

Winter vomiting disease in Florida students

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SUMMARY

On 14 November 1967 an investigation was begun at the University of South Florida to determine the cause of an increased number of gastrointestinal illnesses on campus. An estimated 300 students suddenly became ill with nausea, vomiting, abdominal pain, and diarrhoea. While at first food poisoning was suspected, no common food, water, or toxic exposure could be found. The data collected were most consistent with Winter Vomiting Disease. This entity is characterized by acute gastrointestinal illnesses which may occur in epidemic form in residential schools between September and March. Most investigators suspect that a viral agent is responsible for the illnesses, though attempts to isolate a virus have been unsuccessful. The incubation period in the University outbreak was estimated at 28 hr., and contact was the most likely mode of transmission. The outbreak illustrated that Winter Vomiting Disease in residential institutions may be an accentuation of the gastrointestinal illnesses occurring simultaneously in the community at large.

INTRODUCTION

Winter Vomiting Disease was first described by Zahorsky (1929). Subsequently, several large autumn and winter outbreaks of acute gastrointestinal illness have been reported, in which viral agents were suspected but could not be isolated (Reimann, Hodges & Price, 1945; Reimann, Price & Hodges, 1945; Gordon, Ingraham & Korn, 1947; Kuhns & Wetherbee, 1950; Webster, 1953; Haworth, Tyrrell & Whitehead, 1956; Walker *et al.* 1960; McLean, McNaughton & Wyllie, 1961; Pollock & Clayton, 1964). The incubation period and mode of spread of this disease are not established. The following report describes an outbreak of Winter Vomiting Disease in which the data collected support previous theories implicating contact as the most likely mode of spread, and permit an estimate of the incubation period. In addition, Winter Vomiting Disease in the residential school studied appeared to be only an accentuation of the gastrointestinal illnesses occurring simultaneously in the adjacent community.

DESCRIPTION OF THE OUTBREAK

On Tuesday 14 November 1967, the health department in Hillsborough County, Florida, was notified of a suspected 'food poisoning' epidemic at the University of

South Florida. An estimated 300 students had suddenly become ill with symptoms of nausea, vomiting, abdominal pain, and diarrhoea. Thirteen had been admitted to the infirmary. All thirteen were resident students who ate at the same cafeteria. The university officials suspected food poisoning related to that cafeteria.

MATERIALS AND METHODS

The University of South Florida has 11,500 students including 2750 resident students and 8750 commuters. The resident students live in 12 dormitories which are divided into two complexes: complex A includes the suspect cafeteria and three surrounding dormitories, and complex B includes the 'other' cafeteria and nine surrounding dormitories. Both cafeterias have the same commercial source of raw food products, but they prepare foods independently. According to infirmary records, the average number of students reporting to the infirmary with gastrointestinal symptoms, in previous winters, was eight per week.

Permission was granted to sample one of the three dormitories of complex A. Complex A contained the suspect cafeteria and included a total of 1300 students. Between 15 and 17 November, questionnaires were completed by 360 of 412 students in Alpha dormitory. Alpha dormitory was chosen by a random method from the three dormitories in the complex, and turned out to be a male dormitory. The questionnaire asked if the student had become ill in the last few days, asked for the date and time of onset of illness, and for the symptoms (nausea, vomiting, diarrhoea, chills, fever, stomach pain, other). The student was then asked where he had eaten his meals on Sunday 12 November and Monday 13 November, and a list was given of the foods served at the cafeteria on those days which was to be checked yes, no, or don't know, for each food.

For comparison purposes, similar questionnaires were completed by a dormitory in complex B. Complex B was thought, by the university authorities, to be relatively free from gastrointestinal illnesses. The questionnaires were completed by 245 of 246 students in the comparison dormitory, which had been chosen by a random method from the nine dormitories in the B complex. The comparison dormitory turned out to be a female dormitory.

Interviews were held with each of the ill students in the Alpha and comparison dormitories. To be counted as ill, a student had to indicate on the questionnaire that he had become ill in the last few days and check at least one of the following symptoms: nausea, vomiting, abdominal pain, diarrhoea.

Bacteriological and virological examinations were performed on a representative group of human and environmental specimens. Specimens from patients were obtained within 12 hr. of onset of symptoms.

Rectal swabs, vomitus specimens, and the left-over food were inoculated on to *Salmonella-Shigella* (S.S.) agar plates; Wilson Blair (W.B.) agar plates; tetrathionate broth and brilliant green (B.G.) agar plates. After 24 hr., suspect colonies were subcultured on triple-sugar-iron (T.S.I.) agar slants for identification. Anaerobic cultures were not done.

In addition, the vomitus specimens, swabs of skin lesions, and the left-over food

were inoculated on blood agar plates, eosin-methylene-blue (E.M.B.) plates and tellurite agar plates.

The water specimens were examined by the Millipore membrane filter technique (American Public Health Association, 1965).

Rectal swabs and throat washings (collected in 0.5 % lactalbumin hydrolysate) were inoculated on primary monkey kidney, HEp-2 (continuous human epithelial) and RU-1 (diploid human embryonic lung fibroblast) cells at 37° C. and observations made for cytopathic effect. A second blind passage was made in the HEp-2 and RU-1 cell cultures. The throat washings were also inoculated on primary monkey kidney cells at 32° C. Newborn mice were inoculated with the original specimens and observed for 14 days.

The monkey kidney tissue cultures were tested for haemadsorption with 0.4 % guinea-pig red cells seven days after inoculation of throat and stool specimens. Tissue culture fluids were harvested at nine days from the monkey kidney cultures and tested for hemagglutination with both guinea-pig and human-O red cells. On the ninth day of incubation, the medium was poured off and the monkey kidney tissue culture cells were challenged with 1000 TCID₅₀ of ECHO-11 virus. After incubation for three more days, the tests were read for the presence of an interfering agent.

The acute and convalescent sera were tested against the following antigens: influenza A, influenza B, parainfluenza 1, parainfluenza 3, adenovirus, and *Mycoplasma pneumoniae*.

During the course of the investigation, it was learned that illnesses similar to those at the University were occurring in the community at large. Local physicians were seeing many patients with characteristic acute gastrointestinal illnesses in which repetitive vomiting was a prominent feature. Therefore, a telephone survey was conducted on 20 December to determine the incidence of acute gastrointestinal illnesses in the Temple Terrace community, which surrounds the University. The Temple Terrace population is approximately 6750. A systematic sample, using a random start, was selected, and 500 phone calls were completed. Those called were read the following statement: 'Were you or other members of your immediate family ill since November 1st with symptoms of vomiting, diarrhoea, or abdominal pain lasting about 24 hours.' If the answer was yes, the respondent was asked how many were ill, whether the illness(es) occurred before or after Thanksgiving, and the approximate date. If the respondent himself had been ill, he was asked whether he had had nausea, vomiting, diarrhoea, or abdominal pain, with or without fever.

Statistical significance of differences in attack rates was tested by standard chi-square analysis.

RESULTS

Clinical illnesses

Eighty of the 360 students in Alpha dormitory reported that they had become ill between 10 November and 17 November, and checked at least one of the four symptoms of gastroenteritis listed. Four additional Alpha students reported that they had become ill within the last few days but couldn't remember the date, or

gave it as before 8 November. Of the 84 ill students, 57 (67%) indicated that they had nausea and 63 (75%) reported abdominal pain. Twenty-nine (35%) reported vomiting, which was usually repetitive and extended over a period of 4–6 hr. Forty-eight students (57%) reported diarrhoea, which usually followed the vomiting and consisted of two to three non-bloody, loose stools. Thirty (36%) thought they had had fever and 27 (32%) reported accompanying chills. The illnesses were self-limited and nearly all who were affected felt well within 24 hr. of the onset of symptoms.

The epidemic curve for the 84 ill Alpha students is shown in Fig. 1.

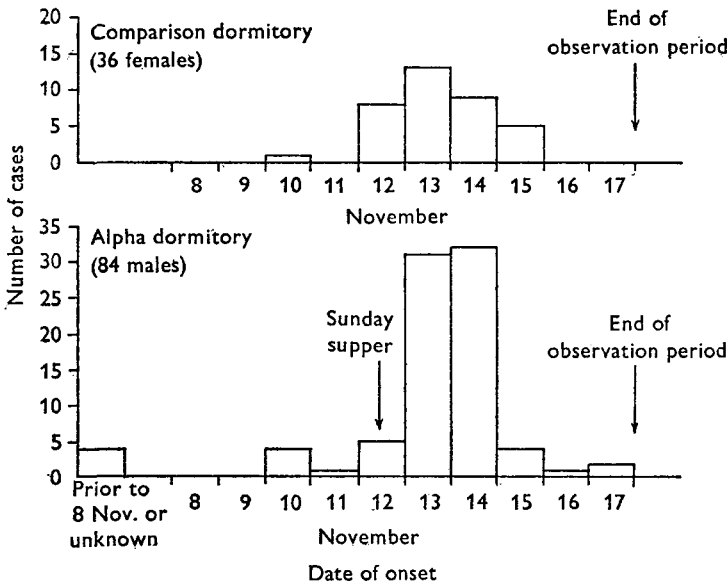


Fig. 1. Gastrointestinal illness by date of onset in resident U.S.F. students—Tampa, Florida, November 1967.

Analysis of the questionnaires obtained in the comparison dormitory revealed that gastrointestinal illnesses had occurred in complex B as well as in complex A. Thirty-six of the 245 (16%) complex B respondents (females) reported illness between 10 and 17 November and checked at least one of the four symptoms of gastroenteritis listed. Nausea (58%) and abdominal pain (77%) were again the most commonly reported symptoms. Seven of the complex B students (20%) reported vomiting, 11 (31%) reported diarrhoea, 11 (31%) reported chills but only two (5.5%) thought they had had fever.

The epidemic curve for the 36 ill students in the comparison dormitory is also shown in Fig. 1. It was noted to be similar to the Alpha epidemic curve, despite the fact that the Alpha students and the students in the comparison dormitory eat in separate cafeterias. Also, additional cases continued to appear despite the fact that all left-over food had been discarded at the beginning of the investigation.

Laboratory studies

Six acutely ill infirmary patients, including five from complex A and one from complex B, had laboratory examinations of rectal swabs, throat washings, and acute and convalescent sera:

The rectal swabs were found negative for *Salmonella* and *Shigella*.

No viral isolate was made from the rectal swabs or throat washings in the culture systems used. The newborn mice likewise remained well throughout the observation period.

No haemagglutination or haemadsorption was noted, and no interfering agents were demonstrated.

No rise in titre was found in the acute and convalescent sera against the viral respiratory antigens tested, though constant titres to one or more antigens were found.

Two vomitus specimens from infirmary patients were found negative for *Salmonella*, *Shigella* and staphylococci.

In addition, rectal swabs on 8 female and 12 male outpatients and on all 30 employees in the suspect cafeteria were negative for *Salmonella* and *Shigella*. Three of the suspect cafeteria employees had small skin lesions. These lesions were cultured and all were positive for coagulase positive-staphylococci. Combination salad was the only food left over from meals served on Sunday and Monday, 12 and 13 November. It was negative for *Salmonella*, *Shigella* and staphylococci.

The University has an independent water supply consisting of five wells interconnected into one system with a one-half million gallon storage tank. Water samples taken, during the week of 13 November, from the suspect and 'other' cafeterias, the Alpha and control residence halls, and from the storage tank were all considered negative since they contained one or fewer coliform organisms per 100 ml.

Epidemiologic associations

No common food, water, or toxic exposure could be found to explain the illnesses in the Alpha dormitory. Attempts to associate illness with several small social events that had taken place at the University over the weekend were unsuccessful.

However, analysis of the 360 Alpha questionnaires for associations between illnesses and places and times of eating revealed one statistically significant ($p < 0.001$) association (Table 1). Among Alpha students, eating supper on campus on Sunday and illness were associated. For those who ate in the suspect cafeteria, the attack rate was 32.5%. For those 40 Alpha students who ate at the 'other' cafeteria, the attack rate was 27.5%. Both attack rates were significantly different from the 10.3% attack rate among those who ate that meal off campus.

Food history analysis was done for each food served at the suspect cafeteria on Monday 13 November and for each food served for supper on Sunday 12 November. The only statistically significant difference ($P < 0.05$) in attack rates between eaters and non-eaters was for tossed salad served for Sunday supper. The food specific attack rates for that meal are shown in Table 2. Of 70 Alpha students who

ate tossed salad, 30 (42.8%) became ill. Twenty-five of 99 (25.2%) non-eaters of tossed salad became ill.

Table 1. *Associations in 360 Alpha (male) students between illness and eating place for supper, Sunday, 12 November, 1967, University of South Florida, Tampa, Florida*

Location of eating place	No. of students with or without acute G.I. illness		Total no. students	Attack rates
	Ill	Not ill		
Suspect cafeteria	54	112	166	32.5
Other cafeteria	11	29	40	27.5
Elsewhere on campus (infirmary, dormitory, coffee shop)	4	4	8	(50.0)
Off campus	15	131	146	10.3
	84	276	360	23.3

Table 2. *Food history analysis for suspect cafeteria, U.S.F., Sunday supper, 12 November*

Food or beverage	Persons who ate specified food				Persons who did not eat specified food			
	Ill	Not ill	Total	Attack rate (%)	Ill	Not ill	Total	Attack rate (%)
Ham	16	29	45	35.6	39	85	124	31.5
Chicken	15	42	57	26.3	40	72	112	36.7
Veal	17	37	54	31.5	38	77	115	33.0
Potatoes	32	68	100	32.0	23	46	69	33.3
Combination salad	9	26	35	25.7	46	88	134	34.3
Tossed salad	30	40	70	42.8	25	74	99	25.2
Carrots	9	18	27	33.3	46	96	142	32.3
Cabbage	10	13	23	43.5	45	101	146	30.8
Corn	20	40	60	33.3	35	74	109	32.1
Cake	19	59	78	24.4	36	55	91	39.6
Pie	14	25	39	35.9	41	89	130	31.5
Pudding	5	12	17	29.4	50	102	152	32.9
Jello	21	43	64	32.8	34	71	105	32.3
Coffee	2	9	11	18.1	53	105	158	33.5
Tea	16	47	63	25.3	39	67	106	36.8
Milk	19	32	51	37.3	36	82	118	30.5
Fruitade	19	30	49	38.8	36	84	120	30.0

In the comparison group no association could be found between illnesses and eating place or between illnesses and foods eaten. Food history analysis done for each food served at the corresponding meals at the 'other' cafeteria on Sunday and Monday, 12 and 13 November, revealed no statistically significant difference ($P < 0.05$) in attack rates between eaters and non-eaters for any food. Individual interviews held with the 36 ill students in the comparison dormitory revealed, however, that 10 of the 36 (28%) were room-mates. Twenty of the 36 (56%) were suite-mates, and 26 of the 36 (72%) were suite-mates or had more than casual

contact with an ill person within 48 hr. of becoming ill herself. Two of the 36 ill students in the comparison group had been off campus for the weekend, returned to an ill room-mate on Sunday night, and became ill an average of 26 hr. after return.

Table 3. Incidence of acute gastrointestinal illness in Temple Terrace, Florida, 1 November, 1967—20 December, 1967*

Population group	Time interval	Number	Incidence	
			No. ill	(%)
Total sample	1-23. xi. 67	1802	199	11.1
Total sample	1. xi.-20. xii. 67	1802	374	20.8
1 and 2 member households	1. xi.-20. xii. 67	276	31	11.2
3 and 4 member households	1. xi.-20. xii. 67	791	175	22.1
5 and more member households	1. xi.-20. xii. 67	735	168	22.9

* Via telephone survey of 500 households, 20 December 1967.

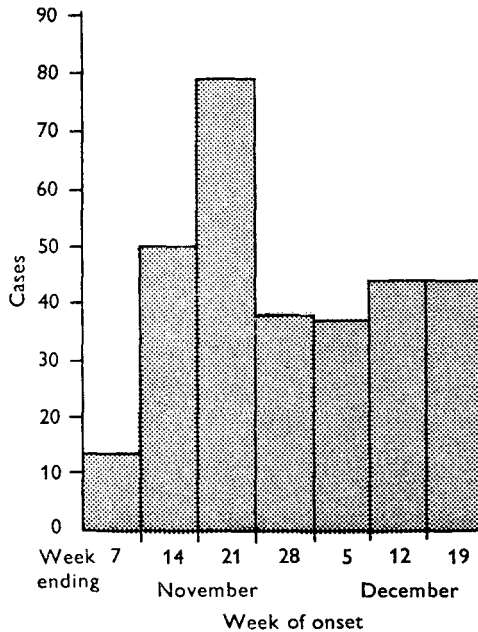


Fig. 2. Gastrointestinal illness by week of onset, Temple Terrace, Florida, 1 November to 20 December 1967.

The results of the community survey are shown in Table 3. One hundred and ninety-nine of 1802 persons questioned (11.1%) reported gastrointestinal symptoms between 1 and 23 November (Thanksgiving). During the 7-week period of 1 November to 20 December, 374 persons (20.8%) reported such symptoms. The attack rates were higher in the larger families. Ninety-three respondents had illnesses themselves. Of these, 55% reported nausea, 63% abdominal pain, 46% vomiting, 71% reported diarrhoea, and 41% reported fever. Of the 374 ill persons an approximate date of the onset of symptoms could be obtained in 305. The

epidemic curve for these 305 persons is shown in Fig. 2. The peak of the curve occurred at the same time that the outbreak occurred at the University.

DISCUSSION

The data collected at the University of South Florida were inconsistent with the diagnosis of food poisoning. The inability to culture a typical pathogen from food or ill students, the finding of a significant number of ill students who had not had a common meal exposure, and the continued appearance of cases, all argued against food poisoning as the explanation for the outbreak. Actually two observations had suggested food poisoning: the staphylococcal isolations from the hands of the three suspect cafeteria employees, and the statistical association of tossed salad with illnesses. However, two of the three employees were not involved in food handling, and the third, a salad maker, was off duty on Sunday 12 November, when the above-mentioned salad was prepared and served. Of the 70 in Alpha who ate tossed salad, 30 became ill. The latter number is insufficient to explain the 84 illnesses in Alpha. Also, the tossed salad, prepared fresh for each meal and kept on ice until served, was an unlikely source of bacterial contamination with human pathogens. Finally, the combination salad, prepared by the same person who prepared the tossed salad, was negative on culture for *Salmonella*, *Shigella* and staphylococci. When the Alpha data were added to the data from the comparison group and both were added to the information obtained from the community at large, no common food, water, social, or toxic exposure could be found to explain the characteristic illnesses which had occurred.

The illnesses seen at the University were most consistent with Winter Vomiting Disease. This syndrome is characterized by acute gastrointestinal illnesses, which may occur in epidemic form in residential schools or institutions. The outbreaks have nearly always taken place between September and March. Numerous reports (Zahorsky, 1929; Miller & Raven, 1936; Gray, 1939; Bradley, 1943; Reimann *et al.* 1945*a*; Gordon *et al.* 1947; Hargreaves, 1947; Kuhns & Wetherbec, 1950; Ingalls & Britten, 1951; Webster, 1953; Simpson, 1954; Haworth *et al.* 1956; Pollock & Clayton, 1964; Cumming & McEvedy, 1969) in the British and American literature over the past three decades have described epidemics of Winter Vomiting Disease (synonyms are epidemic vomiting, epidemic gastroenteritis, epidemic nausea and vomiting, and epidemic diarrhoea and vomiting), and the subject has been reviewed in recent years (Editorial, 1969; Webb & Wallace, 1966). Many of the outbreaks were initially thought to be food poisoning.

The etiology of Winter Vomiting Disease has never been established, though most investigators suspect that a viral agent is responsible. Filtrable agents including the enteroviruses (Cheever, 1967) have been associated with viral dysentery, the term Reimann (1963) uses to include Winter Vomiting Disease and its synonyms, but attempts to isolate and identify a virus in several large-scale autumn and winter school and institutional outbreaks have been unsuccessful (Webster, 1953; Haworth *et al.* 1956; Pollock & Clayton, 1964).

The mode of transmission of Winter Vomiting Disease has not been definitely

established. Hargreaves (1947), discussing an epidemic which occurred in a Cornwall institution, and which affected certain wards but spared others, concluded that the 'infection is airborne'. Ingalls & Britten (1951), describing an epidemic in a school for boys, felt that their data supported dissemination through personal contact. Gordon (1955) agreed that the usual mode of spread was by person to person contact. Cumming & McEvedy (1969), after investigating a recent outbreak at a Reading University residence hall, thought that a food-borne agent was the most likely explanation for Winter Vomiting Disease, but spread by contact was also mentioned as a possibility. Our data suggest contact* as the most likely mode of transmission.

The evidence for contact as the mode of transmission in this University outbreak includes:

(1) The higher attack rate among those Alpha students congregating on campus for supper on Sunday compared with those who ate off campus. Congregating in either cafeteria appeared equally significant.

(2) The high degree of contact among the 36 ill comparison students.

(3) The two comparison students who were off campus for the weekend but who became ill following contact with an ill room-mate.

(4) The higher attack rates in the larger families in the community, characteristic of diseases spread by contact.

Spread by contact, probably via droplet spread in the suspect and 'other' cafeterias on Sunday, would best explain the majority of the Alpha illnesses. Contact would also appear to be the most likely mode of spread in the comparison group.

Clearly not all of the 84 ill Alpha students became infected at the Sunday supper. Fifteen of the 84 were not on campus for that meal. Several of the Alpha students had their onset of symptoms within a few hours of that meal and were most likely infected elsewhere. Yet there was a strong statistical association between illness in Alpha students and eating at the suspect cafeteria for supper on Sunday, which could not be explained by infection from a common food source. It would seem most likely that this association occurred because of the contact between the Alpha students that occurred at that meal. Many students return to the campus for Sunday supper after being off campus for part or all of the weekend. Assuming that contact (direct, indirect, or droplet spread) at that meal produced the association with illness, a rough estimate of the incubation period of Winter Vomiting Disease at the University can be made as follows: There were 54 ill Alpha students who were at that meal; the median incubation period among those who became ill after the meal was 28 hr. The latter figure corresponded to the 26 hr. estimated incubation period in the two control students who were away for the weekend, and to the experimental incubation period of 27 hr. which Jordan, Gordon & Dorrance (1953) found in studies of epidemic non-bacterial gastroenteritis in Cleveland.

Reimann *et al.* (1945*b*) were able to transmit epidemic gastroenteritis to volunteers by both inhalation and ingestion of bacteria-free faecal filtrates. Gordon *et al.*

* Contact as defined by the 10th edition of the *A.P.H.A. Manual, Control of Communicable Diseases in Man*, includes direct contact, indirect contact, and droplet spread.

(1947) were able to transmit the disease from a New York institutional outbreak by feeding bacteria-free stool filtrates to a group of volunteers. Jordan *et al.* (1953), in a series of experiments in a group of Cleveland families, found that they too were able to transmit the disease to volunteers. They concluded that there are at least two different types of acute, infectious, non-bacterial gastroenteritis; an afebrile type and a febrile type.

The afebrile type, produced by the Macey agent obtained in New York (Gordon *et al.* 1947), had an experimental incubation period of 60 hr. It was characterized by profuse, watery diarrhoea and no fever. The illnesses lasted as long as a week.

The febrile type, caused by the (FS) agent obtained in Cleveland, was characterized by fever, abdominal pain, and constitutional symptoms. Vomiting was a prominent feature. The (FS) symptoms were of short duration, with complete recovery usually occurring within 24 hr. of onset. The University of South Florida outbreak and the descriptions of Zahorsky correspond best to Jordan's (FS) type illness. It was the (FS) type illness that Jordan was able to transmit to volunteers with an experimental incubation period of 27 hr. Our estimate in a natural epidemic of an incubation period of 28 hr. would tend to support Jordan's work.

The results of the community survey suggest that Winter Vomiting Disease in the University population was merely an accentuation, probably because of close contact, of the gastrointestinal illnesses which were occurring simultaneously in the community at large. Webb & Wallace (1966) state in their review that 'epidemic gastroenteritis, presumably viral in etiology, is probably the most prevalent gastrointestinal disorder in this country after one year of age'. Thus it is not surprising that the attack rates at the University and in the community were so high.

A complete understanding of Winter Vomiting Disease will probably not be attained until the etiologic agent (or agents) is isolated and identified. Vigorous efforts towards such isolation are advocated by Cheever (1967) in his recent review of the subject of viral agents in gastrointestinal disease.

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