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The ecology and epidemiology of the pig-bel syndrome in man in New Guinea

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Spontaneous gangrene of small intestine, without obvious vascular or mechanical cause, is an extremely rare disease. Bowel gangrene, as such, is rarely associated with the known enteric and dysenteric infections of the bowel. A condition with the pathology of a gangrenous enteritis was first recognized in epidemic numbers in subjects undergoing laparotomy for an 'acute abdomen' at Goroka, New Guinea, early in 1961. The disease was described by the non-specific name of 'necrotizing jejunitis' because initial bacteriological investigations failed to establish a cause (Murrell & Roth, 1963). Beta toxin producing strains of *Clostridium welchii* were subsequently recovered from the bowel contents and faeces of subjects with the condition (Egerton & Walker, 1964). They are considered to be important in the pathogenesis of the disease. 'Pig-bel' has been the name given to the disease because of its aetiological association with the pig-feasting customs of the New Guinea Highlander (Murrell *et al.* 1966).

A similar disease, 'Darmbrand', appeared in epidemic form in north-west Germany in the latter and post-war years, and was believed to be due to *Clostridium welchii* type F (Zeissler & Rassfeld-Sternberg, 1949; Oakley, 1949; Marcuse & König, 1950). A food-borne origin for the disease was established (Hain, 1949), but other workers believed that additional dietary, nutritional and possibly viral factors were associated with the pathogenesis (Pietzonka & Rassfeld-Sternberg, 1950; Hormann, 1948; Siegmund, 1948; Kloos & Brummund, 1951). As a result of the recovery of type C strains from man in New Guinea, the strains of *Cl. welchii* type F, believed to cause 'Darmbrand', have been reclassified on toxicological grounds as a type C variety (Sterne & Warrack, 1964).

Heat resistant type A strains are the only other recognized *Cl. welchii* enteric pathogens of man. These cause food poisoning usually via a medium of re-heated meat dishes (Hobbs *et al.* 1953). These strains produce alpha toxin as the major antigen and are one of the organisms responsible for the ordinary gas gangrene of

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wounds. Strains from other groups, producing different toxins, cause enterotoxaemic diseases in animals, each type having a limited host range. Thus type B is associated chiefly with lamb dysentery (Dalling, 1928), type C with enterotoxaemias of sheep, calves and piglets (McEwen, 1930; Griner & Bracken, 1953; Field & Gibson, 1955), type D with pulpy kidney disease of sheep (Bennetts, 1932), and type E is occasionally found as a saprophyte in the intestines of calves (Bosworth, 1940–43).

This paper describes the epidemiological features of pig-bel and its ramifications with pork consumption in the local population.

MATERIALS AND METHODS

Case records were kept of patients with known and suspected enteritis necroticans during the period January 1961 to November 1964. A total of 210 cases are reviewed in this study, the diagnostic criteria for which have been described previously (Murrell *et al.* 1966; Murrell, 1966).

Bacteriological investigations, except for cases in 1961, were conducted in the Veterinary Laboratory, Port Moresby. Thirty-eight resected specimens of bowel were forwarded packed in ice in a vacuum flask, after tying off a segment proximally and distally. The minimal delay for such specimens in reaching the laboratory was 3 days. Faecal specimens from 107 suspected and contact cases were placed in wide-necked bottles containing buffered glycerol-phosphate transport medium. During 1961, faecal smears were placed in selenite F broth and Robertson's cooked meat media and despatched to the School of Public Health and Tropical Medicine, Sydney, for examination. In the laboratory material from the lumen and necrotic wall were examined for the presence of known aerobic and anaerobic enteric pathogens. Sheep blood agar was inoculated directly from affected bowel surfaces. Material from the lumen (1–2 ml.) was also inoculated into Robertson's cooked meat medium. The latter was placed for 1–2 min. in a boiling water bath and then transferred to the incubator. After 24 hr. subcultures were made on sheep blood agar plates which were incubated anaerobically. Plating on deoxycholate citrate agar plates before and after incubation in enrichment media was used for *Salmonella* and *Shigella* sp. strains. Blood agar plates were incubated aerobically and examined for colonies resembling those of *Bacillus anthracis*. Cultures morphologically and biochemically resembling *Cl. welchii* were forwarded to the Wellcome Research Laboratories for identification by methods described by Egerton & Walker (1964).

Antitoxin titres to the beta toxin of *Cl. welchii* were estimated in 24 proven cases, serial samples being taken from twenty-one persons with the disease. Sera from thirty-eight exposed relatives and from 216 randomly chosen individuals from differing Highland clans were also matched against forty-two control sera taken from Europeans, nineteen in the United Kingdom and twenty-three in New Guinea. The serum was separated, kept under refrigeration and 1 drop of 50% *o*-cresol-ether added to each ml. of serum as a preservative. These were despatched in batches to the Wellcome Research Laboratories for estimation of *Cl. welchii*

beta antitoxin by the method of Glenny, Llewellyn-Jones & Mason, 1931, as modified by Glenny *et al.* 1933.

Epidemiological information was obtained in the field on six different occasions—in June and August 1961 in Upper Asaro; September 1963 in Tambul and Wabag; May and October 1964 in Upper Chimbu; and from June to July 1964 in Chuave, Tari, Lake Kopiago and Baiyer River (Fig. 1). The first three periods coincided

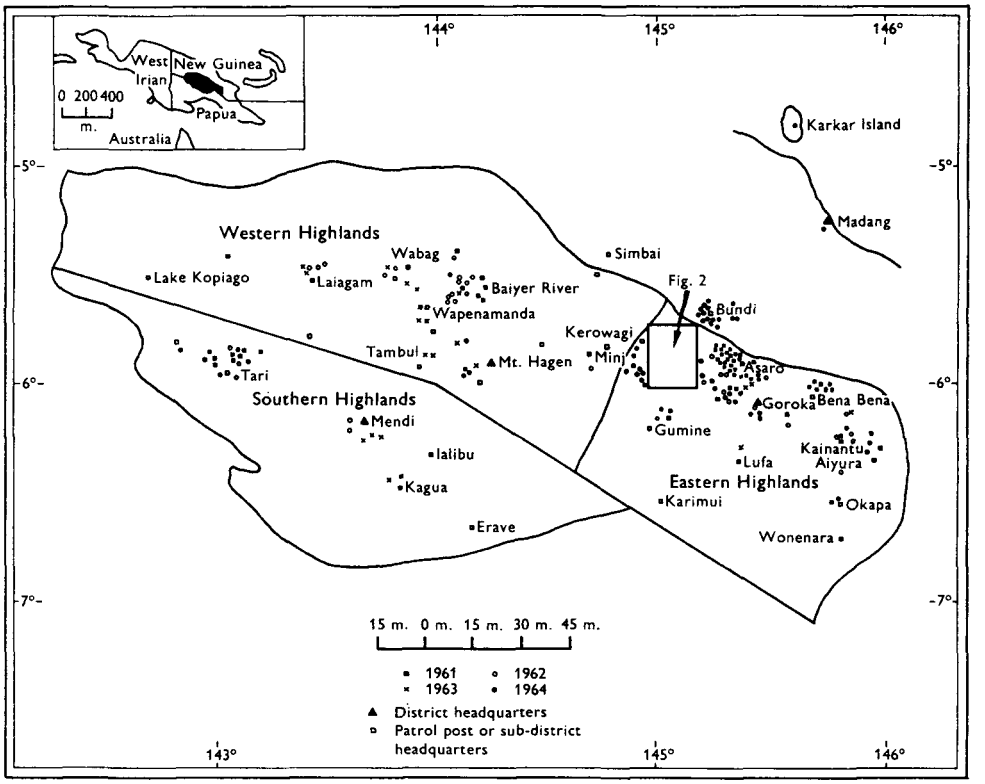

Fig. 1. Geographical distribution of reported cases of pig-bel in the Highlands of New Guinea for the period 1961–1964.

with pig-feasting activities of the local people. Comparative observations of pig-slaughtering methods, preparation of meat for cooking, cooking methods and pork distribution were undertaken. Sources of contamination were looked for: pig carcasses were examined for any noteworthy pathological abnormalities: random samples of faeces and intestinal contents were taken from 322 pigs for bacteriological examination. These were collected aseptically, placed in transport media and sent airfreight to Port Moresby, packed in ice by methods described earlier. Specimens of pig meat in the fresh, cooked and stored state were collected and processed as with other samples. A systematic survey to determine the frequency of *Cl. welchii* type C in the normal population was undertaken during June to July 1964. Clean unsterile waxed paper cups were issued by random sampling to individuals on a basis of a 1 in 5 sample. About 5 g. of faeces were transferred to

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wide-necked sterile bottles containing transport medium and forwarded to Port Moresby and from there to London. In the laboratory this was halved to a 1 in 10 population sample. Four hundred and sixty-eight examinations were thus completed. A record of the foods eaten and estimations of the weight and volume of individual meals were made. Temperatures of the cooking process and cooked meat were taken with a thermocouple.

Blood samples were taken from 216 volunteer subjects. These volunteers were representative of seven different Highland groups. These were from Upper Asaro, Chuave and Upper Chimbu (clans in the Eastern Highlands); Baiyer River, Wabag and Lake Kopiago (clans in the Western Highlands), and one clan near Tari in the Southern Highlands.

A population census was conducted in an endemic area at Goromaugo in the Upper Chimbu in October of 1964. An approximate estimate of the disease incidence was obtained by equating the population figures with persons treated for pig-bel in whom the diagnosis was established bacteriologically or serologically.

Information on diarrhoeal disease patterns and causes of death were obtained from records of the Goroka, Tari, Baiyer River and Kundiawa hospitals for periods between 1 January 1962 and 30 November 1964. From the Goroka hospital records a survey of surgical operations undertaken for acute and subacute intra-abdominal conditions for the 4-year period 1 January 1961 to 30 November 1964 was also made.

RESULTS

Disease incidence

The prevalence of pig-bel varied considerably from area to area in the Highlands (Fig. 1). Owing to the lack of adequate demographic records in the Territory, incidence figures were initially assessed from population census figures and established cases. This ranged from 4 per 10,000 in the Wabag district to 22 per 10,000 in the Upper Asaro area of the Eastern Highlands.

A more accurate assessment of the incidence of the disease was made in a census of five clans at Goromaugo in the Upper Chimbu taken in 1964. Following pork feasting in May and August of the same year, seven persons were known to have contracted pig-bel in a population of 1448. The diagnosis was established bacteriologically in three cases, serologically in two and by autopsy in a further two. The incidence in this area was therefore assessed at 48·3 per 10,000. A high prevalence of *Cl. welchii* beta antitoxin in population groups sampled in the Upper Chimbu supported the impression that Goromaugo represented a high incidence area.

Mortality

Two of the seven persons known to have contracted pig-bel during 1964 at Goromaugo died. This gave a mortality of 13·8 per 10,000 population. This compares with the case mortality rate of 36·0%. Excluding the mild forms of the disease this was 49·8%, which was highest in the acute toxic group (84·6%). The mortality rose significantly in the younger and older age groups.

For all ages, enteritis necroticans accounted for 2·1% of deaths at the Goroka and Kundiawa hospitals and 3·5% at the Baiyer River hospital during the period

1 April 1962 and 30 November 1964. These rates were again higher in the 1 to 12 year age group. At Baiyer River 16·1% of deaths occuring in this age group were due to pig-bel.

Outbreaks and geographical distribution

The geographical distribution of cases over the 4 years 1 January 1961 to 30 November 1964 is shown in Fig. 1. Epidemics occurred in the Upper Asaro

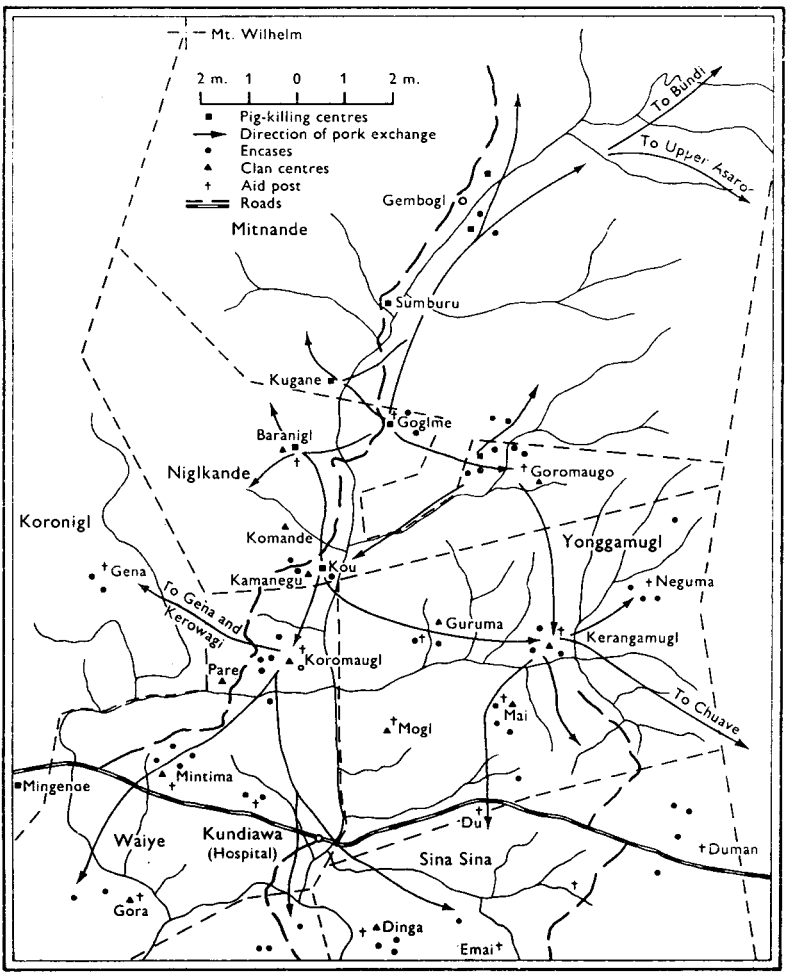

Fig. 2. Local spread of pig-bel in the Upper Chimbu area of the Eastern Highlands. Pig-killing commenced in the Niglkande census division in May of 1964 and concluded in the Sina Sina in October of the same year.

during the months June to September of 1961; in the Tari Basin just prior to this; most of Baiyer River in the Western Highlands in March to June 1962, and in Bundi and the Chimbu throughout 1964.

When pig-feasting activities commenced in May in the Upper Chimbu, pig-bel prevalence rose, as did other diarrhoeal diseases. There was a further increase

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following the pig cycles in the Central Chimbu (Fig. 2). The spread of cases is shown along the directional movements of pork shown in Fig. 2. Following the 'Te' festival of the Enga people of the Western Highlands late in 1963, only six cases of the disease were reported. Serological sampling earlier in the year confirmed the impression that the Wabag-Wapenamanda area was a low incidence area. *Cl. welchii* type C was isolated from cases at the Upper Asaro, Chimbu, Baiyer River, Wabag, Wapenamanda and Mendi.

Evidence of local spread

Following pig killing at Goromaugo there was a bridal exchange of pork between members of a Pagakaune line and one man, Ambane Umba, of a Kurumogl line. Ambane subsequently developed a more protracted form of the disease which was confirmed by a rising beta antitoxin and the recovery of *Cl. welchii* type C from his faeces. Sera taken from eight relatives exposed to the same meal and from the man who had prepared the meat all contained significant amounts of beta antitoxin. A further case occurred in a man at Gena (Upper Chimbu) who had eaten pork originating from another Goromaugo clan. Contacts again had immunological evidence of exposure to beta toxin. However, severe infections, confirmed by autopsy or laparotomy, were seen in more than one member of a family at risk on only three occasions.

Owing to difficulties in communication suspected meal remnants could not be obtained.

Seasonal distribution

The data from hospital records indicated that peaks of pig-bel occurred mostly in the 'dry' season between the months of April and September (Fig. 3). Major epidemics coincided with the larger pig-killing ceremonies held at this time of the year because of the prevailing climate and good harvest.

The relationship of pig-bel and epidemics due to other diseases was not investigated in detail. A widespread influenza epidemic preceded the pig killing in the Upper Chimbu in 1964, and a measles epidemic was known to occur from October to December of 1962 in the Western Highlands. Beecroft (1962) in a personal communication noticed that a gastro-enteritis epidemic was prevalent before the pig killing near Baiyer River in April 1962. The seasonal prevalence of all the infective diarrhoeal diseases showed similar trends in the drier months of the year when admission rates at various Highland hospitals were analysed (Fig. 4).

Race, age and sex distribution

Of the 210 persons reviewed in the case series, only one was a European. All other victims, with one exception, were inhabitants of the areas in which they contracted the disease. The exception was a Mount Hagen native working as a plantation labourer on Kar Kar Island, in the Madang District. The European, a 28-year-old Polish linesman, contracted his disease at Tari following a meal of native pork.

Ages and sexes are listed in Table 1. The greatest number of patients were 2–10 years old (52·3%). Children in the 6 to 10-year group made up the largest segment of overall cases (29·0%). The distribution of persons with the disease under 10 years of age was most significant ($P < 0{\cdot}001$) when compared with other decades.

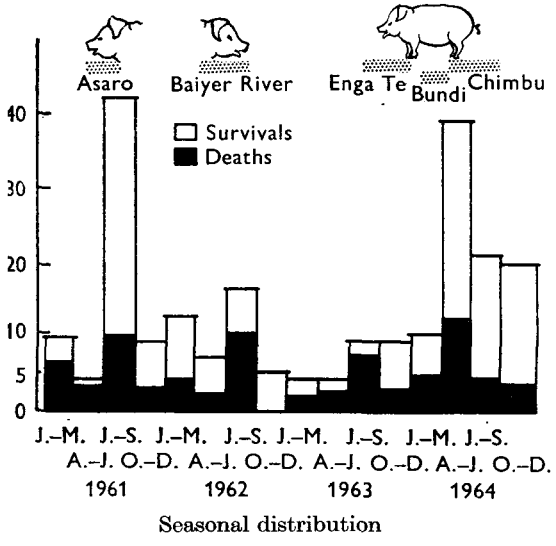

Fig. 3

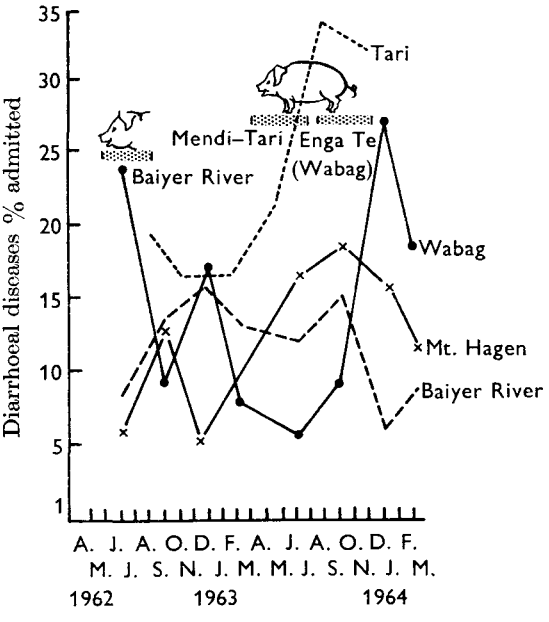

Fig. 4

Fig. 3. Quarterly distribution of pig-bel cases during the period of 1961–64. Pig-feasting activities are indicated at the top of the diagram.

Fig. 4. Prevalence of diarrhoeal disease as measured by percentage of admission for all diseases in four Highland hospitals. Peak rises followed the large pig-kills in the areas served by these hospitals.

Table 1. *Age and sex distribution of* 210 *cases of pig-bel*

Age group	Males	%	Females	%	Total	%
0– 1	1	0·4	2	1·1	3	1·4
2– 5	36	17·1	13	6·2	49	23·3
6–10	46	21·9	15	7·1	61	29·0
11–15	17	8·1	5	2·4	22	10·5
16–20	10	4·8	12	5·7	22	10·5
21–30	13	6·2	8	3·8	21	10·0
31–40	12	5·7	7	3·3	19	9·0
41+	10	4·8	3	1·4	13	6·2
Totals	145	69·0	65	31·0	210	99·9

Males were affected more than females in the ratio of 2·2:1. This ratio was generally maintained in all age groups except in the 16- to 20-year group when more females were recorded with the disease. The numbers recorded in this group, however, were too small to be significant. At Goromaugo, the sex incidence in the

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normal population was 5:2 in favour of males. Of the seven persons found to have contracted pig-bel disease in this survey, four were under 10 years of age.

Pig-feasting investigation

The pig is the central figure in all social events—part of the bride price in weddings, currency in compensations, and the main menu dish in feasts celebrating the epochal events in clan and tribal life. It is offered as a sacrifice to placate the spirits of the deceased: it is offered when crops have failed and given medicinally when sickness occurs: wherever, in fact, the ancestor spirits, good or bad, might be. Ownership of pigs, therefore, is not an asset in regard to food supplies. Rather their possession and circulation are values in themselves in enhancing a man's social status and prestige, and form the basis of a barter economy. Within the framework of this credit system large 'pig-kills' occur in cycles when herd numbers have built up sufficiently. These begin within one lineage group and precipitate progressive killings over the ensuing 6 months along well-defined trade routes within clans and tribes (Meggitt, 1958; Brookfield & Brown, 1959). Up to 10,000 pigs may be killed in a 25-mile radius on such occasions.

Observations of a pig-kill

Preparations months in advance took place when long houses were built to accommodate residents and visitors round central courtyard clearings. Tables were also built in readiness to receive the pork sides and quarters for distribution. In the Eastern Highlands 2–4 weeks before a large pig-kill, a smaller celebration took place, the smaller pigs being sacrificed on such an occasion.

All large pig-killing ceremonies occurred at the time of a full moon. Whenever possible they were held during favourable weather which was usually during the dry season. Dancing and singing festivities were held before the killing began. In the Chimbu the pork preparation and distribution took 3–4 days, whereas only 1–2 days was required in the Tari and Wabag areas. A preliminary count of pigs occurred in the 'Te' festival of the Enga tribes (Plate 1*a*). The tethered pigs were clubbed to death by a near relative of the owner, using a 2–3 in. diameter stick. There was no bleeding of the carcass, although some blood from the head wounds spilt out over the ground. Cassowaries, chickens and dogs were occasionally sacrificed along with the pigs but were cooked separately. The hair of the pig was singed over an open fire and the carcasses then lifted on to leaf mats of banana, breadfruit and tree fern leaves. An intricate dissection in the dorsal position was commenced with double lateral incisions in the anterior axillary line (Plate 1*b*). The outermost continued down behind the anus and the inner two met anterior to it. Here, spillage sometimes occurred and the butcher's hands became contaminated directly by pig faeces. The abdominal skin flaps were dissected up towards the head and the thoracic cage opened laterally by axe cuts. A careful butcher then removed the diaphragm, peritoneal sac and contents *in toto*.

The women took the hollow abdominal viscera in their 'bilums' (string carrying bags slung over the head) to the nearest stream for washing. The Chimbu and

Gahuku (Asaro) women everted the small intestine by intussuscepting a stick into the bowel lumen. They siphoned and forced water through the large bowel, the anal skin and anus acting as a funnel. Enga fashion was a little less crude as the whole bowel was split open and cleaned more thoroughly. The bowels were plaited and wrapped in leaves ready for cooking. Stomach was packed with chopped fat, greens and herbs and cooked in the form of a haggis. It was eaten after 'maturing' for 2 or 3 weeks. While the bowel washing progressed, the men continued the final filleting of the carcase, skull, rib cage and backbone being removed in one section. Here, contamination by feet was most apparent as assistants were required to pull the head and spine forward and ventrally.

The butchering completed, which took about 2–4 hr., the night was given to more singing and dancing. Earth pits were dug: long and shallow for the half sides of meat, and deep and wide for the offal and prime cuts. The ovens were lined with banana leaves, tree fern fronds (*Cyathea contaminans*) and leaves of a variety of breadfruit (*Ficus dammaropni*). The meat, sweet potato, taro, corn, bananas and chopped greens were added with pre-heated stones and included in the festive meal. Men handled the long wooden tongs with great dexterity (Plate 2). Layer by layer the oven filled up until the large leaves roof over a final insulating layer formed by the pig's quarters and flanks. Water was poured in and the oven was sealed by another layer of leaves and mounded up with dirt, so that the food was cooked under steam pressure in its own moisture. The juices of all the contents were thus retained in the cooking process. The Chimbu women also cooked bowel and other morsels in wooden barrels.

Table 2. *Cooking time and procedure. The temperature readings were taken from the centre of oven using a thermocoupled pyrometer*

Cooking process	2. x. 63 Time no. 1	Temp. °C	3. x. 63 Time no. 2	Temp. °C
Commenced putting meat in ovens. Water introduced	11.30	38	11.45	35
Sealed with earth and cooking commenced	11.45	82	12.00	79
Temperature rise	11.58	93	12.11	92
Stationary temperature	13.00	104	13.15	102
Started opening pit to remove partially cooked meat (dirt shovelled off)	13.15	104	13.22	104
Exposed meat	13.25	99	13.34	99
Carcasses now cut in half down spinal centre section, meat extracted for disposal, i.e. head, thoracic cavity and everything removed	13.37	93	13.40	83
Actual cooking time	1 hr. 37 min.		1 hr. 34 min.	
Kau-kau and bananas well cooked, soft and crumbly and eaten almost immediately				

Observations near Wapenamanda, indicated in Table 2, show temperature readings taken using a pyrometer. The thermocouple was placed in the centre of the earth ovens and temperatures taken at set time intervals during the cooking. The centre of a hind quarter immediately after removal from an oven after 2 hrs.

cooking had a mean temperature of 78° C. for five pieces sampled. The stones after cooking were a little hotter than warm and could be handled with bare hands.

The results indicated that large chunks of meat were not thoroughly cooked. After cooking the meat rapidly cooled, further handling occurred and the same sources of contamination were present—feet, flies, dogs and so on. It was after cooking that contamination seemed most significant and the arguments and discussions concerning the distribution would continue for a whole day. In transit to its destination, a half side of pork might change hands two to five times and reach consumption point 1–4 days later. A day or more might elapse before the pork was further cut up and a second distribution arranged. Further cooking under similar circumstances increased the chances of contamination and food poisoning. This probably caused the prolonged upper abdominal pain of which so many people complained after eating pork (Nilles, 1950).

From the foregoing, it may be concluded that the likelihood of disease spread was much greater in the large pig ceremonies than in the much smaller marriage and death distributions, where there was a limit to the number of pigs killed and a smaller number of recipients. Bridal payments took place within the framework of the larger ceremonies and stacks of pork quarters and halves were seen in the Upper Chimbu for this purpose.

Sources of infection

Pig and pork

The only notable disease observed in the slain pigs apart from intestinal helminths was intestinal emphysema (Egerton & Murrell, 1965). Between 1 and 4% had macroscopic evidence of enteritis. No cases of anthrax or trichinosis were found. *Cl. welchii* was recovered from the intestinal contents of 53 of 322 pigs chosen at random from pigs killed at Upper Asaro, Tambul and Upper Chimbu. All strains isolated proved to be type A and no type C strains of this organism were recovered. From 115 samples of cooked and uncooked pork, collected mostly from the medial aspect of the hind-quarters where contamination was considered to be most likely, eighteen isolations of *Cl. welchii* type A were obtained. Again no type C strains were recovered. These samples included eighteen cooked bowel specimens. *Escherichia coli* and *Proteus* were also recovered frequently. Remnants of a pig (hair, bone and fat) suspected as a source of origin for five cases of pig-bel, yielded *Cl. welchii* type A but no type C strains. The failure to enlarge the samples from this latter group was due primarily to the late arrival of cases to hospital and the difficulty in obtaining meal remnants. All traces of meat had gone by the time attempts to get such samples were made. The general reluctance of the people to blame pork as a cause also added to this difficulty. Type A strains were recovered from soil samples collected in the Upper Chimbu.

Human population

Antecedent dietary histories were taken from 140 patients with pig-bel: of these, 133 had a prior pork meal and seven persons firmly denied eating pig. The incubation period ranged from 6 hr. to 6 days with a mean of 24 hr. The shorter the

incubation the greater the severity of the disease. Table 3 shows the results of bacteriological examinations of resected bowel and faeces from persons suspected of having pig-bel. *Cl. welchii* was isolated from the lumen and necrotic wall of thirty-six bowel specimens. Seventeen type C strains and nine type A strains were subsequently identified from the isolates. Ten were not submitted for complete identification. The toxicological analysis of eleven beta-producing strains have been reported earlier (Egerton & Walker, 1964). This organism was not recovered from four intestines resected for other causes of strangulation.

Table 3. *Isolation of* Clostridium welchii *in the bacteriological investigation*

Subjects	Total	Negative	Positive	*Cl. welchii* Type A	*Cl. welchii* Type C	*Cl. welchii* Untyped
Surgical resections (bowel smears)	38	2	36	9	17	10
Suspected mild cases and contacts of proven cases (faecal smears)	107	53	54	45	9	0
Follow-up (faecal smears)	17	10	7	6	1	0
Controls						
Bowel resections from other causes	4	1	3	3	0	0
Normal population	468	351	117	117	0	0

Table 4. Clostridium welchii *in normal New Guinea population*

	Faeces examined	Isolation *Cl. welchii* type A	%
Asaro	100	40	40
Upper Chimbu	100	36	36
Wabag	68	12	18
Tari	100	16	16
Lake Kopiago	100	13	13
Total	468	117	25

From a group of 107 in which no surgical intervention was undertaken and which included fourty-four contacts of known cases, *Cl. welchii* strains were isolated from fifty-four faecal specimens (Table 3). Only nine of these proved to be type C strains with the same toxin production as other strains from bowel. *Shigella flexneri* type 2 was isolated from three persons in this group, once in association with *Cl. welchii* type C and twice as the sole demonstrable pathogen.

The 1 in 10 systematic sample of faeces of a normal human population failed to yield any *Cl. welchii* type C strains. This survey was carried out independently of any pig feasting activity, except in the Upper Chimbu where sampling was undertaken 1 month after the pig killing there. The results are shown in Table 4.

These isolations all proved to be type A strains. No type C strains were recovered from the normal population. The only evidence that type C is of human origin was the recovery of such a strain from a pig-bel patient 12 months after bowel resection (Table 3).

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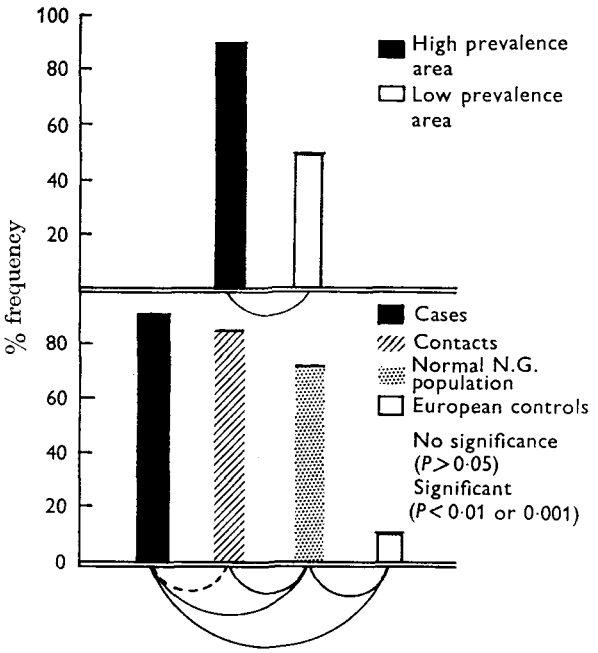
Table 5. *Overall results of beta antitoxin levels*

Group	Place	Units of beta antitoxin per ml. No.	$< \frac{1}{2}$	$\frac{1}{2}$–2	2–5	> 5	$\geqslant \frac{1}{2}$	% $\geqslant \frac{1}{2}$	
A. Known recent pig feasting									
(i) Low prevalence pig-bel	Wabag	66	34	26	3	3	32	48·5	
(ii) High prevalence pig-bel	Upper Chimbu	39	4	29	5	1	35	89·7	
	Total	105	38	55	8	4	67	63·8	
B. Doubtful recent pig feasting									
(i) Low prevalence pig-bel	Watabung	17	9	8	0	0	8	47·1	
(ii) High prevalence pig-bel	Upper Asaro	20	6	12	1	1	14	70·0	
	Tari	34 (71)	5 (11)	21	7	1	29 (60)	85·3	84·5
	Baiyer River	17	0	13	4	0	17	100·0	
	Total	88	20	54	12	2	68	77·3	
(iii) Unknown prevalence. Recent European contact	Lake Kopiago	23	4	17	2	0	19	82.6	
C. Overall N.G. population (A + B)	Grand Total	216	62	126	22	6	154	71·3	
D. Known cases pig-bel		24	1	9	4	10	23	95·8	86·2
E. Suspected mild cases pig-bel		34	7	20	2	6	27	79·4	
F. Relatives of pig-bel cases		38	6	23	6	3	32	84·2	
G. Follow-up of cases of pig-bel ($> \frac{6}{12}$)		9	4	3	1	1	5	55·6	
H. European controls		42	38	4	0	0	4	9·5	

Serological investigations

Sera were examined for *Cl. welchii* beta antitoxin from seven different population groups (A–H, Table 5). A titre greater than or equal to 0·5 of a unit of antitoxin per ml. was regarded as significant. The last group (H) in Table 5 consisted of a control group of forty-two Europeans. There had been no exposure to native pork except in three of these persons. Four, which included the three at risk, had titres of 0·5–1 unit per ml. In the sera of twenty-four cases of pig-bel, twenty-three had detectable



Statistical significance of differences in % frequency of groups of persons with detectable *Cl. welchii* β antitoxin

Fig. 5. Frequency distribution of *Cl. welchii* antitoxin (*a*) normal populations of high and low pig-bel prevalence and (*b*) in four comparative groups representing cases of pig-bel, their contacts, normal New Guinea population and European controls. Amounts of beta antitoxin 0·5 units per ml. were arbitrarily chosen as significant.

Table 6. *Statistical analysis of antitoxin studies of sera listed in Table* 5

		Comparison with	
	Percentage $\geqslant \frac{1}{2}$	High prevalence sample	Low prevalence sample
Row (iii) Group B	82·60	$P > 0{\cdot}05$ $0{\cdot}01 >$	$P > 0{\cdot}001$
Group D	95·83	$P > 0{\cdot}05$	$P < 0{\cdot}001$
Group E	79·41	$P > 0{\cdot}05$ $0{\cdot}01 >$	$P > 0{\cdot}01$
Group F	84·21	$P > 0{\cdot}05$	$P < 0{\cdot}001$
Group G	55·56	$P \simeq 0{\cdot}05$	$P > 0{\cdot}05$
Group H	9·52	$P < 0{\cdot}01$	$P < 0{\cdot}001$

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antitoxin, ten of which were greater than 5 units per ml. In the overall normal population sample of 216 persons (group C = A + B) 154 (71·3%) had significant antibody levels to the beta toxin. The statistical significance of these results is shown diagrammatically in Fig. 5. In the normal population there were significantly different levels between groups chosen from areas where the disease was

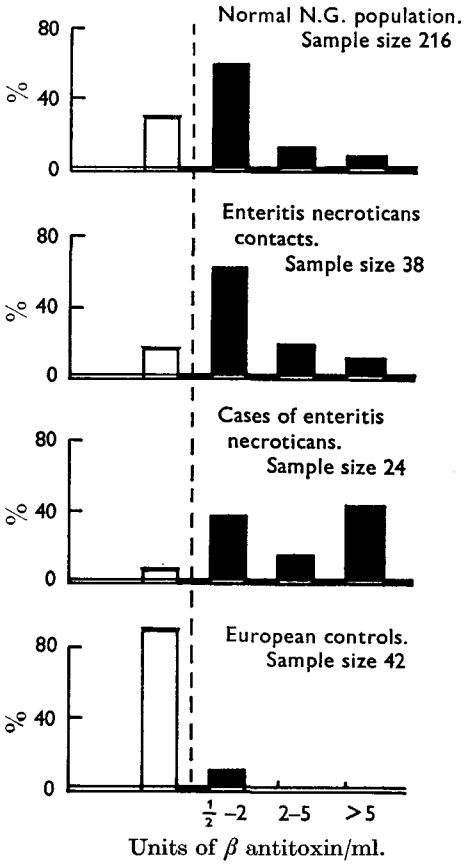

Fig. 6. Quantitative distribution of beta antitoxin in the four groups represented in the lower half of Fig. 8.

thought to have a high and low prevalence respectively, for example, groups A (i) + (ii) and B (i) + (ii). It was not possible to sample a large group of individuals before and after pig feasting. Detectable levels in relatives of persons with known pig-bel were significantly higher than in the normal population, cf. groups C and F (Table 5).

The comparisons of row (iii) of group B representing a 'blind' control group and each of the other groups with the low incidence frequencies and high incidence frequencies of the combined table are shown in Table 6.

Row (iii) group B and groups D, E and F corresponded with high incidence frequencies. Group G corresponded most closely with low incidence frequencies but the percentage $\geqslant \frac{1}{2}$ will be seen to be in a rather equivocal region between the

two extremes. Group H differed significantly from both high and low incidence groups with a percentage of $\geqslant \frac{1}{2}$ of 9·52 which is highly significantly lower than the low incidence percentage.

The number of sera containing more than 2 units/ml. of beta antitoxin was considerably higher in groups D, E and F. This upward trend is shown diagrammatically in Fig. 6. These results suggest that the immune response in individuals in these groups is the result of a greater or more prolonged exposure to beta toxin. They also indicate that antitoxin detection may be diagnostically reliable.

The frequency of detectable beta antitoxin in different age groups is shown in Table 7. It was highest in thirty-one persons above an estimated age of 40 years (90·3%). In the 16- to 20- year age group only 55·6% of those examined had detectable levels. Between these limits the frequencies were evenly distributed between 63·6 and 76·9%. There was no statistical difference in titres detectable in males and females (Table 8).

Table 7. *Age distribution of antitoxin titres in normal population*

Age group	No. examined	Detectable beta antitoxin per ml.	%
0– 5	7	5	71·4
6–10	11	7	63·6
11–15	22	14	63·6
16–20	18	10	55·6
21–30	39	30	76·9
31–40	55	40	72·7
40+	31	28	90·3
Total	183*	134	73·2

* Total population surveyed differs slightly from that in Table 8 because age and sex were not recorded on thirty-three occasions.

Table 8. *Sex distribution of antitoxin titres in the normal New Guinea population*

Sex	No.	Detectable beta antitoxin	%	
M	74	56	75·7	$P > 0{\cdot}05$
F	109	78	71·6	
Total	183	134	73·2	

DISCUSSION

Accurate measurement of the occurrence of enteritis necroticans proved difficult because there was no single pathognomonic criterion of clinical diagnosis. The figure of 48·3 per 10,000 at Goromaugo must serve only as a rough guide to assess the size of the problem.

In North Germany, with the exception of Lübeck, the incidence of Darmbrand ranged from 0·04 per 10,000 in 1946 to 1·77 per 10,000 in 1947. The maximal

incidence was 3·59 in Kiel in 1947 (Kloos & Brummund, 1951). In Lübeck the incidence was much higher, being 16 per 10,000, and the mortality was estimated to be 3·5 per 10,000 (Hansen *et al.* 1949). The mortality figure at Goromaugo was 13·8 per 10,000. The discrepancies between the two German figures were influenced rather by difference in definition and classification of the less severe forms of the disease.

Of 364 Darmbrand cases reviewed by Hansen *et al.* (1949) the overall death rate was 22%. In a series of 355 cases Kloos & Brummund (1951) estimated the case fatality rate at 41·4%. These latter writers were concerned mainly with the severe forms of the disease and included only thirty-eight in the mild group. Three other German authors gave high mortality rates for their case series. Ernst (1948) reported a mortality of 50% in Hamburg, Nissen (1950) a figure of 44% for the Landesteil-Schleswig area, and Griessman (1950) a mortality rate of 46% for 124 cases. These figures compare with an overall New Guinea case fatality rate of 36·0%. This case fatality rate was maximal at 52% in 1963, but fell to 30% in 1964 following the introduction of antiserum therapy in June of that year. The cumulative experience of managing cases preoperatively also helped to bring about this reduction (Murrell & Roth, 1963; Murrell *et al.* 1966).

The German outbreaks of Darmbrand had well defined seasonal distributions over the years 1947–48 and the disease reached pandemic levels during the mid and late summer months (July to September) of those years. The maximum incidence occurred in 1948, and then in the following 2 years declined and the disease disappeared as mysteriously as it had arrived. Jeckeln (1957) who had experience with a large number of patients in Lübeck, expressed the view that this intestinal disease, the most severe he knew, had abated. The seasonal or annual appearance of the disease in New Guinea was generally confined to the middle 6 months of the calendar year. This is in the 'dry' season of the year and as such, gardens were at maximum production and the absence of rain favoured outdoor festivities.

A noteworthy difference in the New Guinea disease from its German counterpart was the age distribution. The number of persons in the 0 to 10 year age groups was significantly higher (53·8%) than in any other decade. This distribution contrasts with Darmbrand, where most cases occurred in the fourth, fifth and sixth decades, although infants under 1 year of age were reported with the condition (Jochims, 1947). The mortality rose in the very young and most elderly patients, which was also the case in New Guinea. The higher incidence in children in New Guinea has similarities with the incidence of enterotoxaemia in sheep, calves, lambs and piglets caused by type D and C strains (Field & Goodwin, 1959). The occurrence of the disease in childhood in New Guinea is readily explained by the dietary practice associated with pork feasting. With the larger pig cycles occurring at 3–7 yearly intervals, it is possible that the younger age groups have an initial exposure to massive pork meals. Infants without teeth are not offered solid foods, and toddlers, who are breast fed up to the age of $2\frac{1}{2}$ to 3 years, are given only token amounts of pork. Only three patients in the series reviewed were under 2 years. The belief that pork imparts strength into the individual is practically demonstrated by encouraging children, especially males, to consume as much

as possible. Particular organs, such as the genitalia, liver and kidneys, are also reserved for children to eat. Burchett (1964—personal communication) reported that in the Baiyer River outbreaks in 1962 children actively refused further meals offered to them by their parents because of their fear of becoming ill.

The reluctance of older people to seek medical care, the general regard that illness in the older generation is due to 'old age' and lower life expectancy, account for the fewer case reports in people over 40 years of age. The life expectancy of the Highlander is probably lower than that estimated in life tables by Scragg (1954) for New Ireland natives of New Guinea.

The sex distribution in New Guinea may be influenced by the greater frequency with which males seek medical care as indicated by a higher bed occupancy rate both in Papua (Campbell & Arthur, 1964) and the Territory as a whole (Department of Public Health, 1964). However, the survey at Goromaugo indicated that twice as many males as females were affected. The women do eat more of the cooked bowel of the pig than the men. If heat resistant spores of the clostridial organisms survive the cooking, as well they might, a higher morbidity of illness should be expected in the females. The New Guinea type C strains did not exhibit the heat resistant quality that the type F strains did in Europe (Egerton & Walker, 1964). More females contracted the disease than males in the 16– to 20-year age group. At marriage feasts the bride and prospective brides are encouraged to consume unusually large amounts of pork to encourage fertility. This possibly explains the distribution of the disease in this group.

The evidence that large scale pig-killing activities influence the prevalence and spread of enteritis necroticans in the Highlands is circumstantial. It would appear from the trends in admission figures that this cultural practice influences the prevalence of all diarrhoeal diseases in the localities of the hospitals reviewed.

The experience of one of us (T.G.C.M.) at Wabag and Kundiawa left the impression that pig-bel varied considerably in density in the two areas. This impression was borne out by the differing titres of beta antitoxin in individuals sampled in the upper Chimbu and Wabag areas after pig feasting. An alternative explanation of the variable prevalence may lie with the actual feasting habits of the tribes concerned. In the Chimbu it became apparent that the admission rate rose as the feasting continued. In the Goroka and Chimbu areas, there was also a small introductory pig-kill which was the overture to the larger massive feast. This took place 2–4 weeks earlier and was not a feature of pig-kills witnessed in the Western Highlands.

A more accurate index of the morbidity and mortality following pig feasting could be obtained by measuring the diarrhoeal attack rate in a population sample along the lines of the WHO team investigations (Ordway, 1960). Difficulties apparent in this approach were found to be a reluctance of the population to implicate pork as a cause of diarrhoea, and the problem of accounting for people before, during and after the feasting, due to the transient migrations which naturally occurred at these times. The proven cases at Goromaugo and the subsequent appearance of documented cases at Kurumogl and Gena after consuming pork prepared by people from Goromaugo supports the argument that

pork is a vector of the disease. A significantly high level of *Cl. welchii* beta antitoxin was found in contact persons at risk. The failure to demonstrate type C strains in other than clinical cases suggests a low incident 'carrier rate' in either the human or porcine population. The prolonged exposure of samples to aerobic conditions before plating probably accounted for this failure because the immunological studies provided strong support for a significant exposure to the beta toxin of *Cl. welchii* in the normal Melanesian population. It is unfortunate that a parallel study was not undertaken in pigs because this may have provided some added information on the origin of *Cl. welchii* type C.

The generally high immune status of the population could explain the absence of massive epidemics of the disease. At Bundi, where many deaths occurred, the previous large pig feast had been held 7 years earlier. Here the population was not as dense as in the Chimbu and a significantly high non-immune population may have built up during the 7 years' absence from massive pork feasting. In this regard, the fluctuations in the disease prevalence simulate those of some other diseases such as chicken pox and measles, their endemicity being dependent on the non-immune pool. In the Chimbu, it was established that the time interval between the larger feasts was only 3 years. This factor could therefore influence the variance of disease from one place to another.

A future investigation of the immune status of new-born infants and toddlers may provide information on the age at which exposure to *Cl. welchii* type C first takes place. Samples taken in the 0- to 5-year age group were too small to make any meaningful conclusions. Examination of faeces for the presence of beta toxin is another important avenue for further elucidation of the epidemiology and aetiology of pig-bel.

In limited experiments with guinea pigs, intraduodenal inoculation of type C strains reproduced the disease whereas feed contamination with this organism failed (Egerton, 1966). Whilst food poisoning seems the most likely cause of pig-bel, this has yet to be demonstrated. Considerable problems face the investigator in determining family outbreaks of food poisoning that may follow pig feasts in New Guinea. The difficulties include the primitive state of the people; the lack of adequate communication, and the deficient pathology services available in Highland hospitals.

The variable incubation period in pig-bel suggests that both exogenous and endogenous infection may be responsible in the pathogenesis. The suggestion of Dische & Elek (1957) that toxic effects of *Cl. welchii* enteritis depend upon the presence of living clostridia in the bowel rather than pre-formed toxin supports this argument.

The pathogenesis probably starts by the action of powerful toxins in the upper intestine and this genesis is then stimulated by additional factors, intrinsic and extrinsic, favouring an overwhelming toxaemic infection of the host. The role that other faecal and oral flora play in this toxaemia requires further investigation. Differential bacteriological analysis of organisms of the bowel flora such as *E. coli*, *Paracolon* sp., *Bacteroides* sp., *Streptococcus faecalis* and *Proteus miribalis* normally present in bowel above obstructed areas (Bishop & Allcock, 1960) needs to be

worked out in the New Guinea disease. It is believed that pig-bel bears an epidemiological relationship to the enterotoxaemias of animals where dietary change and over-feeding play such an important role in their initiation (Bullen & Scarisbrick, 1957). Exogenous infection more probably occurs in the young whilst an endogenous infection by resident gut flora, stimulated by local changes, can be postulated in older age groups. The age distribution of clinical types of pig-bel favours this thesis.

The Highlander eats his pork in hugh quantities over a period of weeks or months during the larger pig festival seasons. Then follows a relative fast until the next celebration. Heavy and continuous pork indulgence therefore occurs only once or at the most twice every 3–7 years or possibly longer. There are occasions, however, when smaller festivities as death, marriage or illness call for a small sacrifice of one or more pigs. The mean weights of pork eaten at such a ceremony were estimated by Venkatachalam (1962) at 29 and 26 g. per day in children in the 10- to 15- and 5- to 10-year age groups respectively. At a large pig-kill at Goglme the mean weight of five pork meals consumed by adults and children was 652 g. Volumetric measurements were not made, but the total engorgement of food at one sitting would approximately amount to between 2 and 3 l., more than half of which was made up of pork. This unaccustomed diet is both qualitatively and quantitatively in excess of the average daily diet.

The diet of the Chimbu people has been studied by several workers (Oomen & Malcom, 1958; Venkatachalam, 1962; Bailey & Whiteman, 1963). This consisted of a daily protein intake of between 25 and 30 g. The calorie intake was between 1850 and 2883, very little of which was made up by fat. The conclusion was that such a low protein intake, mostly arising from vegetable protein, amounted to chronic protein malnutrition, particularly in the very young and elderly. Therefore, an intestinal tract primarily conditioned to a vegetarian diet, the bulk of which is a carbohydrate staple of sweet potato, suddenly becomes confronted with large meals rich in animal protein and fat. The stomach of the Highlander is well conditioned to meals of large bulk, because as much as 1500–2000 g. of sweet potato make up a normal meal (Bailey & Whiteman, 1963). However, in order to handle the qualitative change in dietary content, there must be a change in bacterial flora and enzyme secretion in the gastro-intestinal tract. In children it is possible that this change may induce bowel atony. The high helminthic load, so often present in children of the 4- to 10-year age groups, may adversely influence this change, the nett result being stasis of intestinal content and bowel distension favouring the proliferation of existent and ingested bacteria. Yasnogorodsky (1936) drew attention to the production of paralytic ileus caused by over abundant, unaccustomed, indigestible or spoiled food. In South India, a condition of spontaneous paralytic ileus has also been reported. This is considered to be due to a diet of spoiled millet ragi, the staple food (Roantree, 1949).

Pietzonka & Rassfeld-Sternberg (1950) supported the suggestion of Schütz (1948) that there was a history of an antecedent diet rich in protein, usually of improperly cooked or canned meat preparations, in persons with enteritis necroticans in Germany. Thus in Germany, soon after World War II, a similar situation existed

in the population to that in New Guinea, whereby a population subsisting on meagre rations, was suddenly tempted by an influx of richer foods when hostilities ceased. This unbalanced situation, in which the diet consisted chiefly of carbohydrates, is believed to have been a factor underlying the epidemics in both Norway and Germany.

The success of specific *Cl. welchii* type C antiserum in therapeutic trials (Murrell *et al.* 1966) both supports our hypothesis as to the aetiology of pig-bel and provides an opportunity for the future control of the disease using toxoided preparations. Eradication of the disease and other food borne enteric infections, however, lies in an understanding of the beliefs and customs relating to pig raising and ownership in Highland cultures. The efforts of the social anthropologist, epidemiologist and bacteriologist will have to be married if success is to be achieved in the reduction of the mortality and morbidity which result from unhygienic pork consumption in New Guinea.

SUMMARY

1. Features in the epidemiology of a spontaneous enteric gangrene in the Highlands of New Guinea are described.

2. The disease has been called pig-bel because of its firm association with the pig-feasting practices of the people, which occur in 3–7 year cycles.

3. *Cl. welchii* type C is believed to play an important role in the pathogenesis of the condition. Strains isolated were strongly toxigenic and uniform in their toxin production.

4. A food poisoning aetiology was not proved but circumstantial and immunological evidence suggest that pork may be a vector of the disease.

5. The source of *Cl. welchii* type C was not established.

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EXPLANATION OF PLATES

PLATE 1

(*a*) A preliminary count of pigs at a 'Te' festival near Wabag in the Western Highlands of New Guinea.

(*b*) Dissection and evisceration of singed carcasses in progress. The animals are not bled prior to butchering (Upper Chimbu).

PLATE 2

Half sides of pork are placed in a long leaf lined earth oven and cooking occurs from pre-heated stones. (Enga 'Te' pig-kill at Tambul in the Western Highlands.)

(*a*)

(*b*)

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