. In addition, work absence among foodhandlers due to diarrhea should become the norm [28,29].

Finally, 27 of the 680 (4%) people affected by norovirus were secondary cases, a ten-fold greater proportion than in bacterial outbreaks. Reports of foodborne norovirus outbreaks mention secondary cases [27,31,32], but their frequency is not clear. If a foodborne outbreak is detected when person-to-person transmission has already occurred, this will be erroneously considered the mode of transmission, because they are secondary cases resulting from a point exposure [6,31,33].

In addition, as the incubation period of norovirus infections is very short, even among people exposed to the food vehicle, cases may occur from contact with an infected person and not from the consumption of the food, and therefore the number of secondary cases detected should be considered inferior to the real number. Research into whether cases in foodborne outbreaks are primary or secondary is necessary.

Of the secondary cases detected, most corresponded to residential nursing homes (45%) and schools (10%), where close contact is the norm. It is established that up to 30% of infected people continue to shed the virus for three weeks, even though they have recovered clinically [34]. When an gastroenteritis outbreak of viral etiology is suspected in a residential nursing home or other institution, strict measures with respect to handwashing and disinfection of surfaces should be immediately adopted and compliance checked [35-37].

The main limitations of this study were the small number of samples available to diagnose the outbreak and the passive nature of the reporting on which the study was based.
Although the means for laboratory diagnosis are available, it is not always possible to obtain the minimum four positive samples necessary to attribute the outbreak to norovirus [36,38,39]. In this study we considered that one sample positive for norovirus was sufficient if tests for bacteria and parasites were negative and the clinical signs and epidemiology were compatible [40]. Theoretically, if only one patient tests positive and the other samples are negative or if there is only one sample, the viral etiology may be questioned. However, this is improbable if Kaplan's criteria are considered [40].

The passivity of the surveillance system used during the study period may have resulted in less outbreaks being studied than really occurred. However, it is unlikely that this influenced the comparison between viral and bacterial outbreaks, since once the reports were received by the surveillance teams, the activities carried out were identical. In any case, for the reasons previously stated, it may be considered that underreporting of viral outbreaks was greater than that of bacterial outbreaks. In conclusion, we found that norovirus was the second causal agent of foodborne outbreaks after *Salmonella* and that norovirus outbreaks were larger than bacterial outbreaks, suggesting greater underreporting of norovirus outbreaks. Given that the norovirus has an exclusively human reservoir, a very low infective dose and prolonged persistence in the environment, foodhandlers should receive training on the way they can transmit the infection in different situations [5]. In order to avoid secondary cases, when a foodborne outbreak of viral gastroenteritis in closed or partially-closed institutions is suspected, rapid control measures should be adopted, with a particular emphasis on handwashing and correct disinfection of environmental surfaces [37].
Competing interests: The authors declare that they have no competing interests.

Authors’ contributions: AD and AM carried out the design of the study and drafted the manuscript. NT participated in its design and coordination and helped to draft the manuscript. LR performed the statistical analysis. RB, UP, DF, JB performed the microbiological analysis, IB, NC, JA, RS, PG, SM, AP participated in the acquisition of outbreak data. All authors read and approved the final manuscript.

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Reference List


Figure 1. Monthly distribution of foodborne outbreaks. 15 October 2004 – 14 October 2005
Table 1. Distribution of foodborne outbreaks according to etiology. Catalonia, 15 October 2004- 30 October 2005

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Number of outbreaks (%)</th>
<th>Number of persons affected (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Salmonella</td>
<td>72 (39.8)</td>
<td>605 (29.4)</td>
</tr>
<tr>
<td>Norovirus</td>
<td>30 (16.6)</td>
<td>680 (33.0)</td>
</tr>
<tr>
<td>Other bacteria *</td>
<td>14 (7.7)</td>
<td>413 (20.1)</td>
</tr>
<tr>
<td>Vegetable toxins</td>
<td>16 (8.8)</td>
<td>57 (2.7)</td>
</tr>
<tr>
<td>Other toxic substances</td>
<td>12 (6.6)</td>
<td>39 (1.9)</td>
</tr>
<tr>
<td>Unknown</td>
<td>37 (20.5)</td>
<td>263 (12.8)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>181 (100.0)</strong></td>
<td><strong>2057 (100.0)</strong></td>
</tr>
</tbody>
</table>

* Staphylococcus aureus (6), Clostridium perfringens (6), Campylobacter jejuni (19, Streptococcus pyogenes (1).
Table 2. Distribution of primary and secondary cases in the norovirus and bacterial foodborne outbreaks according to setting.* Catalonia, 15 October 2004- 30 October 2005

<table>
<thead>
<tr>
<th>Setting</th>
<th>Norovirus outbreaks</th>
<th>Bacterial outbreaks</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Primary cases</td>
<td>Secondary cases</td>
</tr>
<tr>
<td>Restaurants</td>
<td>481</td>
<td>6</td>
</tr>
<tr>
<td>Residential nursing homes</td>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td>Hospitals</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Schools</td>
<td>128</td>
<td>14</td>
</tr>
<tr>
<td>Summer camps</td>
<td>38</td>
<td>2</td>
</tr>
<tr>
<td>Cake shops</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>All outbreaks</td>
<td>653</td>
<td>27</td>
</tr>
</tbody>
</table>

Note: Family outbreaks excluded.
Table 3. Clinical characteristics of norovirus and bacterial outbreaks. Catalonia, 15 October 2004- 30 October 2005

<table>
<thead>
<tr>
<th></th>
<th>Norovirus</th>
<th>Bacterial etiology</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nº outbreaks</td>
<td>30</td>
<td>86</td>
<td></td>
</tr>
<tr>
<td>Nº cases</td>
<td>741</td>
<td>1018</td>
<td></td>
</tr>
<tr>
<td>Size of outbreak</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(median and range)</td>
<td>8.5 (2-174)</td>
<td>5.0 (2-123)</td>
<td>0.029a</td>
</tr>
<tr>
<td>Attack rate (median)</td>
<td>0.66</td>
<td>0.70</td>
<td>0.091a</td>
</tr>
<tr>
<td>Abdominal pain (%)</td>
<td>81.5</td>
<td>79.6</td>
<td>&lt; 0.001 b</td>
</tr>
<tr>
<td>Diarrhea (%)</td>
<td>68.5</td>
<td>87.0</td>
<td>&lt; 0.001 b</td>
</tr>
<tr>
<td>Nausea (%)</td>
<td>62.2</td>
<td>41.2</td>
<td>&lt; 0.001 b</td>
</tr>
<tr>
<td>Fever (%)</td>
<td>42.0</td>
<td>52.4</td>
<td>&lt; 0.001 b</td>
</tr>
<tr>
<td>Hospitalizations (%)</td>
<td>0.15</td>
<td>8.6</td>
<td>&lt; 0.001 b</td>
</tr>
</tbody>
</table>

a Mann-Whitney U; b χ²
Table 4. Distribution of foods involved in norovirus and bacterial outbreaks.

Catalonia, 15 October 2004 – 30 October 2005

<table>
<thead>
<tr>
<th></th>
<th>Norovirus outbreaks</th>
<th>Bacterial outbreaks</th>
<th>p value *</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mayonnaise and similar</td>
<td>0</td>
<td>27 (31.4)</td>
<td>0.005</td>
</tr>
<tr>
<td>Other products containing egg</td>
<td>2 (6.7)</td>
<td>19 (22.1)</td>
<td>0.51</td>
</tr>
<tr>
<td>Fish and seafood</td>
<td>8 (26.7)</td>
<td>2 (2.3)</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Meat/sausage</td>
<td>1 (3.3)</td>
<td>6 (7.0)</td>
<td>1.0</td>
</tr>
<tr>
<td>Vegetables</td>
<td>2 (6.6)</td>
<td>0</td>
<td>0.04</td>
</tr>
<tr>
<td>Fowl</td>
<td>0</td>
<td>3 (3.5)</td>
<td>1.0</td>
</tr>
<tr>
<td>Cake shops</td>
<td>3 (10.0)</td>
<td>3 (3.5)</td>
<td>0.06</td>
</tr>
<tr>
<td>Others</td>
<td>1 (3.3)</td>
<td>9 (10.5)</td>
<td>0.67</td>
</tr>
<tr>
<td>Unknown</td>
<td>13 (43.3)</td>
<td>17 (19.8)</td>
<td>0.003</td>
</tr>
</tbody>
</table>

* Fisher test calculated comparing each food with respect to all foods involved in norovirus and bacterial outbreaks.
Figure 1: Bar chart showing the number of norovirus and bacterial outbreaks per month. The x-axis represents the months from January to December, and the y-axis represents the number of outbreaks ranging from 0 to 18. The chart indicates higher incidences in July and August for both types of outbreaks. The legend specifies blue bars for norovirus and light blue bars for bacterial outbreaks.