Chapter 14
Salmonella Infections in Dogs and Cats

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Salmonellosis occurs worldwide in dogs and cats and is of clinical and public-health importance. Infections in these animals are usually asymptomatic, with intermittent excretion of *Salmonella* in faeces. Clinical syndromes, which are comparatively uncommon, are often most severe in young or debilitated animals. These syndromes include enterocolitis, septicaemia and, rarely, abortions. Conjunctivitis has been reported in cats. Outbreaks of salmonellosis have occurred in veterinary hospitals, where predisposing factors, such as immunosuppressive therapy or major surgery, can precipitate disease in a carrier animal. The significance and extent of the carrier–excreter state in dogs and cats was perhaps not fully realized until the early 1950s, when surveys of apparently healthy animals were conducted. One of the earliest comprehensive reviews of *Salmonella* infections in animals was by Buxton (1957), who documented the occurrence of the organisms in cats and dogs.

Epidemiology

Serovars

Numerous *Salmonella* serovars have been isolated from dogs. There are regional and national variations in the occurrence of serovars (Tables 14.1 and 14.2), reflecting those present in the animals' diet or general environment. *S. typhimurium* and *S. enteritidis* appear to be universally distributed, with *S. anatum* a common isolate from dogs in the USA (Morse and Duncan, 1975). No host-adapted serovars are described in dogs or cats. There are rare reports of *S. paratyphi* B isolation from dogs, usually where this serovar is also present in the human population. Some authors have noted a similarity in the distribution of serovars isolated from dogs and humans (Galton *et al.*, 1952). Comparatively few surveys have been carried out in cat populations (Table 14.3). Multiply resistant strains of *S. typhimurium* definitive types (DT) 104 and 204c have been isolated from domestic carnivores. *S. enteritidis* phage type (PT) 4, often associated with poultry and eggs (Humphrey, 1990), has also been recovered from cats.

Excreter state

Isolation rates of *Salmonella* serovars from faeces of clinically normal cats range from 0 to 14% (Center *et al.*, 1995). The prevalence of *Salmonella* in the faeces of asymptomatic dogs under various environmental and husbandry conditions is presented in Table 14.4.

Salmonella excretion rates for asymptomatic dogs ranged from 0 to 43%. Faecal samples from dogs in boarding kennels, dog pounds and veterinary hospitals occasionally yielded more than one serovar from a single sample, four serovars being isolated in one instance (McElrath *et al.*, 1952). Generally, there was a lower excretion

 Salmonella in Domestic Animals (eds C. Wray and A. Wray)

Table 14.1. *Salmonella* serovars isolated from dogs in various countries.

Germany[a]	Iran[b]	Ireland[c]	South Africa[d]	UK[e]
S. agona	*S. adelaide*	*S. agona*	*S. berta*	*S. adelaide*
S. anatum	*S. anatum*	*S. brandenburg*	*S. enteritidis*	*S. alachua*
S. bilthoven	*S. braenderup*	*S. bredeney*	*S. gloucester*	*S. ardwick*
S. brandenburg	*S. derby*	*S. derby*	*S. haardt*	*S. dublin*
S. bredeney	*S. enteritidis*	*S. dublin*	*S. lagos*	*S. enteritidis* (unknown PT)
S. derby	*S. haifa*	*S. enteritidis*	*S. saintpaul*	*S. enteritidis* PT1
S. dublin	*S. havana*	*S. fyris*	*S. tennyson*	*S. goldcoast*
S. enteritidis	*S. heidelberg*	*S. kentucky*	*S. tsevie*	*S. indiana*
S. give	*S. hindmarsh*	*S. montevideo*	*S. typhimurium*	*S. infantis*
S. heidelberg	*S. infantis*	*S. ohio*		*S. kedougou*
S. infantis	*S. kiel*	*S. typhimurium*		*S. montevideo*
S. java	*S. kisangani*			*S. panama*
S. manhattan	*S. manhattan*			*S. rissen*
S. newington	*S. newport*			*S. schwarzengrund*
S. oranienberg	*S. reading*			*S. seftenberg*
S. panama	*S. saintpaul*			*S. singapore*
S. saintpaul	*S.* II *sofia*			*S. stanley*
S. thompson	*S. tallahassee*			*S. typhimurium* (unknown PT)
S. typhimurium	*S. thompson*			*S. typhimurium* DT104
	S. typhimurium			*S. typhimurium* DT204c
				S. virchow
				S. worthington

[a] Förster *et al*. (1974); [b] Shimi *et al*. (1976); [c] P.T. Quinn, M.E. Carter and Y.E. Abbott, Faculty of Veterinary Medicine, Dublin, unpublished data, 1986–1996; [d] Venter (1988); [e] Veterinary Investigation Services (1988–1996) and Neil *et al*. (1981).
PT, phage type; DT, definitive type

Table 14.2. *Salmonella* serovars isolated from dogs in the USA (based on Bruner and Moran, 1949; Adler *et al*., 1951; Ball, 1951; Gorham and Garner, 1951; Galton *et al*., 1952; Bruner, 1973; Uhaa *et al*., 1988).

S. anatum	*S. illinois*	*S. newbrunswick*
S. bareilly	*S. infantis*	*S. newington*
S. bonariensis	*S. inverness*	*S. newport*
S. bovismorbificans	*S. javiana*	*S. norwich*
S. bredeney	*S. johannesburg*	*S. oranienburg*
S. budapest	*S. kentucky*	*S. oregon*
S. california	*S. krefeld*	*S. paratyphi B*
S. canoga	*S. litchfield*	*S. pomona*
S. carrau	*S. livingstone*	*S. poona*
S. cerro	*S. lomita*	*S. pullorum*
S. choleraesuis	*S. luciana*	*S. rubislaw*
S. cubana	*S. macallen*	*S. saintpaul*
S. derby	*S. madelia*	*S. sandiego*
S. duval	*S. manhattan*	*S. seftenberg*
S. enteritidis	*S. meleagridis*	*S. tallahassee*
S. florida	*S. memphis*	*S. tennessee*
S. gaminara	*S. miami*	*S. typhimurium*
S. give	*S. minnesota*	*S. urbana*
S. hartford	*S. montevideo*	*S. weslaco*
S. homosassa	*S. muenchen*	*S. worthington*

Table 14.3. *Salmonella* serovars isolated from cats in Iran, UK and USA.

Iran[a]	UK[b]	USA[c]
S. adelaide	*S. enteritidis* (unknown PT)	*S. anatum*
S. anatum	*S. enteritidis PT4*	*S. bareilly*
S. blockley	*S. enteritidis PT24*	*S. bredeney*
S. braenderup	*S. typhimurium* (unknown PT)	*S. choleraesuis*
S. derby	*S. typhimurium DT12*	*S. derby*
S. gaminara	*S. typhimurium DT49*	*S. donna*
S. havana	*S. typhimurium DT104*	*S. enteritidis*
S. infantis	*S. typhimurium DT193*	*S. javiana*
S. kisangani	*S. typhimurium DT204c*	*S. lomita*
S. livingstone		*S. montevideo*
S. manhattan		*S. pharr*
S. oritamerin		*S. poona*
S. II *sofia*		*S. pullorum*
S. thompson		*S. sandiego*
S. typhimurium		*S. saintjuan*
S. tyresoe		*S. typhimurium*
		S. weslaco

[a] Shimi and Barin (1977); [b] Veterinary Investigation Services (1988–1996); [c] Ball (1951), Bruner (1973) and Bruner and Moran (1949).
PT, phage type; DT, definitive type.

rate in household pet dogs, compared with those confined to kennels. In one survey of pet dogs, a wide range of serovars was obtained and this was attributed to exposure to a variety of sources rather than to cross-infection between animals (Shimi *et al*., 1976). The excretion rate in stray dogs was comparatively high, as these animals presumably survived by scavenging and hunting. Surveys of racing greyhounds in kennels yielded an infection rate of up to 43.5%, with an estimated exposure rate leading to more than one infection every 2 months (Stucker *et al*., 1952). This high level of excretion was attributed to the diet of contaminated raw meat and offal. The infection rate in these greyhounds fell to lower levels (20–30%) during the racing season, indicating that infection was due to ingestion rather than through animal contact at the racing track. Working farm dogs may be exposed to high levels of *Salmonella* in the farm environment, particularly if recent outbreaks of salmonellosis have occurred in the livestock (Carter *et al*., 1983).

Asymptomatic canine excreters appear to shed *Salmonella* in faeces intermittently for comparatively short periods. When the faeces of 49 culture-positive dogs were sampled over a 4–6 week period, only five dogs yielded the same serovar that had been isolated initially. Within this sampling period, seven dogs were found to have acquired a second, and in some instances a third, *Salmonella* serovar (Mackel *et al*., 1952). Tanaka *et al*. (1976a) examined apparently healthy stray dogs for faecal shedding of *Salmonella*. The number of organisms fluctuated from 10^2 to 10^5 *Salmonella* 100 g^{-1} of faeces. Two dogs were found to shed small numbers of organisms (10^2 *Salmonella* 100 g^{-1}) sporadically for up to 74 days. Three other dogs excreted 10^4–10^5 *Salmonella* 100 g^{-1} of faeces initially and shedding continued for up to 115 days.

Transmission

Transmission in dogs and cats usually occurs either directly or indirectly by the faecal–oral route. Tanaka *et al*. (1976b) succeeded in experimentally producing a carrier state in dogs by oral administration of naturally contaminated faeces. Each dog received 39–92 *S. typhimurium* organisms *per os*. These experimentally infected dogs shed *Salmonella* for 18–24 days. None of three dogs dosed with 60 freshly isolated *Salmonella* organisms became shedders or produced antibodies. This apparent loss of virulence of *S.*

Table 14.4. Categories of apparently healthy dogs, from different countries, shedding *Salmonella* serovars.

Categories of dogs sampled	Georaphical region/country	Dogs shedding/ dogs sampled	% shedding	Reference
Household pets	Hawke's Bay/ New Zealand	0/150	0.0	Timbs *et al.* (1975)
	Washington/USA	10/809	1.2	Gorham and Garner (1951)
	Tehran/Iran	21/472	4.4	Shimi *et al.* (1976)
	Florida/USA	244/1626	15.0	Galton *et al.* (1952)
Boarding kennels	Tehran/Iran	28/181	15.5	Shimi *et al.* (1976)
	Florida/USA	21/126	16.6	McElrath *et al.* (1952)
Quarantine kennels (rabies)	Honolulu/Hawaii	38/295	12.9	Adler *et al.* (1951)
	Jacksonville/USA	194/1385	14.0	Mackel *et al.* (1952)
Dog refuges/pounds	Hawke's Bay/ New Zealand	5/150	3.3	Timbs *et al.* (1975)
	Florida/USA	71/895	7.9	McElrath *et al.* (1952)
	Brisbane/Australia	12/138	8.7	Frost *et al.* (1969)
Greyhound kennels	Florida/USA	930/2548	36.5	Galton *et al.* (1952)
	Jacksonville/USA	697/1602	43.5	Stucker *et al.* (1952)
Veterinary hospitals	Brisbane/Australia	7/157	4.5	Frost *et al.* (1969)
	Jacksonville/USA	13/66	19.7	Mackel *et al.* (1952)
Working dogs on sheep farms	Hawke's Bay/ New Zealand	13/300	4.3	Timbs *et al.* (1975)
Working dogs at abattoir	Brisbane/Australia	2/10	20.0	Frost *et al.* (1969)
Stray dogs	Tehran/Iran	3/19	15.8	Shimi *et al.* (1976)
	Khartoum/Sudan	104/442	23.5	Khan (1970)

typhimurium for the intestinal tract following culture of the organisms was also observed in experiments with mice (Tanaka *et al.*, 1977).

Intraocular inoculation of *S. typhimurium* in cats with 9×10^5 colony-forming units (cfu) produced conjunctivitis, with faecal shedding of *Salmonella* organisms, while a lower dose (9×10^3 cfu) initiated an asymptomatic excreter state (Fox *et al.*, 1984).

Rare cases of intrauterine transmission have been reported in both bitches (Redwood and Bell, 1983) and queens (Reilly *et al.*, 1994).

Sources of infection

Domestic carnivores are exposed to diverse sources of *Salmonella* serovars (Fig. 14.1). Dogs are particularly indiscriminate in their eating habits, ingesting food irrespective of quality, freshness or source. Many cats and dogs are allowed to roam and thus have access to carrion or are able to hunt, kill and eat wildlife species. Meat, offal and meat-and-bone-meal, common ingredients in the diets of dogs and cats, are frequently contaminated with *Salmonella*. Offal sold at a wholesale meat market and judged 'fit for consumption' was found to contain 24 different *Salmonella* serovars and 56.6% of 408 samples yielded *Salmonella* organisms (Sinell *et al.*, 1984). When 112 samples of commercial raw meat, used for feeding greyhounds, were cultured for *Salmonella*, 44.6% were positive (Chengappa *et al.*, 1993). In both surveys, *S. typhimurium* was the most common serovar isolated.

Galton *et al.* (1955) isolated *Salmonella* from

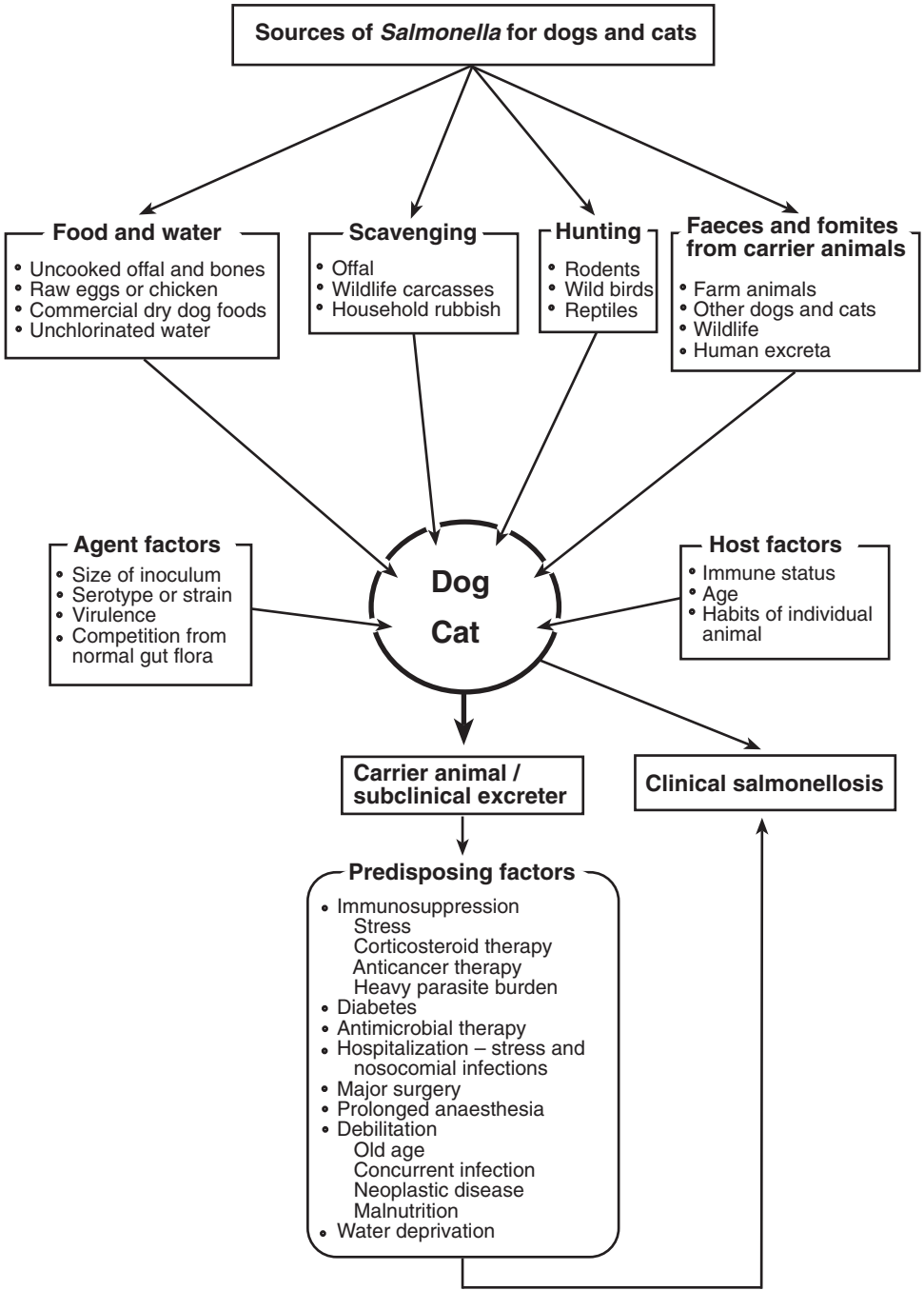

Fig. 14.1. Sources of *Salmonella* for dogs and cats and predisposing factors which may precipitate clinical salmonellosis in a previously asymptomatic carrier animal.

26 (26.5%) of 98 samples of dry dog meal, but found none in pressed foods, such as dog biscuits, or in kibbled products. Two brands of dog meal that failed to yield *Salmonella* serovars had been effectively heat-treated. Day *et al.* (1963) fed contaminated dry dog meal to young dogs and, of the nine serovars recovered from their faeces, seven were also cultured from the meal.

Raw or undercooked poultry meat or eggs can be a source of *Salmonella* for both humans and companion animals. *S. enteritidis* PT4 may be present in the yolk of intact hens' eggs (Humphrey *et al.*, 1989). Storage of eggs at temperatures of 10°C or above can allow the organisms to multiply rapidly to numbers that may permit their survival after cooking (Humphrey, 1990).

Wildlife species are a potential source of *Salmonella* for dogs and cats. Over 50 *Salmonella* serovars have been isolated from mice and rats (Weber, 1982). Wild mice can become infected with *S. enteritidis*. A naturally infected mouse excreted the organism in its faeces for 19 weeks and had 10^4 *Salmonella* g^{-1} of liver and 10^3 organisms g^{-1} of macerated intestine (Davies and Wray, 1995). Deaths from *S. typhimurium* have been recorded in garden birds (Laing, 1990; Kirkwood *et al.*, 1995). The occurrence of *Salmonella* serovars in free-living wild birds was reviewed by Wilson and MacDonald (1967), who concluded that, although salmonellosis in birds is a potential hazard to humans and domestic animals, there is a low incidence of disease in the general wild-bird population. However, rare localized outbreaks of salmonellosis do occur. Both captive and wild reptiles, including lizards, tortoises and terrapins, are notorious for *Salmonella* infections (Borland, 1975) and dogs and cats may acquire infection from these sources.

Coprophagia or ingestion of faecally contaminated food or water may result in infection by *Salmonella* serovars. The organisms are often widespread in soil on farms where outbreaks of salmonellosis have occurred and these organisms can survive for up to 9 months in shaded, moist soil (Carter *et al.*, 1979). A comparative survey of working dogs on sheep properties with and without ovine salmonellosis during the previous 12-month period indicated an excreter rate in dogs of 4.7% and 4.0%, respectively (Timbs *et al.*, 1975). *S. bovismorbificans*, the most common serovar in sheep in the district, was isolated only from dogs on farms where recent outbreaks of salmonellosis in the sheep had occurred. *S. typhimurium* was isolated from the faeces of the two asymptomatic farm dogs where clinical disease attributed to this serovar had occurred in dairy cows (Carter *et al.*, 1983). Two recently acquired parakeets (*Melopsittacus undulatus*) that developed clinical salmonellosis were thought to have been the source of *S. typhimurium* var. Copenhagen infection for a human infant and a 4-year-old Siamese cat in the same household (Madewell and McChesney, 1975).

Carrion is a potentially rich source of *Salmonella* and replication of these organisms may occur in carcasses, depending on ambient temperatures. Fly maggots are a possible source of infection associated with carcasses. Fly eggs placed in moist food contaminated with *S. enteritidis* resulted in infected maggots, pupae and adult flies (Kintner, 1949).

Animal Infection

Pathogenesis

The numbers of *Salmonella* ingested, the serovar or strain involved and its virulence may determine the clinical outcome of infection with *Salmonella* serovars. Non-specific host factors that affect the ability of organisms to establish and produce infection include gastric acidity, peristalsis, quality of intestinal mucus, lysozyme in secretions, lactoferrin in the intestinal tract and the normal resident intestinal flora (Clarke and Gyles, 1993). The greater susceptibility of young animals to salmonellosis may be associated in part with the lack of a well-established normal flora. In addition, a naïve or incompetent immune system renders such animals vulnerable to these enteropathogens.

Salmonella are facultative intracellular pathogens and the ability of these organisms to survive and multiply inside phagocytes is critical to the outcome of infection (Salyers and Whitt, 1994). Endotoxin (lipopolysaccharide) is a major virulence factor of *Salmonella* serovars and is the probable cause of death in animals with septicaemic salmonellosis. Endotoxin also interferes with the beneficial effects of activated comple-

ment. Other toxins produced by *Salmonella* include a heat-labile enterotoxin and a cytotoxin that inhibits protein synthesis (Groisman *et al*., 1990). Some virulence factors are associated with plasmids, such as a 50 MDa plasmid in *S. dublin* (Terakado *et al*., 1983), a 60 MDa plasmid in *S. typhimurium* (Jones *et al*., 1982) and a 36 MDa plasmid in *S. enteritidis* (Nakamura *et al*., 1985).

The distribution of organisms in the asymptomatic carrier state was studied in 17 dogs naturally infected with *S. typhimurium* (Tanaka *et al*., 1976a). *Salmonella* were isolated from the mucosae and contents of most segments of the intestinal tract, including the gastric mucosa in three cases. The mucosae of the caecum, colon and ileum were most commonly found to harbour *Salmonella* particularly at the junction of these intestinal segments. *Salmonella* were present at levels between 10^2 and 10^6 100 g^{-1} of intestinal tissue. Organisms were also recovered from the intestinal lymph nodes, with most isolates from the jejunal node. The carrier state was subsequently produced experimentally with *S. typhimurium* (Tanaka *et al*., 1976b). *Salmonella* were isolated from the mucosa of the middle portion of the ileum, 1 day post-inoculation (PI) and within 1–2 days they had spread to the mucosae of the lower part of the ileum, the caecum and the upper part of the colon. The major site of localization and multiplication in 5–15 days PI was the mucosa adjacent to the ostium ileocaecocolium. The jejunal node yielded *Salmonella* from the second day PI. Detectable serum antibody levels were present from the seventh day PI.

A high percentage of *Salmonella* are eventually cleared by the host's defences and only a small proportion survive for comparatively short periods, leading to the carrier state (Clarke and Gyles, 1993). A range of predisposing causes can precipitate clinical salmonellosis in a previously asymptomatic carrier animal (Fig. 14.1). In enterocolitis, the organisms adhere to the intestinal cells, invade the mucosa and are taken up by mucosa-associated macrophages. They induce toxin-mediated damage to microvilli of apical enterocytes, which results in shortening of villi, degeneration and abnormal extrusion of enterocytes and increased emptying of goblet cells. There is a neutrophilic reaction in the lamina propria, accompanied by transepithelial migration of neutrophils into the lumen (Takeuchi and Sprinz, 1967). Invasive strains of *Salmonella* are able to breach the mucosal barrier and invade underlying tissues. When these strains penetrate underlying tissues, septicaemia follows, with replication in the cells of the reticuloendothelial system. Characteristic systemic signs of fever and vascular damage occur, due to the effects of circulating endotoxin.

Factors predisposing to the development of clinical disease

Clinical salmonellosis is rare in cats and dogs. Healthy adult animals appear to have a high natural resistance to the disease. Young animals and ageing or debilitated dogs and cats are most susceptible (Buxton, 1957). This high natural resistance to clinical disease may be lowered by various immunosuppressive or stress factors (Fig 14.1). The disease is difficult to reproduce experimentally in dogs and cats. This may be due, in part, to the difficulty in experimentally mimicking the stress factors present in situations such as veterinary hospitals (Timoney, 1976). Sporadic cases of salmonellosis can occur in the general cat and dog populations, but outbreaks of the disease tend to occur in confined groups of animals, such as those in kennels, research facilities and veterinary hospitals. Animals in confinement often experience environmental stresses, such as overcrowding and changes in diet, heating, lighting and humidity. Animals in research facilities may be exposed to stress during shipping. Fifteen cats out of 142 (10.6%) in 17 shipments to a research laboratory were found to be infected with *Salmonella* serovars and two subsequently died from the septicaemic form of the disease (Fox and Beaucage, 1979). Additional stresses in veterinary hospitals may include immunosuppressive therapy, surgery, prolonged anaesthesia and antimicrobial therapy. Animals with concurrent disease or suffering from debilitating conditions, such as diabetes, are susceptible. Clinical salmonellosis developed in six dogs with multicentric lymphosarcoma that were subjected to surgery and treated with corticosteroids and anti-cancer drugs. Three of the affected animals subsequently died from salmonellosis (Calvert and Leifer, 1982).

The index case initiating an outbreak of salmonellosis in a veterinary hospital may be an

animal admitted with *Salmonella*-associated diarrhoea or may result from activation of a pre-existing asymptomatic infection. Clinically affected dogs and cats usually excrete larger numbers of *Salmonella* than carrier animals. However, comparatively small numbers of *Salmonella* may be required to cause clinical salmonellosis in animals stressed by surgical or medical treatments (Timoney *et al*., 1978).

Predisposing or risk factors have been investigated in several nosocomial outbreaks of salmonellosis. In an episode involving hospitalized dogs, Calvert (1985) listed pre-existing disease, hospitalization of 5 days or longer, major surgery, glucocorticoid therapy, cytotoxic drug administration and neoplasia as important risk factors leading to the development of clinical salmonellosis. In a nosocomial outbreak of disease in dogs due to *S. krefeld*, Uhaa *et al.* (1988) statistically analysed the risk factors involved. The use of antimicrobial agents, particularly ampicillin, was identified as an important risk factor. This has been attributed to selective elimination by the antibiotic of competing intestinal microflora that are antagonistic to *Salmonella*, thereby creating a more favourable environment for the replication of *Salmonella* serovars. A serious nosocomial outbreak of salmonellosis in cats was reported by Timoney *et al.* (1978). Most of the cats involved were less than 1 year old and had been admitted for routine surgical procedures, medical reasons or for boarding. The animals became ill 2–5 days after admission. *Salmonella* serovars were consistently isolated from the oral cavity, as well as from the faeces, of the affected cats. Organisms in the oral cavity may contaminate the cats' coats during grooming, increasing the hazard of *Salmonella* being transferred to the hands and clothing of personnel caring for the animals.

Clinical signs

The majority of *Salmonella* infections in dogs and cats are asymptomatic. The main clinical syndromes, in both animal species, are an acute enterocolitis and septicaemia, with resultant endotoxaemia. Rare syndromes include conjunctivitis in cats and *in utero* infections in bitches and queens, which result in abortions, stillbirths or the birth of weak offspring.

Acute enterocolitis usually develops within 3–5 days of exposure and is limited to mucosal invasion. It is manifested by watery or mucoid diarrhoea, containing blood in severe cases, accompanied by vomiting, fever (40–41°C), inappetence or anorexia, lethargy, abdominal pain and progressive dehydration. Some cats drool excessively during the illness and often have pale mucous membranes (Timoney, 1976; Center *et al*., 1995). Most animals recover in 3–4 weeks, but a carrier–excreter state may persist for a further 6 weeks.

In a few instances, acute enterocolitis can develop into an overwhelming infection, with septicaemia, endotoxic shock and signs consistent with the development of disseminated intravascular coagulation. Respiratory distress is often reported in these animals. In two cases involving young pups of about 2 months of age, septicaemia associated with *S. dublin* resulted in meningoencephalitis and neurological signs, such as left-sided hemiparesis (Milstein, 1975; Nation, 1984). Septicaemia in cats due to *S. arizonae* (Krum *et al*., 1977) and *S. enteritidis* (Fox and Beaucage, 1979) has been reported.

Abortion attributable to *Salmonella* serovars is rare. Redwood and Bell (1983) reported an *in utero* infection caused by *S. panama* in two boxer bitches in the same breeding establishment. A pure growth of *S. typhimurium* was isolated from stillborn kittens (Reilly *et al*., 1994) and *S. choleraesuis* was cultured from the uterus of a rural cat containing three dead fetuses (Hemsley, 1956).

S. typhimurium-associated unilateral conjunctivitis has been described in a cat (Fox and Galus, 1977). The condition was reproduced experimentally using an intraocular instillation of *S. typhimurium* (Fox *et al*., 1984). Features common to the natural infection and experimental studies included moderate to severe conjunctivitis of several days' duration, with lacrimation, blepharospasm and a prominent nictitating membrane. Scleral injection and mucopurulent ocular discharge continued for several days, with large numbers of neutrophils in conjunctival scrapings, but with no signs of systemic illness. *S. typhimurium* was recovered from the faeces.

Some cats with salmonellosis are pyrexic and have a neutropenia with a left shift and vague non-specific signs of illness but no definite intestinal signs (Dow *et al*., 1989).

An unusual case of *Salmonella*-associated

pneumonia was reported in an 8-year-old cat previously treated for severe dental calculus. The animal appeared to have no enteric manifestation of disease (Rodriguez *et al.*, 1993). *S. typhimurium* was isolated from kidney tissue of an 8-month-old dog with unilateral pyelonephrosis. No other organ systems were involved (Crow *et al.*, 1976). The animal recovered following surgical removal of the kidney and antibiotic therapy. A 7-year-old dog with intermittent epigastric pain, extending over a period of 8 months, was found to have chronic cholecystitis (Timbs *et al.*, 1974). *S. typhimurium* was present in the liver, the bile and a mesenteric lymph node. The main post-mortem finding was severe thickening of the gall-bladder wall. Histopathological findings included numerous inflammatory foci in the liver and mucosal congestion and haemorrhage in the intestines, without evidence of diarrhoea.

Pathological findings

When salmonellosis is confined to the intestinal tract, gross pathological findings may include an acute enteritis with bloodstained luminal contents, limited to the distal ileum, caecum and colon. Histopathological findings vary from catarrhal inflammation to villous atrophy, mucosal erosion and infiltration of neutrophils and macrophages into the lamina propria.

The carcasses of cats that die or are euthanized *in extremis* tend to be emaciated and dehydrated. The mucous membranes are pale and diffuse haemorrhagic enteritis is often present. Most of the parenchymatous organs may be grossly normal but the liver frequently contains small necrotic foci, about 2 mm in diameter. Blood-tinged fluid may be present in the serous cavities (Timoney, 1976). In some animals, the main pathological findings include petechial or ecchymotic haemorrhages, involving most organs including the lungs and epicardial and endocardial surfaces. Thrombosis and haemorrhage are consistent histopathological findings (Krum *et al.*, 1977).

Pups that have died rapidly from septicaemic salmonellosis may have good fat reserves. Multifocal or local extensive areas of consolidation are present in the lungs. The spleen, liver and kidneys may be swollen, pale and friable. Petechial or ecchymotic haemorrhages are often present in many organs. Histopathological findings can include embolic pneumonia, splenitis, myocarditis, nephritis and meningoencephalitis. *Salmonella* localize in the vascular beds of lungs, heart, spleen, liver, kidneys and brain (Nation, 1984).

Diagnosis

Salmonellosis should be suspected in hospitalized dogs or cats developing fever and acute enterocolitis, particularly in those animals that have recently undergone major surgery, those with serious medical conditions or those receiving immunosuppressive therapy. Young pups or kittens with diarrhoea should be investigated for possible infection with *Salmonella*. Bloody diarrhoea and a degenerative left shift of the leucogram are consistent with salmonellosis (Calvert, 1985).

In severe disease, abnormal biochemical changes may include hypoalbuminaemia, elevated serum alkaline phosphatase levels and hypoglycaemia. Calvert (1985) found that a combination of: (i) hypoalbuminaemia with hypoglycaemia, or (ii) hypoalbuminaemia, elevated alkaline phosphatase levels and hypoglycaemia correlated with a poor prognosis.

The definitive diagnosis of salmonellosis requires the isolation of the organisms from affected tissue, blood, cerebral–spinal fluid or transtracheal washings, specimens that do not normally yield *Salmonella* in carrier animals. Because of the comparatively common occurrence of the asymptomatic excreter state, isolation of *Salmonella* from faecal samples does not confirm a causal relationship between the organism and the clinical disease in the animal (Center *et al.*, 1995). However, high numbers of *Salmonella* are usually recovered from animals with *Salmonella*-associated diarrhoea, while asymptomatic carrier animals shed fewer organisms intermittently and usually these organisms can be isolated only after enrichment procedures (Fox, 1991). The presence of large numbers of leucocytes in faeces is indicative of an invasive infection. *Salmonella* can be isolated from conjunctival scrapings from cats with conjunctivitis (Fox *et al.*, 1984), and in abortions and stillbirths the organisms are present in fetal and placental tissues (Redwood and Bell, 1983).

Isolation procedures include the inoculation of selective indicator media, such as modified brilliant green agar, xylose lysine desoxycholate (XLD) or *Salmonella–Shigella* (SS) media, as well as the use of an enrichment broth, such as selenite F, tetrathionate and Rappaport broths, which are selective for *Salmonella* serovars. The media are incubated at 37°C aerobically for 2–3 days (see Chapter 21). Aliquots from enrichment broth are subcultured on to further plates of selective indicator media. An initial presumptive identification of *Salmonella* organisms is based on their colonial appearance. Subsequently, suspect colonies are subcultured in indicator media, such as triple-sugar iron agar and lysine decarboxylase broth. These are incubated at 37°C for 18 h and examined for the characteristic appearance given by *Salmonella* serovars. The serovar can be established using culture from the slant of the triple-sugar iron agar in a slide agglutination test with commercially available typing sera (Quinn *et al.*, 1994). Antibiotic susceptibility tests should be conducted on *Salmonella* isolates associated with the septicaemic form of salmonellosis. Antibiotic-resistant strains of *Salmonella* are not infrequently isolated from cats and dogs (Timoney, 1978). Phage typing of some serovars, such as *S. typhimurium* and *S. enteritidis*, which is usually carried out in reference laboratories, can be useful in epidemiological studies.

A polymerase chain reaction (PCR)-hybridization technique for detecting *S. typhimurium* in canine faeces has been developed. It was found to be more sensitive than standard cultural procedures (Stone *et al.*, 1995).

Standard serologicalal tests may prove useful in groups of animals but have a limited application for individual dogs and cats. Differential diagnosis should include canine parvovirus and coronavirus infections, canine enteric campylobacteriosis, canine haemorrhagic gastroenteritis and feline panleucopenia.

Treatment

Antimicrobial therapy is unnecessary and possibly undesirable in uncomplicated *Salmonella* enterocolitis. Careful management of fluid and electrolyte balances usually promotes recovery (Fox, 1991). Antimicrobial therapy, particularly by the oral route, may alter the normal flora, prolong the shedding period for *Salmonella* and increase the risk of the development of transferable antibiotic resistance. Ampicillin administered parenterally or orally was considered to be a significant risk factor in nosocomial outbreaks of salmonellosis in dogs (Uhaa *et al.*, 1988).

Antimicrobial therapy is required for potentially fatal septicaemic salmonellosis. The choice should be based on isolation and antibiotic susceptibility tests. However, most isolates are susceptible to the fluoroquinolone enrofloxacin, administered at 5 mg kg^{-1} *per os* every 12 h (Rutgers *et al.*, 1994). Chloramphenicol and trimethoprim–sulphonamide combinations may also be effective. Transfusion of plasma can reduce mortality in dogs with signs of severe endotoxaemia. Immunoglobulin for the treatment of endotoxaemia in dogs is available commercially in some countries.

Prevention

Prevention of both the carrier–excreter state and cases of clinical salmonellosis is desirable from the human health aspect, particularly in households with young children. Complete prevention of *Salmonella* infections is difficult, because of the numerous sources of *Salmonella* serovars available to dogs and cats, the viability of the organisms in the environment and the comparatively common occurrence of carrier animals. Because of the wide range of serovars that may infect domestic carnivores, vaccination is not currently a feasible proposition. However, thorough cooking of the animals' food, particularly meat and poultry products, good hygiene and the use of heat-processed commercial food products would eliminate major sources of *Salmonella* for dogs and cats. Uneaten, moist food should not be allowed to remain in food bowls at ambient temperatures for long periods, as *Salmonella* may be able to replicate in the food. Dry meals and biscuits must be stored carefully to avoid contamination by rodents or insects. Dogs and cats should not be allowed access to water from dubious sources or to unpasteurized milk. The hunting and scavenging activities of dogs and cats may increase their exposure to *Salmonella* serovars, but such instinctive behaviour is not easily controlled.

Preventative measures are particularly important in veterinary hospitals, where predis-

posing factors could initiate outbreaks of clinical salmonellosis. In addition to the measures suggested for household dogs and cats, meticulous attention should be paid to routine cleaning and to sterilization or disinfection of cages, food bowls and utensils. Cages should be steam-cleaned or disinfected with sodium hypochlorite (3%) or iodophors (2%). Endoscopic and other equipment likely to become contaminated should be sterilized after use.

Staff can spread *Salmonella* between patients on their hands, footwear or clothing. Routine hand washing, before and after handling hospitalized animals, should be observed and protective clothing must be changed and washed regularly.

If possible, boarders and animals with minor ailments should be separated from animals most at risk, particularly those undergoing major surgery or receiving immunosuppressive therapy. An ideal procedure would be to identify *Salmonella* excreters on arrival by cultural methods on 3 consecutive days, using enrichment procedures for isolation. However, practical considerations, such as emergency surgery, may render this strategy impractical. Any animal admitted with diarrhoea or developing diarrhoea while in the hospital should be isolated, a faecal sample cultured for *Salmonella* and procedures instituted to prevent fomite transmission. Animals with clinical salmonellosis usually excrete large numbers of organisms, thus posing a serious threat to those in their immediate vicinity.

Public Health Aspects

Sources of infection

Cats and dogs tend to be the most popular household pets in developed countries. It has been estimated that, by the beginning of the next millennium, the numbers of pet cats in the UK will reach 7.8 million and the dog population will have declined slightly to 6.8 million (Anon., 1996). These companion animals can spend a large proportion of their lives indoors in close contact with their owners. Some cats frequent or live on food premises as part of a rodent control programme. Because of their agility, felines can gain access to and contaminate food preparation areas. Humans can become infected, usually by the oral route, by *Salmonella* shed in faeces of either carriers or clinically affected animals. *Salmonella* can be present on hands after handling animals, on fomites or in contaminated food. Humans are considered more likely to acquire infection from cats than from dogs. Cats tend to rake soil or litter over their faeces, thus contaminating their paws. Oral swabs from infected cats have revealed the consistent presence of *Salmonella* organisms, leading to the contamination of their fur during grooming (Timoney *et al.*, 1978). This presence of the organisms over the cat's coat increases the potential for transfer of *Salmonella* to the hands and clothing of humans handling such animals. Many reported cases of pet-associated salmonellosis occur in young children, where a direct route of transmission may be of importance, as infants tend to place objects into their mouths indiscriminately (Kaufmann, 1966).

Isolation of the multiply resistant *S. typhimurium* DT104, resistance (R) type ampicillin, chloramphenicol, streptomycin, sulphonamides, tetracycline (ACSSuT) from cats and dogs is of public-health concern. This definitive type has become the second most prevalent *Salmonella*, after *S. enteritidis* PT4, in the human population in England and Wales. During the period from 1991–1995, of 110 *Salmonella* isolated from cats, 70 were *S. typhimurium* and 40 of these were DT104, R type ACSSuT. Four of the strains had additional resistance to trimethoprim (Wall *et al.*, 1996). *S. typhimurium* DT104 was shown to be excreted in the faeces of infected cats for 12 weeks after recovery from an acute episode of enterocolitis (Wall *et al.*, 1995).

Prevention

It is standard policy in many countries to exclude domestic carnivores from premises where food is prepared, stored or served. Animals should not be allowed to eat from the same food dishes as humans. Individuals handling dogs and cats must be made aware of the need to thoroughly wash their hands after contact with animals, especially before serving or consuming food. Because clinical salmonellosis is uncommon in dogs and cats, animals with diarrhoea may often be treated empirically, without submission of a faecal sample

for culture. Veterinarians should consider the possibility of salmonellosis in animals with enterocolitis and advise their clients on the zoonotic potential of such infections.

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References

Adler, H.E., Willers, E.H. and Levine, M. (1951) Incidence of *Salmonella* in apparently healthy dogs. *Journal of the American Veterinary Medical Association* 118, 300–304.

Anon. (1996) Cats as man's best friend. *Veterinary Record* 138, 99.

Ball, M.R. (1951) Salmonellosis in dogs and cats in the Los Angeles, Honolulu and Bermuda areas. *Journal of the American Veterinary Medical Association* 118, 164–166.

Borland, E.D. (1975) *Salmonella* infection in dogs, cats, tortoises and terrapins. *Veterinary Record* 96, 401–402.

Bruner, D.W. (1973) *Salmonella* cultures typed during the years 1950–1971 for the Service Laboratories of New York State Veterinary College at Cornell University. *Cornell Veterinarian* 63, 138–143.

Bruner, D.W. and Moran, A.B. (1949) *Salmonella* infections in domestic animals. *Cornell Veterinarian* 39, 53–63.

Buxton, A. (1957) Symptoms and lesions of *Salmonella* infections in animals: dogs and cats. In: *Salmonellosis in animals: a Review*. Review Series No. 5, Commonwealth Agricultural Bureaux, Farnham Royal, p. 101.

Calvert, C.A. (1985) Salmonella infections in hospitalized dogs: epizootiology, diagnosis and prognosis. *Journal of the American Animal Hospital Association* 21, 499–503.

Calvert, C.A. and Leifer, C.E. (1982) Salmonellosis in dogs with lymphosarcoma. *Journal of the American Veterinary Medical Association* 180, 56–58.

Carter, M.E., Dewes, H.G. and Griffiths, O.V. (1979) Salmonellosis in foals. *Journal of Equine Medicine and Surgery* 3, 78–83.

Carter, M.E., Cordes, D.O. and Carman, M.G. (1983) Observations on acute salmonellosis in four Waikato dairy herds. *New Zealand Veterinary Journal* 31, 10–12.

Center, S.A., Hornbuckle, W.E. and Hoskins, J.D. (1995) The liver and pancreas. In: Hoskins, J.D. (ed.) *Veterinary Pediatrics*. W.B. Saunders, Philadelphia, pp. 212–213.

Chengappa, M.M., Staats, J., Oberst, R.D., Gabbert, N.H. and McVey, S. (1993) Prevalence of *Salmonella* in raw meat used in diets of racing greyhounds. *Journal of Veterinary Diagnostic Investigation* 5, 372–377.

Clarke, R.C. and Gyles, C.L. (1993) *Salmonella*. In: Gyles, C.L. and Thoen, C.O. (eds) *Pathogenesis of Bacterial Infections in Animals*. Iowa State University Press, Ames, pp. 133–153.

Crow, S.E., Lauerman, L.H. and Smith, K.W. (1976) Pyelonephrosis associated with *Salmonella* infection in a dog. *Journal of the American Veterinary Medical Association* 169, 1324–1326.

Davies, R.H. and Wray, C. (1995) Mice as carriers of *Salmonella enteritidis* on persistently infected poultry units. *Veterinary Record* 137, 337–341.

Day, W.H., James, E. and Heather, C.D. (1963) Salmonellosis in the dog. *American Journal of Veterinary Research* 24, 156–158.

Dow, S.W., Jones, R.L., Henik, R.A. and Husted, P.W. (1989) Clinical features of salmonellosis in cats: six cases (1981–1986). *Journal of the American Veterinary Medical Association* 10, 1464–1466.

Förster, D., Holland, U. and Tesfamariam, H. (1974) Occurrence of *Salmonella* in the dog. *Zentralblatt für Veterinärmedizin B* 21, 120–134.

Fox, J.G. (1991) *Campylobacter* infections and salmonellosis. *Seminars in Veterinary Medicine and Surgery (Small Animal)* 6, 212–218.

Fox, J.G. and Beaucage, C.M. (1979) The incidence of *Salmonella* in random-source cats purchased for use in research. *Journal of Infectious Diseases* 139, 362–365.

Fox, J.G. and Galus, C.B. (1977) *Salmonella*-associated conjunctivitis in a cat. *Journal of the American Veterinary Medical Association* 171, 845–847.

Fox, J.G., Beaucage, C.M., Murphy, J.C. and Niemi, S.M. (1984) Experimental *Salmonella*-associated conjunctivitis in cats. *Canadian Journal of Comparative Medicine* 48, 87–91.

Frost, A.J., Eaton, N.F., Gilchrist, D.J. and Moo, D. (1969) The incidence of *Salmonella* infection in the dog. *Australian Veterinary Journal* 45, 109–110.

Galton, M.M., Scatterday, J.E. and Hardy, A.V. (1952) Salmonellosis in dogs. I. Bacteriological, epidemiological and clinical considerations. *Journal of Infectious Diseases* 91, 1–5.

Galton, M.M., Harless, M. and Hardy, A.V. (1955) *Salmonella* isolations from dehydrated dog meals. *Journal of the American Veterinary Medical Association* 126, 57–58.
Gorham, J.R. and Garner, F.M. (1951) The incidence of *Salmonella* infections in dogs and cats in a non-urban area. *American Journal of Veterinary Research* 12, 35–37.
Groisman, E.A., Fields, P.I. and Heffron, F. (1990) Molecular biology of *Salmonella* pathogenesis. In: Iglewski, B.H. and Clark, V.L. (eds) *The Bacteria. XI Molecular Basis of Bacterial Pathogenesis*. Academic Press, San Diego, pp. 251–272.
Hemsley, L.A. (1956) Abortion in two cats, with the isolation of *Salmonella choleraesuis* from one case. *Veterinary Record* 68, 152.
Humphrey, T.J. (1990) Growth of salmonellas in intact shell eggs: influence of storage temperatures. *Veterinary Record* 126, 292.
Humphrey, T.J., Baskerville, A., Chart, H. and Rowe, B. (1989) Infection of egg-laying hens with *Salmonella enteritidis* PT4 by oral inoculation. *Veterinary Record* 125, 531–532.
Jones, G.W., Rabert, D.K. Svinarich, D.M. and Whitfield, H.J. (1982) Association of adhesive, invasive and virulent phenotypes of *Salmonella typhimurium* with autonomous 60-megadalton plasmids. *Infection and Immunity* 38, 476–486.
Kaufmann, A.F. (1966) Pets and *Salmonella* infection. *Journal of the American Veterinary Medical Association* 149, 1655–1661.
Khan, A.Q. (1970) *Salmonella* infections in dogs and cats in the Sudan. *British Veterinary Journal* 126, 607–611.
Kintner, L. (1949) Canine salmonellosis. *Veterinary Medicine* 44, 396–398.
Kirkwood, J.K., Holmes, J.P. and Macgregor, S. (1995) Garden bird mortalities. *Veterinary Record* 136, 372.
Krum, S.H., Stevens, D.R. and Hirsh, D.C. (1977) *Salmonella arizonae* bacteraemia in a cat. *Journal of the American Veterinary Medical Association* 170, 42–44.
Laing, P.W. (1990) *Salmonella typhimurium* in various species. *Veterinary Record* 126, 173.
McElrath, H.B., Galton, M.M. and Hardy, A.V. (1952) Salmonellosis in dogs. III Prevalence in dogs in veterinary hospitals, pounds and boarding kennels. *Journal of Infectious Diseases* 91, 12–14.
Mackel, D.C., Galton, M.M., Gray, H. and Hardy, A.V. (1952) Salmonellosis in dogs. IV Prevalence in normal dogs and their contacts. *Journal of Infectious Diseases* 91, 15–18.
Madewell, B.R. and McChesney, A.E. (1975) Salmonellosis in a human infant, a cat and two parakeets in the same household. *Journal of the American Veterinary Medical Association* 167, 1089–1090.
Milstein, M. (1975) *Salmonella dublin* septicaemia in a Scottish terrier recently imported from England. *Canadian Veterinary Journal* 16, 179–180.
Morse, E.V. and Duncan, M.A. (1975) Canine salmonellosis: prevalence, epizootiology, signs and public health significance. *Journal of the American Veterinary Medical Association* 167, 817–820.
Nakamura, M., Sato, S., Ohya, T., Suzuki, S. and Ikeda, S. (1985) Possible relationship of a 36-megadalton *Salmonella enteritidis* plasmid to virulence in mice. *Infection and Immunity* 47, 831–833.
Nation, P.N. (1984) *Salmonella dublin* septicaemia in two puppies. *Canadian Veterinary Journal* 25, 324–326.
Neill, S.D., McNulty, M.S., Bryson, D.G. and Ellis, W.A. (1981) Microbiological findings in dogs with diarrhoea. *Veterinary Record* 109, 538–539.
Quinn, P.J., Carter, M.E., Markey, B.K. and Carter, G.R. (1994) Enterobacteriaceae. In: Quinn, P.J., Carter, M.E., Markey, B.K. and Carter, G.R. (eds) *Clinical Veterinary Microbiology*. Mosby-Year Book Europe, London, pp. 209–236.
Redwood, D.W. and Bell, D.A. (1983) *Salmonella panama*: isolation from aborted and newborn canine fetuses. *Veterinary Record* 112, 362.
Reilly, G.A.C., Bailie, N.C., Morrow, W.T., McDowell, S.W.J. and Ellis, W.A. (1994) Feline stillbirths associated with mixed *Salmonella typhimurium* and leptospira infection. *Veterinary Record* 135, 608.
Rodriguez, C.O., Moon, M.L. and Leib, M.S. (1993) *Salmonella choleraesuis* pneumonia in a cat without signs of gastrointestinal tract disease. *Journal of the American Veterinary Medical Association* 202, 953–955.
Rutgers, H.C., Stepien, R.L., Elwood, C.M., Simpson, K.W. and Batt, R.M. (1994) Enrofloxacin treatment of Gram-negative infections. *Veterinary Record* 135, 357–359.
Salyers, A.A. and Whitt, D.D. (1994) *Salmonella* infections. In: Salyers, A.A. and Whitt, D.D. (eds) *Bacterial Pathogenesis – a Molecular Approach*. ASM Press, Washington, DC, pp. 229–243.
Shimi, A. and Barin, A. (1977) *Salmonella* in cats. *Journal of Comparative Pathology* 87, 315–318.
Shimi, A., Keyhani, M. and Bolurchi, M. (1976) Salmonellosis in apparently healthy dogs. *Veterinary Record* 98, 110–111.
Sinell, H.J., Klingbeil, H. and Benner, M. (1984) Microflora of edible offal with particular reference to *Salmonella*. *Journal of Food Protection* 47, 481–484.

Stone, G.G., Oberst, R.D., Hays, M.P., McVey, S., Galland, J.C., Curtiss, R., Kelly, S.M. and Chengappa, M.M. (1995) Detection of *Salmonella typhimurium* from rectal swabs of experimentally infected beagles by short cultivation and PCR-hybridization. *Journal of Clinical Microbiology* 33, 1292–1295.

Stucker, C.L., Galton, M.M., Cowdery, J. and Hardy, A.V. (1952) Salmonellosis in dogs. II Prevalence and distribution in greyhounds in Florida. *Journal of Infectious Diseases* 91, 6–11.

Takeuchi, A. and Sprinz, H. (1967) Electron microscope studies of experimental *Salmonella* infections in the preconditioned guinea-pig. 2. Response of the intestinal mucosa to the invasion of *Salmonella typhimurium*. *American Journal of Pathology* 51, 137–146.

Tanaka, Y., Katsube, Y. and Imaizumi, K. (1976a) Distribution of salmonellae in the digestive tract and lymph node of carrier-dogs. *Japanese Journal of Veterinary Science* 38, 215–224.

Tanaka, Y., Katsube, Y. and Imaizumi, K. (1976b) Experimental carrier in dogs produced by oral administration of *Salmonella typhimurium*. *Japanese Journal of Veterinary Science* 38, 569–578.

Tanaka, Y., Katsube, Y. and Imaizumi, K. (1977) Virulence of *Salmonella typhimurium* harboured in feces of carrier-dog for mice. *Japanese Journal of Veterinary Science* 39, 353–356.

Terakado, N., Sekizaki, T., Hashimoto, K. and Naitoh, S. (1983) Correlation between the presence of a fifty-megadalton plasmid in *Salmonella dublin* and virulence for mice. *Infection and Immunity* 41, 443–444.

Timbs, D.V., Durham, P.J.K. and Barnsley, D.G.C. (1974) Chronic cholecystitis in a dog infected with *Salmonella typhimurium*. *New Zealand Veterinary Journal* 22, 100–102.

Timbs, D.V., Davies, G.B., Carter, M.E. and Carman, M.G. (1975) The *Salmonella* excretor incidence of dogs in Hawke's Bay. *New Zealand Veterinary Journal* 23, 54–56.

Timoney, J.F. (1976) Feline salmonellosis. *Veterinary Clinics of North America: Small Animal Practice* 6(3), 395–398.

Timoney, J.F. (1978) The epidemiology and genetics of antibiotic resistance of *Salmonella typhimurium* isolated from diseased animals in New York. *Journal of Infectious Diseases* 137, 67–73.

Timoney, J.F. Neibert, H.C. and Scott, F.W. (1978) Feline salmonellosis: a nosocomial outbreak and experimental studies. *Cornell Veterinarian* 68, 211–219.

Uhaa, I.J., Hird, D.W., Hirsh, D.C. and Jang, S.S. (1988) Case–control study of risk factors associated with nosocomial *Salmonella krefeld* infection in dogs. *American Journal of Veterinary Research* 49, 1501–1505.

Venter, B.J. (1988) Epidemiology of salmonellosis in dogs – a conceptual model. *Acta Veterinaria Scandinavica* 84, 333–336.

Veterinary Investigation Services (1988–1996) Reports, published in the *Veterinary Record*.

Wall, P.G., Davis, S., Threlfall, E.J., Ward, L.R. and Ewbank, A.J. (1995) Chronic carriage of multidrug resistant *Salmonella typhimurium* in a cat. *Journal of Small Animal Practice* 36, 279–281.

Wall, P.G., Threlfall, E.J., Ward, L.R. and Rowe, B. (1996) Multiresistant *Salmonella typhimurium* DT 104 in cats: a public health risk. *Lancet* 348, 471.

Weber, W.J. (1982) Salmonellosis. In: Weber, W.J. *Diseases Transmitted by Rats and Mice*. Thomson Publications, Fresno, California, pp. 46–52.

Wilson, J.E. and MacDonald, J.W. (1967) *Salmonella* infection in wild birds. *British Veterinary Journal* 123, 212–219.