Sources of Calicivirus Contamination in Foodborne Outbreaks in Denmark, 2005–2011—The Role of the Asymptomatic Food Handler

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Background. Norovirus (NoV) is the predominant cause of foodborne disease outbreaks. Virus contamination may occur during all steps of food processing, from production to preparation and serving. The relative importance of these different routes of contamination is unknown.

Methods. The purpose of this study was to estimate the proportions of outbreaks caused by asymptomatic and symptomatic food handlers (FHs). Reports of foodborne NoV and sapovirus outbreaks (n = 191) that occurred over a 7-year period were extracted, reviewed, and categorized according to the available evidence for source of contamination.

Results. In 64 (34%) of the outbreaks, contamination from FHs took place during preparation or serving of food. In the majority of these outbreaks (n = 41; 64%), the FHs were asymptomatic during food handling. Some had been in contact with ill household members before handling the food and remained asymptomatic; others developed symptoms shortly after or were post-symptomatic. In 51 (27%) of the outbreaks, contamination occurred during production of the food, and in 55 (29%) of the outbreaks, contamination had supposedly occurred after serving a guest at a self-serve buffet.

Conclusions. Guidelines regarding exclusion of FHs where household members suffer from gastroenteritis could limit the number of outbreaks.

Keywords. norovirus; food; food handlers; viral gastroenteritis; foodborne transmission; foodborne outbreaks; *Caliciviridae*; sapovirus.

Norovirus (NoV) and sapovirus (SaV) belong to the family *Caliciviridae* [1, 2]. NoV is a major cause of gastroenteritis globally [3] and is often considered the most frequent cause of foodborne outbreaks in developed countries [4, 5]. SaV is less prevalent [6]. Caliciviruses are single-stranded, nonenveloped RNA viruses that are divided into several genogroups, of which NoV GI, GII, and GIV and SaV GI, GII, GIV, and GV are human pathogens. Based on phylogeny, genogroups can be further subdivided into genotypes [1, 2].

NoV and SaV cause similar symptoms, typically vomiting, diarrhea, and abdominal pain. Other symptoms include nausea, fever, and headache [7]. Malaise, nausea, and abdominal gurgling typically begin 12–24 hours before onset of vomiting or diarrhea [8]. NoV GI and GII have a median incubation time of 1.1 days and 1.2 days, respectively, and SaV has a median incubation time of 1.7 days [9]. Asymptomatic infections have been described

for both SaV [10] and NoV; in England, >5% of asymptomatic adults were infected with NoV [11]. Among community cases of gastroenteritis, 4.7% had mixed infections, primarily involving adenovirus, NoV, and SaV [12].

Caliciviruses are transmitted when virus particles are excreted in stool or vomit. Transmission takes place by direct person-to-person contact or indirectly through aerosols, food, water, or environmental contamination [13–16]. The infectious dose can be as low as 200 NoV particles [17], and more than 10^9 virus particles per gram of stool can be shed during an acute infection [10, 18]. Healthy adults usually excrete NoV for about 10 days [19], but shedding for up to 29 days after clearance of symptoms has been described [20]. NoV particles in the environment can be infectious for several weeks, possibly months [21, 22], and inactivation by the use of alcohol-based disinfectants is difficult [23].

Case reports have shown that asymptomatic and post-symptomatic food handlers (FHs) can cause foodborne calicivirus outbreaks [10, 24–26]. The extent of infections caused by asymptomatically infected persons is unknown [11]. Our goal in this study was to describe the sources of virus contamination in foodborne calicivirus outbreaks reported in Denmark during 2005–2011, focusing on the contribution of FHs, who were asymptomatic during the handling of food.

METHODS

Material

Since 2005, foodborne outbreaks investigated in Denmark have been included in a national database, the Danish Food- and Waterborne Outbreak Database. Information on all calicivirus outbreaks was extracted from this database. The following information was obtained: date of outbreak start, number of illnesses, number of exposed individuals, causative agent, setting of outbreak, suspected vehicle, and evidence for the latter. Additional information about the outbreaks was collected from additional outbreak reports and/or other available written material from the original outbreak investigations (results of analytical epidemiological investigations, contamination source, and evidence of this). Outbreaks were only included if illnesses were related to the consumption of a common food item, were caused by NoV or SaV, and at least 2 persons were affected. The etiologic agent was determined by direct virus detection in the implicated food or in stool samples from at least 1 case-patient or by reported fulfillment of the Kaplan criteria [27]. Thus, outbreaks were excluded if there was uncertainty about the fulfillment of the Kaplan criteria, if person-to-person transmission was found to be the most likely transmission route, or if the outbreaks were waterborne.

Contamination sources were classified into 5 main groups (Table 1), and 1 of these groups was further subdivided into 3 groups, as shown in Figure 1 and Table 2.

Outbreaks classified as being caused by asymptomatic FHs were reviewed by 2 of the authors, who reached the same conclusions regarding the classification.

Using the definitions in Tables 1 and 2, 25 outbreaks fell into more than 1 category. To simplify the presentation and to avoid overestimation of the number of outbreaks caused by

Table 1. Classification of Contamination Sources

Contamination Source	Definition
Asymptomatic food handlers[a]	Food handlers without vomiting or diarrhea during the preparation of food
Symptomatic food handlers	Food handlers who were ill with vomiting or diarrhea during handling of food or symptomatic persons, who were present in the kitchen without handling food; they did not necessarily vomit or have diarrhea while in the kitchen
Production	Contamination of food during production or distribution (ie, before reaching the kitchen for preparation)
Guests	Contamination by guests at self-serving buffets
Unsolved	Unknown contamination source

Based on classification of contamination sources in 191 foodborne calicivirus outbreaks in Denmark from 2005 to 2011.

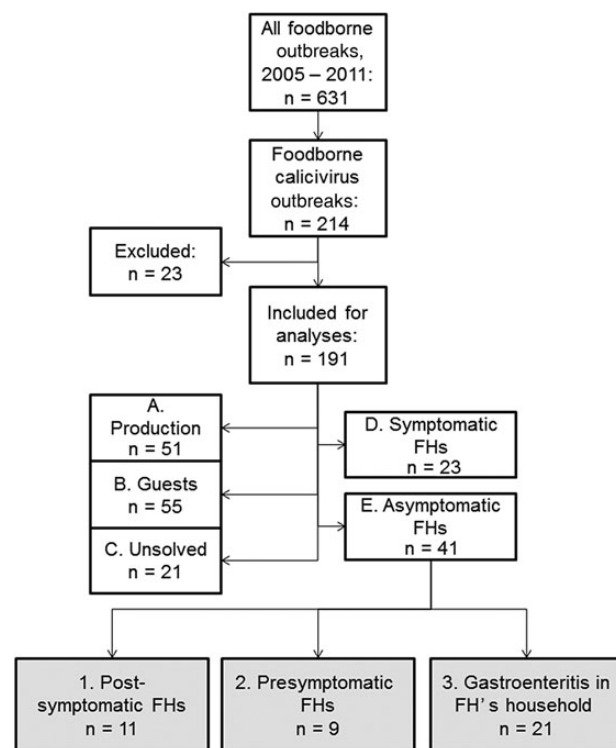[a] Food handlers refer to kitchen staff or serving staff.

Figure 1. Flow diagram for the extraction, inclusion, and classification of foodborne calicivirus outbreaks in Denmark from 2005 to 2011. Abbreviation: FHs, food handlers.

Table 2. Subclassification of Contamination Source in Outbreaks Caused by Asymptomatic Food Handlers

Subclassification	Definition
1. Post-symptomatic food handlers[a]	Food handlers who prepared food within 2 days after last symptom of gastroenteritis
2. Presymptomatic food handlers	Food handlers who did not have symptoms or reported only gastrointestinal discomfort[b] (no vomiting or diarrhea) while preparing the food, but who began to vomit or developed diarrhea within 12 hours after preparation of food
3. Gastroenteritis in food handlers household	Food handlers who did not suffer from vomiting or diarrhea before or after food handling[c], but who had household members with gastroenteritis during or in the days preceding the outbreak

Based on subclassification of contamination sources in 41 foodborne calicivirus outbreaks caused by asymptomatic food handlers in Denmark from 2005 to 2011.

[a] Food handlers refer to kitchen staff or serving staff.

[b] We did not subcategorize "gastrointestinal discomfort without vomiting or diarrhea," as these symptoms had not been systematically recorded.

[c] The length of the symptom-free period had not been systematically recorded.

asymptomatic FHs, the following hierarchical classification was used for these outbreaks:

1. If gastroenteritis symptoms were reported among the guests <12 hours after the suspected meal, the outbreaks were classified as caused by guests.

2. If the kitchen staff or serving staff, their household members, or any other persons in the food-handling area did not report any symptoms of gastroenteritis, the outbreaks were not classified as caused by FHs.

3. If FHs or any other persons present in the food-handling area reported having gastroenteritis symptoms, the outbreaks were classified as caused by symptomatic FHs.

4. Outbreaks classified as being caused by asymptomatic FHs were further subclassified according to the numerical order depicted in Figure 1 and Table 2.

In a sensitivity analysis, an alternative and less conservative hierarchical classification was used for outbreaks that fell into more than 1 category according to the classification described above, as follows: (I) if gastroenteritis was present among the household members of asymptomatic FHs, the outbreaks were classified as caused by this; and (II) the outbreaks were not classified as caused by guests if other contamination sources were possible.

The proportion of asymptomatic FHs was also calculated, when FHs working within 2 days after the last symptom of gastroenteritis were classified as symptomatic.

For case-patients with a recorded Danish personal identification number, vital status was found in the civil registry; if dead, date of death was found in the civil registry. The Danish Data Protection Agency approved the study (2013-54-0503).

Laboratory Analyses

Diagnostic polymerase chain reaction analyses for NoV and SaV in stool samples were performed at several local microbiology laboratories, primarily at Statens Serum Institut, Copenhagen, Denmark, where detection of virus and genotyping were performed as previously described [28, 29]. For 1 NoV outbreak, supplementary sequencing of the hypervariable P2 part of the capsid gene was performed. For a description of primers, cycling conditions, sequencing, analysis of the P2 region, and testing of NoV in food, see Online Technical Supplement.

RESULTS

During 2005–2011, 214 foodborne outbreaks were recorded as caused by caliciviruses; 191 outbreaks (189 NoV and 2 SaV) fulfilled the inclusion criteria. This corresponds to 5.7 reported foodborne calicivirus outbreaks per 1 000 000 inhabitants per year. The outbreaks comprised 6991 recorded illnesses. The median number of exposed individuals was 45 (range, 2–1139; 25 and 75 percentiles, 2–80), and the median attack rate was 55% (range, 5%–100%; 25 and 75 percentiles, 38%–71%), excluding 4 outbreaks with an unknown number of exposed individuals. A personal identification number was available for 490 patients, among whom 4 were found to have died within 1 month after the stool sampling date. Eighteen of the outbreaks have been described elsewhere [30–33].

A total of 94 (49%) outbreaks were laboratory confirmed by detection of calicivirus in patients' samples (n = 74 outbreaks; 39%), food (n = 4 outbreaks; 2%), or both (n = 16 outbreaks, 8%). Of these, 51 (54%), 21 (22%), and 2 (2%) were caused by NoV GII, NoV GI, and SaV, respectively. Combinations of these viruses were detected in 13 (14%) outbreaks as follows: NoV GI and GII (n = 11 outbreaks); NoV GI, GII, and GIV (n = 1 outbreak); and NoV GII and SaV (n = 1 outbreak). Information on NoV genogroup was not available for 7 (8%) outbreaks. Food items were not tested for SaV. For the remaining 97 (51%) outbreaks, calicivirus as the causative agent was based on the Kaplan criteria.

Table 3 shows the number of outbreaks and illnesses with respect to classification of contamination source and the evidence of foodborne transmission. Foodborne transmission was confirmed by the detection of NoV in food in 20 (11%) outbreaks. Identical partial NoV capsid and/or polymerase sequences were detected in samples from FHs and at least 1 case-patient in 10 (5%) outbreaks and further confirmed through sequence comparison of the P2 region in 1 of the 10 outbreaks. In 22 (12%) outbreaks, analytical epidemiologic investigations revealed a statistical association between illness and suspected food items. In the remaining 139 (73%) outbreaks, descriptive

Table 3. Foodborne Transmission With Respect to Suspected Contamination Source

Evidence of Foodborne Transmission	Contamination Source and Number of Outbreaks (Number of Illnesses)					
	Production	Symptomatic FH	Asymptomatic FH	Guest	Unsolved	Total
Norovirus detected in food	20 (1277)					20 (1277)
Identical viral sequences[a]		5 (341)	4 (298)	1 (14)		10 (653)
Analytical epidemiology	10 (209)	4 (398)	5 (543)	1 (21)	2 (22)	22 (1193)
Descriptive epidemiology	21 (422)	14 (368)	32 (1012)	43 (1553)	19 (513)	139 (3868)
Total	51 (1908)	23 (1107)	41 (1853)	55 (1588)	21 (535)	191 (6991)

Based on evidence of foodborne transmission with respect to suspected contamination source in 191 foodborne calicivirus outbreaks in Denmark from 2005 to 2011.

Abbreviation: FH, food handler.

[a] Samples from the FH or FH's family members and from case-patients tested positive for norovirus with identical viral sequences (part of the capsid gene).

epidemiology was the only evidence that food was the vehicle. Examples of the outbreaks that were analyzed in this study and the type of evidence available are presented in Figure 2.

FHs caused 64 (34%) of the 191 outbreaks; in the majority of these cases, the FHs (n = 41) were classified as asymptomatic during the food preparation period (Table 3). Among the asymptomatic FHs, 21 (11% of all reported outbreaks) had household members with gastroenteritis. In these outbreaks, the FHs reported no symptoms of gastroenteritis either before or after the preparation of food. The classification by contamination source in
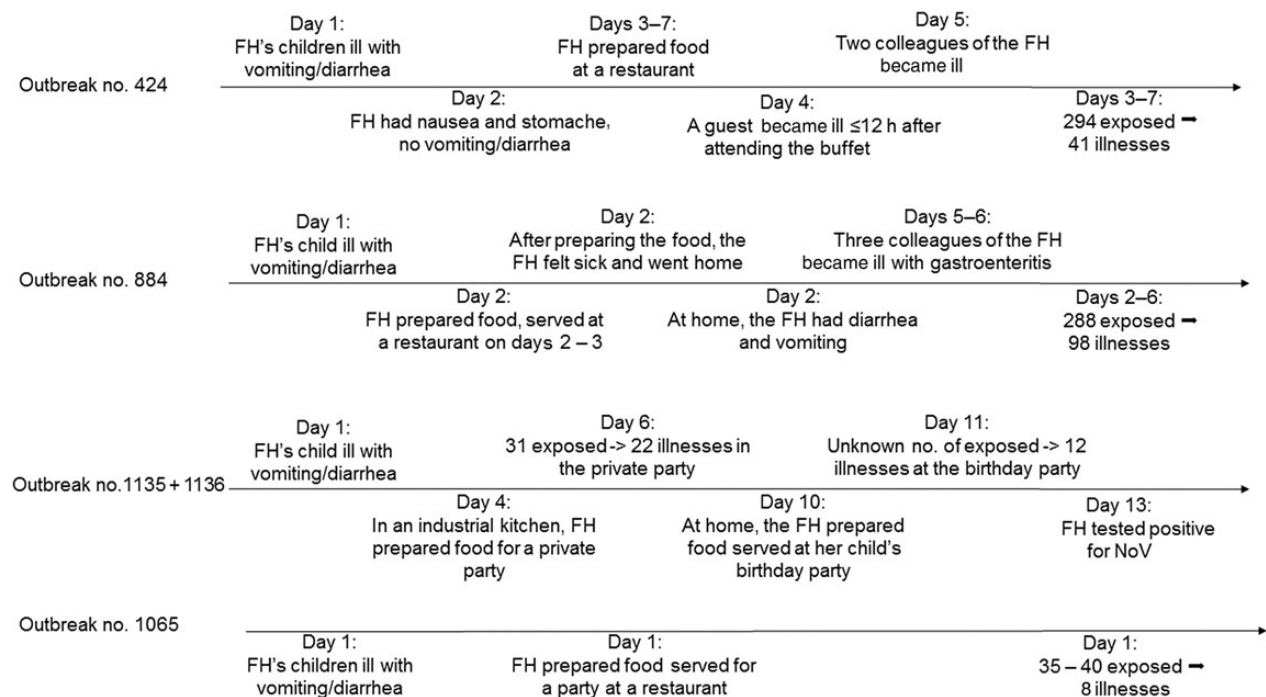

Figure 2. Description of 5 norovirus (NoV) outbreaks caused by asymptomatic food handlers (FHs) in Denmark from 2005 to 2011. Outbreak 424: The FH's child, the FH, 2 FH colleagues, and 5 case-patients tested positive for NoV GII.P7_II.7. Identical P2 sequences were detected in 4 case-patients and the FH. Hypothesis: The FH was infected with NoV with only gastrointestinal discomfort and was post-symptomatically shedding infectious NoV. Outbreak 884: The FH's child, a FH colleague, and 5 case-patients tested positive for NoV GII.Pe. The child and a case-patient also tested positive for GII.4 Osaka_2007 with identical region C sequences. Hypothesis: The FH was presymptomatically shedding infectious NoV. Outbreaks 1135 + 1136: The FH had no signs of gastroenteritis, either before or after food handling. The 2 outbreaks were diagnosed as NoV outbreaks by the use of the Kaplan criteria. Hypothesis: The FH caused the outbreaks by asymptomatic shedding of infectious NoV. Outbreak 1065: The FH had no signs of gastroenteritis, either before or after food handling. One case-patient and 1 of the FH's children tested positive for NoV GII.P7_II.6 with identical region C sequences. Hypothesis: The FH had transferred infectious NoV from the child to the food, or the FH had been asymptomatically infected with NoV.

Table 4. Classification of Contamination Source and Evidence of This

Contamination Source	Molecular Biological Evidence[a]	Descriptive Evidence: Number of Outbreaks (Number of Illnesses)		Total
		Gastroenteritis in Several Parties Receiving Food From the Same Kitchen	Only One Party Received Food From the Same Kitchen	
Symptomatic food handlers	5 (341)	7 (328)	11 (438)	23 (1107)
Asymptomatic food handlers				
Post-symptomatic	1 (141)	5 (415)	5 (101)	11 (657)
Presymptomatic	1 (115)	3 (152)	5 (129)	9 (396)
Household member with gastroenteritis	2 (42)	10 (467)	9 (291)	21 (800)
Total	9 (639)	25 (1362)	30 (959)	64 (2960)

Based on classification of contamination source and evidence of this in 64 foodborne calicivirus outbreaks caused by food handlers in Denmark from 2005 to 2011.

[a] Virus with identical sequences in part of the capsid gene was detected in samples from the food handler or food handler's family member and minimum of 1 case-patient.

food-handling–related outbreaks is presented in Table 4. In 6 of the 9 outbreaks classified as caused by presymptomatic FHs, the FHs had been interviewed personally by 1 of the authors. In 3 of these outbreaks, the FHs reported being completely well during the handling of food; 2 of these FHs became ill with gastroenteritis after the preparation of food and 1 was suspected to have given an incoherent statement. In 2 outbreaks, the FHs did not feel well while preparing the food and became ill with vomiting or diarrhea afterward; in 1 outbreak, the suspected source of contamination was a child who became ill with vomiting a few hours after playing in a private kitchen. In the remaining 3 outbreaks classified as caused by presymptomatic FHs due to the report of gastroenteritis symptoms after the preparation of food, we do not know if the FHs had been questioned regarding symptoms other than vomiting and diarrhea. In 15 of the 64 (23%) food-handling–related outbreaks, stool samples from the FHs or their household members had been tested. In 9 of these outbreaks, identical virus sequences were detected in FHs or members of their household and a case-patient.

Two outbreaks were not classified according to the algorithm described in the "Methods" section. One outbreak (18 illnesses) was classified as caused by a presymptomatic FH, since presumably its source was a child present in the food-handling area who began vomiting a few hours later. The other outbreak (141 illnesses) was classified as caused by a post-symptomatic FH (Figure 2; outbreak 424), even though a guest became ill <12 hours after attending the buffet. However, the FH had experienced nausea and stomachache the day before the preparation of food, and identical sequences (848 nt capsid gene including the entire P2 region) were identified in stools from the FH and a case-patient.

Using the alternative hierarchical outbreak classification, FHs caused 72 (38%) of the outbreaks, and 48 (67%) of them were asymptomatic. Among the asymptomatic FHs, 29 (60%) had household members with gastroenteritis. Guests contaminating

ingested food were the cause of 47 (25%) outbreaks. The numbers of outbreaks caused by contamination during food production or with unsolved contamination source did not change. When post-symptomatic FHs were classified as symptomatic, 30 (42%) food-handling–related outbreaks were caused by asymptomatic FHs.

The settings and implicated food items of the 64 food-handling–related outbreaks are shown in Figure 3. In 83%
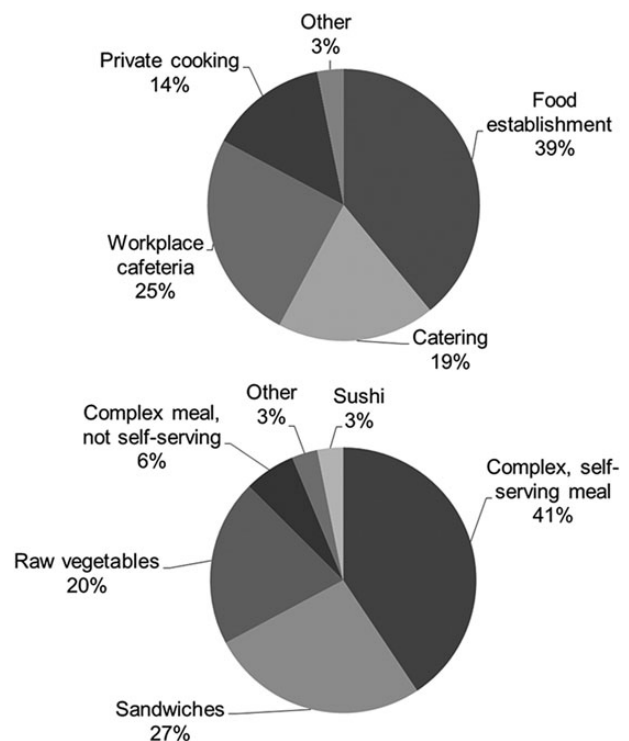

Figure 3. Setting (upper panel) and vehicle (lower panel) of 64 foodborne outbreaks of calicivirus caused by food handlers in Denmark from 2005 to 2011.

(53/64) of the outbreaks, the food was prepared by commercial food establishments, workplace cafeterias, or catering companies. In 14% (9/64) of the outbreaks, the food was prepared in private kitchens. Food that was prepared and served cold (sandwiches, raw vegetables, sushi, and layer cakes) accounted for 52% (33/64) of the contaminated food items. In the 51 outbreaks caused by contamination during production, the implicated food items were frozen raspberries (n = 24, 47%), lettuce (n = 22, 43%), and oysters (n = 5, 10%). Excluding outbreaks caused by guests contaminating self-serve buffets, 47% (64/136) of reported outbreaks were caused by FHs.

DISCUSSION

In this overview of 191 foodborne calicivirus outbreaks in Denmark over a 7-year period, we conclude that almost a quarter of the outbreaks, affecting 1853 persons, were caused by asymptomatic FHs. In 4 of these outbreaks, identical viral sequences were detected in samples from the case-patients and in samples from the FHs or a household member; this is strong support for the suggested contamination source. The outbreaks caused by asymptomatic FHs could be further classified into the following 3 subgroups: (i) outbreaks caused by FHs preparing food within 48 hours after being ill, (ii) outbreaks caused by FHs who developed illness after food preparation or who had gastrointestinal discomfort other than vomiting or diarrhea, and (iii) outbreaks caused by FHs with household members suffering from gastroenteritis.

It has been noted that asymptomatic shedding of NoV particles can occur [11]. In a study by Sukhrie et al, asymptomatic patients were contagious although less likely to contribute to an outbreak than symptomatic patients [34]. Transfer of infectious NoV via environmental contamination is also well documented. NoV can be infectious for at least 5 days in the environment [13] and in water, NoV remained infectious for at least 61 days and detectable for more than 3 years [21].

Outbreaks suggesting both post-symptomatic [24] and asymptomatic transmission of NoV from infected FHs have been described [25, 26]. Molecular biological investigations confirmed the contamination sources in these outbreaks. In 11% of the reported outbreaks, we suspected that household members of the FHs were the contamination sources via asymptomatic infection of the FHs or mechanical transmission of NoV particles from the household members via the FH's hands or clothing. This is similar to the experience in Minnesota from 1981 to 1998, where FHs denied illness in 21 (18%) foodborne NoV outbreaks but reported illnesses among their household members [35]. Daniels et al reported an outbreak that could have been caused by a FH's child, who contaminated the hands and clothing of the FH. Thereafter, the FH involuntarily transferred NoV from the child to the food [36]. Identical NoV sequences were detected in samples from the child and 9 case-patients, and the FH claimed to be asymptomatic. However, the authors doubted this scenario because the FH chose to resign her job rather than submit a stool sample for NoV testing. No other documentation for mechanical transmission of NoV from an infected household member has been published to date. However, since deposited NoV particles can remain infectious for long periods of time [13, 21, 22], mechanical transmission from sick household members to food via the FH's hands or clothes can occur.

In 2 of the outbreaks that we classified as caused by presymptomatic FHs, we know with certainty that the FHs had been questioned about symptoms other than vomiting and diarrhea; both FHs reported feeling completely well during food handling. Other similar outbreaks have not been published to date. Vivancos et al described 2 NoV outbreaks in which a FH felt mildly dizzy during food preparation and developed diarrhea a few hours later [37]. In another outbreak, described by Zomer et al, a FH vomited 2 hours after food preparation; however, it was not known if the FH had suffered from nausea, for example, while handling the food [38]. In a challenge study by Atmar et al, some participants shed NoV in stool 3–14 hours before having clinical symptoms [39], and this has also been observed in a patient during a NoV outbreak in an aged-care facility [40]. Thus, there is evidence of calicivirus-infected persons being contagious for several hours before onset of symptoms.

We found 5.7 foodborne NoV outbreaks per 1 000 000 inhabitants yearly in Denmark, which is higher than the reported 1.2 outbreaks/1 000 000 person-years in the United States [15]. FHs were the suspected source in 53% of the US outbreaks, but the state-to-state variation in the number of reported outbreaks was substantial. Differences in reporting practice, both at the consumer level and among health inspectors, may be a factor influencing the number and type of reported outbreaks. An example of this is outbreaks caused by guests contaminating self-serve buffets, which is a type of outbreak that has been included in our study and classified as foodborne. In a review of published foodborne outbreaks performed by Mathijs et al [41], none of the foodborne outbreaks were caused by guests contaminating food at self-serve buffets. Thus, presumably these sorts of outbreaks were not classified as foodborne in the study by Hall et al [15]. This is understandable, as it may be difficult to distinguish foodborne person-to-person transmission in these types of outbreaks. Excluding the 55 outbreaks caused by guests after serving, 47% of outbreaks presented in our study were caused by FHs.

Many of the outbreaks covered in this review were, for various reasons, not investigated to the point where no doubt remained concerning the mechanism of transmission. In many instances, important information that formed the basis of our conclusions regarding the outbreak vehicles came from statements obtained from FHs. Doubt may remain as to the validity of these statements. There are, however, a number of reasons

why we believe that the conclusions we reached using our classification method are a reflection of the outbreak mechanisms that actually occurred. Following is a discussion of these reasons: (I) In contrast to previously published individual outbreak reports, the information presented here is substantial and suggests a trend; while some outbreaks may have been misclassified, this is unlikely to have happened at the general level. (II) The reports covered are not primarily based on second-hand information, as most outbreaks were investigated in the Copenhagen area by 1 of the authors. If the authors were uncertain as to whether the FHs could be trusted, this was noted and taken into consideration when classifying the outbreak according to contamination source. (III) Persons employed in food businesses in Denmark receive either wages or a financial refund during short-term sick leave. FHs therefore normally do not have a financial incentive to go to work if they are ill with gastrointestinal symptoms. (IV) An effort was made in the classification phase not to overestimate the number of outbreaks caused by FHs. If illness was reported among the guests ≤12 hours after the meal of suspected exposure, the outbreaks were not classified as caused by FHs.

We found that 49% of the presented outbreaks were laboratory confirmed, while stool samples from the FHs were available in 23% of the food-handling–related outbreaks. Incomplete diagnostics in calicivirus outbreaks is a frequently encountered problem; 58% of reported foodborne NoV outbreaks in the United States from 2001 to 2008 were laboratory confirmed [15]. When laboratory testing was unavailable, the Kaplan criteria can often be used [27]; these evaluations have been shown to effectively distinguish NoV outbreaks from outbreaks caused by bacteria [42]. In our study, laboratory testing of samples from a larger proportion of the presented outbreaks would have resulted in more certain conclusions regarding contamination sources. There are several explanations for the low number of stool samples submitted for diagnostic testing in foodborne outbreaks. For example, because NoV infections are usually self-limiting and of short duration, patients rarely seek medical assistance and, even if they do, their physicians may choose not to request a test for NoV.

Healthy adults excrete NoV for about 10 days [5]. In 2 of the outbreaks with unknown contamination source, the involved FHs had gastroenteritis symptoms >48 hours before food handling (data not presented). As FHs have not been systematically questioned about symptoms >48 hours before food handling, other outbreaks classified as caused by unknown contamination sources may have been caused by post-symptomatic FHs. Due to the very thorough interviews, we consider it unlikely that outbreaks classified as caused by asymptomatic FHs with gastroenteritis among household members were actually caused by post-symptomatic FHs with long-term shedding of infectious NoV.

Existing guidelines recommend exclusion of symptomatic and post-symptomatic FHs and strict hand hygiene when household members of FHs are ill with gastroenteritis [43]. It appears that guidelines are not always followed in Denmark, and published reports suggest that this is true in other developed countries as well. Furthermore, existing guidelines may not have been written bearing in mind the mechanisms of transmission of NoV described here. Symptoms compatible with NoV infection among household members, especially children, of FHs should be taken into account, as mechanical transfer of virus particles from private homes to industrial kitchens appears to be an important cause of outbreaks. Shedding by pre- or nonsymptomatic FHs may, however, be difficult to prevent as no warning signs are available. This makes general hygienic precautions such as thorough hand hygiene and use of gloves and protective clothing in kitchen areas very important. Exclusion of FHs with gastroenteritis among household members could be considered. Cost–benefit analyses that consider local settings would be among the tools that could clarify the value hereof. If FHs develop gastroenteritis after food preparation, instruction for swift cleaning of kitchen environments and disposal of possibly contaminated food could also have effect.

In conclusion, this review of a large number of investigated foodborne NoV outbreaks clearly shows that FHs are responsible for a huge burden of illnesses through outbreaks. Furthermore, it shows that transmission by FHs who are asymptomatic while preparing the food constitutes a substantial number of these outbreaks. This has important implications for advice on public health and regulation of food-handling advice.

Supplementary Data

Supplementary materials are available at *The Journal of Infectious Diseases* online (http://jid.oxfordjournals.org/). Supplementary materials consist of data provided by the author that are published to benefit the reader. The posted materials are not copyedited. The contents of all supplementary data are the sole responsibility of the authors. Questions or messages regarding errors should be addressed to the author.

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