In: Diseases of Children in the Subtropics and Tropics. D.B.Jelliffe and J. Paget-Stanfield (editors),3rd ed. Edward Arnold(London), p. 492-498, 1978.

Torres-Pinedo, R., Rivera, C. and Rodriguez, H. (1971) *Ann. N.Y. Acad. Sci.*, **176**, 284.
Townley, R. R. (1956) *Pediatrics*, **38**, 127.
WHO (1974) *Treatment and Prevention of Dehydration in Diarrhoeal Disease*. Geneva: WHO.
Young, V. M. *et al.* (1962) *Amer. J. trop. Med.*, **11**, 380.
Yow, M. D. *et al.* (1970) *Amer. J. Epidem.*, **92**, 33.

2 ShigellusiS

L. J. 1l iattl anti J. J Urrutia'

Shi,,-,cllosis is an infectious disease caused by Shi; ellae, which are bacilli with capacity to invade the mucosa of the colon and less frequently the ileum. The disease occurs sporadically and in epidemics with mild to severe clinical manifestations. The principal symptoms in *severe* cases (bacillary dysentery) arc abdominal cramps, fever, tenesmus, **and** diarrhoea. Toticosis and dehydration arc common in young children. Asymptomatic infections (healthy carriers) arc rare; convalescent carriers are common, particularly in children with growth retardation or protein-energy malnutrition (Mata *et ah, z966).*

Pathogenicity

All shigellae are capable of inducing diarrhoea but certain *Sh. ffexrreri* and Sh. *dysenteriae are* more pathogenic. Mechanisms responsible for the disease arc not clear. The endotoxin of these bacteria elicits a physiological response in man and animals indistinguishable from *that* evoked by other Gram-negative bacteria. When inoculated into modified animals, the resulting disease resembles the dysentery of man. Labrec *et al.* (1964) showed that virulent *SJntrlla* strains penetrate the lamina propria with production of diarrhoea or dysentery; this does not occur with the nonpathogenic variants. Multiplication of the organisms in *the* lamina propria is associated with synthesis of significant amounts of endotoxin.

Slit:gena dysentery primarily is a disease of humans, but dogs and primates can be *attacked.*

Geographical distribution -.

Shigellac have a world-wide occurrence. There have been no major changes reported in distribution of strains since the report of Young (1947) who emphasized localization of Sh. *flexneri* 1 and 4 in the USA and India, while serotypes **2** and 3 are more cosmopolitan. *Sh. **snrmci is*** most frequent in North America and Eastern Europe. Felsenfcld (z965) suggests that this distribution of Sir. *sontrri* may be due to the greater resistance of this *species* to antibacterial treatment. In recent times the Shiga bacillus *(Sir. dysenteriae* a) has emerged again as an important scrotype, predominating over other serotypes during large outbreaks in Central America (Gangarosa *et al.,* 1971; Mata el *al., 1974*

Environmental factors largely determine distribution of these organisms. Deficient sanitary conditions, poor housing and overcrowding, together with improper disposal of wastes, are contributing factors. Lack of *a* sufficient and safe water supply appears to *be* the most important determinant in rural **areas.**

Pathology

The pathological features of shigellosis vary considerably, depending upon the age of the child, nutritional status, intercurrent infection, duration of the disease, and whether the child is breast-fed or receives artificial feeding. In severely ill infants dying of dehydration and she_k, lesions **may** bear little correlation with severity of clinical manifestations. Stowens (19$7) seldom found ulrcration in infant shigellosis, and in fulminating cases with death occurring within 4S-72 hours, usually there was little more than diffuse hyperaemia of the mucosa, with or w•:thour oedema. However, **a** *WHO Expert Committer* (r964) described mucosal ulceration as common. Lesions predominate in the proximal and distal colon, but they may be found in the terminal ileum and in the large intestine **as** well. The evolution lesions includes an increase in int1ainination, oedema of the subtnucosa and muscularis, ant cellular mfiltration with polvmorphonucicar lcucocyrcs. At this stage considerable haemor rlrage may occur. In cases of long duration, hypertrophy of the lymphoid tissue is a consta finding. Development of pseudo-diphtheritic membranes or passage of tubular casts have occasionally been observed in chronic or relapsing cases. Degenerative or toxic changes ha· been observed in the liver, spleen, and kidneys. The fatty degeneration described in the !iv in chronic cases may be due to starvation. Pathologic changes in Shiga dysentery tend to b **more** pronounced and varied. The advanced colonic lesion shows varying degrees of coagulation necrosis of the mucosa and sometimes the submucosa. Toxic ntegacolon is found in 5% of autopsy cases. intravascular coagulation phenomena arc found in :4% of cases in a variety of organs. particularly in kidneys, adrenals, pancreas, and liver (Mata and Castro. 1974),

Clinical manifestations

There is a great variation in the clinical picture of shigellosis. Symptoms may be virtuall absent and recognizable only by isolation of the organism; *fever,* profuse diarrhoea, vomiting, and inflammation characterize the dysentery fonts; the latter often leads to death. Studies in Egyptian and Guatemalan villages revealed that shigellae are highly endemic (Higgins *et al.,* /955; Gordon *et a1.,* 1962), although they could not be recovers from a high percentage of children with diarrhoea! symptoms. The attack rate in infants less than 6 months of age was lower than for older infants and young children (Mata *et a* 1969). Breast feeding is customary in such regions and the nursling infant has good nutrition and a greater resistance to intestinal infection (Mata and Urrutia, 1974 He **is** endowed with passive maternal resistance and possesses a series of immune mechanisms derived from breast milk (Wyatt *et al.,* 1972; Hanson *et al., 1975). With the* introduction of supplemental foods prepared under unhygienic conditions, *the* risk of exposure increases as the nutritional status deteriorates (Gordon et a1., 1963). The course of *Shtgella* infection in kwashiorkor and other nutritional deficiencies, or with concurrent diseases, b usually more severe. Heavy infection with parasites, and multiple and mixed infections with *Salmonella,* enteropathogenic *E. coli,* and viruses *are* more common in *the* tropics (Young *et al.,* 1962; **Mata** *et at,* 7967). In such cases it is difficult to assess the contribution of the shigcllae to the svmpromatology observed.

The average incubation period is **2** to s days, with extremes of to $_7$ **days.** Some believe that children seldom experience **a** prodromal phase with marked and sudden diarrhoea and vomiting. Several authors (Hardy, 5954) showed that slight *fever,* abdominal discomfort, and nausea may precede diarrhoea) symptoms by u hours or more, Convulsions or central nervous manifestations may announce an attack in the **very** young.

Dysentery is **a** syndrome of watery and foul diarrhoea evolving rapidly into an acute, painful, and sometimes fulminating disease with passage of many stools of small size containing blood, mucus, and pus. Cramps and tencsmus arc characteristic and may **or** may not be accompanied by prostration. Vomiting is observed in as *many* as a third of the cases. Rectal prolapse is observed sometimes. Fever is often found with general toxic manifestations. Attacks arc self-limited and of short evolution.

Acute dysentery particularly due to the Shiga bacillus is usually preceded by vomiting, abdominal colic, and an increasingly frequent urge to defecate. Stools of decreasing amounts arc passed more and more often until bloody mucus and pus may be all that is evacuated. It is in this stage of the disease in children that dehydration, loss of electrolytes, and toxicosis assume great importance. Fever and prostration arc almost always

present. Cramps, tenesnms, and straining also usually occur. The duration of symptoms is dependent upon the severity of the disease and the success of therapy. In a small percentage of rases chronic recurrent diarrhoea with bouts of acute dysentery alternates with periods of apparent health. Such a prolonged illness in children who continue to excrete Ship bacilli in their stools is accompanied by chronic malnutrition (Mata *et al.,* 1966).

Fulminating dysentery develops as a *severe* diarrhoea accompanied by vomiting and a sharp rise in temperature. The infant becomes very dehydrated and goes into shock. Convulsions may be frequent in sonic areas, but in Central America these are not an important feature of the disease. The course is serious and response to therapy may be $_t$+oar. Fulminating dysentery is not frequent, except under special conditions, for instance, during the regional epidemic of Shiga dysentery in 1969-72 (Mara and Castro, 1974).

Choleraiform dysentery is also a fulminating foram which is characterized by profuse diarrhoea, subnormal temperature, and exhaustion and collapse. The outcome of this form is almost always fatal.

In chronic bacillary dysentery, symptoms may subside but do not disappear completely for a considerable period during which an attack may recur. In other cases remissions of symptoms are interspersed with repeated attacks. Extensive ulcer formation may result in scars and strictures. However, intestinal perforation is rare. There is considerable interference with digestion and absorption, and in an infant already weakened by malnutrition, prognosis is bad unless supportive therapy is provided.

Some children experience little or no diarrhoea, but have marked toxic systemic manifestations. Japanese observers differentiated between frank bacillary dysentery **and** *ckiri.* Sakamoto *et al.* (1956) described the two cardinal characteristics of *eiriri* as **central** nervous symptoms (particularly convulsions, and sensory disturbances) and circulatory alterations (especially cyanosis, vomiting, and duodenal haemorrhage). The severe form of *ckiri* is almost t00% fatal within 48 hours. The condition is now much less common in Japan.

Shigellosis simulates other diseases only in rare instances. Severe atypical forms of the disease may resemble pneumonia, agranulocytosis, meningitis, or appendicitis.

Laboratory diagnosis

A definitive diagnosis can be made by the recovery of shigellae from the stool, or by demonstration of a high titre of passive haemagglutinating antibody (Neter et *al.,* 1957; Young *ct al.,* 196o; Haltalin *et aL,* 1966; Lee et al., 1961; Ciiccres and Mata, r974). BIood cultures are not recommended because septicaemia seldom occurs. Stool examination for other possible aetiologic agents should be made because of the frequency of multiple infections among children (Young *et al.,* $1_9$64

Rectal swabs or faecal samples are collected and streaked as soon as possible on primary plating media_ Care should be exercised that swabs arc not allowed to dry, or the stool allowed to stand, because die-off rates for shigellac generally arc high. When the faecal specimen cannot be cultured within a short time, a 30%„ buffered glycerol-saline should be used as a preservative. If possible, flecks of bloody mucus should be selected for inoculation as they are most apt to contain large numbers of shigellac. Because some strains of shigellac are sensitive to inhibitory agents, it is best to use one or more mildly selective media such as Leviné s eosin methylene blue agar, MacConkey agar, or xylose lysine desoxycholate citrate agar, as well as more selective media such as Salmonella--Shi,>ella agar or desoxycholate citrate agar. Tergirol 7 with triphenyl terrazolium chloride has become popular in recent times and is optimal for isolation of the Shiga bacillus (Mata *a al.,* 197o). *Shigclla* colonies can be picked for biochemical and serological tests (Edwards and Ewing, 1962). polyvalent antisera arc used to establish the group and serotype of *Shigclla.*

A$_g$glutination tests performed on die patient's serum arc frequently misleading. Hacrnag-elutinatiou rests, however, provide a much more reliable means of antibody detection (Neter *a aL,* 1957; Young *ct al.,* 1964 1iacnragglutinatiou uses formalinized red cells sensitized with ' O' polysaccharide :antigens of several of *the* most prevalent shigellae. Acute and convalescent sera are tested for rises in antibody titre. Results obtained on a single scrum are not meaningful unless the titre is significantly elevated. Any titre above t : 40 is important in assessing the epidemiology of a particular serotype, more specifically of those to which *the test is* specific, namely, *Sim. dysenteriae 1, Slr. dysenteriae 2, Sh. flexrieri* 4 and 6, and *A. sourrci* (C3ceres and *Mata, 1974).*

Microscopic examination of the stool specimen in bacillary dysentery may be informative because it contains large numbers of leucocytes **and mucus.** Red blood cells, if' present, tend to clump. Large amounts of mucus are a constant finding in children, **and** blood is present less frequently. After administration of broad-spectrum antibiotics large numbers of yeasts, in the absence of other flora, can be seen.

In cases of dehydration or shock, levels of serum sodium, potassium, and CO_t **combining** power may be significantly altered. Leucocytosis may or may not **be** present during the early stages of acute shigellosis, **but** often occurs when secondary infection is involved. However, severe lcueocytosis is a common finding in patients with Shiga dysentery; leucocyte counts as high as so 000 have been reported (Basagoitia, 1974).

Differential diagnosis

The diagnosis of shigellosis can be wade with certainty only by culturing shigellae front the stool. Various other diseases capable of producing dysentery can be ruled out by **adequate** parasitological and bacteriological examinations, which will also reveal the cases of mixed infection. Among the parasitic diseases, giardiasis, balantidiasis, aunocbiasis, strongyloidiasis, and schistosomiasis can be recognized by microscopic examination of the stool, and malaria by identification of the blood parasites.

Diarrhoea caused by salmonellae or enteropathogenic *E. call* can be differentiated **by** proper bacteriologic techniques.

Coccal food poisoning may occasionally be mistaken for shigellosis, from which it usually differs in so far as outbreaks are small, vomiting is more prominent and **stool** cultures arc negative. Food poisoning can occasionally be caused by *Shigella* spp. (Felsenfield and Young, 195s) and attempts should be made to isolate the organisms from contaminated food and from the patients.

Convulsions, stupor, severe headaches, violent vomiting, and rigidity of the neck arc sometimes encountered in infants and young children, and may be confused with an attack of meningitis. Such cases of shigellosis yield a negative spinal fluid culture and the fluid generally has a normal cytology.

The presenting symptoms of acute intussusception on occasion are diarrhoea and vomiting, but the considerable quantity of blood present in the stool in most of these instances should suggest the proper diagnosis, confirmed by palpation of the typical **mass.**

Treatment

Therapy must he varied according ro the severity of the disease and should be symptomatic and specific. Symptomatic treatment deals with dehydration, 'intoxication', **and** possible shock. Of primary importance is the restoration of proper fluid and electrolyte

balance. The drugs frequently employed for relief ordiarrhocal symptoms in older children and adults are not indicated in infant cases. Purgatives are definitely contraindicated. Tincture of opium wakes the child less irritable, but does not stop diarrhoea, and is not recommended, except in cases of intense pain.

Various feeding regimens may be advocated. Foods arc usually withheld during the first 12 hours of disease, though some clinicians recommend the use of small feedings of glucose solution; milk feedings are resumed gradually, diluted initially. Breast feeding, if interrupted, should be recommenced as soon as possible. Solid foods are not given in the first 6 to t z hours.

Most cases of shigellosis subside with symptomatic treatment, adequate handling of the diet, and proper care (moral support from another and attendants). Rehydration definitely plays a significant role.' If mucosal involvement is significant and toxicosis, exhaustion, and other serious manifestations arc developing or have become established, antimicrobials should be instituted without hesitation. Such is the case in explosive *Shigella* diarrhoeas and dysenteries, particularly the varieties caused by *Sit. dysenteriae* r and certain flexneri sero-types. Sulfonamides, chlortetracycline, and chloramphenicol arc frequently recommended. In the last decade a significant proportion of *Shigella* organisms have become resistant to these drugs, often through acquisition of episomes by conjugation. Ampicillin has been found to be excellent (Haltalin *el* at, 1967) and nalidixic acid, and more recently trimethoprim-sulphainethoxazole have been found excellent for severe shigellosis, particularly for the Shiga variety (Mata *et aL, 1971).* The nutritional status and age of the child must be carefully considered when deciding on the type of drug and **its** dosage. Testing for sensitivity to antibiotics is of paramount importance since *Shigella* resistance to some of these drugs (for instance nalidixic acid, and ampicillin) has been reported.

Prognosis

,The prognosis for infants and young children has improved since the advent of specific drug and antibiotic therapy, and of better methods for rehydration and restoration of electrolyte balance. Prior to their use, fatality rates of 30-40% in infants under 1 year were common. At present, only fulminating cases with acute sudden onsets, with headaches, convulsions, irritability, and/or shock, are fatal. Proper treatment lowers the fatality rate of Shiga dysentery to less than 1%.

The well-nourished child over 1 year of age is able to withstand an attack of acute bacillary dysentery. However, after the weaning period, such an attack may constitute a hazard to the pre-school child, at risk of increasing malnutrition and exposure to infection in arcs of poor hygiene.

Prevention

Shigellosis must be regarded as a disease of societies living under inadequate conditions of hygiene. It occurs as a result of direct or indirect faecal ingestion. It is rarely transmitted by water, milk, or insects. Thus, the human host is the chief source of infection. Human to human transfer is common in mental institutions, prisons, and refugee camps, and where crowding and low sanitation arc present. Sub-clinical cases or true carriers arc **as** potentially dangerous as arc clinical cases. The importance of easy availability of water for washing purposes was pointed out by Stewart *et al.* (1955) who found the highest infection rates where water was least available. In areas with dense fly populations, **a** notable parallelism in seasonal incidence of diarrhoea and flies has been noted. Outbreaks of food-borne shigellosis do occur. They arc relatively prominent in industrial societies where person-to-person spread is rare due to the significant improvement in sanitation.

Chemnoprophylaxis with sulphonamides and antibiotics is of no value and should not be encouraged on ethical grounds (Rosenberg *et* al., 1974). Its use would favour bacterial acquisition of R factors responsible for antibiotic resistance, and other pathogenic properties. Vaccines have thus far only limited application (DuPont *et al.,* 1974) as in many areas many *Shigella* serotypes are found at any one time. Colostrum and breast milk provide *a* powerful resistance against *Shigella* infection through, the secreted lgA, high content of lysozyme, and the inhibitory influence of the Grain positive lactobacillary flora maintained by breast milk.

Rehydration is extremely important, particularly to replenish water and electrolytes lost primarily during the cholera-like phase of shigellosis, The fluid to be employed for rehydration is the **same** recommended for cholera patients (sec section **5** of this chapter). Rehydration should be by cup, or spoon, or by nasogastric tube if necessary. Intravenous rehydration is justified only *for* severe cases.

References

Basagoitia, J. S. (1974) *PAHO Scientific Publications,* **No. 283,** p. 5.

Cáceres, A. and Mata, L. J. (197$_4$) *J. infect.* Dis., **129,** 439.

DuPont, H. L., Nornick, R. B., Snyder, M. J., Libonati, J. P., Formal, S. B. and Gangarosa, E. J. 0974) *PAHO Scientific Publications,* No. z83, p. **129.**

Edwards, P. R. and Ewing, W. H. 0962) *Errerobacteriaceae.* Minneapolis, Minn.: Burgas Publishing.

Felsenficld, O. (1965) *Synopsis of Clinical Tropical Medicine.* St Louis, Mo.: C. **V. Mosby.**

Felsenfield, O. and Young, V. M. (1951) *Salntoneliosis, Shigellosis, Clinical Tropical Medicine.* St Louis, Mo.: C. V. Mosby.

Gangarosa, E. *J., Mata,* L. J., Perera, D. R., Relief., L. B. and Mcndizabal-Morris, C. (1971) *Proc. Vlth Int. Scient. Meet.,* Primosten, Yugoslavia, p. 259.

Gordon, J. E., Chitkara, I. D. and Wyon, J. B. (1963) *Am. J. med. Sri.,* **²45, 345.**

Gordon, J. E., Pierce, V., Ascoli, W. and Scrimshaw, *N.* S. (1962) *An,. J. trap. Med. Ha.,* **11, 389.**

Haltalin, K. C., Matteck, B. M. and Nelson, J. D. (1966) *J. Juwmn., 97, 517.*

Haltalin, K. C., Nelson, J. D., King, R., Sladoje, M. and Hinton, L. V. (1967) *J. Pediat., 7o,* 970.

Hanson, L. A., Carlsson, B., Ahlstedt, S., Svanborg, C. and Kaijser, B. (1975) *Mod. Prob1. Paediat.,* **15,** 63.

Hardy, A. V. (1954) *XIV Pan American Sanitary Conference Topic, Ii,* B (ii).

Higgins, A. R., Floyd, T. M. and Kader, M. A. (1955) *Am. J. trap. Med. Hyg., 4,* **27t.**

Labrec, E. H., Schneider, H., Magnani, T. J. and Formal, S. B. (1964) *J. Ban.,* **88, 1503.**

Lee, M. R., ikari, N. S., Branche, W. C. and Young, V. M. (1966) *J. Bacterial., 91,* **463.**

Mata, L. J. and Castro, F. (1974) *Industry and Tropical Health. VIII,* p. 30. Boston, **Mass.:** Harvard School of Public Health.

Mata, L. J. and Urrutia, J. J. (197r) *Ann. N.Y. Acad. Sci., 176,* 93•

Mata, L. J., Cáceres, A. and Torres, M. F. (1971) *Lancet, i,* 600.

Mata, L. J., Catslán, M. A. and Gordon, J. E. (1966) *Am. J. trop. Med. Hyg.,* **15, 632.**

Mato, L. *J.,* Fcrnández, R. and Urrutia, J. J. (t969) *Rev. lat.-aaner. Microbial. Parasitol.,* **II, 102.**

Mata, L. J., Urrutia, J. J. and Gordon, J. E. (1967) *Trop. geogr. rued., 19.* 247.

Mata, L. J., Gangarosa, E. J., Cáceres, A., Perera, D. R. and Mejicanos, M. L. (197o) *J. inf. Dis.,* **122, 170.**

Netcr, E. and Dunphy, D. 4957) *Pedlar.,* **20,** 78.

Rosenberg, i4H., Bciscl, W. R., Gordon, J. E., Katz, M., Keusch, G. T., Luckey, D. and **Mat.,** L. J. (1974) *Am. J. clin. Niue.,* **27,** 3^{0}4.

Sakamoto, A. and Komo, S. (1956) *Ann. Pardiat.,* **186.** a.

Stewart, *W. If.,* Mccabe, L. J., Jr., Hemphill, E. C. and Decapito, T. (1955) *Am. J. trap. Med.,* **4, 718.**

Stowens, D. Maj. (1937) Personal communication.

WHO (1964) *Tech, Rep. Ser. W14 Hlth Org.,* No. 288, p. 1 t.

Wyatt, R. G., García, B., Cáceres, A. and Mata, L. J. (1972) *Archos lat.-am. Nutr.*, **22**, 629.
Young, V. M. (1947) *Am. J. trop. Med.*, **27**, 293.
Young, V. M., Sochard, M. R., Gillem, H. C. and Ross, S. (1960) *Proc. Soc. exp. Biol. Med* **105**, 638.
Young, V. M., Lindberg, R. B., Ortiz, O., Jahiel, D., Sochard, M. R. and Hemphill, J. J. (1962) *Am. J. trop. Med. Hyg.*, **11**, 380.

3 Salmonellosis

P. E. Soysa

The term salmonellosis is not intended to cover typhoid and paratyphoid infections. Salmonellosis is a global problem though differences in incidence appear to exist between countries, depending in part on the interest in case finding and on laboratory techniques employed. There has been a progressive rise in the frequency of *Salmonella* infections in recent years. This trend has been linked with international traffic in animals and food-stuffs, with industrial food production methods, with large scale intensive stock breeding, and with communal restaurants, all of which arc conducive to the introduction of new serotypes and the spread of *Salmonella.* On the other hand in Sri Lanka, the incidence is declining steadily. An early combined study by the Department of Paediatrics and the Medical Research Institute in 1951 revealed that 32.2% of the so-called non-specific diarrhoeas were ***due to*** *Salmonella.* More recent annual statistics from the Medical Research Institute are as follows: 13.3% of all stool cultures were positive for *Salmonella* in 1970; 9% in 1971; and 7.8% in 1972. The only dairy products presently imported are butter and powdered infant milk foods. No meats or fish arc imported now. This could be one of the reasons for the reduction in incidence.

Serotypes and transmission of infection

Though the host range for *Salmonella* includes many wild animals, the threat ***to man*** arises from his predilection for domestic animals, such as chicken, ducks, geese, turkeys, pigs, and cattle.

The main transmission of bovine salmonellosis is by direct contact with infected cattle, contaminated milk and dairy products (also ice-cream), meat (particularly processed minced meat), and river water. Shellfish, eggs and egg products (including mayonnaise and cakes), and sausages together with communal feeding habits in the modern world ***are*** responsible for recent outbreaks. Epidemics also attack crowds on the move as in the case of wars and pilgrimages. This is also true of animals transported to markets and abattoirs, due to bad hygiene and poor environment during transport.

Animals play an epidemiological role in the spread of salmonellosis. *S.* typhimuriunt accounts for over 7o% of isolations in many parts of Europe. Dogs, cats, rats, and wild mammals (hares, wild boars, foxes, etc.) arc infected by serotypes most commonly found in man such as *S. typhimurium* and *S. enteritidis.* Pet caged birds and pct tortoises are important sources of infection. Some serotypes that easily adapt to man are regularly associated with the same product or the same animal: *S. infant's, S. derby,* and *S. panama* with pigs, pork products, and sausages; *S. dublin* with beef and veal; *S. infantis, S. enteritidis, S. agora,* and *S. thompson* with chickens; and *S. saint paul* with turkeys.

Hospital outbreaks often occur in children's wards and psychiatric wards. In Sri Lanka in 1972, cross-infection in a children's ward of a General Hospital accounted for a high incidence of .5. *san diego* and *S. thompson.*