AN EPIDEMIC OF INFANTILE GASTRO-ENTERITIS IN QUEENSLAND CAUSED BY *SALMONELLA BOVIS-MORBIFICANS* (BASENAU)

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(With 6 Figures in the Text)

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INTRODUCTION

Gastro-enteritis in young children is still a major problem in Queensland, as, indeed, it is throughout Australia and in many other parts of the world. Arden (1948) considers that, in this State, it 'now heads the list of killing diseases of infancy'. Its general course over the past 32 years is shown in Fig. 1. In earlier years 'summer diarrhoea' was the predominant infection, but its incidence decreased after the 1914–18 war (Turner, 1938), leaving a non-seasonal residue, which swelled into the succession of peaks shown during the recent war years.

With all this material available and so much to gain from precise knowledge, it is surprising that no detailed bacteriological investigation has been reported previously. Turner (in discussion of Litchfield, 1921) 'thought the early summer epidemics were dysentery and were fly-borne, but lacked bacteriological evidence'. Croll (1946) agreed in retrospect with this view, and North (1935) included at least two young children in the series of Flexner dysenteries he reported from Rockhampton. Arden (1948), on the other hand, considers that bacillary dysentery is not now a common type of infantile diarrhoea, and mentions an outbreak of neonatal diarrhoea in 1943 caused by 'paratyphoid B' bacilli. Atkinson, Woodroofe & Macbeth (1947) list only three infections in children among the strains of *Salmonella* received from Queensland, namely, two cases of meningitis due to *S. typhi-murium* and one of gastro-enteritis caused by *S. bovis-morbificans*. The experience of ourselves and the General Hospital Laboratory during 1947 and 1948 indicates that Salmonella infections are quite prevalent (Mackerras & Mackerras, 1949*b*), that dysenteries are relatively uncommon, and that the majority of infections in infants must still be relegated to the unsatisfactory 'uncertain etiology' group.

Superimposed on this general level of endemic infantile gastro-enteritis, on which a great deal of work obviously remains to be done, there came the clearly defined epidemic, which it is the purpose of this paper to describe.

THE EPIDEMIC WAVE

Fig. 1 shows that there was a great increase in the number of cases of gastro-enteritis treated in hospital from 1942–3 onwards, which Stable & Philpott (1948) associated with 'a vast increase in city population and resultant overcrowding of the city area'. The mortality, however, remained low until 1946–7, when a sharp rise occurred. These authors considered also, on clinical grounds, that the epidemic developed in two phases: a phase of gradually increasing severity of infection from November 1946, and the frankly virulent epidemic from April 1947. We have not found numerical evidence of the first phase, but the second is shown in Fig. 2, in which the data are entered by months of onset in order to give a true picture of the children who became ill each month and their fate.

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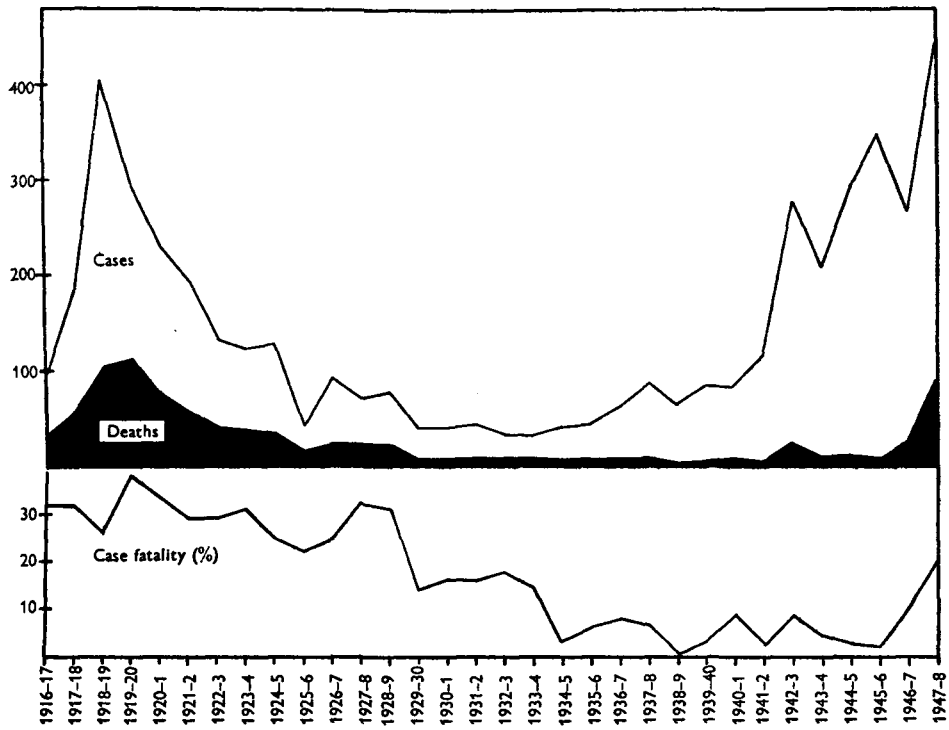

Fig. 1. Incidence, deaths, and case mortality of gastro-enteritis in children under 2 years old from 1 July 1916, to 30 June 1948. (From the records of the Brisbane Children's Hospital.)

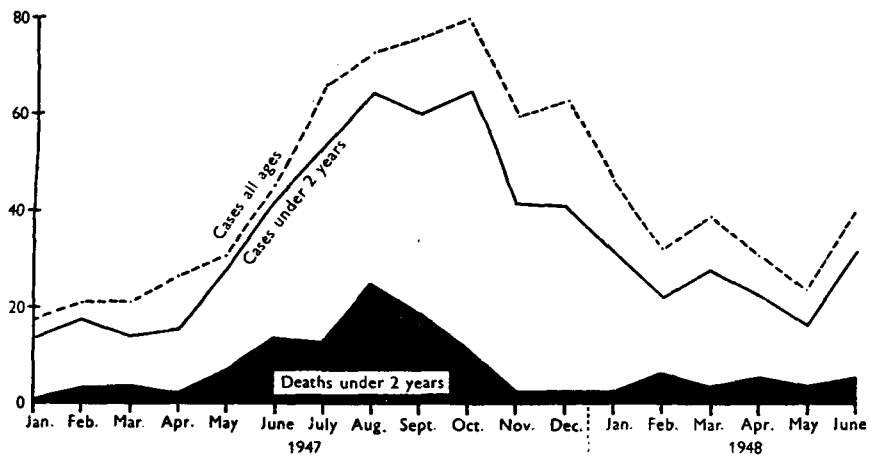

Fig. 2. The 1947 epidemic as recorded by month of onset. *Note.* There was one death of a child over 2 years old; it is not included in the graph.

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It is apparent from Fig. 2:

(1) That clinical infection was largely restricted to children under 2 years of age.

(2) That new infections continued to be numerous until October, and the curve of incidence was roughly symmetrical about the month of September.

(3) That substantially the whole of the increased mortality of the past two fiscal years was compressed into the 6 months from May to October 1947, the highest fatality rate being in cases with onset in August, after which there was a steady decline.

RELATION OF *SALMONELLA BOVIS-MORBIFICANS* TO THE EPIDEMIC

Bacillus bovis-morbificans was described by Basenau in 1894. He isolated it at Amsterdam from a cow, which had developed metritis after calving, and he gave a detailed description of its behaviour in the then available culture media and in experimental animals. Since then, it has been found in other parts of Europe, in food poisoning in man in Great Britain (twenty-two reports in as many years—Haines and Wilson in M.R.C., 1947) and South Africa (Henning & Greenfield, 1937), once in India (Hayes & Freeman, 1945), and in a few human infections in the United States (Seligmann, Saphra & Wassermann, 1943, 1946; Angrist & Mollov, 1946). The last named described an outbreak of six mild infections in premature infants. Hormaeche, Surraco, Peluffo & Aleppo (1943) did not record it among the 537 strains of *Salmonella* isolated from children in Uruguay.

The first record of *S. bovis-morbificans* in Australia was by Stewart (1940*a*, *b*), who identified it initially from sheep and later from pigs with pneumonia. Atkinson *et al.* (1944, 1947) list the following infections in man identified by them prior to the present outbreak: children with gastro-enteritis—eleven cases in South Australia, one in Victoria, and one in Queensland, which was isolated in Brisbane in April 1945; adults with gastro-enteritis—two in New South Wales, four in North Queensland. They also found it twice in sheep's kidneys in South Australia, while Albiston (1947) records it from a foal and from the faeces of a cow in Victoria. Mr G. C. Simmons (personal communication) has found it in a duck in South Queensland.

Thus *S. bovis-morbificans* is widely distributed in Australia, and is known from a variety of domestic animals as well as man. Miss Atkinson informs us (personal communication) that it comes next after *S. typhi-murium* in general frequency of occurrence, and that most of the human infections of which she has records have been relatively mild.

Evidence of pathogenicity

When a species of *Salmonella* is frequently isolated from a series of patients, it is customary to regard them as suffering from some form of clinical salmonellosis, but this is not always true. The evidence that *S. bovis-morbificans* was an active pathogen during the epidemic under discussion is:

(1) The frequency of its occurrence in typical severe cases recognized clinically during the epidemic (Figs. 3 and 4; Stable & Philpott, 1948.*

(2) The occurrence of agglutinins for its 'H' antigens (though not for the 'O' antigens) in sera collected from some of the cases during the epidemic (Miss Atkinson, personal communication).

(3) Occasional positive blood cultures in life, and frequent post-mortem evidence of widespread parenteral infection with associated severe liver damage (Mackerras & Mackerras, 1949*b*).

Numerical relation to the epidemic

Two sets of data are available for this analysis, those relating to the total number of cases, and those relating to the deaths. Restricting observations to the period May to December 1947, and to the age group under 2 years, there were 391 cases of gastro-enteritis, among which 128 *S. bovis-morbificans* infections were recognized. The details are set out in Table 1, and the chronological sequence of the *bovis-morbificans* infections in Fig. 3.

Table 1. *Gastro-enteritis, May–December* 1947

Group	No.	Deaths	Mortality (%)
S. bovis-morbificans	128	49	38
Other *Salmonella*	24	2	8
No pathogens isolated	112	21	19
Not cultured	127	17	13
Total	391	89	23

It is apparent from Table 1 that those cases from which *S. bovis-morbificans* was isolated were not a representative sample of the whole, but that many less fatal infections were occurring at the same time. On the other hand, there is evidence that not all the *bovis-morbificans* infections were identified bacteriologically. The investigation did not begin until mid-July, so the early records are undoubtedly incomplete, and are therefore enclosed in broken lines in Fig. 3. Even in the later stages, we did not receive material from every clinically typical case, nor did we always isolate *S. bovis-morbificans* when we expected to, as the following example will indicate:

P.W., aged 16 months, had a severe, prolonged attack of gastro-enteritis; two brothers had proven

* Other organisms were present in many specimens, alone or in company with *Salmonella*. Species of *Proteus* were almost universal, without regard to clinical type or stage of infection; several kinds of paracolon bacilli were frequent; *Pseudomonas aeruginosa* sometimes occurred. They often appeared in small 'waves', but none showed any consistent relationship to the clinical picture or the epidemic wave. We lacked means to search for a virus.

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S. bovis-morbificans infections (one died), a sister died of acute gastro-enteritis (not diagnosed bacteriologically). His stools were examined three times, with negative results. Miss Atkinson found agglutinins for the 'H' antigens of *S. bovis-morbificans* in his serum to the following titres:

	17 July (ill)	5 Sept. (convalescent)
Specific	1 : 320	1 : 40
Group	1 : 160	1 : 20

caused by *S. bovis-morbificans*, making a total of nearly 200 cases in the epidemic proper, the remaining nearly 200 being a continuation of the enhanced war-time level of endemic gastro-enteritis in the community.

More definite evidence that *S. bovis-morbificans* played a major part in the epidemic is derived from consideration of the fatal infections. There were eighty-nine deaths in the group; for seventeen there is no bacteriological or post-mortem information, leaving seventy-two for analysis. Of these: forty-

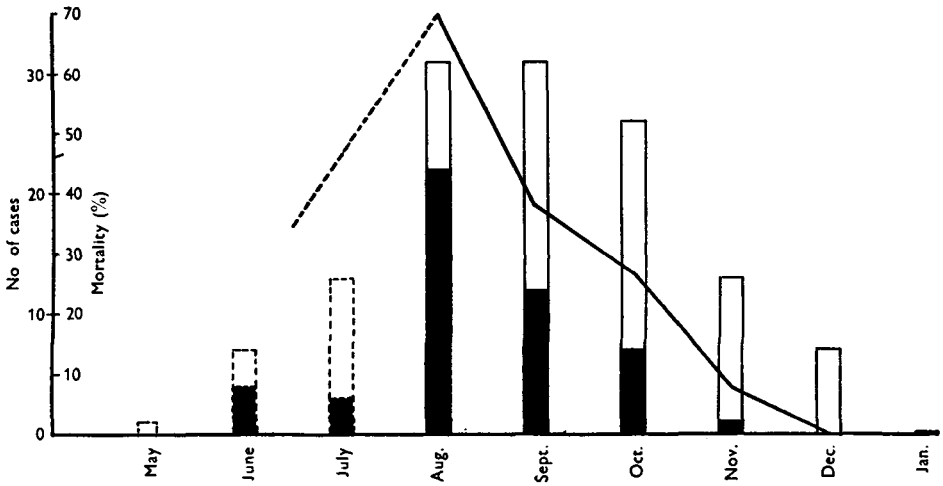

Fig. 3. *S. bovis-morbificans* infections in children under 2 years old with gastro-enteritis, arranged by months of onset. The histogram shows numbers of cases, with deaths in black; the line indicates case fatality rate. Only three infections in this age group have been found in 1948, two in March and one in June; none was fatal.

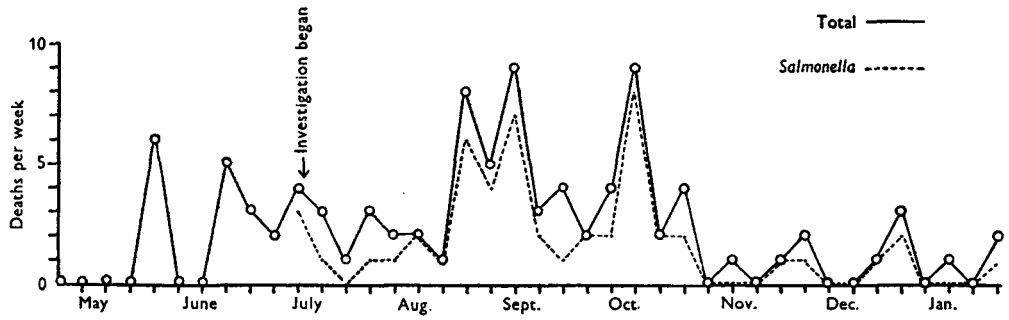

Fig. 4. Weekly deaths from gastro-enteritis. The solid line shows all cases; the broken line the Salmonella infections.

We have analysed the reliability of the bacteriological methods elsewhere (1949*a*).

It is difficult to estimate how many *bovis-morbificans* infections are hidden in the unidentified groups. If, as seems reasonable, it is assumed that the fatality rate of the other infections did not change from that prevailing before the epidemic, then a comparison of the rates suggests that about one-quarter of the 239 unidentified cases (including about two-thirds of the deaths) were probably

eight had *S. bovis-morbificans* infections; one had *S. typhi-murium* with a superadded *S. bovis-morbificans*; two had *S. typhi-murium* alone; seven had clinical and post-mortem signs compatible with Salmonella infection, but negative post-mortem cultures (one had 'H' agglutinins in the serum); and the remaining fourteen were classed as indefinite (either negative cultures in life and no post-mortem permitted, or negative post-mortem cultures and equivocal clinical and post-mortem findings). The

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inference from these findings is supported by the weekly distribution of deaths, which is set out in Fig. 4.

Severity of infection

Not only were the *bovis-morbificans* infections generally more severe than others occurring at the same time, but it is apparent from Fig. 3 that the fatality rate waxed and waned during the course of the epidemic. Thus, the appalling rate of 71 % for cases with onset in August was significantly higher than the combined rate for June–July or that for September ($P < 0{\cdot}02$ and $< 0{\cdot}01$ respectively). It is important to note, too, that the decline in September occurred when new infections were still numerous and the dosage of infection was still undoubtedly high.

Rubbo (1948) and Mushin (1948) considered that the severity of the infections with *S. derby*, which they studied in Melbourne, was influenced by other diseases from which the patients were already suffering, and observed that healthy children might have no more than a mild, transitory attack. There was no evidence of any such effect in our epidemic series, and a statistical comparison of mortality in those infected in hospital and those infected outside showed no difference between the two groups.

Improvements in treatment and ward hygiene began towards the end of September, and could have influenced the later mortality. Their effect on cases with onset in September was not expected to be large, but it was considered desirable to have a separate check. All cases were therefore classed as 'severe' or 'mild' on the basis of whether or not they were given intravenous fluid, the results being set out in Table 2. These figures are independent of the improvements indicated, and again the difference between the August and September groups is highly significant ($P = 0{\cdot}001$).

Table 2. Salmonella bovis-morbificans *infections in children under 2 years old*

	June–July	Aug.	Sept.	Oct.	Nov.	Dec.
Mild	6	2	13	9	4	5
Severe	13	29	18	17	9	2

Discussion

Taking all the evidence into account, it is reasonable to conclude that *S. bovis-morbificans* was in fact highly pathogenic, and that it was responsible for at least half the cases of gastro-enteritis and the great majority of the deaths during the epidemic period. There is, further, a strong suggestion that the epidemic was due to the development of a transitory epidemic strain of enhanced virulence and high infectivity, in *S. bovis-mortificans*, which both before and since has been of comparatively mild behaviour. But, if this is so, we still cannot explain why virulence was increased only for a particular age group; nor can we offer any direct evidence of increased infectivity, because there was during the period no observation which could not be as readily explained by the accumulation of infective material where it could be transferred to other children, as by an increased ability of the organism to bridge gaps previously too wide for it.

SOME GENERAL FEATURES OF THE INFECTION

We do not propose to discuss the clinical features of the infection, an account of which has been given by Stable & Philpott (1948), nor the pathology, which we have outlined briefly elsewhere (1949*b*). There are, however, some observations which require mention because they are significant in relation to epidemiology.

Age incidence

The general relation of the epidemic to age is indicated in Fig. 2, and the figures for clinically active *S. bovis-morbificans* infections in children up to 8 years old are set out in Fig. 5. It is apparent:

(1) That 77 % of cases were in children under 1 year, 19 % in those between 1 and 2 years old, and 4 % in those 2–8 years old. The highest incidence was in the group 6–8 months old.

(2) That the fatality rate rose slightly in the first 12 months of life (trend not statistically significant), and thereafter declined rapidly. These figures are supported by an analysis of severity of infection, using the same criterion of severity as before, namely, the receipt of intravenous fluid (Table 3). The difference between the first and second year of life is highly significant ($P < 0{\cdot}001$).*

The figures suggest: (1) that there was little or no passive protection from maternal immunity in the early months; (2) that there was a rapid decrease in susceptibility to infection after the first year of life. Dosage of organisms would not account for the whole of the latter phenomenon, because it would have been as high (possibly higher, owing to the greater activity of the older babies) in the second year as in the first, though it would probably have decreased considerably thereafter. Whatever the explanation, the essential epidemiological point is that the infection was confined for all practical purposes to the 'napkin age', that is, to those children whose toilet was performed for them by an adult.

* The criterion of severity may not be as reliable here as in Table 2, because clinicians may tend to give intravenous fluid more readily to the younger babies. The figures are, however, in accord with general clinical observation.

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There is one further point about age. A scatter diagram was constructed to show age in relation to time during the epidemic. The distribution was uniform, and it may be taken therefore that the data in Fig. 3 were not influenced by variations in age distribution of the patients.

Relation to feeding

We may assume that all babies more than 6 months old received at least some supplementary feeding. Of forty-four up to 6 months of age, for whom information is available, only one was recorded as fully breast-fed. Dr H. C. Murphy, Director of Maternal and Child Welfare, informs us that about 70 % of babies of this age group attending the clinics in Brisbane are fully breast-fed, so it is clear that artificial feeding was a significant aetiological factor in the epidemic.

Duration of infection

The duration of clinical illness was difficult to determine, because the onset was sometimes insidious, and many cases showed a marked tendency to improve and relapse, often dragging on intermittently for 2 or 3 months. Not infrequently they were discharged in an intermission, only to be readmitted a few days later. The following examples illustrate what happened:

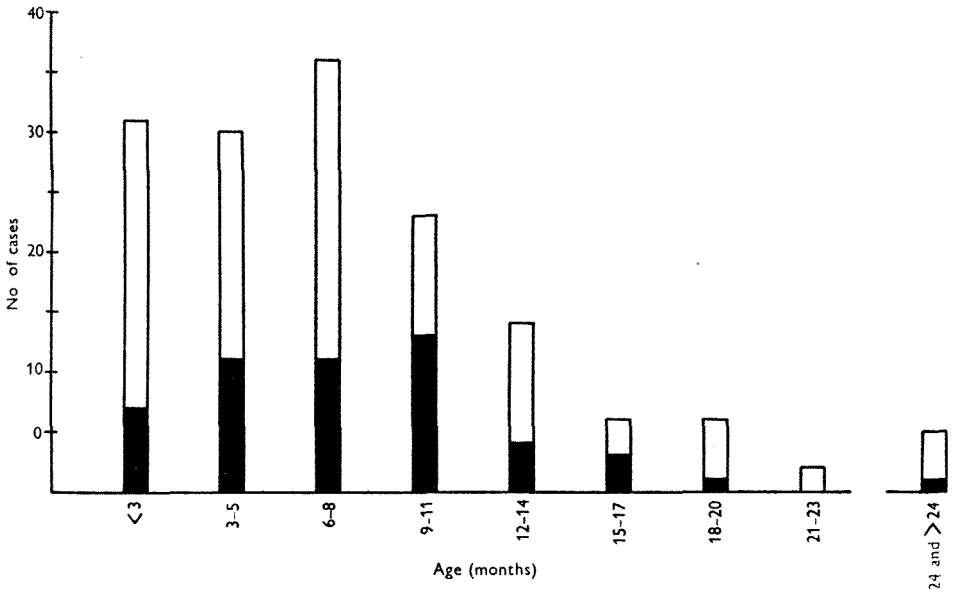

Fig. 5. Clinical infections with *S. bovis-morbificans* from May to December 1947, tabulated by age at onset, deaths being shown in black. Ages are grouped in 3-monthly periods, from and including the first 3 months of life.

Table 3. *Age distribution and type of primary infections with* Salmonella bovis-morbificans, *May to December* 1947

Age group	Carriers	Cases		Deaths	Total primary infections
		Mild	Severe		
Less than 1 year	1	24	78	41	103
1–2 years	3	15	10	8	28
2–8 years*	3	4	1	1	8
Adults	3	1	0	0	4

* No infections were found in children more than 8 years old.

S.W., aged 9 months. Onset about 1 August, admitted 13 August, 'made good recovery' and discharged 25 August; second onset 27 August, readmitted 30 August, *S. bovis-morbificans* isolated 8 September, died 30 September.

R.C., aged 11 months. Onset 30 August, admitted 4 September, *S. bovis-morbificans* isolated 8 September, improved after intravenous drip saline

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and discharged 10 September; readmitted 12 September with recrudescence, discharged relieved on 20 September; *S. bovis-morbificans* isolated from faeces repeatedly until 16 December.

D.T., aged 9 months. Onset 30 August, admitted 2 September, *S. bovis-morbificans* isolated 9 September; discharged ('does not look very ill') 10 September; readmitted with recurrence 15 September, died 4 October.

There was, thus, difficulty in determining duration of illness even in those that died, seven out of fifty fatal cases being of somewhat uncertain duration. Making allowance for these, the duration of fatal infections varied from 4 to 107 days, with a mean of $26 \pm 2{\cdot}6$ days. The mode also lay in the fourth week, and three-fourths of the deaths occurred in the first 4 weeks.

It soon became apparent (see, for example, R.C. above; also Draper, 1944; Foley, 1947) that infection, probably augmented by reinfection in earlier cases, often continued for considerable periods after clinical cure, and it was arranged that no patient would be discharged until three consecutive negative stool cultures had been obtained. Many of these babies were apparently quite well and producing perfectly normal stools; yet they repeatedly gave abundant pure cultures, suggesting that the Salmonella may have been present in very large numbers. We have no evidence on the anatomical site of this residual infection.

Broadly, we may say that about two-fifths of the patients lost their infections within 1 month, one-quarter were infective for 1–2 months, and one-third were infective for more than 2 months. The details are set out in Fig. 6, and the last case is worth a special note.

Baby B. Born 28 November, onset of enteritis on the fourth day after birth, *S. bovis morbificans* isolated same day; discharged at mother's request, quite well, 21 January. *S. bovis-morbificans* isolated fifteen times out of sixteen cultures of faeces between 2 December 1947 and 25 June 1948; the family then went to the country, the baby perfectly well but still a carrier, having been infected for at least 206 of the 210 days since his birth.

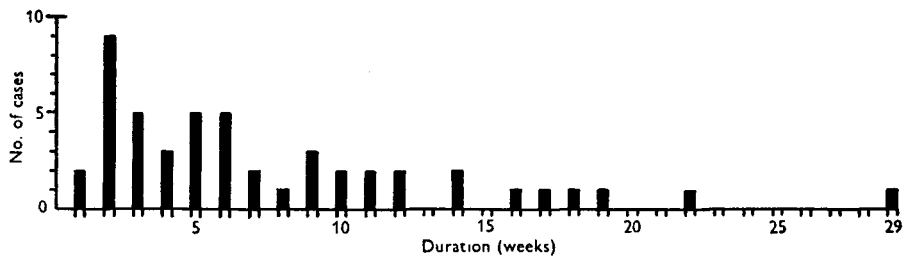

Fig. 6. Duration of infection from onset of illness in forty-nine of the children under 2 years old who recovered from *S. bovis-morbificans* infections.

The numbers are not large enough for a detailed analysis of age, but it is noteworthy that all the infections which lasted more than 6 weeks were in babies under 1 year old. Duration in the 1 to 2-year-old group was 2–6 weeks; while, of the five children over 2 years old with clinical infections, one died on the twenty-sixth day of illness, one was in hospital for 17 days and was only examined once, two were cleared bacteriologically within 2 weeks, and one was in hospital for 6 days with a very mild illness and was not followed after one subsequent negative stool culture.

The inapparent infections in older children and adults (Table 4) appear also to have been of short duration. Three adults and two children, from whom *S. bovis-morbificans* had been cultured, were re-examined after a fortnight's interval, and none was found to be still infected: three consecutive negative samples were obtained from four of them, two from the fifth.

For comparison, we may mention that the duration of infections with other species of *Salmonella* studied during the same period varied from a maximum of 9 weeks in clinical infections in babies to a few days in inapparent infections of older people.

Distribution of infections

All the known infections occurred in the greater Brisbane area; it was important from the point of view of prevention to discover, if possible, where they were originating. Before these studies began, there was a tendency to focus attention on the temporary housing areas, former Service camps, where considerable numbers of families were living a rather crowded existence under conditions of discipline and hygiene which were certainly not up to Service standards. However, there was also plenty of published evidence (for example, Campbell, 1945; Angrist & Mollov, 1946; Wright & Wright, 1946*a*, *b*; Rubbo, 1948) concerning diarrhoea in children's hospitals and other institutions, and experiences such as the following led us to examine more closely the possibility of institutional infections:

D.C., aged 6 months, was admitted to the gastro-

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enteritis ward on 16 July with a *S. typhi-murium* infection; progressed well until 29 July, then relapsed, and died 12 August. At post-mortem *S. typhi-murium* was found in liver and spleen, *S. bovis-morbificans* in intestine.

J.T., aged 7 months, was admitted on 3 August to surgical ward for division of webbed figers; vomiting and diarrhoea began 15 August; *S. bovis-morbificans* isolated from faeces 19 August, and from liver and spleen post-mortem on 28 August.

S.J., aged 3 weeks. Abandoned by mother, was admitted to hospital 27 July suffering from exposure; onset of vomiting and diarrhoea 10 August. *S. bovis-morbificans* isolated: recovered.

It is easy to cull convincing examples, but more difficult to assess series, for that requires fairly precise estimation of the incubation period. The incubation period is generally believed to be short in acute primary Salmonella gastro-enteritis (Bornstein, 1943). Stable & Philpott (1948) give it as 2–10 days for the present series of *S. bovis-morbificans* infections, and with this we would generally agree. Three neonatal infections with onset 2 days after birth indicate the lower limit, and one baby, with clinical onset 14 days after it was found, in the course of a ward survey, to be infected, is the longest incubation we have encountered. As there will usually be a lag between exposure and actual infection, the most frequent incubation periods are probably nearer the lower than the upper limit. To be within safe limits for the present analysis, we have accepted onset of symptoms 3 days or more after arriving in a place, or within 3 days after leaving it, as indicating that infection occurred in that place. On this basis, the *bovis-morbificans* infections in children under 2 years old (including two intercurrent on other intestinal infections) fell into the following groups:

Maternity hospital (neonatal)	11
Children's hospital	59
Other institutions for children	13
Temporary housing camps	17
City and suburban	23
Origin uncertain	10
Total	133

Thus, 62 % of infections were institutional, and the ten uncertain cases also had some institutional association, though not sufficiently close to comply with the standards we adopted. Some of the 'outside' infections had an indirect hospital association, too. Thus K.A., with onset 1 day after discharge, was listed as a hospital infection, but his sister, with onset 3 days later, was not so listed, because she was a home contact. In a different category are cases admitted with gastro-intestinal symptoms, and therefore classed as 'outside' infections, but in whom the organism was not found for a considerable time, the most extreme being a boy aged 8 months from whom fifteen negative cultures were obtained in 5 weeks before *S. bovis-morbificans* was isolated from his faeces. Rubbo suspected that similar cases in Melbourne were probably intercurrent Salmonella infections acquired after admission, and we would agree, but they are not so listed unless the cause of the primary infection is known.

Clearly the first task in prevention was to reduce spread within institutions and dispersal from them into the general community, especially into places where the infant population was high and secondary centres of infection might develop.

The results described above differ materially from what happened in *S. bovis-morbificans* infections in older children and in infections with other species of *Salmonella*.

(*a*) Of ten *S. bovis-morbificans* infections in children 2–8 years old in 1947, three developed while the patients were in the gastro-enteritis ward with other intestinal infections, and the other seven originated in widely scattered parts of Brisbane.

(*b*) Of thirty-two clinical infections with other species of *Salmonella* identified in 1947, twenty-five were in the under-2-year-old group, three only originating in institutions.

(*c*) Of thirty-seven infections with nine species of *Salmonella* in 1948 (including five *S. bovis-morbificans*, three in children under 2 years old, two in older children) only five originated in institutions.

These contrasting findings in endemic Salmonella infections lend support to our impression that the epidemic was predominantly institutional, because its development was due to institutional life.

SOURCES OF INFECTION

Having established where the infections were occurring, the next major step was to determine their sources. Were sick infants the chief source of infection, as at first appeared? Or were there hidden reservoirs in the human or animal population, from which the level of infection in the infants was continually being replenished? It is to be remembered that the epidemic was already well developed when these investigations began, so that we were inquiring into the sources of *epidemic* spread. The sources of the scattered *endemic* infections, and the actual origin of the epidemic itself, are different problems, about which we can say nothing at present.

Infants with clinical infections

Our findings in this group are described in other sections, so it is only necessary to emphasize the salient points:

(1) 96 % of clinical infections were in babies less than 2 years old (Fig. 5).

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(2) They were infective, not only while ill, but also in the incubation period, and often for long periods after clinical recovery (Fig. 6).

The faeces of these infants provided an enormous mass of infected material in the wards and the homes throughout the epidemic period. Our own sampling alone gave 274 positive cultures from the faeces of eighty-eight children in 6 months, apart from numerous isolations by the General Hospital Laboratory. The clinical infections in older chidren and adults, few in number and brief in duration, were insignificant by comparison. Moreover, disposal of their faeces did not present anything like such a serious problem in nursing technique.

Attention was concentrated on the faeces as the most probable infective material. We did not examine pharyngeal secretion (see Hormaeche *et al.* 1943; Colbeck & Neisser, 1948), but three swabbings from faeces and one culture of vomit from babies with active *S. bovis-morbificans* infections were negative. Of urines from eight of the male patients (not catheter specimens), one gave a pure culture of *S. bovis-morbificans*.

Table 4. *Surveys for carriers*

Group	Ages	No. of individuals	No. of specimens	*Salmonella* isolated
		1947. Epidemic series		
Ward survey	Under 2	26	32	6 *S. bovis-morbificans*
	2–11	22	24	2 *S. bovis-morbificans*
State Home for Children	Under 2	36	50	1 *S. bovis-morbificans* 2 *S. adelaide* 1 *S. pullorum*
	2–19	36	36	Nil
	Adult	20	20	Nil
Outside contacts, etc.	Under 2	17	23	1 *S. adelaide*
	2–16	53	57	1 *S. bovis-morbificans* 1 *S. paratyphi* C 1 *S. adelaide*
	Adult	47	50	1 *S. newington* 1 *S. adelaide*
		1947–8. Maternity hospital series		
Staff	Adult	115	140	2 *S. bovis-morbificans* 1 *S. typhi-murium* 1 *S. derby*
Mothers	Adult	113	127	1 *S. bovis-morbificans* 1 *S. kottbus* 1 *S. muenchen*
		1948. Post-epidemic series		
Ward survey	Under 2	27	37	2 *S. typhi-murium*
	2–13	55	57	Nil
State Home (repeat)	3 mth. to 9 yr.	49	49	Nil
M. and C. Welfare	Under 2	292	392	1 *S. paratyphi* B 5 *S. typhi-murium* 1 *S. derby* 1 *S. chester* 1 *S. bovis-morbificans*

Ward survey: medical and surgical wards, cases not diagnosed as gastro-enteritis.

State Home: had contributed several proven infections to the series (included in 'other institutions' on p. 173).

Outside contacts: mainly from housing camps, some from suburbs, generally from places where groups of cases had occurred.

M. and C. Welfare: a running survey from March to December, 1948, of babies seen by the Maternal and Child Welfare Service, undertaken to discover the post-epidemic incidence of salmonellosis in babies not sick enough to send to hospital.

Carriers

Our surveys for carriers are summarized in Table 4, the first part relating to the main investigation, the second to the problem of neonatal infections, and the third to post-epidemic surveys undertaken more recently.

Pre-clinical carriers. Three of the twenty-six babies under 2 years old in the first hospital survey fall in this group, their clinical onsets being 1, 3 and 14 days, respectively, after *S. bovis-morbificans* was

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found in their faeces. They had been in hospital for 6–13 days when examined, so were presumably infected in the wards. The incidence was high, considering the smallness of the series, and the group is obviously a dangerous one, as it could contribute materially to dissemination of the infection before clinical warning appeared.

Post-clinical carriers. This large group has been recorded above (Fig. 6), and these cases are not included in Table 4, although some of them were followed into their homes after discharge from the hospital. The remaining *S. bovis-morbificans* infections in children under 2 years old in the first section of Table 4 (three hospital, one State Home) are presumably also post-clinical carriers, as all had histories of gastro-enteritis during the previous few months. In fact, no baby with *S. bovis-morbificans* in this age group could be said to have had a completely inapparent infection during the epidemic.

The situation was different with the other species of *Salmonella* found in surveys during and after the epidemic. Some of the *typhi-murium* infections had a history of past gastro-enteritis, and others were mildly ill, though not severe enough to be sent to hospital, but the *adelaide*, *pullorum* and *chester* infections were practically symptomless, as were two neonatal infections with *S. muenchen* which are not included in the Table. These other Salmonella infections might as appropriately have been classed as healthy carriers, but were younger than others so classed, and were generally infective for longer periods.

Healthy carriers. This group includes children over 2 years old and adults. It is distinguished by the absence of any record of previous or subsequent gastro-enteritis, and by the apparently transitory nature of the infection (p. 174). It includes a surprising number and variety of Salmonellas, but only three *bovis-morbificans* infections in the epidemic series and three in the maternity hospital series. Two of the former were found in the ward survey in children who had been in-patients for 11 and 40 days respectively, and the third was in a child who had recently been in the gastro-enteritis ward with Flexner dysentery. These infections, though numerically insignificant and brief in duration as compared with the younger convalescents, may still have been important in spreading the organism from the hospital to the home.

Animals

As stated earlier, *S. bovis-morbificans* is known in Australia from sheep, cattle, pigs, horse and duck. Mr Simmons (personal communication), however, did not find it among the seven species of *Salmonella* isolated from the considerable series of fowls he has examined. It was difficult for us to add a survey of animals killed at the abattoir to the tasks in hand, and it appeared to be relatively unimportant, because the path from meat to infants would necessarily be via older persons, who were already covered in the investigation. We therefore concentrated mainly on examining rodents and cockroaches, both of which are widely distributed in Brisbane. Cultures were usually made from the liver, spleen, small intestine and colon of the vertebrates, and from the intestine or the faeces only of the cockroaches. The results are set out in Table 5.

It is apparent that neither the general rodent population nor the cockroaches outside hospital constituted a sufficient reservoir of infection with *S. bovis-morbificans* to contribute significantly to the epidemic spread. By contrast, the high incidence of infection in hospital mice and cockroaches could be regarded as a reflexion of the opportunities they had to acquire and disseminate infections in the wards (Mackerras & Mackerras, 1948).

The infected cat was from the same suburb as the rat with *S. bovis-morbificans*. A few human infections occurred in this area, but none in the family who owned the cat. It was suffering from acute enteritis when killed for examination, and we felt that it was more likely to have been a victim than a reservoir of infection. There were no cats in the institutions which provided most of the cases. None of the rodents showed obvious post-mortem signs attributable to their infections.

Other sources of infection

Salmonellas are usually regarded as essentially parasitic organisms, and a long period of existence outside the body of the host is not generally attributed to them. Nevertheless, water-borne infections with *S. typhi* are well known, and various Salmonellas have been isolated from sewage and found to contaminate bathing areas (Bornstein, 1943). Moreover, Welch, Ostrolenk & Bartram (1941) found experimentally that *S. enteritidis* would remain viable in the faeces of rats for 148 days at room temperature. The recent work of Mushin (1948) and Rubbo (1948) also suggests that dust may remain infective for considerable periods, and may therefore constitute what may be termed a 'reservoir' of infection in wards.

Our attention was rather concentrated on the living hosts, and we may not have given these other possible sources the attention they deserved. All we can say is that we failed to find infection in dust samples from hospital wards (Table 6), or from two drain seepages, six creek waters and one sewage effluent. The Government Microbiological Laboratory also examined thirty-five samples of milk (including twenty-seven from vendors supplying the Children's Hospital and a temporary housing camp) during the same period, with negative results.

METHODS OF TRANSMISSION

Having decided that infected infants were the chief source of infective material, it remained to establish how the infection was transferred from infant to infant. As most new infections were occurring in the Children's Hospital, and we were given every facility for study, our attention was naturally focused mainly on what was happening in the wards.

General observations

The salient features in ward procedures and conditions bearing on the inquiry were:

(1) Disposal of faeces was grossly insanitary.* The soiled napkins were placed in a pail, with or without some disinfectant in the bottom. When a round was finished, they were taken to the pan-room and rinsed out by hand, the faeces being brushed off with a scrubbing brush, before the napkins were sent to the laundry, where they were effectively boiled. Gloves were not worn, either when changing or washing the napkins, and separate hand-basins and nail-brushes for the 'change-up' nurses were not usually provided. As far as possible, 'change-up' nurses did not handle food in the same period of duty, but this rule was sometimes broken owing to shortage of staff, especially at night.

(2) Food preparation and handling were much as described by Rubbo (1948). Feeds were prepared in a special room, and transported in open metal containers covered by a towel. Bottles were cleaned, boiled and filled in the ward kitchen, and nurses scrubbed their hands before feeding the babies.

(3) The general handling and management of the babies provided frequent opportunities to contaminate the infant's hands or clothing, or even its face, if a nurse had organisms on her own hands. The exploratory activities of the babies, clutching at everything in sight and putting their hands into their mouths every few minutes, impressed us as particularly potent in adding to their intestinal flora.

(4) The wards were not specially designed to facilitate cleaning, dust was present in moderate amount, and no special precautions were taken, either to sterilize it or to prevent it moving about.

Table 5. Salmonella *infections in animals*

Species	Place	No. examined	*Salmonella*
	1947. Epidemic period		
Brown rats (*Rattus norvegicus*)	Hospital	16	1 *S. chester* 1 *S. paratyphi* C
	City and suburbs	224	1 *S. typhi-murium* 1 *S. bovis-morbificans* 1 *S. meleagridis* 1 *S. adelaide*
Mice (*Mus musculus*)	Hospital	53	3 *S. bovis-morbificans* 1 *S. orientalis*
	City and suburbs	47	Nil
Cats	Suburban	5	1 *S. bovis-morbificans*
Cockroaches*	Hospital	106	3 *S. bovis-morbificans* 1 *S. typhi-murium*
	City and suburbs	56	Nil
	1948. Post-epidemic period		
Brown rats	City and suburbs	207	1 *S. paratyphi* C
Mice	Hospital	83	Nil
Cockroaches*	City and suburbs†	230	Nil

* *Periplaneta australasiae, P. americana, P. ignota, Nauphoeta cinerea, Blatella germanica* and *Supella supellectilium.*

† Including fifty-four *Periplaneta americana* from sewers.

One of us has since visited the hospital where Mushin & Rubbo made their survey, and did not find that there had been any great difference between the two institutions in these respects.

(5) There were moderate numbers of mice and cockroaches in the wards. House-flies were also present in small numbers (winter and spring), and none of the wards was fly-proofed. Later in the epidemic, the babies in the gastro-enteritis ward were kept under nets.

Distribution of organisms in the wards

There were two surveys, one undertaken during the epidemic, when *S. bovis-morbificans* was the prevalent organism, and the other more recently, when there was a small 'flare-up' of infections with

* This is not a criticism of this hospital in particular. It applied, so far as we could discover, to every institution and every home in Brisbane.

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S. typhi-murium. The results were complementary, and they have been amalgamated in the notes that follow.

Whenever possible, the primary cultures were made in tetrathionate broth, although direct plates were also spread on some occasions. The variety of bacteria grown was consequently limited, and we have restricted it still further in Table 6 to include only strains of *Salmonella*, *Proteus*, Coliforms, Paracolon bacilli and *Pseudomonas*, which were culturally similar to organisms isolated from the faeces of scrub the fingers. The technique seemed sound, but unfortunately it was not developed before improvements in ward hygiene had been made as a result of the positive findings in sinks, brushes and vermin.

Our results with dust (including the exposed plates) are in contrast with those of Mushin (1948), who isolated *S. derby* from five out of eleven dust samples in Melbourne. It may be noted, however, that a 'spring cleaning' (which, of course, includes increased awareness as well as decreased dust) has been followed on several occasions, by a reduction in

Table 6. *Surveys of wards*

Situation	No. examined	Organisms isolated
Sinks in sanitary annexes	16	*S. typhi-murium* (1),* Coliforms (8), *Proteus* (4), Paracolons (2), *Pseudomonas* (2)
Scrubbing brushes in sanitary annexes	8	*S. bovis-morbificans* (1), Coliforms (1), *Proteus* (2), Paracolon (1), *Pseudomonas* (3)
Hand-basins in wards	26	*S. bovis-morbificans* (1), Coliforms (11), *Proteus* (4), Paracolons (3), *Pseudomonas* (8)
Hand-basin and nail-brush in ward (combined swabbing)	1	*S. bovis-morbificans* (1)
Nail-brushes in wards	16	*S. typhi-murium* (1), Coliforms (10), *Proteus* (5), Paracolons (8), *Pseudomonas* (1)
Towels in wards	3	Nil
Hands (83 nurses, 5 masseuses, 5 doctors in pooled groups)	29 gps.	Coliforms (6), *Proteus* (1), Paracolons (3), *Pseudomonas* (4)
Ward-kitchen sinks, brushes, draining board, etc.	14	Coliforms (7), *Proteus* (1), Paracolons (4), *Pseudomonas* (2)
Teats for feeding bottles	9	Coliforms (1)
Prepared infant feeds (10 in pooled groups)	3 gps.	Coliforms (1), Paracolons (3)
Ward baths and scales	8	Coliforms (2), *Proteus* (2), Paracolon (1), *Pseudomonas* (1)
Dust from wards	14	*Pseudomonas* (1)
Exposed McConkey and 'SS' plates (15 min.–3 hr.)	29	Coliforms (1), Paracolon (1), *Pseudomonas* (1)

* The figures in brackets are the numbers of isolations.

infants during the period. None, except the Salmonellas, can be strictly accepted, however, as indicating faecal contamination.

It will be seen that we followed Salmonellas as far as the ward hand basins and nail brushes, and we regard the last observation as particularly important, for it showed how the 'clean' nurses could be infected from the 'dirty' ones, and how medical officers, moving from ward to ward, could spread an infection about. Beyond that point, we failed to find Salmonella, although we obtained some evidence which might suggest faecal contamination, and an indication that the food preparation line may not have been as clean as we had expected.

A word about the hands may be of interest. We found that the easiest way to overcome reluctance for this examination was to culture the nurses in groups. Small pails containing about 100 ml. of tetrathionate broth were prepared, and the nurses, without prior warning, were marshalled, and rinsed their hands in turn in a pail, using a sterilized test-tube brush to the ward infections (Dr D. C. Fison, personal communication).

The role of vermin in transmission

We have shown (Table 5) that neither mice nor cockroaches were primary reservoirs of infection. Nevertheless, both were found infected in the wards, and there was experimental evidence (Mackerras & Pope, 1948) that infected cockroaches could become 'chronic carriers' for periods of 2–7 weeks. Infected materials were freely available in the sinks, where they would be sought avidly as food at least by the cockroaches and probably also by mice. Actually, the incidence of infection is even more striking than the Table indicates, for there were three *S. bovis-morbificans* infections in 15 mice and three in 16 cockroaches collected in children's wards during the epidemic, but none in 85 mice and 146 cockroaches from other hospitals or outside in the same period. Clearly these vermin could have acted as secondary reservoirs in the wards, and could have disseminated

the organisms in their faeces in ward dust and on food and utensils in the preparation rooms. No rats were caught in the children's wards.

Flies were not numerous at the time of the outbreak, but a miscellaneous series was examined, a few from the hospital, most from a nearby housing camp which was a minor centre of infection. The following is the list, the Drosophilids and Psychodids being from surface drains leading from a hut in the camp. No Salmonellas were isolated from any.

Musca domestica	30	*Sarcophaga* sp.	1
Fannia canicularis	6	*Drosophila* sp.	12
Calliphora augur	4	*Psychoda* sp.	30

Discussion

So far as the epidemic in Queensland is concerned, the findings reported here, though incomplete, favour the first of the pathways of infection illustrated by Rubbo (1948) more than the second. In the previous section, it was shown that the infected babies provided the overwhelming bulk of infective material; the evidence from the ward surveys given in this section supports the thesis that it was carried from baby to baby by the adults who cared for them. Mushin (1948) found towels, we found nail-brushes, as a link between 'dirty' and 'clean' nurses. We think that transmission in hospital could probably have occurred as easily directly by handling the babies as indirectly by contaminating their food or feeding utensils. Under the prevailing conditions, the dosage by either route was likely to have been adequate.

No doubt, attendants could have infected themselves as well as the babies, and this might have provided an additional line of infection as indicated by Rubbo, though we were unable to check the point in the children's wards (see, however, middle section of Table 4). Indirect contamination of food by vermin probably occurred too. Both factors could have contributed to maintaining the general level of infection in the community during the epidemic, but it is unnecessary to postulate them as more than subsidiary aids in spreading the disease among the babies. Outside, where less care in feeding was usual, the importance of this pathway would be greater, as indicated by the inverse relation of cases to breast feeding (p. 171).

NEONATAL INFECTIONS

The neonatal infections with *S. bovis-morbificans* merit some separate consideration. There were eleven recognized cases in the group, with four deaths. Ages at onset varied from 2 to 13 days, five being in the first week of life and six in the second. All occurred in one large maternity hospital, the dates of onset of diarrhoea being: 9, (22), 28 September; 2, 6, 12, 14, 23 October; 3 November; 2, 10 December. There is an element of doubt about the second case, as it was not diagnosed bacteriologically until 17 October. The first five cases remained in the maternity hospital for 2–14 days after onset, the remainder were transferred immediately they became ill. If case 2 be accepted, there is an almost complete overlapping series of infections from 9 September to 14 October. Nurseries were closed and 'spring cleaned' on two occasions.

It may be, in view of recent findings, that, once introduced, the organisms persisted in the nurseries; but we were impressed with the vigour of the cleansing operations undertaken, and we also regarded these newborn infants as a highly susceptible group, which should be a delicate indicator of persisting infection. We were, therefore, more inclined to believe that there had been at least two, probably three, separate introductions of infection into the hospital. The important question, therefore, was: how did it get in?

The younger age groups could be excluded, because the babies were born there and young visiting were forbidden. Animal surveys were not promising (p. 175), although the rat infected with *S. chester* was caught in the kitchen of this hospital. We therefore surveyed the staff and the women patients, with the results shown in the middle section of Table 4. It is apparent that either group could have provided the sources we were seeking.

We were inclined to discount the nurses, because trained nurses (one of them with Army experience) might be expected to be careful in their personal habits, even if they shared the prevailing superstition about the innocence of infants' faeces. On the other hand, there was published evidence of the transfer of infection from mother to infant at birth (Witkowski, 1935) or while suckling it (Snyder, 1940; Cataldi & Müller, 1946). It was unfortunate, though not surprising in view of the generally transitory nature of the carrier state in adults, that our results merely represented a cross-section of what was occurring, not a connected sequence of events.

There the matter had to rest until February, when the following interesting sequence occurred: Mrs O. had a mild attack of diarrhoea when admitted on 19 February, and *S. muenchen* was isolated from her faeces on three occasions during the following week; Baby O. born 19 February, was found to be infected with *S. muenchen* on 23 February; Baby R., born 18 February, in same nursery as Baby O., was found infected with *S. muenchen* on 2 March. These were the only *muenchen* infections encountered during the investigation. It is particularly interesting from the point of view of general epidemiology, that the infections in these infants were almost completely inapparent,* and that one retained its infection for

* We doubt whether this should affect inferences regarding *S. bovis-morbificans* in the earlier series, when the epidemic, though past its peak, was still highly virulent.

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2 weeks, while the other was still infective when lost sight of 32 days after the organism was first isolated.

We have given much attention to the entry of the infection into this hospital, for that was the crucial question. Once in, the mechanism of its spread in the nurseries was clearly the same as in other institutions. General standards of care of the babies, including their toilet, were high, but the same pernicious practice of rinsing the soiled napkins in the nursery annexes prevailed as elsewhere, and we felt that the sequence of events already described would inevitably follow. It may be significant that only sporadic cases occurred after improved napkin hygiene was instituted in the latter half of October.

PREVENTION

We are concerned here only with the prevention of Salmonella infections in infants. We do not propose to review the whole system of control of gastro-enteritis, about which a considerable literature has grown up (see, for example, M.R.C., 1944; Cummings, 1947; Rubbo, 1948; ourselves, 1949*c*), but simply to emphasize those aspects, which our findings indicate to be particularly important in relation to our specific problem. Also, we will not attempt to assess their effect on our epidemic, because it was already past its peak when they were put into operation.

So far as Salmonella infections are concerned, there are two fundamental considerations, which must underlie any plan of control. They are:

(1) That there is a clinically more or less inapparent carrier rate in the infant community, which is even more significant than an equivalent dysentery carrier rate in an adult community.

(2) That the spread of infection, rising to outbreak or epidemic level, is essentially an institutional phenomenon.

Unsuspected infections in babies have undoubtedly entered institutions on various occasions, and have sometimes spread, producing small institutional outbreaks (e.g. *S. typhi-murium* in 1948). The *S. derby* outbreak in Melbourne described by Rubbo (1948) seems to represent just this sort of thing on a greatly enlarged scale, and our epidemic presumably began in the same way.

The first practical point, then, is that the faeces of *all babies* should be treated as potentially dangerous. We feel strongly that no effective progress can be made until the universal feminine superstition of the innocence of babies' faeces is broken down. We have shown that these remarks apply even to the newly born, and our findings in this respect are supported by the record of Angrist & Mollov (1946) of inapparent neonatal infections with *S. bovis-morbificans* in America.

Practical implementation is not difficult, once the problem is appreciated and superstition destroyed. It involves a permanently established routine that:

(*a*) The duties of 'change-up' nurses shall be completely segregated from those of 'clean' nurses (Nabarro & Signy, 1932).

(*b*) That 'change-up' nurses shall have completely separate washing facilities from 'clean' nurses and doctors.

(*c*) That changing soiled napkins (or other soiled linen) shall be treated in the same way as changing a soiled dressing on a septic wound.

(*d*) That the filthy practice of rinsing soiled napkins in the wards be banished for ever from institutions which profess to care for babies.

If soiled napkins must be dealt with, as in private homes and small hospitals, they should be boiled without preliminary rinsing. We found that, provided there is enough water for them to swirl about freely in the container, and soap powder or any convenient detergent is used, there is no difficulty with smell or stain; the faeces become emulsified, and subsequent washing is made easier. If the napkins are packed into the boiler, both sterilization and cleansing are nullified. Strong feminine objections were raised at first, but the method has been proved practicable both in institutions and by individual mothers.

There are several ancillary measures, which complete the system, and for which reference may be made to the papers we have cited. The most important is aseptic preparation of babies' feeds, and delivery to the wards fully prepared, capped, and sterilized, as described by Cummings (1947) and illustrated by Rubbo (1948, pl. 6, fig. 9), for this ensures that no organisms enter the baby's gut with its food other than those already present on its lips or fauces.

The test of efficiency lies, as Sauer (1935) has pointed out, not in attempting to keep infected babies from entering institutions, which is manifestly impossible, but in the amount of diarrhoea that develops within the institution. Sauer demonstrated a notable success, which we feel confident can be duplicated elsewhere if equal care is taken. Breaks in technique will doubtless occur, and may pave the way for epidemics unless quickly detected. We feel that those who have emphasized 'parenteral diarrhoea' have done a disservice to prevention, for we have seen it become an epidemiological red herring, diverting attention from the true nature of an outbreak. The only safe rule in institutions is to regard all attacks of diarrhoea arising in the course of other illnesses as intercurrent contaminative infections until they are proved otherwise.

SUMMARY

There were nearly 400 cases of gastro-enteritis in Brisbane from May to October 1947. About half represented the endemic level then prevailing, and the remainder formed a virulent epidemic, which was responsible for most of the deaths. *Salmonella bovis-morbificans* was isolated from a sufficient proportion of these cases to justify the belief that it was the cause of the epidemic.

Of recognized infections with *S. bovis-morbificans* 77 % were in babies under 1 year old, 19 % in those 1–2 years old, and 4 % in older children and adults. The infection was most severe in the under-1-year-old group, and severity varied during the epidemic, reaching a maximum in August and thereafter declining steadily.

Nearly two-thirds of the infections were institutional.

Infants were found to be infective during the incubation period and also for periods up to 6 months after clinical recovery. Their faeces provided the greatest mass of infective material. Infections in older age groups were relatively unimportant in relation to the rate of epidemic spread, but significant secondary centres of infection developed in mice and cockroaches in the hospital wards.

The path of infection was traced from the faeces to the sinks and brushes used for cleansing soiled napkins, to mice and cockroaches which had access to the sinks, and to ward wash-basins and nail-brushes used by the nursing staff. Possible indicators of faecal contamination were found on nurses' hands, in ward kitchens, and in milk mixtures prepared for babies' feeds.

Control is believed to be practicable and to depend, first, on making medical officers and nurses aware of the dangers; and secondly, on improved ward hygiene, of which elimination of washing napkins in wards, reduction of contamination when changing napkins, rigid segregation of 'clean' and 'dirty' nurses, and improvements in food preparation and administration, are regarded as the most important.

We have received a great deal of help in many ways in the course of this work. First we would thank Dr A. D. D. Pye, Dr D. C. Fison, and Prof. G. Shedden Adam, Medical Superintendents respectively of the Brisbane General Hospital, the Brisbane Children's Hospital, and the Brisbane Women's Hospital, for entry to their wards, for arranging for material to be collected for us, and for access to their records. Fig. 1 is based on data prepared by Miss L. E. Knowles in Dr Fison's office. Dr J. V. Duhig and his staff in the General Hospital Laboratory have been most generous in passing over to us all the strains they isolated (about one-third of the human infections recorded here), and in collecting post-mortem material for our use. We would thank also Dr A. Fryberg, Director-General of Health, and officers of his Department for additional cultures and much help with the surveys, particularly Dr J. I. Tonge, Director of the Microbiological Laboratory, Dr T. H. R. Mathewson and later Dr H. C. Murphy, Director of Maternal and Child Welfare, Miss C. A. Johnson, Matron of the State Home for Children, and Mrs V. Wills and Mr J. J. Forbes of the central office.

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REFERENCES

Albiston, H. E. (1947). Salmonella food poisoning. *Bull. Dep. Hlth, Victoria*, nos. 89–90, pp. 2412–13.

Angrist, A. & Mollov, M. (1946). Bacteriologic, clinical and pathologic experience with 86 sporadic cases of Salmonella infection. *Amer. J. Med. Sci.* **212**, 336–46.

Arden, F. (1948). Recent progress in pediatrics. *Med. J. Aust.* **1**, 133–6.

Atkinson, N., Woodroofe, G. M. & Macbeth, A. M. (1944). The occurrence of Salmonella types in Australia. 2. *Aust. J. Exp. Biol. Med. Sci.* **22**, 201–4.

Atkinson, N., Woodroofe, G. M. & Macbeth, A. M. (1947). The occurrence of Salmonella types in Australia. 3. *Aust. J. Exp. Biol. Med. Sci.* **25**, 25–31.

Basenau, F. (1894). Ueber eine in Fleisch gefundene infectioese Bacterie. *Arch. Hyg., Berl.*, **20**, 242–94.

Bornstein, S. (1943). The state of the Salmonella problem. *J. Immunol.* **46**, 439–96.

Campbell, K. I. (1945). Report of an outbreak of epidemic diarrhoea of the newborn. *Med. J. Aust.* **1**, 79–84.

Cataldi, M. S. & Müller, A. (1946). Microflora del calostro y de la superficie cutánea del seno materno en relación con la del tubo digestivo del racién nacido. *Rev. Asoc. argent. Dietologia*, **4**, 296–300.

Colbeck, J. C. & Neisser, H. (1948). The presence of *Salm. typhi-murium* in the sputum of an asthmatic. *Mon. Bull. Min. Hlth Publ. Hlth Lab. Serv.* **7**, 66–8.

CROLL, D. G. (1946). Looking backward: A quarter-century of pediatrics. I. Gastro-enteritis. *Med. J. Aust.* **2**, 371.

CUMMINGS, G. D. (1947). Epidemic diarrhoea of the newborn from the point of view of the epidemiologist and bacteriologist. *J. Pediat.* **30**, 706–10.

DRAPER, F. (1944). Gastro-enteritis in children: A report on the bacteriology of twenty cases caused by *Bacterium typhi-murium* and two cases caused by *Bacterium reading*. *Med. J. Aust.* **1**, 533–7.

FOLEY, A. R. (1947). An outbreak of paratyphoid B fever in the nursery of a small hospital. *Canad. Publ. Hlth J.* **38**, 73–5.

HAYES, W. & FREEMAN, J. F. (1945). The incidence, type and bacteriology of Salmonella infections in the Army in India. *Indian J. Med. Res.* **33**, 177–93.

HENNING, M. W. & GREENFIELD, C. G. (1937). *Salmonella bovis-morbificans* (Basenau) from an outbreak of food-poisoning in the Cape Province. *Onderstepoort J. vet. Sci.* **8**, 351–6.

HORMAECHE, E., SURRACO, N. L., PELUFFO, C. A. & ALEPPO, P. L. (1943). Causes of infantile summer diarrhoea. *Amer. J. Dis. Child.* **66**, 539–51.

LITCHFIELD, W. F. (1921). The gastro-intestinal troubles if infancy. *Aust. med. Congr.* 11th Sess. (1920), 475–80.

MACKERRAS, I. M. & MACKERRAS, M. J. (1949*a*). The bacteriological diagnosis of Salmonella infections. *Med. J. Aust.* **1**, 1–3.

MACKERRAS, I. M. & POPE, P. (1948). Experimental Salmonella infections in Australian cockroaches. *Aust. J. Exp. Biol. Med. Sci.* **26**, 465–70.

MACKERRAS, M. J. & MACKERRAS, I. M. (1948). Salmonella infections in Australian cockroaches. *Aust. J. Sci.* **10**, 115.

MACKERRAS, M. J. & MACKERRAS, I. M. (1949*b*). Salmonella infections in Queensland. *Aust. J. Exp. Biol. Med. Sci.* **27**, 163–71.

MACKERRAS, M. J. & MACKERRAS, I. M. (1949*c*). The prevention of gastro-enteritis in infants. *Med. J. Aust.* **1**, 477–82.

M.R.C. (1944). The control of cross-infection in hospitals. *War Mem. Med. Res. Coun., Lond.*, no. 11, 34 pp.

M.R.C. (1947). The bacteriology of spray-dried egg, with particular reference to food poisoning. *Spec. Rep. Ser. Med. Res. Coun., Lond.*, no. 260, 66 pp.

MUSHIN, R. (1948). An outbreak of gastro-enteritis due to *Salmonella derby*. *J. Hyg., Camb.*, **46**, 151–7.

NABARRO, D. & SIGNY, A. G. (1932). Observations on dysentery in children. *Arch. Dis. Child.* **7**, 327–34.

NORTH, E. A. (1935). Bacillary dysentery in Rockhampton, with an account of the distribution and type of bacillary dysentery in other States. *Med. J. Aust.* **1**, 519–22.

RUBBO, S. D. (1948). Cross-infection in hospital due to *Salmonella derby*. *J. Hyg., Camb.*, **46**, 158–63.

SAUER, L. (1935). Enteritis in infants: prevention of its spread. *J. Pediat.* **6**, 753–62.

SELIGMANN, E., SAPHRA, I. & WASSERMANN, M. (1943). Salmonella infections in man. An analysis of 1000 cases bacteriologically identified by the New York Salmonella center. *Amer. J. Hyg.* **38**, 226–49.

SELIGMANN, E., SAPHRA, I. & WASSERMANN, M. (1946). Salmonella infections in the U.S.A. A second series of 2000 infections recorded by the N.Y. Salmonella Center. *J. Immunol.* **54**, 69–87.

SNYDER, M. L. (1940). The normal fecal flora of infants between two weeks and one year of age. *J. Infect. Dis.* **66**, 1–16.

STABLE, G. & PHILPOTT, I. G. (1948). An epidemic of gastro-enteritis in infants, with special reference to treatment. *Med. J. Aust.* **2**, 63–9.

STEWART, D. F. (1940*a*). Two mortalities among sheep due to infection with species of the Salmonella group of bacteria. *Aust. Vet. J.* **16**, 169–72.

STEWART, D. F. (1940*b*). Further Salmonella infections in pigs and acute paratyphoid septicaemia in horses. *Aust. Vet. J.* **16**, 266–7.

TURNER, A. J. (1938). The Jackson Lecture. Experiences in preventive medicine. *Med. J. Aust.* **2**, 805–13.

WELCH, H., OSTROLENK, M. & BARTRAM, M. T. (1941). Role of rats in the spread of food poisoning bacteria of the Salmonella group. *Amer. J. Publ. Hlth*, **31**, 332–40.

WITKOWSKI, R. (1935). Aerobe Mundhöhlenflore bei Mutter und Kind. *Zbl. Bakt.* (1), **133**, 334.

WRIGHT, G. P. & WRIGHT, H. P. (1946*a*). Diarrhoea and enteritis among infants in the London area, 1930–8. *J. Hyg., Camb.*, **44**, 480–90.

WRIGHT, G. P. & WRIGHT, H. P. (1946*b*). Cross-infection in children's wards. *Brit. Med. J.* **1**, 673–7.

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