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A review of Shiga toxin *Escherichia coli* and *Salmonella enterica* in cattle and free-ranging birds: potential association and epidemiological links

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Abstract: Cattle are the main reservoir for human infection by pathogenic *Escherichia coli* and *Salmonella enterica*. To prevent entry of these foodborne pathogens into the human food chain, management factors at the farm level must be identified and controlled. External sources of contamination, such as birds, should be considered as potential sources of transmission over long distances. In this review, we focus on the epidemiology of infection by *E. coli* and *S. enterica* and the consequences of birds acting as disseminators of these pathogens at dairy farms in terms of cattle health and the subsequent effects on human health.

Key words: birds, cattle, *Escherichia coli*, prevalence, *Salmonella enterica*, salmonellosis, STEC

Two members of the family *Enterobacteriacea*, *Escherichia coli* and *Salmonella enterica*, are ubiquitous microorganisms that cause intestinal infection in humans and animals (Farmer 2003). Although innocuous *E. coli* predominate among the normal flora of the vertebrate intestine, pathogenic forms exist that cause disease of varying severity in humans and other animals (Lewis 1997, Bell and Kyriakides 1998). Similarly, *S. enterica* may live harmlessly in the intestinal tract, but it is one of the most common foodborne illnesses in humans (Lewis 1997), and it is associated with illness in animals. Because cattle are the main reservoir for human infection by pathogenic *E. coli* and *S. enterica* (Wells et al. 2001), it is assumed that minimizing the presence of these pathogens in the herd will reduce the number of infected cattle sent to slaughter, thus minimizing disease risk to humans (Herriott et al. 1998). As a consequence, research on farm management practices has attempted to identify factors affecting pathogen prevalence in cattle (Hancock et al. 1994, Hancock et al. 1997, Kabagambe et al. 2000). However, 1 source of farm management and biosecurity assessment that has received little attention has been the role that peridomestic and free-ranging birds have in pathogen transmission. In this paper, we review the epidemiology of infection by a pathogenic group of *E. coli* known as shiga toxin *E. coli* (STEC) and *S. enterica* in free-ranging birds and cattle as these bacteria relate to herd management and human health.

The STEC group of *E. coli* produces toxins similar to those produced by *Shigella dysenteriae* (Wray and Woodward 1997). STEC, sometimes referred to as verocytotoxin-producing *E. coli* (VTEC), are capable of causing hemorrhagic colitis (inflammation of the colon resulting in bloody diarrhea), hemolytic uremic syndrome (bloody diarrhea followed by renal failure), hemolytic anemia (fragmented red blood cells), and thrombotic thrombocytopenic purpura (a blood disorder characterized by low platelets, low red blood cell count, abnormalities in kidney and neurological functions (Lewis 1997, Saunders et al. 1999, Donnenberg and Nataro 2000, Murinda et al. 2002) in humans. STEC also have been identified as the causative agents of diarrhea and disease in other animals including cattle, pigs, sheep, goats, cats, and dogs (Willshaw et al. 1997, Wray and Woodward 1997, Zschöck et al. 2000).

The epidemiology of outbreaks of STEC in humans is linked to the consumption of contaminated bovine products (Griffin 1995, Zhao et al. 1995, Mechie et al. 1997, Wray et al. 2000). Dairy cattle have been identified as the main reservoir of STEC (Wells et al. 1991, Faith et al. 1996, Shere et al. 1998, Schmidt et al. 2000). As a result, research has been dedicated to understanding infection in cattle and identifying management factors that can be modified to minimize the occurrence of STEC in the herd. Efforts also have been directed

toward preventing infection in calves because their immature immune systems cause greater morbidity and mortality from STEC infection than in adults (Frank and Kaneene 1993).

The genus *Salmonella* consists of more than 2,600 serotypes that cause a wide spectrum of illness in humans (García-Del Portillo 2000). Most serotypes are not host-specific, although some serotypes are associated more commonly with infection in humans (*S. typhi*, *S. paratyphi*, Table 1), sheep (*S. abortovis*), and poultry (*S. gallinarum*) (Varnam 1991, Daniels et al. 1993, Bell and Kyriakides 2002).

Table 1. The top 10 most frequently reported *Salmonella* serotypes from cattle and humans in Colorado reported to Centers for Disease Control (CDC) in 2003 (2004).

Human	Cattle
S. typhimurium	*S. newport*
S. enteritidis	*S. typhimurium* var. 5-
S. newport	*S. typhimurium*
S. heidelberg	*S. montevideo*
S. javiana	*S. agona*
S. montevideo	*S. dublin*
S. saintpaul	*S. kentucky*
S. muenchen	*S. muenster*
S. oranienburg	*S. uganda*
S. infantis	*S. anatum*

S. enterica have been identified as the causative agent of disease in many animals including sheep, cattle, pigs, reptiles, and various bird species (Girdwood et al. 1985, Gay and Hunsaker 1993, Hudson et al. 2000, Winfield and Groisman 2003). Because cattle represent a major reservoir for human foodborne infection (Wells et al. 2001), research efforts have been directed towards reducing *S. enterica* in the herd to prevent contamination at the slaughterhouse and subsequent entry into the human food chain. Prevention of salmonellosis in cattle is a critical factor for dairy producers to reduce economic losses incurred from culling, reduced feed efficiency, decreased weight gain, decreased milk production, abortion, and mortality (Huston et al. 2002).

Sources of infection

Various food products have been implicated in STEC and *S. enterica* infection in humans. Implicated products include raw seeds (Murinda et al. 2002), fruit juice (Griffin 1995, Pell 1997, Zschöck et al. 2000, Bell and Kyriakides 2002), salad (Murinda et al. 2002), fresh fruits (Bacon et al. 2002, Murinda et al. 2002, Winfield and Groisman 2003), alfalfa sprouts (Barak et al. 2002, Bell and Kyriakides 2002), melon (Fukushima et al. 1999), well water (Pell 1997, Fukushima et al. 1999, Zschöck et al. 2000, Murinda et al. 2002), pork (Bacon et al. 2002), chicken (Bacon et al. 2002), seafood (Fukushima et al. 1999), eggs (Pell 1997, Bacon et al. 2002), bean sprouts (Barak et al. 2002, Bell and Kyriakides 2002), salami (Bell and Kyriakides 2002), chocolate (Bell and Kyriakides 2002), vegetables (Pell 1997, Zschöck et al. 2000, Winfield and Groisman 2003), milk and milk products (Griffin 1995, Zhao et al. 1995, Zschöck et al. 2000, Bacon et al. 2002), and undercooked ground beef and other bovine products (Hancock et al. 1994, Zhao et al. 1995, Mechie et al. 1997, Troutt and Osburn 1997). Although outbreaks attributed to contaminated non-animal products are important sources of human infection, fecal contamination of meat by infected cattle is the most common source of STEC infection (DebRoy and Maddox 2001, Troutt et al. 2001) and a major cause of *S. enterica* infection in humans (Wells et al. 2001).

Human health risks associated with dairy cattle

Fecal shedding of *E. coli* O157 occurs in the most cattle herds, with an increase in prevalence occurring during summer months when environmental conditions such as temperature and moisture are conducive to growth of *E. coli* (Herriott et al. 1998, Van Donkersgoed et al. 1999). Shedding of *S. enterica* is common in cattle during late summer and early fall (McEvoy et al. 2003). Infected animals often appear asymptomatic and shed STEC and *S. enterica* intermittently, making detection and diagnosis based on clinical signs more difficult (Kabagambe et al. 2000, Sargeant et al. 2000, Wray and Davies 2000, Yilmaz et al. 2002). Limiting shedding in cattle is essential to prevent dissemination of the bacteria throughout the herd and to reduce the risk of carcass contamination with infected fecal matter at the slaughterhouse, and subsequent entry of STEC or *S. enterica* into the human food chain.

Cattle were identified as the primary reservoir of *E. coli* strain O157:H7 (Montenegro et al. 1990, Cobbold and Desmarchelier 2000, Kobayashi et al. 2001, Yilmaz et al. 2002), soon after it was first recognized in 1982 as an important disease-causing pathogen in humans (Zhao et al. 1995, Faith et al. 1996, Troutt and Osburn 1997). Initially, *E. coli* O157 was thought to be the only serogroup of *E. coli* important in human disease, but since then many non-O157 STEC pathogenic strains of *E. coli* have been identified (Griffin 1995, Beutin et al. 1998, Appendix). Cattle are also major reservoirs of *S.*

enterica (Wells et al. 2001), but unlike *E. coli*, all strains of *S. enterica* are potentially pathogenic to humans (Lewis 1997, McEvoy et al. 2003)

Most dairy cattle enter the meat market when milk production declines, reproduction fails, or mastitis, disease or injury becomes debilitating (Troutt and Osburn 1997). According to the USDA's National Animal Health Monitoring System 1996 Dairy Study, 30.9% of cull (market) dairy cattle were shedding verotoxogenic *E. coli* O157, and 66.7% were shedding *Salmonella* (Wells et al. 1998*a*). Transportation, modified food rations, and poor health are stressors that induce *S. enterica* and STEC shedding (Daniels et al. 1993, Fedorka-Cray et al. 1998, Galland et al. 2001), and consequently create additional opportunities for contamination of carcasses with enteric bacteria or interchange of bacteria between carcasses (Bettelheim 1997). Contamination at the slaughterhouse increases the risk that the consumer will eat tainted meat, especially if it is undercooked. Limiting contamination is desirable because approximately 17% of the nation's ground beef supply is produced from culled dairy cattle (Troutt and Osburn 1997, Wells et al. 1998*b*), and the infectious dose of STEC for humans is as low as 10 bacteria (Fukushima et al. 1999).

Various modes of indirect transmission pose a threat to cattle health. For example, manure slurry often is applied as fertilizer to agricultural crops and pastures used for grazing (Mechie et al. 1997). Due to the long-term survival of STEC and *S. enterica* in feces (Pell 1997, Fukushima et al. 1999, Huston et al. 2002), cattle may become infected indirectly by STEC or *S. enterica* while grazing on pastures fertilized with contaminated slurry or by eating silage or other feed harvested from fields treated with slurry (Mechie et al. 1997, Himathongkham et al. 1999). The length of time that STEC and *S. enterica* are able to survive in the environment depends on the manure's source, pH, dry matter content, age and chemical composition (Pell 1997). *E. coli* can survive in the environment anywhere from 1 (Winfield and Groisman 2003) to 105 days (Himathongkham et al. 1999), whereas *S. enterica* can survive up to a year in soil (Winfield and Groisman 2003). In a prevalence survey of 60 dairy cattle herds, a tentative association was found between the number of days between application of manure and grazing or hay harvesting and *E. coli* O157:H7 status in the herd (Hancock et al. 1994). The survey revealed that the longer slurry-treated fields are left before grazing or harvesting, the lower the risk that cattle will become infected. Contaminated manure slurry used as fertilizer also poses a threat to other animals that graze on crops while STEC or *S. enterica* are still viable. Proper management of manure slurry and an understanding of the factors affecting survival of STEC and *S. enterica* in slurry are essential to prevent exposure.

Another indirect mode of transmission at dairy farms is through cattle drinking water. *S. enterica* has been isolated frequently from water sources, which may serve as bacterial reservoirs (Winfield and Groisman 2003). *E. coli* was demonstrated to persist in cattle drinking water several days after initial contamination (Rice and Johnson 2000). The ability of the bacteria to survive in water not only presents an opportunity for cattle to become infected, but birds or other animals that use water troughs also may become infected and spread pathogens within the farm or to neighboring farms (Figure 1).

Free-ranging birds as sources of infection in cattle

Many management factors such as herd size, grouping, manure management, equipment sanitation, feed composition, and feed additives have been associated with STEC and *S. enterica* prevalence in cattle herds (Bender 1995, Van Donkersgoed et al. 1999). Unfortunately, little attention has been given to external sources of contamination such as veterinarians, introduction of new cattle into the herd, wildlife, rendering trucks, feed, etc. (Figure 1). Although rats and mice may prolong persistence of these microorganisms on the farm by acquiring

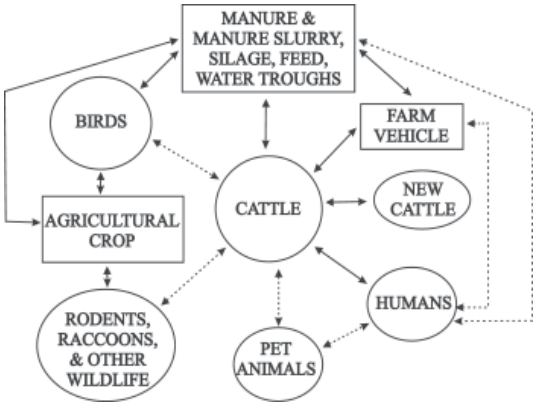
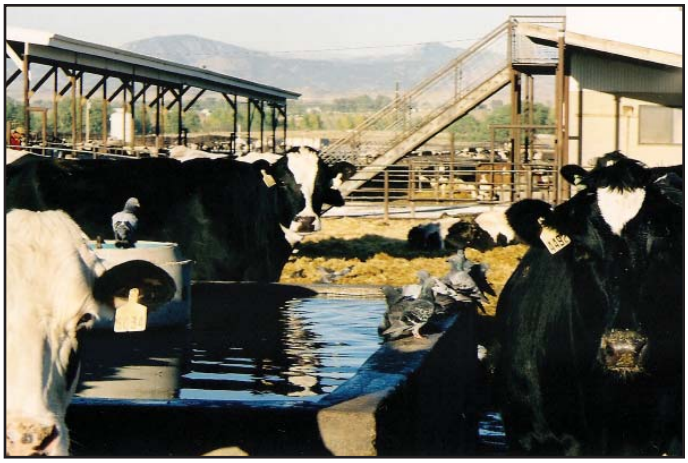


Figure 1. Potential modes of transmission of *Escherichia coli* and *Salmonella enterica*. Dotted lines represent indirect transmission. Cattle manure or bird feces are direct methods of contamination that could lead to infection in birds and cattle. Transmission to humans occurs after consuming contaminated meat, unpasteurized milk, or other dairy products.

infection (Henzler and Opitz 1992), available evidence suggests that they do not play an important role in amplifying transmission (Wray and Davies 2000). Though the literature is sparse, free-ranging birds have been identified as carriers of various strains of STEC (Wallace et al. 1997, Makino et al. 2000) and *S. enterica* (Scholtens and Caroli 1971, Pennycott et al. 1998, Kirk et al. 2002), suggesting that birds are a potential source of external contamination that deserve further scrutiny. Because birds can transport and disseminate pathogens over long distances, their importance as carriers and their role in transmission of STEC and *S. enterica* to cattle has been suggested (Wallace et al. 1997). Birds not only have the potential to transmit disease, but they also consume feed meant for cattle resulting in an estimated $100 million annual loss to the U.S. agriculture industry (Lee 2005). Contaminated feed can also pose a hazard to human health when microorganisms colonize the animal and multiply on the food product or when toxic products persist as a residue in the meat (Hinton 2000).



Pigeons and cattle.

E. coli O157 was first isolated from birds in fecal samples collected from free-ranging birds (mainly gulls) in 1997 (Wallace et al. 1997). Since then, gulls have been identified as carriers of additional strains of STEC (O136:H16 and O153:H$^-$) (Makino et al. 2000). This carrier role highlights the possibility that gulls could contaminate water or crops with their feces, or they may become infected by foraging on farmland fertilized with manure slurry, thereby, circulating STEC into the environment.

In Italy, feral pigeons (*Columba livia*) were confirmed as natural reservoirs of STEC by isolation of STEC from fecal samples recovered from captured pigeons (Dell'Omo et al. 1998, Morabito et al. 2001). All of the strains produced shiga toxins, and most of the strains contained the *eae* gene and produced cytolethal distending toxin (virulence factors produced by a pathogen and necessary for causing disease in a host). Other studies have isolated STEC strains revealing toxins produced by the *eae* gene in pigeons (Wada et al. 1995, Schmidt et al. 2000, Pedersen 2004). Genomic subtyping has linked both pigeon and environmental isolates of *E. coli* O157:H7 to a common source in some instances (Shere et al. 1998). Because pigeons often live in close association with cattle year-round, the possibility of cross-contamination is high.

STEC have been detected in various tissues collected from nestlings of house sparrows (*Passer domesticus*) and tree sparrows (*P. montanus*) (Pawiak et al. 1991). Other bacterial species including *Salmonella* spp., *Campylobacter jejuni*, and *Clostridium perfringens* have been identified in fecal samples of house sparrows collected near broiler chicken houses, suggesting that birds gaining access to livestock facilities may transmit bacteria (Craven et al. 2000). The potential for transmission is important because house sparrows, like pigeons, often are found year-round at livestock facilities (Kirk et al. 2002).

Infections with the bacteria *E. coli* O86:K61 and *Salmonella typhimurium* DT40 were identified as the most common causes of mortality in various species of free-ranging finches of the family Fringillidae in Britain (Foster et al. 1998, Pennycott et al. 1998). The *eae* gene and cytolethal distending toxin production were detected in the strains of *E. coli* O86 isolated from the finches, indicating the potential of the organism to cause disease and mortality in the finches. *E. coli* O86:K61 also has been associated with disease in humans, cattle, and other animals (Foster et al. 1998). Given their propensity to congregate in large numbers at birds feeders, finches may pose a threat to other birds and humans that come in contact with fecal-contaminated bird feeders. Although the free-ranging finches examined in Britain were submitted from households that provided supplementary food (Pennycott et al. 1998), house finches are commonly observed at dairies (Kirk et al. 2002). Consequently, infected finch populations have the potential to disseminate these pathogens to domesticated livestock.

Outbreaks of salmonellosis attributed to serotype *S. typhimurium* in bird populations have been responsible for large-scale mortality

in many species of domestic and free-ranging birds (Faddoul and Fellows 1966, Goodchild and Tucker 1968, Grimes 1979, Hudson et al. 2000, Daniels et al. 2003). *Salmonella enterica* also have been identified in asymptomatic birds (Adesiyun et al. 1998, Kirk et al. 2002). *S. enterica* infection often spreads via backyard bird feeders where high densities of free-ranging birds congregate during winter when food is scarce (Pennycott et al. 2002). Free-ranging birds such as European starlings (*Sturnus vulgaris*), great-tailed grackles (*Cassidix mexicanus*), common grackles (*Quiscalus quiscula*), and brown-headed cowbirds (*Molothrus ater*) (Clark and McLean 2003) flock to dairy farms (as many as 5,000 to 10,000 birds on some dairies) during fall and winter (Kirk et al. 2002). This high concentration of birds increases the risk of transmission of *S. enterica* to other birds and cattle, especially in feed bunks. Free-ranging birds have been implicated in contamination of livestock feed while it is stored at the farm (Daniels et al. 2003) and immediately after the feed is placed into feed bunks (Smith and House 1992). The risk that cattle will become infected is magnified by the presence of free-ranging birds, which may transport *S. enterica* to the farm from outside sources or recirculate the microbe within the farm to uninfected animals. *S. enterica* survive in the environment for extended periods, increasing the likelihood for birds to come into contact with the bacteria (Ĉižek et al. 1994) and subsequently infect other animals. Serotypes of *S. enterica* commonly associated with infection in humans and animals have been identified in asymptomatic free-ranging birds, suggesting that transmission across species is possible (Sambyal and Sharma 1972).

Even though pigeons and other birds have been clearly identified as important carriers of STEC and *S. enterica*, little is known about the length of time that STEC or *S. enterica* are shed or remain viable in bird droppings. Pigeons infected experimentally with *E. coli* O157 shed the pathogen for up to 29 days depending on the infective dose (Ĉižek et al. 2000). Research suggests that *E. coli* coliform counts in Canada geese (*Branta canadensis*) droppings increase upon excretion (Feare et al. 1999). If this is true also for pigeons and other birds that live in close association with cattle, the risk of indirect transmission would increase.

Migratory birds are important contributors to losses attributed to consumption of feed. However, peridomestic birds, such as pigeons and house sparrows, may be more culpable of disease transmission. Migratory birds are generally absent from dairies during the summer when a seasonal fluctuation in prevalence of STEC is observed in cattle (Herriott et al. 1998, Garber et al. 1999, Van Donkersgoed et al. 1999). In contrast, peridomestic birds are present during this time and often loaf and roost in or near cattle barns, thus increasing the potential for disease transmission.

Another concern is that indirect transmission will occur when birds or cattle forage or consume silage or feed grown on slurry-treated fields contaminated with STEC or *S. enterica*. Once infected, birds may redistribute STEC or *S. enterica* within the farm or disseminate STEC or *S. enterica* to previously uncontaminated farms. Cattle may also facilitate the cycle by transmitting bacteria to other cattle or birds after becoming infected from contaminated feed sources. In general, farmers do not implement rigorous bird control methods because the economic loss associated with bird consumption of cattle feed is perceived to be less than the intensive labor and financial expenditures associated with an effective bird control program. This perception may change, however, because the additive effects of feed loss and bacterial transmission as a precursor to disease may be sufficient evidence to justify a bird control program.

Conclusions

Because STEC and *S. enterica* are able to survive adverse environmental conditions (Pell 1997, Winfield and Groisman 2003), the bacteria have many opportunities to infect new hosts and continue the cycle via indirect transmission. Indirect transmission of STEC or *S. enterica* to cattle or birds may occur through contaminated domestic animals or wildlife that defecate in drinking water or food sources that are later ingested by other animals (Figure 1). Crops fertilized with contaminated manure slurry may pose a threat if conditions are conducive to bacterial growth (Himathongkham et al. 1999). These crops then present a source of infection for birds such as gulls or Canada geese, that forage on farmland or to cattle that are fed harvested grains or silage that has been grown and stored in conditions that support bacterial growth. In addition, contaminated manure slurry, if not managed properly, may seep into ground water and pose a threat to human health.

Migratory birds, such as European starlings, common grackles, and red-winged blackbirds (*Agelaius phoenieceus*) are found in large numbers at dairies during the fall and winter and may amplify transmission of diseases by spreading the bacteria from 1 farm to another. Peridomestic birds, such as pigeons and sparrows, may

be more important in recirculation of STEC within the farm. Once birds are infected, the risk of cross contamination between cattle and peridomestic birds increases because of their close association.

Birds may be more susceptible than cattle to indirect contamination because of their mobility. Nonetheless, cattle are vulnerable to infection through various outside sources. Many external sources of contamination may influence prevalence of STEC and *S. enterica* in cattle and birds (Figure 1).

Knowledge is paramount in understanding the epidemiological links between STEC and *S. enterica* infection in cattle and free-ranging birds and successful management of both dairy herds and bird populations to minimize cross-infections. Ultimately, the producer loses money if free-ranging birds play a role in disseminating and redistributing STEC or *S. enterica*. The benefit to exploring all potential modes of transmission and cross-contamination lies not only in adapting management efforts to minimize economic loss to the producer, but more importantly, in preventing entry of STEC and *S. enterica* into the human food chain. Epidemiological knowledge is necessary to make educated management decisions that will minimize bacterial prevalence in dairy herds and protect human health via safer bovine products.

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Appendix

STEC serogroups identified in wild birds, cattle, and humans.

STEC	Birds	Cattle	Humans	References
O1	X	X	X	Sussman 1997, Bopp et al. 2003, Pedersen 2004
O2		X	X	Sussman 1997, Willshaw et al. 1997, Bell and Kyriakides 1998, Beutin 1999, Bopp et al. 2003
O4		X	X	Sussman 1997, Bell and Kyriakides 1998, Beutin 1999
O5	X	X	X	Sussman 1997, Willshaw et al. 1997, Bell and Kyriakides 1998, Beutin et al. 1998, DebRoy and Maddox 2001, Kullas et al. 2002, Bopp et al. 2003, Pedersen 2004
O6	X	X	X	Sussman 1997, Willshaw et al. 1997, Bell and Kyriakides 1998, Pedersen 2004
O8	X	X	X	Sussman 1997, Willshaw et al. 1997, Bell and Kyriakides 1998, Beutin 1999, DebRoy and Maddox 2001, Pedersen 2004
O15	X	X	X	Wells et al. 1991, Willshaw et al. 1997, Bell and Kyriakides 1998, Beutin 1999, Pedersen 2004
O18	X	X	X	Sussman 1997, Bell and Kyriakides 1998, Beutin et al. 1998, Morabito et al. 2001, Kullas et al. 2002
O22		X	X	Montenegro et al. 1990, Wells et al. 1991, Willshaw et al. 1997, Bell and Kyriakides 1998, Beutin 1999, Bopp et al. 2003
O23		X	X	Willshaw et al. 1997, Bell and Kyriakides 1998
O25	X		X	Sussman 1997, Willshaw et al. 1997, Kullas et al. 2002, Pedersen 2004
O26		X	X	Wells et al. 1991, Griffin 1995, Willshaw et al. 1997, Bell and Kyriakides 1998, Beutin et al. 1998, Fukushima et al. 1999, Cobbold and Desmarchelier 2000, DebRoy and Maddox 2001, Bopp et al. 2003
O45	X	X	X	Wells et al. 1991, Sussman 1997, Morabito et al. 2001, Bopp et al. 2003, Pedersen 2004
O55		X	X	Willshaw et al. 1997, Bell and Kyriakides 1998, Bopp et al. 2003
O75	X	X	X	Willshaw et al. 1997, Bell and Kyriakides 1998, Beutin 1999, Morabito et al. 2001
O79		X	X	Bopp et al. 2003
O91	X	X	X	Montenegro et al. 1990, Willshaw et al. 1997, Bell and Kyriakides 1998, Beutin et al. 1998, Beutin 1999, Kullas et al. 2002, Bopp et al. 2003, Pedersen 2004
O103	X	X	X	Wells et al. 1991, Griffin 1995, Willshaw et al. 1997, Bell and Kyriakides 1998, Beutin et al. 1998, DebRoy and Maddox 2001, Bopp et al. 2003, Pedersen 2004
O104		X	X	Willshaw et al. 1997, Bell and Kyriakides 1998, Beutin 1999, Bopp et al. 2003

O105	X	X	X	Willshaw et al. 1997, Bell and Kyriakides 1998, Beutin 1999, Pedersen 2004
O111		X	X	Wells et al. 1991, Griffin 1995, Willshaw et al. 1997, Bell and Kyriakides 1998, Beutin et al. 1998, Fukushima et al. 1999, DebRoy and Maddox 2001
O113		X	X	Montenegro et al. 1990, Griffin 1995, Willshaw et al. 1997, Bell and Kyriakides 1998, Beutin et al. 1998, Beutin 1999, Bopp et al. 2003,
O114		X	X	Willshaw et al. 1997, Bell and Kyriakides 1998
O117	X	X	X	Willshaw et al. 1997, Bell and Kyriakides 1998, Pedersen 2004
O118		X	X	Bell and Kyriakides 1998, Beutin et al. 1998, DebRoy and Maddox 2001, Kullas et al. 2002, Bopp et al. 2003
O121		X	X	Wells et al. 1991, Willshaw et al. 1997, Bell and Kyriakides 1998, Bopp et al. 2003
O128	X	X	X	Willshaw et al. 1997, Bell and Kyriakides 1998, Beutin et al. 1998, Bopp et al. 2003, Pedersen 2004
O136	X	X		Montenegro et al. 1990, Willshaw et al. 1997, Beutin 1999, Makino et al. 2000
O145	X	X	X	Wells et al. 1991, Willshaw et al. 1997, Bell and Kyriakides 1998, Beutin et al. 1998, DebRoy and Maddox 2001, Bopp et al. 2003, Pedersen 2004
O153	X	X	X	Wells et al. 1991, Bell and Kyriakides 1998, Beutin 1999, Makino et al. 2000, Bopp et al. 2003
O157	X	X	X	Wells et al. 1991, Wallace et al. 1997, Willshaw et al. 1997, Bell and Kyriakides 1998, Herriott et al. 1998, Shere et al. 1998, Fukushima et al. 1999, Beutin 1999, Čižek et al. 2000, Barham et al. 2002, Bopp et al. 2003, Pedersen 2004
O163		X	X	Wells et al. 1991, Willshaw et al. 1997, Bell and Kyriakides 1998, Beutin et al. 1998, Bopp et al. 2003
O165	X	X	X	Parma et al. 2000, Bopp et al. 2003, Pedersen 2004
O168		X	X	Willshaw et al. 1997, Bell and Kyriakides 1998, Beutin 1999
O172	X	X	X	Bopp et al. 2003, Pedersen 2004

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