growth hormone (4, 9). Not only do genes need to be identified, but they must be transferred, integrated, expressed and regulated without deleterious effects. This will require much more research, especially for traits affected by multiple genes.

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Epidemiology of Bovine Salmonellosis

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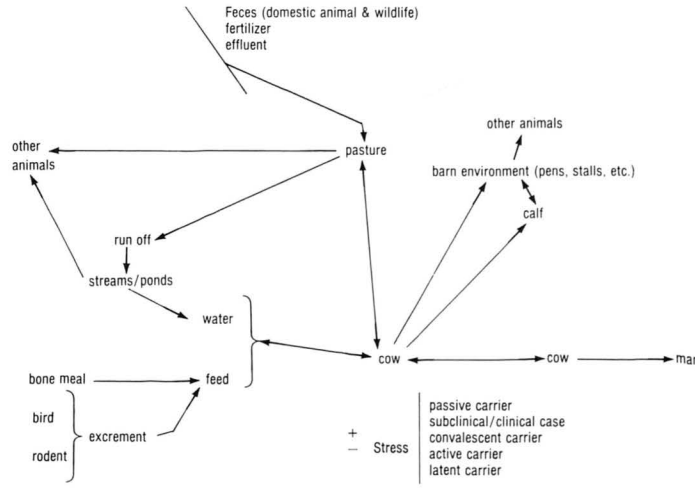
Salmonellosis is a disease with great economic and public health impact. The burdens placed upon the animal industry each year due to salmonellosis amount to millions of dollars. Depending upon which salmonella serotype is involved in the disease process, there are also other associated problems: high morbidity in affected herds, abortion sequels, the problems of carrier cattle and the reoccurrence of salmonellosis in herds. Moreover, survivors of outbreaks are often unthrifty and take longer to reach marketable weight or do not regain lost milk production. Along with the lost revenues from decreased milk production are the costs of therapy and replacement animals, and public health problems leading to restrictions on the sale of milk and/or meat products.

Because of the ubiquity of the many salmonella serotypes in the environment, water, birds, rodents, etc., in feed stuffs that are easily contaminated, and because carrier animals are common, it is unlikely that salmonellosis will be eradicated. The best defense against salmonellosis, then, is prevention and control. Our efforts must be directed toward understanding the epidemiology of salmonellosis with the aim of breaking the cycles of infection. This research summary is a brief discussion of the epidemiology of salmonellosis in cattle, including the effects of stress and infection, the changing pattern of salmonellosis in cattle, and the use of modern epidemiological techniques that enable the fingerprinting of strains of Salmonella.

Epidemiology

The general epidemiological pattern of bovine salmonellosis is depicted in Figure 1. In many outbreaks it is not possible to definitely identify the source(s) of herd infection because site investigations are often conducted well after an outbreak has started (11, 12). Sometimes, however, through extensive bacterial cultures of animals, feed, water, and the environment early in an outbreak, a source may be identified (9). A cow may become infected in many ways. Once infected an acutely ill animal can rapidly contaminate the environment and serves as a focus to infect other cattle (24). The resulting widespread environmental contamination can make it very difficult for the epidemiologist to sort out the problem.

FIGURE 1. Epidemiology of Bovine Salmonellosis — Adult Cattle.



From Figure 1 it is easy to see how a dairy calf may become infected by Salmonella. Exposure to a rapidly contaminated environment and/or udder teat surfaces are ready avenues of infection for the calf (7, 26). Even under optimal conditions, calves that are born indoors during colder months in a "clean" freshening stall that is physically apart from the rest of the herd may become infected. Usually a breakdown in management occurs that has allowed the calf to be exposed to contaminated manure on boots, clothing, etc. A seemingly healthy but subclinically infected carrier cow may also infect her calf when the "stress" of parturition causes her to become a clinical case (31). Calves born at pasture during warmer months of the year are at lesser risk of developing salmonellosis because the lower density of cattle results in exposure to lower numbers of salmonella. Once again management breakdowns that allow increased exposure of the neonate to contaminated manure (e.g., spreading slurry onto pasture rather than cropland) will negate the benefits of low density grazing on pasture. Veal calves, in contrast to dairy calves, are at extreme risk of salmonellosis due to transport stress, feed practices, concurrent disease and the many stresses of intensive husbandry systems (Figure 2) (14, 21, 22, 26, 27).

A spectrum of disease occurs in salmonellosis, i.e., once exposed an animal may go through stages of infection—from an incubating, subclinical state—to a subacute or acute clinical case—to convalescence or death. Some animals never progress beyond the subclinical stage. Convalescent cases may ultimately be free of salmonella infection or may become chronically infected animals or carriers. Young calves are more likely to develop acute septicemia, especially if they are colostrum-deprived. Adult cattle may have a localized gastrointestinal infection unless severely stressed (8, 10, 18).

The various classifications of animals with salmonellosis may seem academic, but they do serve to identify animals that may be shedding Salmonella (22) (Table 1). The period of communicability or shedding of Salmonella can vary for different salmonella serotypes (9, 23, 25, 32). The concept of the carrier state in bovine salmonellosis is very important as it relates to herd infection, stress, and to prevention and control aspects of salmonellosis (see below). Carriers serve as continuing foci of infection for the herd. Carriers are difficult to detect without doing multiple fecal cultures in the herd.

The reader should beware of the scientific literature on salmonellosis. A great deal of the literature on bovine salmonellosis discusses the European situation with *Salmonella dublin.* It is important to "think" serotype when discussing Salmonellosis, because the pattern of disease is different for different serotypes (23, 24). The Salmonella problem in the Northeastern U.S.A. involves *S. typhimurium* predominantly. In New York State increasing numbers of outbreaks are caused by salmonellae from serogroup E, e.g., *S. anatum, S. muenster* (15, 17). *Salmonella dublin* is slowly making its way across the U.S.A. from

FIGURE 2. Epidemiology of Bovine Salmonellosis — Veal Calf.

cow to calf:	on farm of origin directly — transplacental: S. dublin milk excretion fecal-udder contamination indirectly — contamination of barn floors, buckets, feed, water by feces.
transportation:	increased exposure in trucks; crowding in sale yards; tendency for calves to suckle each other.
increased susceptibility of the neonate:	questionable immune status — has calf received colostrum, was it of good quality, too little — too late?
problem in veal unit:	poor husbandry! stress! diet — is milk replacer of good quality?; problem of denatured milk proteins. crowding — poor ventilation (high humidity; ammonia vapor in air from urine buildup in drains: effect on respiratory defense mechanisms). intercurrent disease — (parasitism; colibacillosis; enteric viruses; viral and bacterial pneumonias).
in general:	in conditions of intensified husbandry with stress factors, a rapid buildup of Salmonella and other organisms will occur in the environment. This is in association with a compromised host population of neonatal calves whose immune status is questionable.

TABLE 1. Animals that Shed Salmonella.

classification		comment
passive carrier		— This animal is a "living fomite", and is not actually infected. Contaminated feed is passing through its intestinal tract. Many parameters interact to determine if a host animal will become infected including dose of Salmonella ingested. Nevertheless such animals contaminate their environment. Passive carriers are at risk of infection.
case	incubating (subclinical) case ↓ ↓ clinical case ↓ ↓ convalescent carrier and chronic cases	— Part of a spectrum of disease: these animals are truly infected, i.e., Salmonella have invaded and multiplied in the intestinal mucosa, and may be shedding varying numbers of Salmonella. Animals that are recovering from disease may still be shedding for varying periods of time.
active carrier		Seemingly healthy animals that shed intermittently without apparent stress.
latent carrier		These apparently healthy animals shed only when stressed. Both active and latent carriers may be subclinical cases or recovering animals.

the west. It is only a matter of time before it could become established in the dairy herds of the Northeast. A brief comparison of some features of *S. typhimurium* and *S. dublin* infections in the bovine is given in Table 2.

TABLE 2. S. dublin versus S. typhimurium infections in the bovine.

	S. dublin	**S. typhimurium**
Serogroup	D	B
Host Range	A host-adapted serotyle found mostly in cattle.	A non-host adapted serotype affecting many species; ubiquitous in the environment.
Carrier State	Once infected, adult cattle are carriers for life and are foci of infection for the herd; calf cases are usually associated with presence of adult carrier.	Infected adult cattle shed less than 6 or so months; adult cattle **and** other animal species are important in the cycle of infection for calves.
Abortions	commonly occur	less common

Risk Factors Affecting Severity of Infection and Stress

Bovine salmonellosis results from the interaction of the disease agent (Salmonella), the host animal (a colostrum-deprived calf or stressed cow), and the environment (crowding, temperature extremes)—the well known epidemiological triad of disease. A dynamic cycle of infection exists and many factors interact including management and stress (see below) to determine the severity of infection in each animal. A few of the risk factors affecting the severity of infection in an exposed animal are found in Table 3. The carrier cow or a newly exposed cow also is at greater risk of developing clinical salmonellosis if the animal is "stressed" (10, 22). Stress has been variously defined as referring to not any single or even a set of body reactions, but to a heterogeneous assortment of phenomena that may predispose an animal to infection; also stress may be a profound physiological change in an animal's condition generally leading to disease (6). From the experience of salmonellosis in New York State cattle over the past twelve years, stress has played a significant role in the development of disease (15). In fact, in some herds, without the added stress factor(s), some disease outbreaks might not have occurred (9). Table 4 lists some of the stress factors thought to interact to contribute to the development of bovine salmonellosis.

TABLE 3. Risk Factors of Bovine Salmonellosis.

1. salmonella serotype involved (its relative virulence).
2. dose ingested.
3. route of exposure.
4. age of host (neonate and immature calves at greatest risk).
5. pre-existing infection(s)
6. prior exposure to Salmonella and immunological status of host (colostrum-deprived calf at risk; BVD immunosuppressed cow at risk; previous exposure may confer some degree of immunity).
7. nutritional plane of host (affects overall well-being of animal, including the immune system; starvation may lower the volatile fatty acids of rumen and large intestine which are a protective factor in the gastrointestinal tract).
8. stress.

TABLE 4. Stress Factors and Bovine Salmonellosis.

1. shipment (crowding, exhaustion, dehydration, starvation).
2. weather extremes (especially sudden changes).
3. parturition.
4. surgery and associated procedures (shipment to hospital, food and water deprivation, antimicrobials, anesthesia).
5. vaccination (MLV-BVD immunosuppression).
6. concurrent disease.
7. parasite load.
8. poor nutrition (resulting in starvation, indigestion from poor quality feeds, moldy feeds, overheated feed, frosted feed, grain excess, concentrate excess).
9. sudden food or water deprivation.
10. feed changes (especially today with so many additives and custom-made diets; resulting in rumen fatty acid changes and changes in resident microbial flora).
11. contaminated feed (toxic materials: herbicides, mycotoxins and effect on immnne system).
12. oral administration of drugs (antimicrobials, pH-altering drugs and their effect on resident microbial flora).
13. crowding.
14. poor ventilation (humidity, ammonia fumes).

Pattern of Disease

In New York state surveillance of salmonellosis by the diagnostic and clinical laboratories at the New York State College of Veterinary Medicine has revealed an apparent increased evidence of bovine salmonellosis since 1978 (Table 5). Also there are some indications that the observed pattern of disease may be changing from that of sporadic cases of one or two animals to epidemics (Table 5). This is what occurred in Northwest England in the 1970's and was associated with a change in housing from tie stalls to loose housing (25). The typical pattern of disease in New York state is either a 1) sporadic case—where a stressed carrier cow two to four days post-parturition, or post-shipment, or post-feed change develops salmonellosis and, depending on the management practices (ability to isolate cases, etc.) and

TABLE 5. Culture-confirmed Salmonellosis in Cattle in New York State.

	1978	**1979**	**1980**	**1981**
Dairy	4[a] (75)[b]	18 (72)	28 (61)	29 (55)
Veal	1 (100)	5 (100)	1 (100)	1 (100)
Other	—	—	2	6
Total	5	23	31	37

[a] these represent only herds from cases accessioned at the New York State Diagnostic Laboratory at the New York State College of Veterinary Medicine; separate premises only are listed.

[b] number in parentheses indicates percentage of total dairy herds in New York State that had outbreaks of salmonellosis rather than individual cases.

type of housing, the infection is either confined to that one case or 2) it spreads throughout the herd to become epidemic (15). Epidemics may also occur when there is a point source of infection (feed) or exposure from the environment and many animals are infected at once. Stress factors also have been involved in New York State epidemics (15). The apparent higher incidence of salmonellosis and the greater number of outbreaks in cattle in New York State may be the result of modern management practices of increased herd size, more intensive husbandry, and the popularity of free-stall housing that permits greater chances of spread of disease once introduced into the herd.

Salmonella Serotypes

The major serotype of Salmonella isolated from New York State cattle has remained *S. typhimurium,* including its antigenic variant Copenhagen (4, 17). Since 1979 many new serotypes have emerged in New York State cattle. Most of these new serotypes have been serogroup E salmonellas such as *S. anatum* or *S. senftenberg* (17). All of these new serotypes had not been reported in New York cattle by Dr. D. W. Bruner while serotyping salmonellae during the period 1950-1971 (4). Possible reasons for the occurrence of new salmonella serotypes in the bovine population are the introduction of contaminated feed stuffs and infected animals or birds into New York from other geographical areas. There is evidence that the reservoir of some of the serogroup E salmonellas is wild bird populations (9, 15).

Clonal Groups of S. typhimurium in New York State Animals

Using modern epidemiological techniques, it is possible to differentiate strains of apparently similar salmonellae by fingerprinting the strains. Through a combination of phage typing (13), biotyping (5), and the analysis of antibiogram patterns (3, 30) and plasmids (19), there is evidence that clonal groups of *S. typhimurium* exist in the cattle population of New York State (16). Strains of Salmonella that share matching fingerprint characters (phage type, biotype, plasmids) can often be designated clonal lines (2, 20). In fact strains isolated from different locations, from different sources but sharing many fingerprint traits may have a common origin. The ability to distinguish clonal lines of *S. typhimurium* has enabled the epidemiologist to study the phylogeny and spread of strains of *S. typhimurium* through different animal populations by confirming the clonal relationship between strains obtained from widely separated sources of the clone.

The major reason for fingerprinting a strain of *S. typhimurium,* then, is to distinguish it from, or as part of, a clonal line. The best example of the usefulness of the clonal concept can be illustrated by British studies of the outbreak of *S. typhimurium* phage type 49-204-193 (28, 29). In this *S. typhimurium* spread from calves to the human population. Phage typing alone indicated that three different strains of *S. typhimurium* were involved in this widespread outbreak, but subsequent biotyping studies showed that 98% of the strains of phage types 49 and 204 were the same biotype 26a. Such close association of two phage types, 49 and 204, with one biotype 26a provided supportive evidence to show that phage type 204 was derived from phage type 49 after 49 acquired plasmids (1). While many phage types were found in the cattle in New York State, phage types U275, 49, 10, and 2 were most common (16). The combination of biotyping with phage typing showed that phage type/biotypes U275/26ei, 49/26ei, 10/3a, and 2/3a were most common. U275/26ei and 49/26ei were widespread in New York from 1973 to 1976 and were found mainly in calves, especially veal. The decline of these two phage type/biotypes, perhaps, as the predominant clones after 1976 was associated with the decline of veal calf salmonellosis noted statewide. In contrast, phage type/biotype 10/3a was seen sporadically from 1973 to 1978 in other animal species, and after 1978 was widespread mostly in adult cattle, not calves. In contrast to U275/26ei and 49/26ei which had many plasmids and were antibiotic resistant, 10/3a was relatively plasmidless and antibiotic sensitive. Phage type/biotype 2/3a was found from 1973 to 1981 but was not widespread. After 1976 2/3a was seen mostly in adult cattle, not calves. As with 10/3a, 2/3a was antibiotic sensitive and relatively plasmidless. It is unknown why different clones of *S. typhimurium* have occurred in New York state cattle or why some clones predominate more than others in different animal groups. Possibly the widespread exchange of animals and feedstuffs between states and Canada can account for the clonal changes.

With surveillance schemes at the state and national levels, phage and biotyping of strains of *S. typhimurium* and plasmid analysis for all salmonella serotypes will help us to monitor the creation of new epidemic strains. Once Salmonella has become established in an animal population, epidemiological studies provide information needed to pinpoint sources of infection and to coordinate control measures to prevent further disease. The cooperation of clinicians, researchers, and diagnosticians is needed to control salmonellosis in domestic animals. This paper has presented the basic epidemiology of bovine salmonellosis in light of recent epidemiological research of changing patterns of bovine salmonellosis in New York state.

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Comparison of Oral and Intravenous Fluid Therapy in Neonatal Calves with Experimental Colibacillosis

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Fluid therapy is widely recognized as the primary means of correcting the abnormalities of volume, electrolyte and acid-base status that occur in severe diarrhea. Commercially available oral electrolyte preparations are commonly used and likely prevent the loss of many dehydrated, scouring calves. Intravenous fluid therapy is effective in restoring body fluid and electrolytes but has the disadvantages of 1) greater expense, 2) less availability of supplies to farmers, 3) more skill needed to administer and 4) more time required for proper delivery. Most would agree that calves in coma or severe shock will not respond favorably to oral therapy. These clearly require immediate intravenous fluids. Those calves that are dehydrated and depressed but not yet shocky represent a grey area. Will oral fluids be utilized adequately or must therapy be more aggressive? These questions prompted our study comparing oral and intravenous therapy in experimentally produced diarrhea.

The Model

Male Holstein calves (n=25) were purchased from nearby farms and obtained within 3 hours of birth. Blood samples were collected and a fixed quantity of pooled colostrum given. The calves were transported to an isolation unit and prepared for quantitative collection of feces and urine. At 12 hours of age they were inoculated orally with a log-phase broth culture of *Escherichia coli* 0101:K(A), originally isolated from a calf with severe diarrhea. Calves were allotted in advance to 1 of 4 treatment groups. Treatments