THE AMERICAN JOURNAL OF TROPICAL MEDICINE AND HYGIENE
Vol. 22, No. 1

BALANTIDIASIS OUTBREAK IN TRUK*

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Abstract. In May and June 1971, an epidemic of balantidiasis involving 110 persons occurred on Truk following a devastating typhoon on 1 May. The disease was mild, self-limited, and lacked distinguishing clinical features. The outbreak was multifocal in origin, occurred in all age groups and with equal sex distribution, and terminated spontaneously by early July. The people of Truk live in close association with pigs, and the epidemic probably resulted from widespread contamination by pig feces of ground and surface water supplies, which the people were forced to use after their catchment systems were destroyed by the typhoon.

Balantidium coli is the largest protozoon and the only ciliate of medical importance parasitizing man. Its habitat is the large intestine. Although the distribution of *B. coli* is cosmopolitan, human infection is uncommon. Estimates of worldwide prevalence have ranged from 0.07 percent to 0.77 percent,[1,2] and more recent studies have shown similar figures.[3,4] Prior to 1960, only 722 cases of balantidiasis had been described.[5]

Thirty-three animal hosts are known to harbor species of the genus *Balantidium*, and species morphologically indistinguishable from *B. coli* have been found in the pig, rat, monkey, and guinea pig.[2] The pig is generally considered to be the principal natural reservoir of the parasite and is the most frequently mentioned source of infection in man. However, the role of the pig in human balantidiasis is controversial and the lack of knowledge concerning the epidemiology and pathogenicity of *B. coli* has recently been emphasized.[6]

This paper describes an outbreak of balantidiasis on the islands of Truk in May and June, 1971, following a devastating typhoon. The people of Truk live in close association with pigs, and the epidemic probably resulted from widespread contamination by pig feces of ground and surface water supplies, which the people were forced to use after their catchment systems were destroyed by the typhoon.

BACKGROUND

Truk, situated at 7° 25′ N and 151° 47′ E among the Caroline Islands, is one of six administrative districts of the Trust Territory of the Pacific Islands, a United Nations trust territory under the control of the United States.[7] The total land area of Truk is 45.7 square miles and its population of 29,208 is the largest of all districts.[8] Most of the people on Truk live on islands of the Truk lagoon, which is 40 miles in diameter and is the world's largest reef-bound body of water. Our investigation centered on Moen, the most densely populated of these islands (population 5,687) and the site of the district government.

The climate on Truk is tropical. Moen has an average temperature of 80.7° F with little seasonal variation, humidity is 77 to 86%, and the average yearly rainfall is 137.52 inches.[8,9] Typhoons, an annual threat throughout most of the Trust Territory, have rarely been a problem on Truk.

The people of Truk are classified as Micro-

Accepted 8 July 1972.

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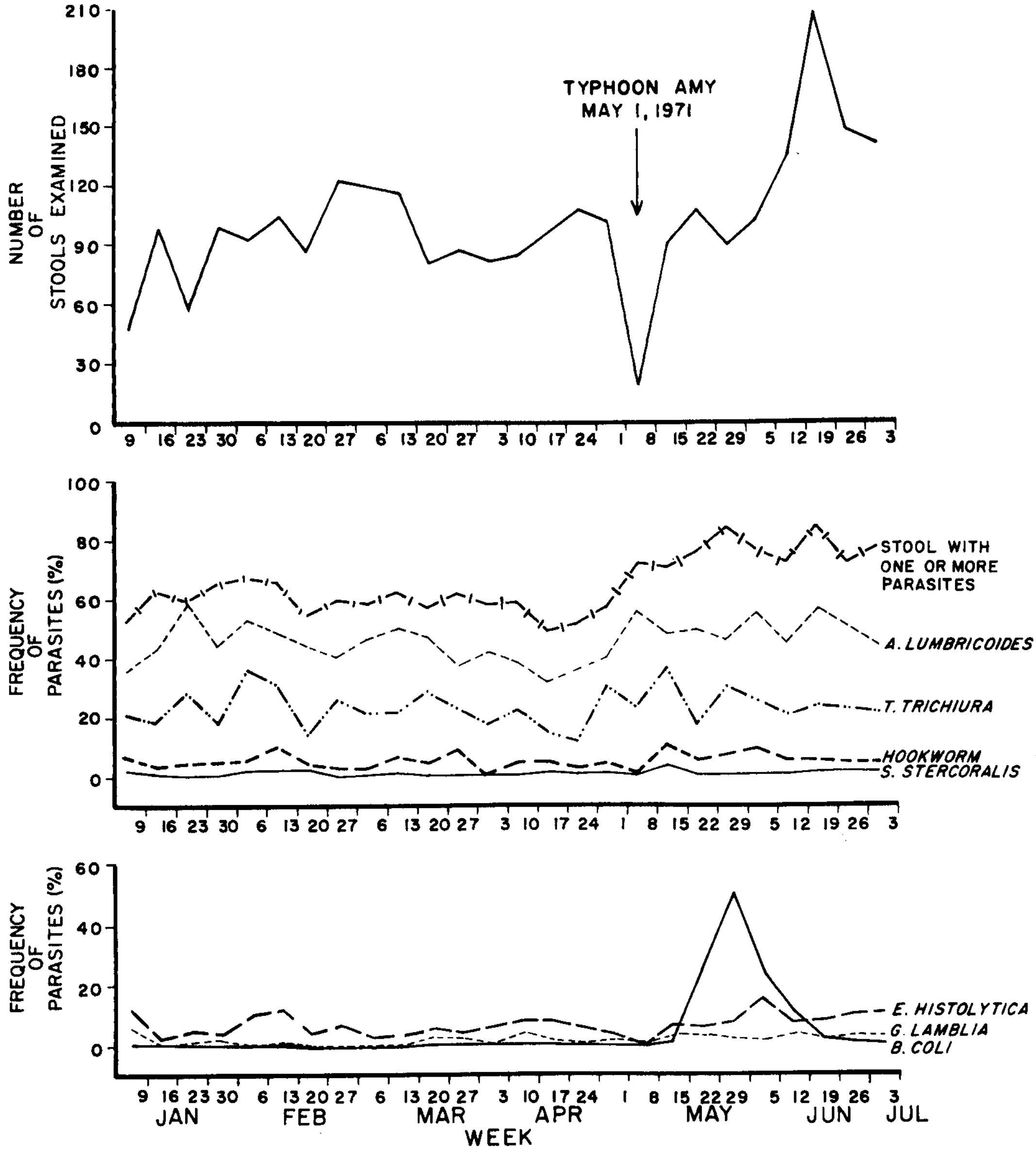

FIGURE 1. Number of stools examined, by week and frequency of parasites demonstrated, 1 January through 1 July 1971.

nesians and speak Trukese, one of the nine major languages of the Trust Territory. The basic diet consists of seafood and such tropical plants as taro, bread-fruit and coconuts. An efficient air service links Truk to the other districts and boat service links neighboring islands. Verbal communications are mainly by radio telephone. Moen has a radio station which often serves as a vehicle for public announcements, since most of the people have radios. Medical care is provided mainly at the new 170-bed hospital on Moen and through dispensaries staffed by health aides on neighboring islands. Large-scale immunization projects have been successfully carried out. The major public health problems for Truk and the entire Trust Territory are tuberculosis and intestinal parasites. Truk accounted for 30 of the 120 cases of tuberculosis and 410 of the 1,361 cases

TABLE 1
*Attack rates of balantidiasis for Moen by age and sex**

Age group (years)	Male			Female			Total		
	No. cases	Population	Attack rate per 1,000	No. cases	Population	Attack rate per 1,000	No. cases	Population	Attack rate per 1,000
<1	0	119	0	0	117	0	0	236	0
1–4	8	337	23.7	11	380	29.0	19	717	26.5
5–9	4	428	9.3	3	408	7.4	7	836	8.4
10–19	4	658	6.1	3	638	4.7	7	1,296	5.4
20–29	3	338	8.9	3	353	8.5	6	691	8.7
30–39	7	318	22.0	5	291	17.2	12	609	19.7
40–49	1	241	4.1	3	213	14.1	4	454	8.8
50–59	5	131	38.2	3	119	25.2	8	250	32.0
60+	2	131	15.3	1	109	9.2	3	240	12.5
Unknown/other	0	201	0	1	157	6.4	1	358	2.8
TOTAL	34	2,902	11.7	33	2,785	11.8	67	5,687	11.8

* Attack rate based on laboratory isolates of *B. coli.*

of amebiasis (the only reportable intestinal parasite) reported in the Trust Territory during 1970.[8]

THE EPIDEMIC

On 1 May 1971, Typhoon Amy swept through Truk with winds of 78 knots and during the 24-hour period dumped 5.6 inches of rain, more than one-third the total rainfall for May. Airport facilities, communications, and many homes were destroyed or disrupted. Beginning in mid-May, the Moen hospital laboratory noted a distinct increase in the incidence of *B. coli* isolates in stools examined, and by early June, over 100 isolations had been reported. Because of this and of the rarity of outbreaks of balantidiasis, the Center for Disease Control (CDC) was invited to conduct an epidemiologic investigation.

THE INVESTIGATION

The investigation began on 9 June and centered around the hospital on Moen, which has the only laboratory facilities for the Truk district and where all cases of balantidiasis were diagnosed and treated. Laboratory records and procedures were carefully studied and hospital physicians were interviewed. *B. coli* patients were asked over the local radio station to return to the hospital for follow-up questioning and stool examinations.

Field investigations included on-site inspection of homes of *B. coli* patients and parasitologic stool survey of household contacts. Comparable studies were performed on other homes in the community. Stool samples were placed in Burrows' phenol-alcohol-formalin (PAF) fixative usually within 2 to 3 hours of collection and transported back to the CDC for examination. Specimens were initially examined both by direct smear and concentration procedures, but the latter was abandoned when it became clear that the yield of *B. coli* isolates was not increased.

RESULTS

Clinical and Epidemiologic Features

A review of hospital laboratory records indicated that normally about 100 stool specimens are examined for eggs and parasites each week (Fig. 1). Stool samples are usually obtained from only the most symptomatic patients; some specimens are submitted routinely from hospital admissions or from persons who must fulfill requirements for foreign travel or selected occupations. Conversations with laboratory workers revealed no recent changes in laboratory personnel, level of personnel training, or in stool processing or examination technique. Stools are examined for parasites by direct saline smear only; culturing facilities for bacteria or viruses are unavailable.

The laboratory recorded only one isolation of *B. coli* from stool during 1971 (in February) prior to the typhoon, and only four in 1970. About 60% of the specimens examined before 1 May 1971 were found to contain one or more intestinal parasites (Fig. 1). *Ascaris lumbricoides* and *Trichuris trichiura* were the most frequently reported helminths. While *Entamoeba histolytica*

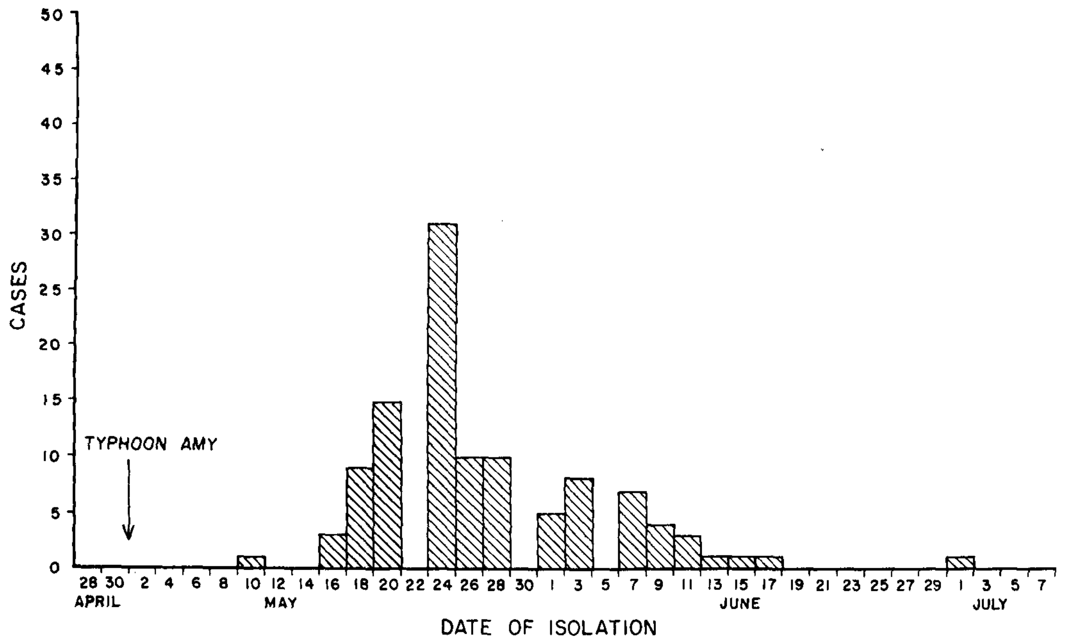

FIGURE 2. Cases of balantidiasis, by date of laboratory isolation, 28 April through 7 July 1971.

appeared to be slightly more common than other intestinal protozoa, no effort was made to differentiate *E. hartmanni* or to record the frequency of other nonpathogens.

Between May 10th and July 2nd, there were 120 isolations of *B. coli* from 110 patients (Figs. 1, 2). The number of stool examinations per week increased markedly during June (2 to 3 weeks after the peak of *B. coli* isolations) and probably reflected the effects of the epidemiologic investigation. The percentage of stools containing parasites increased after the typhoon, but the relative frequencies of individual parasites (except *B. coli*) remained relatively constant. Three additional *B. coli* isolates, one each in August, September, and November, were reported, but it was impossible to determine whether they were related to the outbreak.

The laboratory reported finding only trophozoites of *B. coli*. The low power setting on the microscope was particularly useful in the diagnosis because *B. coli* trophozoites were very motile. We confirmed these observations in a few instances.

Efforts to obtain clinical and epidemiologic data on *B. coli* patients were hampered by the paucity of medical records and the limited communications system. Since most hospital charts contained only the date of isolation of *B. coli*, it was impossible to determine the type of symptoms or treatment given from a chart review. Some patients are listed by first name only, and the addresses are birthplace rather than current residence. Thus, laboratory reports of *B. coli* isolates were the most reliable source of information and the basis of the epidemic curve (Fig. 2). (The breaks in the curve and slight skewing to the right represent weekends, when the laboratory was closed.)

The overall attack rate of balantidiasis by stool isolation for the Truk district was 3.7 cases per 1,000 population. Cases were equally divided among the sexes and occurred in all age groups. The youngest patient was 2 months old, and the oldest was 70 years old. At least 67 patients resided on Moen, an attack rate of 11.8 per 1,000, 11 persons lived on three other islands, and the current address of 32 could not be ascertained.

The Moen attack rate probably more accurately reflects the extent of the epidemic because residents here had easiest access to medical facilities. The highest attack rates on Moen were for the 1 to 4, 30 to 39, and 50 to 59-year-old age groups (Table 1). Cases on Moen were widely scattered throughout the island without clustering in time (Fig. 3). There were at least two instances of

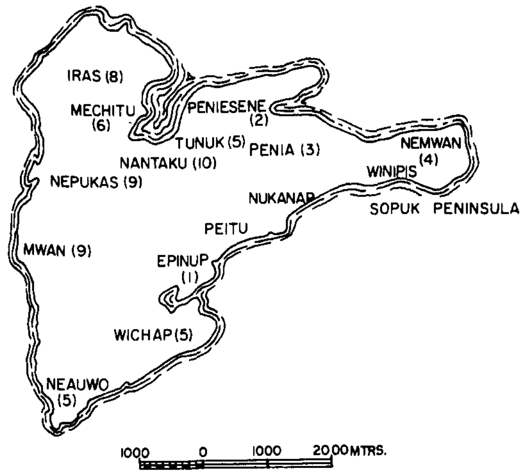

FIGURE 3. Cases of balantidiasis, Moen, 1971.

multiple infection of *B. coli* occurring in one household: 1) two patients from a family on Moen; 2) three Peace Corps Volunteers living in close association on a neighboring island.

Thirty-two patients responded to our radio broadcast and returned for follow-up. They were interviewed through interpreters using a uniform questionnaire. The epidemic curve by date of stool isolate (Fig. 4A) is similar to that of the entire group of *B. coli* patients but the epidemic curve by onset of symptoms (Fig. 4B) is somewhat different. Symptoms preceded laboratory diagnosis by an average of 6.2 days (median 5.0 days, range 0–27 days).

The majority of *B. coli* patients harbored other intestinal parasites (Table 2). In order to evaluate the role of these parasites in symptomatology, 53 consecutive patients submitting stool specimens for egg and parasite analysis over a 2-day period served as unmatched controls, and were interviewed using the same questionnaire. None of these persons had *B. coli* in their stools. This group differed from the *B. coli* patients in terms of mean age (17.8 years vs. 21.8 years), proportion of females (76% vs. 50%), and slightly higher frequency of other intestinal parasites, particularly *Ascaris lumbricoides* (Table 2). However, gastrointestinal symptoms occurred in over 90% of both interviewed *B. coli* patients and controls; nor was it possible to distinguish either group on the basis of type or severity of these symptoms. The laboratory's inability to perform bacterial or viral studies made it impossible to

TABLE 2

Frequency of other intestinal parasites in patients with balantidiasis and unmatched controls

Parasite	All *B. coli* patients (107)*	Interviewed *B. coli* patients (31)†	Controls (53)
A. lumbricoides	50%	45%	74%
T. trichiura	23%	16%	26%
E. histolytica	8%	13%	15%
Hookworm	8%	13%	11%
G. lamblia	4%	3%	6%
S. stercoralis	0	0	2%
None	34%	42%	15%

* Data missing on 3 patients.
† Data missing on 1 patient.

determine the role of these agents in symptomatology.

Interviews with hospital physicians also failed to indicate any distinguishing features about the *B. coli* patients. The outbreak seemed relatively mild—no deaths or excess morbidity were reported—and terminated spontaneously by early July. The lack of a characteristic clinical picture limited our efforts to find additional cases on the basis of history.

Although precise information concerning treatment of *B. coli* patients was scarce, tetracycline appeared to be the most commonly used drug. Some persons received only symptomatic therapy or treatment of other concurrent intestinal parasites. Metronidazole, which has been widely employed in Truk for amebiasis, was given in doses currently recommended for amebiasis,[10] but failed to eradicate *B. coli* in 3 of 5 patients who submitted follow-up stool specimens.

Community Investigations

The typical Trukese home lacks a foundation and has a dirt, wooden or concrete floor; some have glass windows but almost none have screens; the walls are usually of wood and roofs are either of thatch, wood, or thin sheet metal. Houses are often very close together and crowding is a problem; on Moen, we found approximately 15 persons per household.

There are virtually no toilets, and since most of the homes are along the shore, "benjos" are used for defecation. A benjo is a privy serving several families, that opens into the lagoon below. People, particularly, children, swim in the lagoon. Children often defecate indiscriminately around

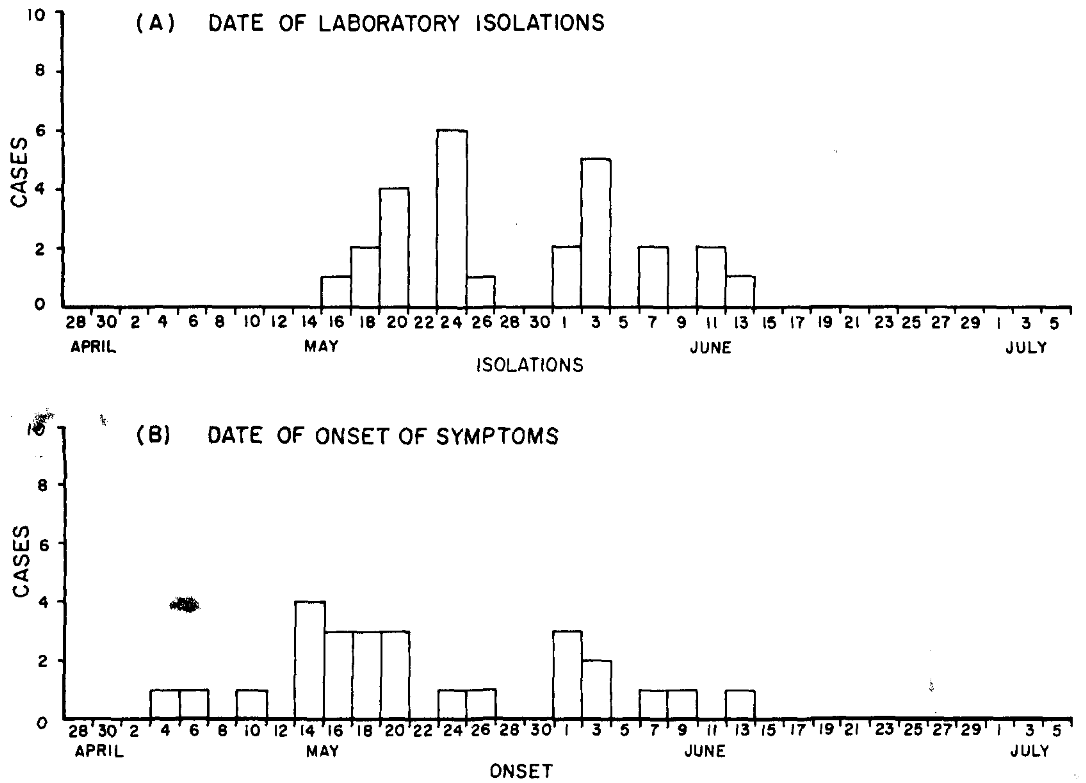

FIGURE 4. Cases of balantidiasis, by laboratory isolation and onset of symptoms, 28 April through 5 July 1971.

their homes. Families commonly were obesrved bathing and washing clothes in streams that are used as sources of drinking water by other families. Animals, especially pigs, but also dogs, goats and chickens, can be found at almost any home. The law specifying that pigs should be kept in pens is not enforced. Samples of pig feces were easy to find at most homes visited. Most houses appeared to have refuse containers, and we did not observe large collections of garbage on lawns.

Drinking water is obtained in several ways: 1) in the majority of homes by catchment from the roof; 2) from a spring, or rarely, a well; 3) by pipe from a nearby stream; 4) a small portion of Moen (e.g. hospital and government offices) has municipal water which is chlorinated and filtered. Public health authorities have long been advocating that people routinely boil all drinking water, but few comply.

The household contacts of six patients with balantidiasis recently (i.e., since 1 June) were surveyed. Eight homes selected either at random or because the inhabitants were known to be cooperative served as controls. We later learned that three of the control families had cases of *B. coli* infection, so these families were included in the balantidiasis group. Of 134 household contacts of *B. coli* patients, only 7 reported recent or current gastrointestinal symptoms; 9 of 91 members of control families reported illness.

Fresh stool specimens of 10 symptomatic persons from homes of patients with balantidiasis and of controls failed to demonstrate *B. coli*. Also negative were stool samples from 20 of 32 balantidiasis patients who had returned for follow-up, from 66 household contacts of *B. coli* patients, and from 37 members control families; these were put in PAF and examined at CDC. The parasite was not found in water samples taken from three homes and poured through a millipore filter. However, pig feces at the homes of balantidiasis patients and of controls readily demonstrated cysts and trophozoites of *B. coli*; the organism was found in 4 of 6 freshly examined specimens and in 3 of 6 specimens examined at CDC.

DISCUSSION

The 110 cases on Truk constitute the largest outbreak of balantidiasis ever recorded. The relative infrequency of *B. coli* (compared with other intestinal parasites) among the Trukese people prior to the typhoon, the lack of recent changes in laboratory personnel or in methods of examining stools, and the fact that the increase in number of stool specimens examined each week came after the peak increase in *B. coli* isolates argue against laboratory error or other factors causing a false epidemic. Only two other outbreaks of balantidiasis, involving 9 and 31 cases, respectively, have been found in the literature.[11, 12] They both occurred in mental hospitals, where poor personal hygiene and coprophagy among patients implicated person-to-person spread as the mode of transmission.

Definitive conclusions about clinical features (or the role of *B. coli*) cannot be drawn because of the lack of medical records, presence of other intestinal parasites, and the inability to rule out bacterial or viral etiologies. However, the absence of observable mortality or excess morbidity and the similarity of symptoms among *B. coli* patients and controls imply that at least some (and perhaps the majority) cases were mild or asymptomatic. Swartzwelder has described three types of balantidiasis: asymptomatic, chronic (recurrent), and acute (dysenteric).[13] Most cases of human *B. coli* infection, particularly in endemic areas,[14] are of one of the first two types. While treatment with antibiotics has considerably improved survival in the dysenteric form, fatalities still occur.[1, 15–23] Tetracycline compounds, the current treatment of choice for balantidiasis,[24] were the most commonly used drugs in the outbreak. Recent studies suggest that metronidazole might also be useful in therapy,[25, 26] but the lack of success in 3 of 5 patients here injects a note of caution.

The temporal relationship of the epidemic to Typhoon Amy strongly implicates the latter as a primary etiologic factor. Although brief outbursts of heavy precipitation (with up to 6 inches of rain in 24 hours) occur several times a year on Truk,[9] typhoons are very rare and much more devastating. The only report found relating *B. coli* infection to precipitation comes from Guatemala where the highest incidence of balantidiasis was during or shortly after the rainy season.[27] Pigs also have to be considered in the etiology because of their great numbers and close contact with the Trukese people. Presumably, then, the typhoon's disruption of primitive catchment water supplies forced people to use ground or surface facilities already contaminated by pig feces; the destruction of pig pens and benjos led to more intimate contact between pigs and people and even more widespread contamination of water supplies.

The role of human carriers in the outbreak was probably small, judging from the very low prevalence of *B. coli* among the population prior to the typhoon. Person-to-person spread may have accounted for some cases, but we found more than one case of balantidiasis in only two families and a very low frequency of gastrointestinal illness among household contacts of *B. coli* patients. Where contact with pigs is lacking, rats have been suggested as the source of *B. coli* infection,[17, 18, 28, 29] and flies as mechanical vectors both in human cases[30, 31] and in an outbreak of balantidiasis among primates in a zoo.[32]

Further support for the fecal contamination of water supplies hypothesis comes from the fact that this epidemic was clearly multifocal in origin. Cases occurred almost simultaneously in widely scattered areas of Moen and on neighboring islands whose only common features were the typhoon, pigs, and primitive catchment water systems.

The brevity of the outbreak could have been due to the high resistance of the Trukese people to *B. coli* infection, the low pathogenicity of *B. coli*, or restoration of original ecologic balance between human host and parasite once the typhoon's damage was repaired. The absence of *B. coli* in stool samples from the community survey supports all these hypotheses. It is possible that the PAF fixative, which was designed especially to preserve morphology of trophozoites,[33] was faulty, but the finding of cysts and trophozoites of *B. coli* in pig feces preserved in PAF and examined freshly makes this unlikely.

Considerable debate exists, however, over the role of the pig in human balantidiasis. Numerous surveys have demonstrated the frequent occurrence of *B. coli* in pigs.[31, 34–37] Arean and Koppisch found that over 50% of human patients with balantidiasis reported in the literature gave a history of contact with pigs, and cite an instance in which a family contracted balantidiasis after eating raw pork sausage.[1] The reported infrequency of *B. coli* infection in man is at least partly due to the failure of laboratories to examine fresh stool specimens (the trophozoites disintegrate within several hours after evacuation) and to the inability of physicians to recognize the disease.[1, 6, 38, 39] The highest prevalence rates of *B. coli* isolates (20–30%) have been found in portions of New Guinea and this may be due to the number of pigs and to environmental conditions that favor contact between people and pigs.[40, 41]

Against the exclusive role of the pig in the transmission of the disease is the occurrence and transmission of balantidiasis in mental institutions[11, 12, 42] and among Moslems, who have no contact with pigs.[43, 44] *B. coli* infection has not been found in people who work with swine[45] even where poor sanitary conditions are present.[46] While *B. coli* can be transmitted experimentally from man to animals (including pigs) and among various animals, attempts to infect human volunteers have failed.[47, 48] Antigenic differences can be found in a number of species of *Balantidium*,[49] and between human and porcine strains of *B. coli*.[50]

Human infection with *B. coli* can be related to other factors, such as low protein and high carbohydrate diet, achlorhydria, alcohol, and infection with bacteria or other parasites.[1] Humidity and temperature also may play a role, since most cases of balantidiasis have been reported from the tropics. In a moist environment trophozoites, the form found most frequently in human cases (including on Truk), survive for 10 days at 22° C; cysts, thought to be the infective form, can survive for several weeks.[2]

Thus, the epidemiology of balantidiasis is complex and incompletely understood. The outbreak in Truk supports the view that man is usually highly resistant to *B. coli* infection even when living in close contact with pigs under poor sanitary conditions. However, under unusual circumstances the pig can be a source of widespread human infection, but the disease is mild and self-limited.

ACKNOWLEDGMENTS

We thank the following persons for their assistance in this investigation: Ann Barrett, William M. Peck, Juan Sablan, Kiosi Aniol, Harunanga Sonis, Charles Jones, John W. Beasley, Koichi Sana, and Mr. Peter.

We thank Stem Salli for his extensive aid in the field work of this investigation.

REFERENCES

1. Areán, V. C., and Koppisch, E., 1956. Balantidiasis. A review and report of cases. *Am. J. Pathol.*, *32:* 1089–1115.
2. Belding, D. L., 1965. The ciliata of man. Pages 109–116 *in* D. L. Belding, *Textbook of Parasitology*, 3rd edition. Appleton-Century-Crofts, New York.
3. Kotcher, E., Hunter, G. W., Villarejos, V. M., Swartzwelder, J. C., Cruz, D. de la, Esquivel R., R., Alfaro B., M., Rodríguez M., C., and Zúñiga R., J. A., 1967. Estudios epidemiologicos de protozoos intestinales en Costa Rica. *Bol. Of. Sanit. Panam.*, *63:* 431–437.
4. Vallada, E. P., 1967. *Balantidium coli* em Itapetininga. Comunicação de 12 casos. *Hospital*, *72:* 821–824.
5. Woody, N. C., and Woody, H. B., 1960. Balantidiasis in infancy. Review of the literature and report of a case. *J. Pediatr.*, *56:* 485–489.
6. Biagi, F., 1970. Unusual isolates from clinical material—*Balantidium coli*. *Ann. NY Acad. Sci.*, *174:* 1023–1026.
7. Worldmark Encyclopedia of the Nations, 1967. Harper and Row, New York, pp. 361–362.
8. Trust Territory of the Pacific Islands: Vital Statistics (Census) 1970.
9. Weather Station, Moen, Truk: Statistics.
10. Powell, S. J., and Elsdon-Dew, R., 1971. Evaluation of metronidazole and MK-910 in invasive amebiasis. *Am. J. Trop. Med. Hyg.*, *20:* 839–841.
11. Young, M. D., 1939. Balantidiasis. *JAMA*, *113:* 580–584.
12. Ferri, L. V., 1942. (Contribution to the epidemiology of balantidiasis.) *Med. Parazitol. (Mosk.)*, *11:* 108–112.
13. Swartzwelder, J. C., 1950. Balantidiasis. *Am. J. Dig. Dis.*, *17:* 173–179.
14. Matabeli, G. V., 1955. (Clinical course, treatment and prophylaxis of balantidiasis). *Med. Parazitol. (Mosk.)*, *24:* 322–326.
15. Cespedes, R., and Morera, P., 1955. Balantidiosis. *Rev. Biol. Trop.*, *3:* 161–170.
16. Kechker, A. S., 1956. (Perforation of the caecum as a complication of balantidial colitis.) *Med. Parazitol. (Mosk.)*, *25:* 308–309.
17. Browne, S. G., 1957. Un cas mortel de dysenterie balantidienne. *Ann. Soc. Belg. Med. Trop.*, *37:* 341–346.
18. Netik, J., Lariviere, M., and Quenum, G., 1958. Un cas de dysenterie balantidienne mortelle. *Bull. Med. de l'A.O.F.*, *3:* 136–140.
19. Garcia-Pont, P. H., and Ramirez de Arellano, G., 1966. Fatal balantidial colitis. *Bol. Asoc. Med. PR*, *58:* 195–199.

20. Wenger, F., 1967. Absceso hepático producido por el *Balantidium coli*. *Kasmera, 2:* 433–441.
21. Alvarez Valverde, R., and Garcia Torres, R., 1967. Estudio de un caso mortel de balantidiosis humana. *Rev. Invest. Salud Publica, 27:* 217–224.
22. Cěspedes, R., Rodríguez, O., Valverde, O., Fernández, J., González, U. F., and Jara, P. J. W., 1967. Estudio de un caso anatomoclínico masivo con lesions y presencia del parásito en el intestino delgado y pleura. *Acta Med. Costarric., 10:* 135–151.
23. Kamat, R. S., 1968. *Balantidium coli* colitis. *J. Postgrad. Med., 14:* 46–48.
24. The Medical Letter on Drugs and Therapeutics, 1969. Volume 11, pages 21–28.
25. Zaman, V., and Natarajan, P. N., 1969. *In vitro* trials of metronidazole against *Balantidium coli*. *Trans. R. Soc. Trop. Med. Hyg., 63:* 152.
26. Zrubec, J., 1971. (Effect of metronidazole and paromomycin [Humatin] on *Balantidium coli* in in vitro and in vivo experiments.) *Cas. Lek. Cesk., 110:* 712–716.
27. Aguilar, R., 1925. The treatment of *Balantidium coli*. *14th Ann. Rep. United Fruit Co. Med. Dept.*, 246–248.
28. Awakian, A., 1937. Studies on the intestinal protozoa of rats. II.—Rats as carriers of *Balantidium*. *Trans. R. Soc. Trop. Med. Hyg., 31:* 93–98.
29. Bogdanovich, V. V., 1955. (Spontaneous balantidiasis in rats.) *Med. Parazitol. (Mosk.), 24:* 326–329.
30. Tsuchiya, T., and Kenamore, B., 1945. Report on a case of balantidiasis. *Am. J. Trop. Med., 25:* 513–514.
31. Kennedy, C. C., and Stewart, R. C., 1957. Balantidial dysentery: A human case in Northern Ireland. *Trans. R. Soc. Trop. Med. Hyg., 51:* 549–558.
32. Cockburn, T. A., 1948. *Balantidium* infection associated with diarrhoea in primates. *Trans. R. Soc. Trop. Med. Hyg., 42:* 291–293.
33. Burrows, R. B., 1967. A new fixative and technics for the diagnosis of intestinal parasites. *Am. J. Clin. Pathol., 48:* 342–346.
34. De Carneri, I., 1958. The frequency of balantidiasis in Milan pigs. *Trans. R. Soc. Trop. Med. Hyg., 52:* 475–476.
35. Coutinho, J. O., and Rabello, E. X., 1958. Contribuicao para o estudo dos protozoarios intestinais do porco (*Sus scrofa domesticus*). *Arq. Fac. Hig. Saude Pub. Univ. Sao Paulo, 12:* 67–78.
36. Larivière, M., Hocquet, P., Lapierre, J., and Camerlynck, P., 1960. Présence de *Balantidium coli* chez les porcs d'un village de Sérère; découverte d'un cas humain. *Bull. Soc. Med. Afr. Noire Lang. Fr., 5:* 176–179.
37. Abdou, A. H., El Sherif, A. F., and Tadros, G., 1961. Internal parasites of swine in Egypt. *J. Arab. Vet. Med. Assoc., 21:* 57–83.
38. Stshensnovitsh, V., 1941. (On the occurrence of *Balantidium coli* and other intestinal protozoa in man.) *Med. Parazitol. (Mosk.), 10:* 252–260.
39. De Jongh, R. T., and Laarman, J. J., 1961. Two cases of *Balantidium* infection in Liberia. *Trop. Geogr. Med., 13:* 203–206.
40. Van der Hoeven, J. A., and Rijpstra, A. C., 1957. Intestinal parasites in the Central Mountain District of Netherlands New-Guinea. An important focus of *Balantidium coli*. *Doc. Med. Geogr. Trop., 9:* 225–228.
41. Couvee, L. M. J., and Rijpstra, A. C., 1961. The prevalence of *Balantidium coli* in the Central Highlands of Western New-Guinea. *Trop. Geogr. Med., 13:* 284–286.
42. Baskerville, L., Ahmed, Y., and Ramchand, S., 1970. *Balantidium colitis*. *Am. J. Dig. Dis., 15:* 727–731.
43. McCarey, A. G., 1952. Balantidiasis in South Persia. *Br. Med. J., 1:* 629–631.
44. Forsyth, D. M., 1954. Balantidiasis. *Lancet, 1:* 628–629.
45. Ostroumov, V. G., 1946. (Materials on the problem of identity of *Balantidium suis* and *B. coli*.) *Med. Parazitol. (Mosk.), 15:* 43–44.
46. Dzbenski, T., 1964. (In search for balantidiasis.) *Bull. Inst. Mar. Med. Gdansk., 15:* 137–141.
47. Knowles, R., and Gupta, B. M. D., 1934. Some observations on *Balantidium coli* and *Entamoeba histolytica* of macaques. *Indian Med. Gaz., 69:* 390–392.
48. Young, M. D., 1950. Attempts to transmit human *Balantidium coli*. *Am. J. Trop. Med., 30:* 71–72.
49. Krascheninnikow, S., and Jeska, E. L., 1961. Agar diffusion studies on the species specificity of *Balantidium coli*, *B. caviae* and *B. wenrichi*. *Immunology, 4:* 282–288.
50. Zaman, V., 1964. Studies on the immobilization reaction in the genus *Balantidium*. *Trans. R. Soc. Trop. Med. Hyg., 58:* 255–259.