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A norovirus outbreak associated with environmental contamination at a hotel

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SUMMARY

In December 2006, an outbreak of gastroenteritis occurred involving 372 guests and 72 employees at a hotel after a guest vomited in corridors on the third (F3) and 25th (F25) floors. Norovirus with identical genotype was confirmed by real-time reverse transcription–polymerase chain reaction in faecal samples from guest cases and employees. Spread of the outbreak on F25 was compared with that on F3. The attack rate in the guests who visited F25 alone (15·0%, 106/708 guests) was significantly higher than in those who visited F3 alone (3·5%, 163/4710 guests) (relative risk 4·3, 95% confidence interval 3·4–5·5, $P<0·001$). The outbreak on F3 ended within 2 days, while that on F25 extended over 7 days. The environmental ratios of F3 to F25 were 7·4 for volume, 6·9 for floor area and 7·6 for ventilation rate. This outbreak suggests that environmental differences can affect the propagation and persistence of a norovirus outbreak following environmental contamination.

Key words: Environmental management, gastroenteritis, infectious disease control, infectious disease epidemiology, Norwalk agent and related viruses.

INTRODUCTION

Noroviruses are major causes of outbreaks of viral gastroenteritis. They are transmitted primarily through the faecal–oral route, either by direct person-to-person spread or by consumption of contaminated food or water [1–4]. In a closed environment they may be spread by airborne viral particles originating from vomitus [5–12]. A review of the attack rates (AR) in relation to the location of vomit and vomit-contaminated formites suggests that inhaling and swallowing dust from desiccated vomit and faeces are important factors [12]. Noroviruses have been detected in environmental swabs by reverse transcription–polymerase chain reaction (RT–PCR) [13, 14]. This suggests that environmental contamination is a major source of norovirus outbreaks in closed or semi-closed settings [13–17]. However, it is not sufficiently known how the viruses spread in buildings or what affects the development of an outbreak.

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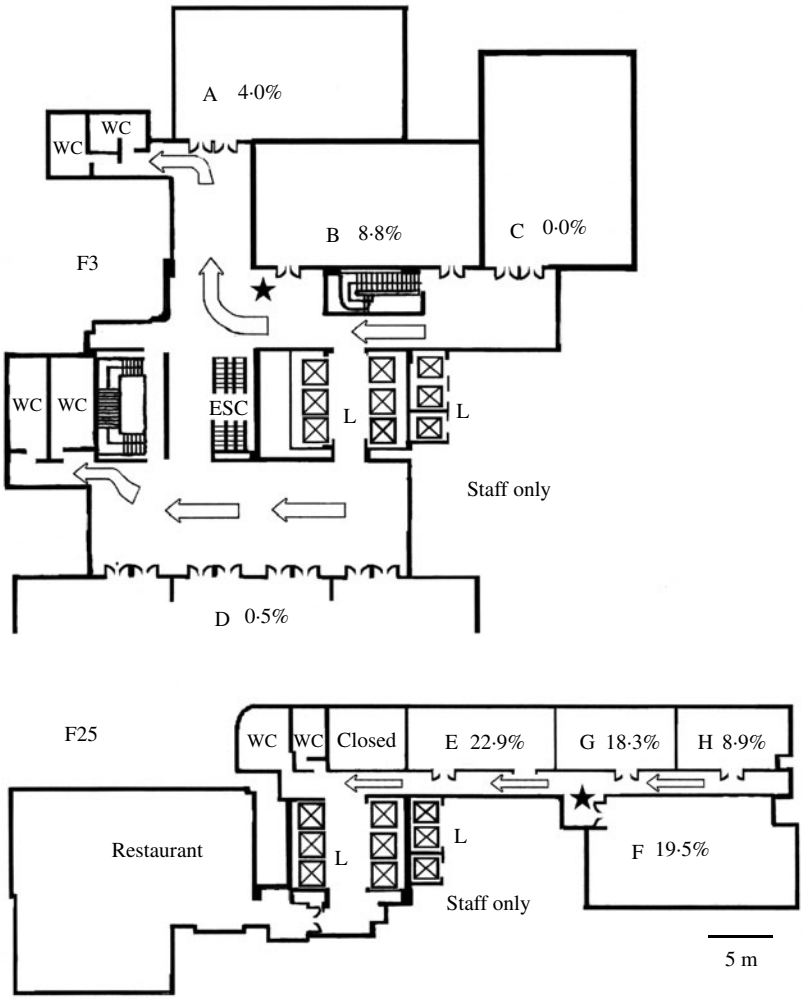

Fig. 1. Relationship between the spatial distribution of gastroenteritis in the hotel guests and the vomit locations on the two different floors at a hotel. L, Lift; ESC, escalator; WC (water closet/toilet). The black stars indicate the locations on the third floor (F3) and the 25th floor (F25) where a guest vomited before mid-day on 2 December 2006. The figures indicate the attack rate in the guests who visited the hall between 2 and 10 December. 'Staff only' areas are marked. Arrows indicate the approximate air current produced by the air-conditioning system. The air supplies were installed on the ceiling board of the corridor, and the air vents of the air conditioners were installed in the toilets of each floor.

We encountered an outbreak of norovirus in a hotel that occurred following vomiting incidents by a guest on two floors with different environments. This outbreak is an example of how environmental factors, such as space or ventilation, can affect the propagation and persistence of norovirus outbreaks following environmental contamination.

METHODS

Epidemiological investigation

Description of the outbreak

On 5 December 2006, a hotel notified the Public Health Centre that several party guests had complained of developing nausea, vomiting and diarrhoea 1–2 days following their visit to the hotel. Disinfection of restaurants, kitchens, and toilets in the staff area was performed on and after 5 December. The main kitchen on floor 3 (F3) and the restaurant on floor 25 (F25) were closed between 6 and 13 December. On 7 December, we obtained some information from various employees. Before mid-day on 2 December a female guest (index case) had vomited in the corridor in front of hall B (waiting room) on F3 (Fig. 1). She then went to a wedding reception on F25 and vomited again in the corridor near to the entrance of hall F. A waiter on F25 (employee B) attended to her and cleaned up the vomit using table napkins, which he discarded in a

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waste bin within the staff area on F25. The waste was incinerated that night. The guest used toilets on both the floors after vomiting and left the hotel without returning to the reception. A cleaner (employee A) quickly removed the carpet stains on F3 and then F25 using a brush and soapsuds while wearing rubber gloves. On 7 December, we conducted a thorough disinfection of the environment including the vomit locations and toilets for guests. The last date of onset among the guests was 10 December, then the outbreak ended.

Environmental investigation and meal service in the hotel

This hotel had 25 floors. Floors 2, 3, 4 and 25 contained halls for receptions. Halls on the two floors were often jointly utilized for one wedding reception. Floors 5 to 24 contained 815 bedrooms. Floor 3 had lifts, an escalator to the ground floor (F1) and a staircase to floor 4 (F4), but F25 had only lifts except for an emergency staircase. All the floors of the corridors and halls were covered with carpet. The corridor on each floor was cleaned every night using vacuum cleaners that were also used in the halls. Separate toilets and lifts were provided for guests and employees. Toilet facilities for guests had automatic taps and the entrance did not have a door. The corridors and halls were separately ventilated. The volume of the corridor on F3 was 2128 m^3 (floor area 733·7 m^2, ceiling height 2·9 m, width 7·2 m) and that on F25 was 288 m^3 (floor area 106·7 m^2, ceiling height 2·7 m, width 2 m). The corridor of F3 had eight air outlets within the toilets and 20 air inlets on the ceiling board, whereas the corridor of F25 had three air outlets and one air inlet. Ventilation volumes of F3 corridor were 11 860 $m^3\,h^{-1}$ and, of F25, 210 $m^3\,h^{-1}$. The ventilation rate, i.e. the ratio of the air volume entering the room per hour to the room volume, equalled the exhaust airflow (the ventilation volume) divided by the room volume [18]. The ventilation rate for the corridor of F3 was 5·6 h^{-1} and that of F25 was 0·7 h^{-1}. The ratios of F3 to F25 were 7·4 for volume, 6·9 for floor area, and 7·6 for ventilation rate.

All the meals for parties at halls on floors 2, 3, 4 and 25 were prepared within the main kitchen on F3 and supplied to each floor via lifts used only by staff. Restaurants on F1, floor 2 (F2) and F25 prepared meals for their own guests. A cafeteria for staff was in the basement (B1).

Hotel guests

The hotel reported the number of guests who complained of gastrointestinal symptoms, the date of their visit to the hotel, and the floor number and the hall or restaurant that they used. Any guest who developed acute gastroenteritis (vomiting and/or diarrhoea) within 1–3 days of their visit to the hotel between 2 and 10 December 2006 was defined as a guest case. Lists of the party schedules, including the number of participants, the hall used, the timetable and the menus were sought from the hotel. Many guests who attended the wedding reception or year-end party did not stay overnight. For guest cases, we administered a questionnaire, including details of age, sex, food history and onset time and duration of symptoms. We failed to ask them which toilets they had used and the routes they had taken within the hotel.

To determine the source of infection, party guests were classified into two groups: those with vomiting who had accessed F3 and/or F25, and those without vomiting who had accessed F2 and/or F4. To study the environmental factors that might have affected the extent and progress of the outbreak, the guests who had accessed F3 and/or F25 were classified into three groups (Table 1): those who had accessed F3 alone but not F25; those who had accessed F25 alone but not F3, and those (including the index case) who had accessed both floors. The attack rate (AR) in the guests was calculated for each group. Furthermore, the AR in guests who had accessed each hall was also calculated (Fig. 1). The guests who visited hall E on F25 included those from hall D on F3. The guests who visited hall F on F25 included those from halls A or B on F3 (Fig. 1, Table 1). To address the possible date of exposure to virus to the floor, we used the date of visit to the hotel (not the onset) as x axes in the epidemic curves (Figs 2, 3).

Hotel employees

The hotel provided us with the number of employees who had worked between 2 and 10 December 2006, details of their sections and whether or not they had become ill. Those who had worked during this period and developed nausea, diarrhoea and vomiting were defined as employee cases. The hotel also reported whether these cases had consumed any meals from the staff cafeteria.

Table 1. *Attack rate and relative risk on the different floors with a vomiting incident between 2 and 10 December 2006*

Guests visited floor	F3 alone				Both F3 & F25		F25 alone	
Ill guests/total guests	163/4710				82/267		106/708	
Attack rate (%)	3·5				30·7*		15·0*	
Relative risk (95% CI)	1				8·9 (7·0–11·2)		4·3 (3·4–5·5)	
Guests visited hall	A	B	C	D	E†	F†	G	H
Ill guests/total guests	18/446	134/1530	0/536	11/2198	70/306	83/426	26/142	9/101
Attack rate (%)	4·0	8·8	0·0	0·5	22·9	19·5	18·3	8·9
Relative risk (95% CI)	1 (1·3–3·5)	2·2 (–)	0·0 (0·06–0·26)	0·12	5·7 (3·4–9·3)	4·8 (3·0–7·9)	4·5 (2·6–8·0)	2·6 (1·4–4·9)

CI, Confidence interval.
* Significantly different from the attack rate in the guests who visited floor 3 (F3) alone ($P<0·001$, χ^2 test).
† The hall includes the guests who visited both floors 3 and 25 (Both F3 & F25). Of the 30 guests who visited both halls D and E, eight became ill. Of the 237 guests who visited halls A or B and hall F, 74 became ill.

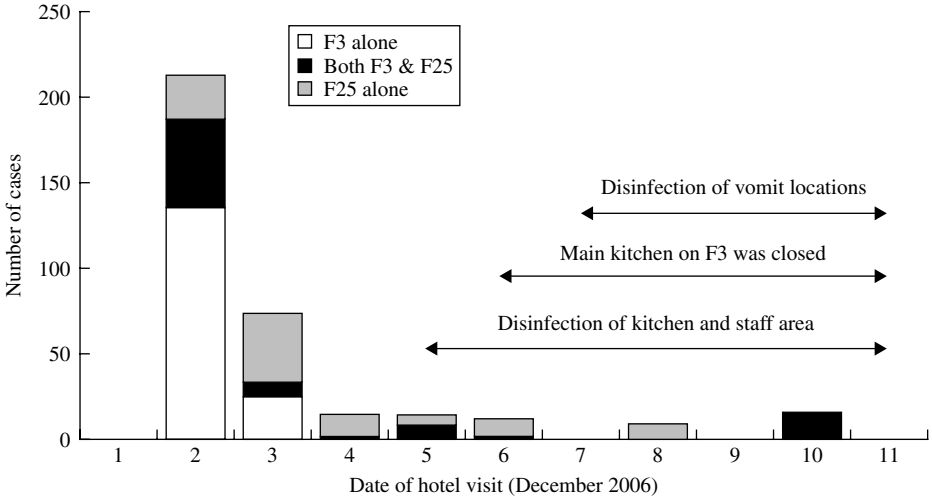

Fig. 2. Epidemic curves for the number of the gastroenteritis cases among the party guests who visited the hotel between 2 and 10 December 2006.

Laboratory investigations

Stool specimens from 92/372 guest cases and from 98 employees (including 85 without symptoms) were examined for 11 bacteria, including *Salmonella* and *Shigella* and norovirus. Of the 98 employees, 79 were food handlers, 18 were waiters and one was a cleaner (Table 2). Of the 79 food handlers, 60 worked in the main kitchen, 10 in the restaurant on F25, and nine in the staff cafeteria. Environmental swabs from 44 positions in the main kitchen and 27 samples from the residual food served at parties on 2 and 3 December 2006 were examined for bacteria. Eight cold samples, including sliced raw fish and sweets, were examined for norovirus. However, we did not collect environmental swabs [13–17] for norovirus from the corridor area with a vomiting incident (carpets, female toilets and lift buttons) or from the dust of the vacuum

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Table 2. *Results of the real time-PCR performed on the 98 employees*

		Positive*		Negative		
PCR	Clinical symptoms …	Positive	Negative	Positive	Negative	Total
Food handlers (AR 5·1%)						
Party foods (main kitchen)		0	0	3	57	60
Restaurant (F25) for guests		4	0	0	6	10
Cafeteria (B1) for stuff		0	0	1	8	9
Waiters (AR 50·0%)		5	4	2	7	18
Cleaner		0	0	0	1	1
Total		9†	4	6†‡	79	98

PCR, Polymerase chain reaction; AR, attack rate.
* Onsets of clinical symptoms of employees were between 3 and 10 December 2006.
† 100% homologous to the pilot strain among the guest cases on F3.
‡ The rate of asymptomatic infection was estimated to be 31·6% (6/19).

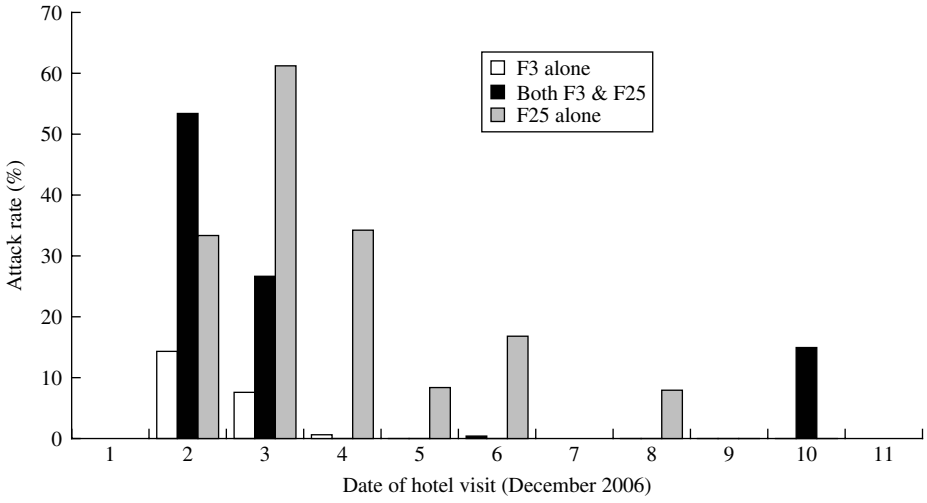

Fig. 3. Attack rate in the party guests who visited different floors with a vomiting incident between 2 and 10 December 2006.

cleaners, because we were technically unable to detect norovirus in environmental swabs. To detect noroviruses from faecal and food samples, a real-time RT–PCR was performed according to the slightly modified protocol of Kageyama *et al.* [19, 20].

The stool specimens from 61/92 guest cases and from all the 98 employees were examined at the Division of Virology, Tokyo Metropolitan Institute of Public Health. The remaining stool specimens from 31 guest cases were examined at other institutes. The employee who tested positive for norovirus but had no clinical symptoms was defined as an asymptomatic employee.

A sequence analysis of the conserved capsid domain (G2SKF/G2SKR) of genogroup II norovirus (GII NV) was further performed on all 59 GII NV-positive specimens (from 44 guest cases and 15 employees). Cycle sequencing was performed with PCR products using a thermal cycler (GeneAmp PCR system 9700) and BigDye Terminator v. 1.1 Cycle Sequencing kit (Applied Biosystems, USA), and the sequences were determined with the ABI Prism 3130 Genetic Analyser. The first GII NV-positive specimen was used as a pilot strain for homology test because no material was obtained from the index case. Phylogenetical analysis of the virus was performed with a method reported by Katayama *et al.* [21]. Sequencing of a hypervariable region encoding the P2 domain [22] was tried on one GII NV-positive specimen.

Statistical analysis

Relative risks (RR) with 95% confidence intervals (CI) were calculated for the AR in the guests who had visited F25 compared to those who had visited F3

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alone, and for the AR in each hall compared to hall A. χ^2 tests were performed on numbers of ill or not ill guests on each floor.

RESULTS

Epidemiological investigation

Hotel guest cases

Of the guests, 372 (including the index case) met the case definition for this outbreak. Of these, one stayed at the hotel and 18 had dinner at the restaurant (four on F1, three on F2, and 11 on F25) but did not attend a party in the halls. Denominators for these cases were unknown. The remaining 353 guest cases were included in the 6846 guests who had mostly attended wedding receptions or year-end parties in the halls. Two (0·2%) of the 1161 guests who had visited F2 and/or F4 (floors without a vomiting incident), and 351 (6·2%) of the 5685 guests who had visited F3 and/or F25 (floors with a vomiting incident), met the case definition. There was a significant difference between the AR on F3 and/or F25 and that on F2 and/or F4 (RR 35·8, 95% CI 8·9–143·7, $P<0·001$).

One hundred sixty-three (3·5%) of 4710 guests on F3 alone, 106 (15·0%) of 708 guests on F25 alone, and 82 (30·7%) of 267 guests on both F3 and F25 met the case definition. Of 163 cases on F3 alone, 161 (99%) occurred in the first 2 days of the outbreak, whereas the onset in cases on F25 alone ranged over 7 days and, on both floors, over 9 days (Fig. 2). The AR in the guests on F25 alone (RR 4·3, 95% CI 3·4–5·5, $P<0·001$) and the guests on both F3 and F25 (RR 8·9, 95% CI 7·0–11·2, $P<0·001$) were significantly higher than those in the guests on F3 alone (Table 1).

There were regional differences in the AR in the guests who had visited halls A, B, C or D on F3, and halls E, F, G or H on F25 (Table 1). The AR in those who had visited hall B was significantly higher than that in those who had visited halls A, C or D. On the other hand, the AR in the guests who had visited hall E was highest and significantly different from that of the guests who had visited hall H. Eight (26·7%) of the 30 guests who visited both hall D on F3 and hall E on F25 became ill. Seventy-four (31·2%) of the 237 guests who visited both halls A or B and hall F became ill (Table 1).

Of 372 guest cases, 199 (response rate 53%) including the index case, returned a completed questionnaire. The index case had not consumed any hotel meals. The interval between the beginning of the party and the onset of gastroenteritis was defined as the incubation period. The first onset of guest cases other than the index case was at midnight on 2 December 2006, at the guest's home. The mean incubation period of 198 guest cases, excluding the index case, was $35·9 \pm 15·5$ (mean $\pm$ S.D.) h. Although the party guests who visited floors 2, 3, 4 or 25 had consumed various meals made by the same food handlers in the main kitchen, the restaurant guests had not eaten any party foods.

Hotel employee cases

Of 838 employees, 72 (AR 8·6%) met the case definition for this outbreak. No staff in the main kitchen and the restaurants had reported any illness on or prior to 2 December 2006. The first onset in an employee was on 3 December. Employee A did not become ill, but employee B became ill on 4 December, 37 h after contact with the vomit. The AR of employees was highest in those in the restaurant on F25 (29·7%, 11/37). The AR in waiters was 11·5% (19/165) and in food handlers 3·7% (6/163). The six food handlers became ill between 4 and 7 December – four of them had worked in the restaurant on F25 (Table 2) and the others in the restaurant on F1 or F2. Of 72 employee cases, 14 (19·4%) reported that they had not consumed any meals in the staff cafeteria.

Laboratory results

GII norovirus was detected by real-time RT–PCR in stool specimens obtained from 71 (77·2%) of 92 guest cases, nine (69·2%) of 13 employee cases, and six (7·1%) of 85 employees without symptoms. The nine symptomatic employees included four food handlers in the restaurant on F25 and five waiters, including employee B (Table 2). Employee A (cleaner) tested negative. The six asymptomatic employees included three food handlers in the main kitchen, another in the staff cafeteria, and two waiters (Table 2). The food handlers in the main kitchen and restaurant on F25 had not consumed food and drink in the staff cafeteria. If the total number of symptomatic and asymptomatic employees is regarded as the total number of infections, the AR for asymptomatic infection is estimated as 31·6% (6/13).

The results of homology test for the PCR products from 58/59 GII NV-positive specimens (from 44 guest cases and 15 employees), showed 100% homology

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with the pilot strain (a guest on F3) except for one case (a guest on F25). Forty-four GII NV-positive guest cases included 38 party guests in halls on F3 and/or F25 and six guests at the restaurant on F25. Sequence analysis of the conserved capsid domain (G2SKF/G2SKR) of the virus revealed that the genotype from 58/59 GII NV patients was GII/4 EUb DenHaag/06/NL and another genotype from only one case was GII/12 SaU1/04/JP. The result of sequencing the hypervariable region encoding the P2 domain for a GII NV-positive specimen showed that the genotype was also GII/4 EUb DenHaag/06/NL. No norovirus was detected in the cold foods and no pathogenic bacteria were detected in stool specimens.

DISCUSSION

This paper describes an explosive outbreak of norovirus gastroenteritis following two vomiting incidents by an index case on two different floors ('affected floors') in a hotel. No specimen was obtained from the index case. We compared the AR in the affected and unaffected floors.

The AR and number of gastroenteritis cases in the guests who visited halls on the affected floors (F3 and F25) were overwhelmingly higher than those on the unaffected floors (F2 and F4). Consequently, the two locations with a vomiting incident on the two floors were considered as major infection sources of this outbreak. The strain GII/4 norovirus was isolated from most of the guest cases and the employees. The development of disease and transmission was facilitated by the low infectious dose (i.e. <100 viral particles) and the resistance of these viruses to various environments and the standard cleaning and disinfection agents [2, 3, 8, 10]. Infection in the guest cases spread widely on the two floors after cleaning the vomit locations (Figs 1, 2). We believe that environmental contamination [13–17] played a significant role in sustaining the outbreak, although we were unable to identify the virus in the environmental swabs.

Norovirus with identical genotype was confirmed by real-time RT–PCR in all the positive specimens from 43 guest cases and 15 employees, including four asymptomatic food handlers, except for one guest case on F25. These facts suggest that this outbreak was caused by a single infectious source. The guest with a different virus genotype was probably infected elsewhere.

Although many party guests on the different floors had eaten the party foods made by the same food handlers (including asymptomatic food handlers) within the main kitchen, there was a clear difference between the AR on the affected and unaffected floors. Nevertheless, the 18 guests from the restaurants on F1, F2 and F25 had not had any party foods but still became ill. Furthermore, the genotype of norovirus detected from them was identical to that from the party guests. The food handlers in the main kitchen and restaurant on F25 had not eaten the meals made by an asymptomatic food handler in the staff cafeteria, but the genotypes of norovirus detected from all food handlers was identical (Table 2). Therefore, the cause of this outbreak could not have been a specific menu nor any foods contaminated by asymptomatic food handlers. However, a few of the guest cases who complained of gastroenteritis could have been infected with the same genotype of the virus outside the hotel, because strain GII/4 has been predominant as the cause of outbreaks worldwide since the mid-1990s. To clarify this point, we could have sequenced the hypervariable region encoding the P2 domain [22] but were unable to do so except for one case.

We studied the development of outbreaks on the two affected floors in detail. The main observation in this outbreak was that the AR in the guests who visited F25 was significantly higher than that in those who visited only F3. Furthermore, the outbreak on F3 was rapidly terminated, while the onset of gastroenteritis in the guests who visited F25 spread over 9 days (Figs 2, 3). This suggests that some environmental differences in the two floors had caused this. We hypothesized that the differences in the structure of the building, floor area, volume and ventilation rate of the corridors were the important environmental factors that led to the difference in the durations of infection. At the first day of the outbreak, the amount of noroviruses involved in the vomit residue on both the floors is unknown, but it was clearly sufficient to cause the total number of guest cases on F3 alone ($n=163$), which was comparable to that on F25 ($n=188$).

To explain how norovirus spread in the buildings, we considered two possibilities. First, viruses may be carried by contact with vomit residue or dust. There is a report that carpets may harbour viable virus for at least 12 days and that the virus is not removed by routine vacuum cleaning [23]. People who walked on vomit residue might have carried the viruses

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elsewhere on shoes or long dresses. The density of viruses on the floor surface of corridor F3 would more rapidly decrease than that on F25, because more people walked through this corridor, where the floor area was 6·9 times larger than that on F25. The highest AR was in hall B (Fig. 1) on F3; this could be explained by exposure to the directly contaminated area in front of the hall. In contrast, the density of virus on the surface of corridor F25 may have been kept high because of its narrowness. Hall E showed the highest AR (Fig. 1) on F25, but could be visited without passing through the vomit area. The corridor around hall E could have been most contaminated by people walking through the vomit area from halls F, G and H to the toilet or lift.

Second, viruses might be spread as airborne dust. This possibility would be supported by a study that examined 144 environmental swabs using nested RT–PCR. We showed that the highest proportion of positive samples were detected in directly contaminated carpets, but noroviruses were also detected from environmental swabs in elevated sites, such as mantelpieces or light fittings, unlikely to have been touched. This suggests that airborne dissemination occurred [14]. The vomit residue, desiccated in the dry environment of the hotel, would be disseminated as dust, and possibly into the air by people traffic or vacuum cleaning. The airborne dust containing noroviruses might have been moved by airflow to the toilets with outlet of air. The ratios of F3 to F25 for volume and ventilation rate of the corridor are 7·4 and 7·6, respectively. Therefore, the airborne viruses in the corridor air of F3 could have been rapidly diluted with the larger air volume and higher ventilation rate [18]. This hypothesis could also explain the rapid decline of the outbreak on F3 and the highest AR in hall E on F25.

Although the employees had been working in the hotel for a long period, the AR in food handlers in the main kitchen on F3 was remarkably lower than that in waiters. It is speculated that the waiters often accessed the contaminated corridors, while the food handlers were working within the kitchen area away from the corridors on F3 and frequently washed their hands. However, the staff area on F25 might have been contaminated by employee B, who was in direct contact when cleaning up the vomit using napkins, leading to the higher AR in the employees in restaurant F25. The female toilets and lift buttons could also have been directly contaminated by the hands of the index case. From experimentally contaminated surfaces, noroviruses can be readily transferred to other fomites via hands [24]. However, there was no gender difference in the guest cases. The lift button to F25 might have been contaminated by the index case and pushed by many guests going to F25, but we did not investigate this.

This outbreak demonstrates that environmental factors such as floor area, volume and air ventilation in the building can affect the extent and progress in norovirus outbreaks with environmental contamination. This outbreak also taught us that the vomit and the area around it should be thoroughly disinfected before desiccation, as brushing and vacuum cleaning, especially in a closed or semi-closed setting with carpeted floors, releases viruses into the air.

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DECLARATION OF INTEREST

None.

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