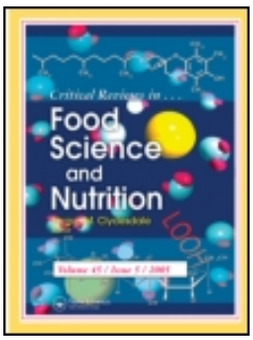

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Transmission of Bacterial Zoonotic Pathogens between Pets and Humans: The Role of Pet Food

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Recent Salmonella *outbreaks associated with dry pet food and treats raised the level of concern for these products as vehicle of pathogen exposure for both pets and their owners. The need to characterize the microbiological and risk profiles of this class of products is currently not supported by sufficient specific data. This systematic review summarizes existing data on the main variables needed to support an ingredients-to-consumer quantitative risk model to (1) describe the microbial ecology of bacterial pathogens in the dry pet food production chain, (2) estimate pet exposure to pathogens through dry food consumption, and (3) assess human exposure and illness incidence due to contact with pet food and pets in the household. Risk models populated with the data here summarized will provide a tool to quantitatively address the emerging public health concerns associated with pet food and the effectiveness of mitigation measures. Results of such models can provide a basis for improvements in production processes, risk communication to consumers, and regulatory action.*

Keywords Zoonotic pathogens, pet food, pets, *Salmonella*, risk assessment, household exposure

INTRODUCTION

Emerging and re-emerging zoonotic pathogens continue to cause significant disease burden in both developed and developing countries. Sixty percent of all emerging pathogens to date are estimated to be zoonotic (IOM and NRC, 2009; Cutler et al., 2010). It has been estimated that direct costs due to zoonotic diseases amount to more than $20 billion, and indirect costs to more than $200 billion (World Bank, 2010). Some of the most widespread of these pathogens can be transmitted between humans, wildlife, urban wildlife, livestock, and pets. Pathogen transmission involving pets, which can occur in both wild and urban settings, and which is becoming more frequent due to built environments encroaching into wildlife habitat, is an important component of "One Health" disease risk (Day et al., 2011). This is the case with several strains of *Escherichia coli* and *Salmonella enterica*. However, except for few organisms, such as the rabies virus (Zinsstag et al., 2009), there is little quantitative information available on the risk of infectious disease transmission between pets, humans, and the domestic environment they share. More specifically, no information is available on the role of pet food as vehicle of direct and indirect exposure to humans and pets.

Dogs and cats are the most common companion animals in several countries and cultures. In the United States, 56–62% of households own at least one pet (AVMA, 2012). In 2011–2012, the U.S. dog population was estimated to be 69.9–78.2 million (1.7 dogs per household, 39% of households own at least one dog), while the U.S. cat population was 74.1–86.4 million (2.2 cats per household, 33% of households own at least one cat) (AVMA, 2012; APPA, 2013). In Europe, 70 million households own at least one pet, with a dog population of 74 million (26% of households own at least one dog), and a cat population of 85 million (25% of households own at least one cat) (FEDIAF, 2010). Individual or communal ownership of dogs is also common in Africa (Kitala et al., 2001; Knobel et al., 2008; Kaare et al., 2009; Davlin and VonVille, 2012), Asia (Hsu et al., 2003; Kongkaew et al., 2004; Yen et al., 2010), the Americas (Ortega-Pacheco et al., 2007; Suzuki et al., 2008), and Oceania (Kobelt et al., 2003; Toribio et al., 2009; ACAC 2010). Owning a pet is associated with documented psychological and health benefits, which include lower anxiety and loneliness, increased physical activity, socialization with other pet owners, faster recovery from

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illness, positive effects on children emotional development, and the effectiveness of pet-facilitated therapy (Brodie and Biley, 1999; Barker and Wolen, 2008; Dotson and Hyatt, 2008; Endenburg and van Lith, 2011), although some physical and mental health effects are unclear (McNicholas et al., 2005; Barker and Wolen, 2008; Chur-Hansen et al., 2010). However, the enjoyment of such benefits needs to be coupled with controlling the risk of infectious diseases that could be acquired through pets. As the pet population is likely to increase, and competition for land use makes interactions between humans, pets, and wildlife habitat more frequent, the role of pets as potential carriers of zoonotic pathogens needs to be better addressed and quantified.

Compared to livestock or wild animals, the role of dogs and cats as pathogen carriers is unique, in that they can interact with urban, wilderness, and domestic environments. In addition, dogs and cats are in close contact with their owners on a daily basis, often sharing food preparation and sleeping environments. Pets mostly eat food prepared specifically for them, commonly industrially produced in large batches. Exposure and health risks associated with this complex interaction network are not well understood. The emerging role of pet food as pathogen carrier is of particular concern, due to recent multi-state outbreaks and recalls (U.S. FDA 2013a), and the involvement of multiple stakeholders in the production and use of the food.

The goal of this study is to systematically review the available information on pathogen transfer at the human-pet interface, with focus on mammalian pets (specifically dogs and cats), bacterial pathogens (e.g., *Salmonella enterica, E. coli, Shigella, Brucella*, and *Listeria*), and the role of pet food as initial vehicle of household contamination. Existing quantitative data on pathogen ecology and exposure associated with pet food are examined through a quantitative review for subsequent input into risk assessment models.

HUMAN PATHOGEN EXPOSURE AND DISEASES BURDEN DUE TO CONTACT WITH PETS

Pets can be a vector of zoonotic enteric diseases to humans. Numerous sporadic and epidemic cases of bacterial, viral, protozoan, and parasitic diseases have been attributed to contact with pets (Plaut et al., 1996; Sanyal et al., 1997; Schröter et al., 2004; Pickering et al., 2008). In the United States, between the years 2000 and 2010 contact with domesticated or wild animals was estimated to be the cause of 14% of illness cases associated with seven common bacterial and protozoan pathogen groups, namely *Campylobacter, Cryptosporidium*, Shiga toxin-producing (STEC) *E. coli* O157 and non-O157, *Listeria monocytogenes*, nontyphoidal *Salmonella*, and *Yersinia enterocolitica* (Hale et al., 2012). Of the approximately 445,000 animal-related cases due to these seven pathogens, nontyphoidal *Salmonella* was estimated to be responsible for 29% of cases, 48% of hospitalizations, and 62% of deaths. Such illnesses are also responsible for a considerable economic burden (Stehr-Green and Schantz, 1987; Buzby and Roberts, 2009). Dogs and cats pose a particular concern due to their popularity as pets, the fact that they live in close proximity to humans, and their direct contact with humans repeated on a daily basis over several years (Chang et al., 2007; Baxter and Leck, 1984; Bowman and Lucio-Forster, 2010; Deplazes et al., 2011; Holst and Englund, 2003; Holt, 1981; Schauder, 1999; Tan, 1997; Weese et al., 2006).

Humans can be exposed to pet-associated zoonotic pathogens via several routes (Figure 1). Dogs and cats that spend time outdoors can acquire pathogens from soil, water, wild animals, or other pets' feces. Pets can also come in contact with pathogens already present in the house environment or in their food, and spread contamination to other surfaces. As a result, pets can carry pathogens internally in their digestive tract as well as externally on their fur, paws, or head. Humans can be exposed by directly touching the animals with their hands or face, or indirectly by touching household surfaces previously in contact with a pet, such as floors, rugs, beds and couches, toys, pet food bowls, and pet litter. Directly coming in contact with a pet's feces or saliva could be of additional health concern to humans, due to the unknown and potentially high levels of pathogens if the animal's digestive tract is colonized. Humans can also be exposed to pathogens by handling pet food contaminated during its production, or pet food that has been contaminated by contact with an infected animal.

There is substantial evidence of human enteric illness associated with household contact with pets, including mammals and reptiles (Plaut et al., 1996; Swanson et al., 2007; Pickering et al., 2008; Harris et al., 2009; Hoelzer et al., 2011; Lowther et al., 2011). Several outbreaks of *E. coli* O157:H7 and other pathogens associated with visiting petting zoos provide evidence that touching animals with hands, which includes

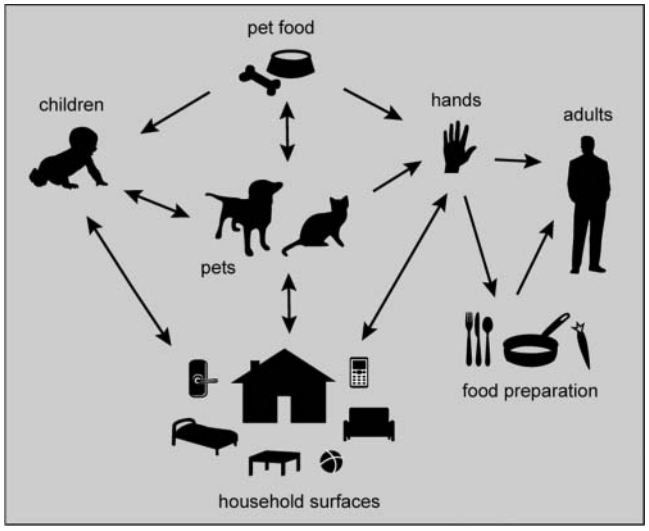

Figure 1 Major routes of pathogen transfer between pets, pet food, and humans in the household environment.

petting, feeding, and being licked, can be an effective exposure route (Heuvelink et al., 2002; Warshawsky et al., 2002; CDC, 2005; CDC, 2009; Stirling et al., 2008; Goode et al., 2009; Moller-Stray et al., 2012). In contrast with the sporadic contact at a zoo or fair, contact between pets and pet owners can occur multiple times a day for several years, and can be mediated by a variety of objects and surfaces. Evidence suggesting pathogen transmission from dogs to humans due to direct animal-human contact or mediated by household environments has been observed for *Staphylococcus* (Duijkeren et al., 2011; Davis et al., 2012), *Enterococcus faecium* (Damborg et al., 2009b), *Campylobacter* spp. (Salfield and Pugh, 1987; Kapperud et al., 1992; Altekruse et al., 1999; Wolfs et al., 2001; Parsons et al., 2010), *Clostridium difficile* (Borriello et al., 1983; Keessen and Lipman, 2012), *Salmonella* (Sato et al., 2000; Varga et al., 2012), and enteropathogenic *E. coli* (Rodrigues et al., 2004). Weese et al., (2010), however, found no association between *Campylobacter* strains in humans and in dogs living in close quarters. Dog-human interactions that can play a role in zoonosis transmission include playing, greeting, feeding, disposing of pet feces, and sharing sleeping areas (Westgarth et al., 2008). Such contact can lead to pathogen exposure through ingestion, often mediated by hands, inhalation (e.g., methicillin-resistant *Staphylococcus aureus*, MRSA), or skin (e.g., dermatitis). In addition, pet bites can inoculate pathogens directly into the skin, muscle tissue, or blood stream (Talan et al., 1999). Most evidence of illness due to contact with cats (Kravetz and Federman, 2002) involves toxoplasmosis, caused by the protozoan parasite *Toxoplasma gondii* (Dubey and Jones, 2008; Dubey et al., 2009), and cat-scratch disease, caused by the bacterium *Bartonella henselae* (Jackson et al., 1993; Athanasiou et al., 2012; Zangwill, 2013). Contact with cats has also been directly linked to human outbreaks of *Campylobacter* illness (Svedhem and Norkrans, 1980; Altekruse et al., 1999), and is a significant risk factor for sporadic and endemic cases (Deming et al., 1987; Hopkins et al., 1984). Cats can also carry pathogens such as *Salmonella* or pathogenic *E. coli*, but limited documentation of direct transmission to humans exists (Busch et al., 2007; Hoelzer et al., 2011).

Different human populations and age groups may face different exposure and risk levels due to contact with pets. For example, children interact with pets and their environment differently than adults in both frequency and extent of contact (Filiatre et al., 1986; Millot et al., 1988; Mathers et al., 2010). In addition, children and adult may perceive risk differently, apply different mitigation strategies, and have a different immunological responses to pathogen exposure. Evidence of gastroenteric disease transmission from pets to children include *Salmonella* from cats, dogs (Morse et al., 1976; Sato et al., 2000; Hoelzer et al., 2011), and reptiles (Mermin et al., 1997), and *Campylobacter* from young dogs (Salfield and Pugh, 1987). Ownership of reptiles was a significant risk factors for *Salmonella* infection in children in Michigan, U.S. (Younus et al., 2010). Ownership of pet puppies was found to be a significant risk factor for *Campylobacter* infection in infants and young children (Tenkate et al., 2001). The occurrence of similar *Giardia* genotypes in dogs and children living in the same locality also suggests zoonotic transmission (Eligio-García et al., 2005; van Keulen et al., 2002; Dado et al., 2012). In infants, salmonellosis was significantly associated with exposure to reptiles (Jones et al., 2006), and in sporadic cases to dogs (Sato et al., 2000). However, overall dog or cat ownership was associated with a lower risk of gastroenteritis in Australian children (Heyworth et al., 2006).

While microbial contamination in environments frequented by pets has been observed extensively, little direct epidemiological evidence exist of zoonotic infections acquired exclusively through surfaces and objects that had previously come in contact with pets. In most epidemiological studies and outbreak reports, the risk factor "contact with animal" includes several routes of transmission, direct and indirect (Heuvelink et al., 2002; Warshawsky et al., 2002; CDC, 2009; Goode et al., 2009). For example, in public settings such as zoos and fairs contact with potentially contaminated surfaces is a concern, in addition to direct human-animal contact, and it includes touching fences, bringing food and drink containers inside animal petting areas, and touching soil and animal feces (McMillian et al., 2007; Erdozain et al., 2013). Insects, such as flies (Diptera), can also carry and transfer pathogens from animals to humans and the environment (Sulaiman et al., 2000; Buma et al., 2006; Nmorsi et al., 2006). In different contexts it has been shown that contact with fomites could cause infection and disease in humans. For example, norovirus has been associated with several outbreaks in ships and hospitals, and hand-mediated contact with surfaces and objects (fomites) was demonstrated to be an important transmission route (Jones et al., 2007; Repp and Keene, 2012). Also, the transmission of methicillin-resistant *S. aureus* from surfaces to people has been observed in hospitals and other public settings (Layton et al., 1993; Boyce, 2007; Otter et al., 2011).

Recent human illness outbreaks have also highlighted the importance of pet food and treats as pathogen vehicles (Behravesh et al., 2010; CDC, 2008a; Harker et al., 2011; Pitout et al., 2003; Finley et al., 2006; CDC, 2012). Feeds for production animal nutrition have also been associated with human illness (Jones, 2011). In these cases, it is unclear if the primary route of exposure was direct contact with the food, direct contact with a pet, or contact with contaminated house surfaces and objects. Overall, the extent of risk posed by pet food and the relative importance of different food and nonfood transmission routes are currently unknown.

INFECTION OCCURRENCE IN PETS

Pets such as dogs and cats can be exposed to pathogens through a variety of routes, such as food, water, contact with outdoor environments, direct contact with other animals or their feces, and contact with humans and indoor house

environments. Both pet dogs and cats have been observed to carry a wide range of zoonotic pathogens including *Salmonella* (Morse et al., 1976; Nastasi et al., 1986; Kwaga et al., 1989; Hill et al., 2000; Sockett and Rodgers, 2001; Boqvist et al., 2003; Bagcigil et al., 2007; Ojo and Adetosoye, 2009), *Listeria monocytogenes* (Marien et al., 2007), *Brucella, Shigella* (Kwaga et al., 1989), *E. coli* (Peeters, 1994; Damborg et al., 2009a; Nielsen, et al., 2009; Morato et al., 2009), *Campylobacter* (Hill et al., 2000; Engvall et al., 2003; Chaban et al., 2010; Parsons et al., 2010), *Yersinia* (Nastasi et al., 1986), *Clostridium difficile* (Riley et al., 1991), *Pasteurella* (Chang et al., 2007), *Bartonella* (Duncan et al., 2007), *Helicobacter* (Craven et al., 2011), rabies (Adeiga and Audu, 1996), enteric viruses (Schulz et al., 2008), *Giardia* (Horejs and Koudela, 1994; Hill et al., 2000; Thompson et al., 2008), and *Cryptosporidium* (Hill et al., 2000; Thompson et al., 2008).

Infection by zoonotic viruses, bacteria, protozoa, and helmints can lead to symptomatic illness in dogs and cats (Warner et al., 1984; Njaa 2008; Vaissaire 2000; Schotte et al., 2007; Mesquita and Nascimento 2012; Schulz et al., 2008; Beutin 1999; Schlegel et al., 2012; Brown et al., 1999). For example, salmonellosis symptoms in dogs can include abdominal pain, loss of appetite, weakness, fever, vomiting, and diarrhea, with symptom severity being very variable (Marks and Kather, 2003). Acute gastrointestinal symptoms in dogs have been linked to pathogenic *E. coli* (Wang et al., 2006; Paula and Marin, 2008), including a case of hemolytic uremic syndrome (HUS) potentially—but not conclusively—linked to *E. coli* O157:H7 (Dell'Orco et al., 2005). Symptoms usually resolve spontaneously in less than a week. When symptomatic illness is reported, therapy is usually supportive, and administration of antimicrobials is not recommended (Marks et al., 2011). Younger dogs have been reported to become infected and ill due to *Salmonella* more commonly than adult dogs (Beutin, 1999; Marks and Kather, 2003; Marks et al., 2011). Also, *Campylobacter* prevalence is higher in young dogs (Parsons et al., 2010), and enteropathogenic (EPEC) and enterotoxigenic (ETEC) strains of *E. coli* have been more commonly associated with symptomatic illness in younger dogs (Beutin, 1999). Cats can also present illness symptoms associated with pathogens such as *Salmonella* (Stiver et al., 2003; Philbey et al., 2008, 2009). Pet outbreaks, however, are unlikely to be detected. Even when illness is recognized and the pet is taken to a veterinary care facility for diagnosis or treatment, by law most pet illnesses do not need to be reported to public health authorities (Moore and Lund 2009; Allen, 2012). Most documented pet outbreaks involve situations where numerous animals are confined to the same location, such as kennels and pounds (Schotte et al., 2007; Selmi et al., 2011), or have visited the same veterinary clinic (Ketaren et al., 1981; CDC, 2001; Cherry et al., 2004).

While it is known that dogs and cats can be susceptible to zoonotic pathogens, no dose–response relationships for either infection or illness has been established for these species, which limits the ability to estimate risk for pets. Nevertheless, dose–response relationships have been derived by feeding trials for other animal species, namely for enterohemorragic *E. coli* in pigs (Cornick and Helgerson, 2004), *L. monocytogenes* in mice (Conner et al., 1989; Golnazarian et al., 1989), nontyphoidal *Salmonella* in mice (Meynell and Meynell, 2009), *Yersinia pestis* in mice (Lathem et al., 2005), (nonzoonotic) porcine enterovirus type 3 and 7 in pigs (Cliver, 1981), and rotavirus in pigs (Payment and Morin, 1990). A feeding study of *Salmonella* Enteritidis in mice observed a highly variable LD_{50} ranging from 16 to more than 10^8 organisms (Lu et al., 1999). Data from these studies, as well as from human dose–response studies, could be extrapolated to mammalian pets when performing risk assessments.

The presence of pathogens in the intestinal tract of an animal is often not associated with symptomatic illness. Asymptomatic intestinal colonization or infection has been commonly observed in dogs for various *Salmonella* strains at a prevalence of 1–4% (Morse and Duncan, 1975; Shimi et al., 1976; Fukata et al., 2002; Parungao et al., 2010; Verma et al., 2011; Tupler et al., 2012), as well as for pathogenic *E. coli* (Staats et al., 2003; Sancak et al., 2004; Pestana de Castro et al., 2010). Asymptomatic infection or colonization has also been observed in populations with higher overall *Salmonella* prevalence and morbidity, such as racing sled dogs (McKenzie et al., 2010; Cantor et al., 1997). Other pathogens such as *Clostridium difficile* (Borriello et al., 1983; Riley et al., 1991; Marks et al., 2002; Weese et al., 2010; Goldstein et al., 2012; Schneeberg et al., 2012; Tupler et al., 2012), *Campylobacter* (McOrist and Browning, 1982; Baker et al., 1999; Engvall et al., 2003; Wieland et al., 2005; Rossi et al., 2008; Chaban et al., 2010; Parsons et al., 2010; Salihu et al., 2010; Rahimi et al., 2012), canine enteric viruses (Schulz et al., 2008), *Giardia* (Tupler et al., 2012), and *Cryptosporidium* (Tupler et al., 2012; Santin, 2013) have been detected in the feces of asymptomatic dogs (Marks and Kather 2003; Struble et al., 1994; Schneeberg et al., 2012; Goldstein et al., 2012; Rossi et al., 2008). However, symptomatic dogs were significantly more likely to be infected with one or more enteric pathogens than nonsymptomatic dogs (Tupler et al., 2012). In particular, *Clostridium perfringens* and *difficile* strains are significantly associated with diarrheic symptoms in dogs (Weese et al., 2001; Marks et al., 2002). Several *Salmonella* strains have also been isolated from both sick and healthy cats, with a prevalence in healthy cats usually in the order of 1–2% (Hill et al., 2000; Spain et al., 2001; Van Immerseel et al., 2004), up to 4% in one study (Sabshin et al., 2012). Other pathogens including *Campylobacter, Yersinia*, and hemolythic *E. coli* have also been isolated from both symptomatic and asymptomatic cats (Queen et al., 2012; Blanco et al., 1993; Kaneuchi et al., 1987). Often such infections or illnesses remain undetected and are not treated, potentially leading to a prolonged spread of pathogens in the household and in outdoor environments.

Pets presenting gastroenteric infection or colonization, symptomatic or not, can shed pathogens into the environment,

particularly through feces (Nastasi et al., 1986; Struble et al., 1994; Van Immerseel et al., 2004; Bagcigil et al., 2007; Finley et al., 2007; Marien et al., 2007). While the duration of shedding after colonization or infection is mostly unknown, chronic carriage of *Salmonella* has been observed in cats (Wall et al., 1995). Fecal *Salmonella* shedding in naturally infected dogs has been observed to last for at least 6 weeks, and up to 117 days in experimentally infected animals (Day et al., 1963). The odds of *Salmonella* infection and shedding was found to be considerably higher—with prevalences up to 69%—in healthy dogs fed raw meat diets (Lefebvre et al., 2008; Finley et al., 2007; Cantor et al., 1997). Infection and shedding prevalence may depend on the animal's age. *Campylobacter* prevalence has been observed to be higher in young dogs (Parsons et al., 2010; Rahimi et al., 2012), and dogs between 3 months and 2 years of age were observed to shed *Campylobacter* strains at high prevalence and for several months (Hald et al., 2004). However, it is unknown if higher infection prevalence in younger animals corresponds to shedding at higher concentrations. A higher prevalence of *Campylobacter* shedding has been also observed in cats younger than 36 months (Wieland et al., 2005; Marks et al., 2011), although other studies observed no age difference (Rahimi et al., 2012) or an opposite trend (Hald and Madsen, 1997; Salihu et al., 2010). Several studies measured pathogen prevalence in animal populations, but few also measured concentrations. One notable example is Chaban et al., (2010), who observed concentrations of *Campylobacter* species in the range 10^3–10^8 CFU/g in the feces of diarrheic dogs. Enteric pathogens have also been observed in the saliva of dogs and cats, but no concentration information is available (Rollof et al., 1989; Kikuchi et al., 2004; Duncan et al., 2007; Tan 1997; Houf et al., 2008). Shedding animals can also spread pathogens to their fur and paws, for example through grooming behavior or commonly used surfaces. Pathogens have indeed been observed on dog and cat fur (Overgaauw et al., 2009), although the route of pathogen transport to fur is unclear, and could include both self-infection and environmental exposure. Shedding rates and duration for other pathogens or for nonsymptomatic animals are not known.

HUMAN AND PET EXPOSURE AND ILLNESS ASSOCIATED WITH PET FOOD

Contaminated pet food could result in human illness through direct and indirect handling, and by infecting a pet that can subsequently infect humans. Direct contact with pet food can occur on a regular basis, for example when handling the food while feeding the pet, or less frequently, for example when transferring the food between storage or handling containers. Indirect contact can occur between human and objects that have come in contact with the food, such as bowls, the floor, or scoops. In both situations, adult humans get in contact with contaminated surfaces primarily through hands. Hands in turn, if not properly washed or sanitized, can transfer pathogens to the person's mouth, eyes, human food, kitchen utensils, or other house objects (Fig. 1). Children and infants can get in contact with pet food with hands or by directly ingesting kibble. They can also contact house surfaces with hands or mouth (Fig. 1). In addition, pets themselves can spread foodborne pathogens to humans or household surfaces after touching the food with their mouth or paws during eating, even before or in the absence of infection. If the pet gets infected, its feces, and potentially its saliva, may contain high pathogen numbers that can spread to humans and household surfaces.

While the occurrence of zoonotic pathogens such as *Salmonella* in pet food has been observed for decades (Galton et al., 1955; Pace et al., 1977; Daoust, 1978), in the last ten years the level of concern has been raised due to the detection of several human outbreaks and large-scale recalls linked to commercial pet food and treats (Table 1) (Finley et al., 2006; Behravesh et al., 2010; Buchanan et al., 2011; U.S. FDA, 2013a). Most notably, between January 2006 and December 2007 a multistate outbreak of *Salmonella* Schwarzengrund caused by dry pet food sickened 79 people in 21 U.S. states (CDC 2008a; CDC 2008b). While the specific routes of exposure were not determined, it is possible that directly hand-touching pet food and surfaces that were in contact with the food could have spread *Salmonella* to humans and the household environment. No outbreak in dogs was reported in relation to the human outbreak, and the few samples of dog feces examined were

Table 1 Human outbreaks of gastroenteritis associated with pet food and treats

Product	Outbreak year	Pathogen	Location	Magnitude	Illness in pets	References
Dry treats (pig ears)	1999	*Salmonella* Infantis	Canada	12 cases	18.5% (5/27) symptomatic dogs	LCDC, 2000; Clark et al., 2001
Beef-patty dry dog treats	2002	*Salmonella* Newport	U.S. (Texas)	At least 5	Undetermined	Pitout, 2003
Dry treats	2005	*Salmonella* Thompson	U.S., Canada	8 cases	1 suspect case, 1 asymptomatic dog positive	CDC, 2006; Health Canada, 2006
Dry dog and cat food	2006–07	*Salmonella* Schwarzengrund	U.S. (21 states)	79 cases	None reported, fecal samples negative	CDC, 2008a; CDC, 2008b
Dry dog food	2012	*Salmonella* Infantis	U.S. (20 states), Canada	49 cases, 10 hospitalized	1 symptomatic and 1 asymptomatic dogs positive	CDC, 2012
Chicken jerky treats	2013	*Salmonella*	U.S. (New Hampshire)	21 cases	None reported	NH DHHS, 2013

negative (CDC 2008a; CDC 2008b; Behravesh et al., 2010). In 2005, the investigation of a pet treat outbreak in western Canada and the U.S. state of Washington detected *Salmonella* Thompson in eight human cases, symptomatic and asymptomatic dogs, and pet treats made of dehydrated beef and salmon. *Salmonella* concentration in salmon treats at the time of the investigation was up to 8×10^4 CFU/g (CDC, 2006; Health Canada, 2006). The latest incident, a 2012 *Salmonella* Infantis outbreak linked to dry dog food sickened 49 individuals in 20 U.S. states, leading to 10 hospitalizations (CDC, 2012). Other two North America outbreaks related to dry pet treats had been reported earlier: one in 1999 associated with *Salmonella* Infantis in pig ear dog treats produced in Canada (Clark et al., 2001; LCDC, 2000), and the other in 2002 associated with *Salmonella* Newport in beef-patty dog treats produced in Texas (Pitout et al., 2003). Significant human illness burden has also been associated with feed for production animals (McConnell Clark et al., 1973; Hinton, 2000; Crump et al., 2002; Hald et al., 2012).

Contaminated food can also sicken pets. Outbreaks of salmonellosis due to dry food have been documented in dogs (Schotte et al., 2007; Selmi et al., 2011). In one outbreak, only 11.3% of the population developed symptoms, although 63.8% of 80 exposed dogs had fecal samples positive for *Salmonella* (Schotte et al., 2007). In the second large outbreak, which occurred in a pound, 60.9% of the 41 dogs were positive for at least one *Salmonella* strain, and several (at least 5 out of 41) presented diarrhea or hemorrhagic diarrhea symptoms (Selmi et al., 2011). In the 1999 Canada pig ear outbreak that sickened 12 people with *Salmonella* Infantis, five out of 27 (18.5%) exposed dogs developed symptoms (Health Canada, 2000). However, outbreaks in household pets are likely undetected. More evidence exists for pet illnesses associated with raw diets, such as yersiniosis (Fredriksson-Ahomaa et al., 2001) and salmonellosis (Finley et al., 2007), due to the higher pathogen prevalence in raw meat (Joffe and Schlesinger, 2002). Salmonellosis associated with food has also been observed in cats (Stiver et al., 2003; Philbey et al., 2009).

CONTAMINATION IN INGREDIENTS DURING PRODUCTION AND IN FINISHED PRODUCT

For food safety purposes, meat-containing pet diets can be categorized as: (1) raw diets, (2) processed heat-treated wet food, (3) processed heat-treated dry food and treats, and (4) uncooked or minimally processed dry food and treats. In terms of moisture content, pet food is classified as either dry (moisture content below 11%), semi-moist (moisture of 25–35%), or wet (moisture of 60–87%) (Zicker, 2008; Crane et al., 2010). Nutritionally, in the United States, the adequacy of commercial dog and cat food as "complete and balanced" diet is determined by voluntary compliance with Association of American Feed Control Officials (AAFCO) guidelines (AAFCO. Association of American Feed Control Officials, 2008; Dzanis, 2009), based on recommendations by the U.S. National Research Coucil (NRC, 2006). In raw diets, the meat does not undergo any heating or other treatment aimed at reducing pathogen loads. In processed wet food, usually sold in cans or pouches, meat, and other ingredients are either placed in a hermetically sealed container and then thermally processed to achieve commercial sterilization, or are heated to achieve commercial sterilization and then aseptically packaged in presterilized packages. Once the package is opened the food can support rapid microbial growth if not properly refrigerated. Processed dry food, usually sold in bags in the form of kibble, contains ingredients that underwent a cooking step aimed at reducing possible pathogens, was dehydrated, and then packed in sealed containers to prevent rehydaration. Its low moisture prevents growth during storage, but the product is not commercially sterile and may contain low levels of a variety of microorganisms. Processed dry treats also undergo a heating step, and are usually sold in small portions for occasional use. Dry minimally-processed animal-derived treats, such as jerky treats and pig ears, are not cooked. Pathogens such as *Salmonella*, while unlikely to grow on this matrix in dry conditions, can persist and grow if the treat is wetted. These four food categories present very different risk profiles, and none is risk free.

Raw meat diets can potentially harbor pathogens more frequently than cooked diets. *Salmonella*, for example, is a common contaminant in raw meat, including meat fed raw to pets (Campos et al., 2008; Finley et al., 2007; Joffe and Schlesinger, 2002; Li et al., 2012; Nakajima et al., 1992; Ossiprandi et al., 2003; Strohmeyer et al., 2006; Eblen et al., 2006). A study found 64% (15/25) of raw meat diet samples positive for generic *E. coli*, and 20% (5/25) for *Salmonella* (Weese et al., 2005). In other studies on raw canine diets, *Salmonella* was detected at a prevalence of 44.6% (50/112) in raw meat (Chengappa et al., 1993), 80% (8/10) in chicken meat (Joffe and Schlesinger, 2002), and 21% (35/166) in assorted commercial raw food diets (Finley et al., 2008). Other microrganisms, such as generic and pathogenic *E. coli* (Gonzalez Garcia, 2002; Weese et al., 2005; Humphrey et al., 2007), *Listeria* (Thevenot et al., 2006; Rhoades et al., 2009), *Campylobacter* (Kotula and Stern, 1984; Stern et al., 1985; Humphrey et al., 2007), and protozoa (Strohmeyer et al., 2006) are also often observed in raw meat for human consumption sold at retail (Gonzalez Garcia, 2002; Kotula and Stern, 1984; Rhoades et al., 2009; Thevenot et al., 2006). Pathogen contamination has also been observed in meat—and to a lesser extent in dry food—for zoo animals (Richter and al-Sheddy, 1990). Some pathogens are more likely to be associated with specific meat animals and production processes. For example, *Campylobacter* and *Salmonella* are commonly detected at higher prevalence in poultry than in red meats (Humphrey et al., 2007; Little et al., 2008a, 2008b). A higher prevalence of infection and illness has been observed in dogs fed raw diets in populations of urban household dogs (Joffe and Schlesinger, 2002), racing dogs (Cantor et al., 1997; McKenzie et al., 2010), and

therapy dogs (Lefebvre et al., 2008), although none of these studies followed a rigorous approach to control for other risk factors. One study observed that even being fed cooked home-made diets, as well as a recent change in diet, was associated with higher risk of diarrhea in dogs, possibly hinting at issues with wet food (Stavisky et al., 2011). No human illness has been associated with raw pet food to-date. Hence, the actual risk posed to pets and owners by raw and home-made diets is still debated (Schlesinger and Joffe, 2011; Remillard, 2008).

Commercial pet food has evolved over time (Bontempo, 2005). The first processed dog food, developed by James Spratt in England in 1860, was a dry biscuit made of beef blood, vegetables, and grains. After World War I a surplus of horse meat led to the increased popularity of canned pet food. During World War II, due to meat shortage pet food production shifted again towards dry food, which made up 80% of the U.S. pet food market in 1946 (Corbin, 2003; Pet Food Institute, 2013). The first extruded pet food was produced in the 1950s (Riaz, 2003). Currently, a variety of industrial diets are available, targeting the nutritional needs of pets of different breed, size, age, and health status (Morris and Rogers, 1994). Snack treats have also become more popular (Cipollini and Zaghini, 2007). Currently dry food makes up more than 60% of the U.S. market. Industrial pet food production has been growing significantly in the last decades, reaching a worldwide market value of U.S.$ 56 billion in 2011 (PMMI, 2013). Currently the U.S. production holds 30% of the world market (U.S. $ 20 billion in 2011, projected to grow to U.S. $ 27 billion by 2015). Five producers account for more than 70% of the U.S. pet food production. The industry is also growing rapidly in Brazil, India, and China. In 2013, 32% of the growing world demand is estimated to be "natural" and premium pet foods, while wet dog food production is not projected to grow significantly (PMMI, 2013).

The current production process of industrial dry kibble for pet nutrition involves several steps: dry ingredients are first mixed, then wetted and briefly cooked in a preconditioner, extruded at high temperature, cut into kibble, dried, coated, cooled, packaged, and distributed to retailers. Before processing, in between processing steps, and after packaging, ingredients or the finished product are stored at various temperatures and for variable times. While the mix of dry ingredients undergoes a preconditioning and an extrusion step at high temperatures, the fats, vitamin mixes, and flavorings added during coating are not treated at high temperature. Several processing steps are carried out at temperatures that, if other conditions were favorable, would lead to bacterial growth. For example, fats are stored at temperatures in the order of 150°F (66°C) to keep the ingredient fluid, coating occurs at temperatures of 100–120°F (38–49°C), after which a cooling step slowly brings the product to room temperature. Furthermore, both dry ingredients and the final product are usually stored at room temperature, which in summer could be a growth-favorable temperature of 90–100°F (32–38°C).

Contamination by human bacterial pathogens, as well as by toxins of microbial origin, has been observed in processed pet food (Table 2a) (Adley et al., 2011; Brazis et al., 2008; Fernandez Juri et al., 2009; Finley et al., 2008; Leung et al., 2006; Li et al., 2012; Martins et al., 2003; Nakajima et al., 1992). In extensive surveys carried out by the U.S. FDA Center for Veterinary Medicine, *Salmonella* was observed on finished pet food and treats at a prevalence of 6.1–12.4% between 2002 and 2009, with a marked decline from 18.1% in 2002–2006 to 8.4% in 2007–2009 (Li et al., 2012). Prevalence in pet supplements was 7–18% (Li et al., 2012). While raw meat is often assumed to pose the highest risk of *Salmonella* exposure, this pathogen has also been observed in dry meat-based pet edible products, either uncooked such as pig ears and chew treats (Clark et al., 2001; Adley et al., 2011; White et al., 2003), or heat-treated such as dry food (Weber et al., 2002; Nakajima et al., 1992; Martins et al., 2003; Brazis et al., 2008). Notably, two of the recent human outbreaks have been associated with *Salmonella* in dry dog kibble (CDC, 2006, 2008a, 2008b, 2012; Behravesh et al., 2010), and three with *Salmonella* in treats (LCDC, 2000; Pitout et al., 2003; CDC, 2006), while no outbreak to-date has been associated with canned wet pet food. The origin of contamination is largely unknown. From November 2005 to April 2013, there were 1129 pet food recalls or voluntary withdrawals in the U.S. (U.S. FDA, 2013b). A high proportion of these recalls were due to product samples positive for presumptive *Salmonella* (e.g., 56%, 25 out of 45 in 2012) (U.S. FDA, 2013b), in addition to recalls due to chemical contamination or misformulation (Bischoff, 2012). In the European Union, the RASFF (Rapid Alert System for Food and Feed) database cites 328 incidents related to pet food between January 2006 and March

Table 2a Evidence of pathogen contamination in finished pet food and treats

Product	Location	Prevalence	Result highlights	References
Dry food, treats	USA	6.1–12.4%	From 12.4% in 2002–06 to 6.1% in 2007–09. Pet supplements: 7.1–18.8%	Li et al., 2012
Pig ear treats	USA	41%	65/158 samples	White et al., 2003
Pig ear treats	Ireland	24.5%	25/102 pig ears positive with culture isolation, 29/102 (28.4%) by PCR	Adley et al., 2011
Pig ear treats	Canada	29–51%	49/171 from production plant, 48/94 retail samples	Clark et al., 2001
Pig ear treats	Canada	4%	12/295 samples	Finley, 2004; Finley et al., 2006
Treats	NZ	5.3–6.7%	6.7% in domestic treats, 5.3% in imported. 12 serotypes.	Wong et al., 2007
Dog chews	UK, imported	7.8%	184/2369 imported treats positive, 29 serotypes in 1998–2000.	Willis, 2001
Dry pet food	Poland	0.15–1.7%	From 2007 to 2010: 0.15%, 1.07%, 1.30%, 1.74% (655–3049 samples/year)	Kukier et al., 2012
Pet food	Poland	1%	22/2271. No *Salmonella* in 18 canned food samples.	Wojdat et al., 2004

2013, 151 of which (46%) due to *Salmonella* contamination (European Commission, 2013).

The available evidence of pathogen contamination in feed for production animals is reviewed in Table 2b. Evidence of contamination in pet food ingredients and similar products is reviewed in Tables 2c–g. Processed dog and cat food is usually composed of meat, cereals, vegetables, animal and vegetable fats, additives such as vitamins and minerals, and flavors added to increase palatability. Cat food can often also contain fish and seafood. The meat portion of dry pet foods is usually a dry granular by-product of the meat industry obtained by rendering, and sold under the name of "meal." Although rendering involves high temperatures to separate fat from meat and bones, the final meal product is often contaminated by bacteria (Table 2c). Also pork and poultry products for human consumption, either at retail or more frequently at the

Table 2b Evidence of pathogen contamination in animal feeds

Feed type	Pathogen	Country	Prevalence summary	Result highlights	References
Animal feed	*E. coli* (STEC)	UK	0%	STEC in fresh grass samples: 6.3% (5/79), silages: 0%; heat-processed feeds: 0%	Hutchison et al., 2006
Plant-protein feed	*E. coli*	USA	5.7%	9/158 samples (4 in maize, 1 peanuts, 1 whole sunflower, 1 pelleted feed, 2 milk replacer powder)	Myint et al., 2007
Poultry feed	*Enterobacteriacee*	Netherlands	1.3–4.3%	6/60 samples	Veldman et al., 1995
Pelleted poultry feed	*Listeria* spp.	Ireland	13.3%	2/15 samples	Whyte et al., 2003
Animal feed	*Salmonella*	Spain	3.3%	Feed materials: 3.5%; compound feed: 3.3%; dust in feed mills: 12.5%; feed mills: 27.5%(144/523); total samples: 4.8%(185/3844)	Torres et al., 2011
Animal feed	*Salmonella*	Sweden	1.1%	Imported feeds of animal origin. Years 1978–1982. 172 strains isolated	Martensson et al., 1984
Animal feed	*Salmonella*	UK	34.5–38.9%	Cattle feed: 38.5% (347/902); pig feed: 38.9% (474/1220); poultry feed: 34.5% (833/2411)	Papadopoulou et al., 2009
Animal feed	*Salmonella*	USA	5.6–9.4%	5.6–9.4% in feed; 19.4–30.9% in ingredients	Li et al., 2012
Animal feed	*Salmonella*	USA	23.5%	4/17 samples	Stott et al., 1975
Animal feed	*Salmonella*	USA	3.3%	1/30 samples feed; 0/6 liquid fat	Jones, 2008, Jones, 2011
Animal feed ingredients	*Salmonella*	USA	8.8%	Brewers grains: 0% (0/3); corn: 5.26% (1/19); cottonseed meal: 100%(2/2); fish meal: 100%(1/1); limestone: 0 (0/1); meat and bone meal: 0%(0/1); soybean hulls:0%(0/5); soybean, meal:10%(1/10); wheat: 0%(0/1); whey: 0%(0/1); wheat middlings: 4.17%(1/24)	Jones and Richardson, 2004
Compound animal feed	*Salmonella*	EU	0.5–0.7%	10,838 samples	EFSA et al., 2012
Compound animal feed	*Salmonella*	EU	0.5–0.9%	Averages of 0.5% (range 0–3.6%) cattle feed, 0.6% pig feed, 0.9% (range 0–8.3%) poultry feed	EFSA et al., 2010
Compound animal feed	*Salmonella*	Poland	0.8–1.8%	All animal feeds: 0.98%, 1.42%, 0.80%, 1.81%. Sample no. range: 3,091–7,280	Kukier et al., 2012
Compound animal feed	*Salmonella*	USA	4.2–8.3%	8.8% mash feed, 4.2% pelleted feed	Threlfall et al., 2003
Compound cattle feed at farms	*Salmonella*	USA	9.8%	29/295 samples in 6 farms	Krytenburg et al., 1998
Compound cow feeds and ingredients at farms	*Salmonella*	USA	42%	21/50 piles presumptive positive	Kidd et al., 2002
Compound pig feed	*Salmonella*	USA	0%	0/221 samples	Barber et al., 2002
Compound pig feed at farms	*Salmonella*	USA	2.8%	36/1264 samples in 30 swine farms	Blaser et al., 1982
Farm feed	*Salmonella*	South Africa	2.9–6.1%	5.18% (577/11141) of farm feed sample; 9.54% (260/2726) by product sample. Cattle: 6.13%; calf: 5.48%; pig: 6.10%; sheep: 5.77%; poultry: 5.65%; miscellaneous: 2.94%. Years 1982–1988.	Durand et al., 1990
Layer feed	*Salmonella*	Japan	3.3%	146/4418 samples	Shirota et al., 2000
Pelleted poultry feed	*Salmonella*	Ireland	0%	0/15 samples	Whyte et al., 2002
Plant-protein feed	*Salmonella*	USA	0.6%	1/158 (whole sunflower)	Myint et al., 2007
Poultry feed	*Salmonella*	Netherlands	2–27%	Mash feeds: 21%; pelleted feeds: 1.4%; fish meal: 31%; meat and bone: 4%; tapioca: 2%; maize grits: 27%	Veldman et al., 1995
Poultry feeds	*Salmonella*	Greece	6.6–20.6%	1/15 pelleted feed, 7/35 broiler feed, 6/29 layers feed.	Zdragas et al., 2001
Animal feed	*Salmonella*	USA		Review article	Crump et al., 2002
Animal feed	*Salmonella*	USA		Review article	Jones et al., 2011
Poultry feed	*Salmonella*	USA		Review article	Maciorowski et al., 2004
Poultry feed	*Salmonella*	USA		Review article	Williams, 1981

Table 2c Evidence of pathogen contamination in meals derived from rendering processes

Product	Pathogen	Product use	Location	Prevalence	Result highlights	Enumeration	References
Meat and bone meal	*Salmonella*	NA	Australia	69.5%	114/164 finished product samples. 53.8% (35/65) along production line	0.2–34.8 CFU/g in meal	Bensink, 1979
Animal protein meals	*Salmonella*	Feed	Brazil	37.7%	77/204 samples: meat meal, feather meal, viscera meal, bone meal	NA	Berchieri et al., 1984
Meat and bone meal	*Salmonella*	Feed	EU	1–2.3%	Years 2006–2008	NA	EFSA, 2010
Meat and bone meal	*Salmonella*	Feed	EU	0.6–1.4%	By year: 33/5436 (0.6%) in 2010, 84/6015 (1.4%) in 2009, 84/8399 (1.0%) in 2008.	NA	EFSA and ECDC, 2012
Meat and bone meal	*Salmonella*	Poultry feed	Netherlands	4%	Also fish meal: 31%	NA	Veldman et al., 1995
Animal protein meals	*Salmonella*	NA	Poland	0–1.2%	Poultry meal: 0%, Meat and bone meal: 0–1.2%; fish meal: 0–2%	NA	Kukier and Kwiatek, 2011
Carcass, bone, and blood meals	*Salmonella*	NA	South Africa	3–17%	Carcass meal: 17%; Blood meal: 3%; Bone meal: 8%	NA	Van der Made et al., 1980
Animal protein meals	*Salmonella*	NA	USA	~ 25%		Range in positive samples: 0.03–1,100 MPN/g (skewed, mean 16.3)	Franco, 2005
Rendered animal products	*Salmonella*	NA	USA	0–13.7%	Meat meal: 7.1%, Meat and Bone Meal (MBM): 8.1%, Poultry: 13.7, Blood: 0%, MBM-Poultry: 0%, Feather: 6.7%, total: 8.7%	NA	Kinley et al., 2010
Rendered protein feed	*Salmonella*	Feed	USA	56%	57/101 samples, year 1993	NA	McChesney et al., 1995
Meat and bone meal	*Salmonella*	Poultry feed	USA	14%	6/43 meat and bone meal. 5% blended meal, 0% poultry and feather meal	NA	Hofacre et al., 2001
Rendered protein feed	*Salmonella*	Feed	USA	25%	22/89 samples, year 1994	NA	Crump et al., 2002
Meat and bone meal	*Salmonella*	Poultry feed	Greece	24.2%	8/33 samples	NA	Zdragas et al., 2001
Meat and bone meal	*Salmonella*	Feed	USA	38.9%	In all rendered products: 34.4 (42/122)	NA	Ge et al., 2013
Meat and bone meal	*E. coli*	Feed	USA	36.1%	In all rendered products: 36.9 (45/122)	NA	Ge et al., 2013
Carcass, bone, and blood meals	*E. coli*	NA	South Africa	54–68%	Carcass meal: 59%; Blood meal: 68%; Bone meal: 54%	NA	van Der Made et al., 1980

slaughterhouse, have been found contaminated by pathogenic bacteria such as *Salmonella, Campylobacter*, and pathogenic *E. coli*. Prevalence varies widely in different studies, with a range of 1–85% for *Salmonella* in pork carcasses and 0–17% in pork meat (Table 2d). For poultry products, observed *Salmonella* prevalence ranged from 0 to 100% (Table 2e). Neither carcasses nor retail meat are direct inputs into the pet food production chain. However, animal by-products used as pet food ingredients are usually of lower quality and undergo less scrutiny than those destined for human consumption, hence their level of contamination is likely higher. Furthermore, while meat has historically been considered the ingredient most at risk for bacterial contamination, other ingredients can also harbor enteric pathogens. For example, contamination by *Salmonella* and pathogenic *E. coli* has been observed at low prevalence levels in wheat and flour (Table 2f) (Berghofer

Table 2d Evidence of pathogen contamination in pork carcasses and meat

Pathogen	Production stage	Location	Prevalence range	Result highlights	References
Campylobacter	Carcass	Canada	16.9%	78/463 samples, years 1983–1986	Lammerding et al., 1988
Campylobacter	Carcass	Netherlands	9.1%	9.1% at slaughter, no detection after cooling or in minced meat. Mean levels in intestine contents: 4,000 CFU/g	Oosterom et al., 1985
Campylobacter	Ground meat	Ireland	22%	40/179 samples	Lynch et al., 2011
Salmonella	Carcass	Australia	9.3–28%	0.28 (42/150), 0.128 (19/148), 0.093 (14/150) at 18 hr, 42 hr, and 66 hr of lairage	Morgan et al., 1987
Salmonella	Carcass	Belgium	37%	137/370 (37%) before chilling, 12/75 (16%) in chilling room. Range: 0–70%, and 3–52% in same facility	Botteldoorn et al., 2003
Salmonella	Carcass	Brasil	62.5–85%	500–680 of 800 carcasses	Schwarz et al., 2009
Salmonella	Carcass	Canada	17.5%	104/596 samples, years 1983–86	Lammerding et al., 1988
Salmonella	Carcass	Denmark	9.7%	161/1665 samples	Sorensen et al., 2004
Salmonella	Carcass	Denmark	9.4%	11/117 samples	Kranker et al., 2003
Salmonella	Carcass	Europe	5.3%	185/3485 samples	Hald et al., 2003
Salmonella	Carcass	Germany	0.3–1.1%	Overall: 1.8% by VIDAS, 1.1% by culture; cuts: 1.0% VIDAS, 0.3% culture (865 samples); carcass: 1.7% VIDAS, 1.1% culture (1,569 samples)	Meyer et al., 2010
Salmonella	Carcass	Germany	10.3%	43/419 samples	Quirke et al., 2001
Salmonella	Carcass	Germany	1.7%		Fehlhaber et al., 1996
Salmonella	Carcass	Ireland	1%	1.04% (2 of 193) in meat	Duggan et al., 2010
Salmonella	Carcass	Ireland	3.3–15.2%	Joints 3.3%, eviscerated carcasses: 15.2% (29/191)	Gonzales Barron et al., 2009
Salmonella	Carcass	Netherlands	12.9%	27/210 carcass samples. Mean levels in intestine contents: 10 CFU/g.	Oosterom et al., 1985
Salmonella	Carcass	Netherlands	1.4%	3/213 carcasses, but overall 47% of 1114 pigs (25.6% rectal samples, 19.6% tonsils, 9.3% liver, tongues, and lymph nodes).	Swanenburg et al., 2001
Salmonella	Carcass	North Ireland, UK	40%	205/513 carcass swabs, also 31.4% caecal content	McDowel et al., 2007
Salmonella	Carcass	Spain	39.7%	356/896 samples	Arguello et al., 2012
Salmonella	Carcass	UK	5.3%	134/2509 samples, years 1999–2000	Davies et al., 2004
Salmonella	Carcass	USA	3.7%	At prescald, preevisceration, and after chilling: 91.2%, 19.1%, 3.7% (enumerable *Salmonella* 37.7%, 4.8%, 0.6%). 294 prescald carcasses >1.9 log CFU/100 cm^2 (not equally distributed btw two plants)	Schmidt et al., 2012
Salmonella	Carcass	Germany	15.5–16.4%	16.4% in intestine/lymph nodes, 15.5% tonsils. 383 animals sampled	Nowak et al., 2007
Salmonella	Meat at slaughterhouse	Netherlands	13.0%	33/248 samples	Oosterom et al., 1985
Salmonella	Retail meat	Ireland	1.1–2.4%	Years 2005–2009	Duggan et al., 2012
Salmonella	Retail meat	Ireland	2.6%	Concentrations range: <0.03 to 2.10 MPN/g	Prendergast et al., 2009
Salmonella	Retail meat	Italy	4.9%	Years 2000–2001	Busani et al., 2005
Salmonella	Retail meat	Mexico	17.3	14/81 samples	Miranda et al., 2009
Salmonella	Retail meat	New Zealand	0%	Years 2003–2005	Wong et al., 2007
Salmonella	Retail meat	UK	3.9%	Overall 3.9% ($n = 1440$); muscle: 1.9% (25/1309); offal: 23.6% (31/131). Years 2003–2005	Little et al., 2008a
Salmonella	Retail meat	USA	3.3%	7/209 samples	Zhao et al., 2001
Salmonella Heidelberg	Retail pork chops	USA	3.4%	10/298 samples, years 2002–2006	Zhao et al., 2008

et al., 2003; Eglezos, 2010). In a survey of 4,000 wheat samples, 12.8% were positive for nonpathogenic *E. coli*, and 1.3% for *Salmonella* (Richter et al., 1993). Other studies found *Salmonella* prevalence ranging from 0 to 6.5%, and generic *E. coli* from 1 to 12% in wheat and flour (Table 2f). An extensive survey of ingredients for pet food and animal feed production detected *Salmonella* in 30.9% (104 out of 337) and 19.4% (40 out of 206) of samples in the years 2002–2006 and 2007–2009, respectively, with prevalence significantly higher in animal than plant-derived ingredients (Li et al., 2012). Microbial safety guidelines for milled grains have been developed (Sperber, 2007). Conversely, very little information on

Table 2e Evidence of pathogen contamination in poultry carcasses and meat

Pathogen	Product/ production stage	Country	Prevalence range	Result highlights	References
Campylobacter	Carcasses or portions at retail	Australia	87.8–93.2%	482/549 (New South Wales), 289/310 (South Australia), range 80–100% depending on retail type. Avg 0.61–1.24 Log CFU/cm^2 (NSW), 0.72–0.90 Log CFU/cm^2 (SA)	Pointon et al., 2008
Campylobacter	Carcass	Canada	38.2%	157/410 samples, years 1983–86	Lammerding et al., 1988
Campylobacter	Meat at slaughterhouse	Croatia	0%	0/75 swabs	Granic et al., 2009
Campylobacter	Raw retail meat	Croatia	66.6%	10/15 meat swabs	Granic et al., 2009
Campylobacter	Raw chicken legs	Germany	11.3–67.8%	Carcass surface: 67.8% (48/71); internal parts: 11.3% (8/71)	Luber et al., 2005
Campylobacter	Carcass at retail	Greece	0%	100 samples conventional, 60 free-grazing. Mean levels: non detected for conventional; non detected for free grazing	Voidarou et al., 2011
Campylobacter	Carcass at retail	Senegal	56.0%	168/300; 76% prevalence in fresh, 53% in refrigerated, 28% in frozen	Cardinale et al., 2003
Campylobacter	Raw retail products	Ireland	84.3%	430/510 samples	Madden et al., 2011
Campylobacter	Raw retail products	Ireland	16%	30/185 samples	Lynch et al., 2011
Campylobacter	Raw retail meat	Japan	21.6–60.3%	Review paper. Meat: 58.8%; By-products: 60.3%; Ground meat: 21.6%; Frozen: 23.6%	Suzuki and Yamamoto, 2009b
Campylobacter	Raw retail products	New Zealand	44.8%	73/163 samples	Chrystal et al., 2008
Campylobacter	Raw retail products	Multiple (review)	35.1%	At processing: 82.8%; raw products in retail: 35.1%	Mataragas et al., 2008
Campylobacter	Raw retail meat	Spain	49.5%	98/198 samples	Dominguez et al., 2002
Campylobacter	Raw retail products	UK	70.8%	523/739 samples fresh: 70.2% frozen: 72.6%; butchers: 70.4% retailers: 70.9%	Meldrum et al., 2004
Campylobacter	Raw retail meat	UK	60.9%	1281/2104 samples	Little et al., 2008a
Campylobacter	Raw carcass at retail	UK	83%	199/241 samples	Jorgensen et al., 2002
Campylobacter	Raw carcass at retail	UK	73.10%	538/736 samples fresh: 73.5% frozen: 71.9%	Meldrum et al., 2005
Campylobacter	Carcass	USA	83–100%	83%–100% (40–48/48) for different ages; 90%–92% (65–66/72) for feed withdrawal; 99% (71/72) before chilling; 83% (60/72) after chilling	Northcutt et al., 2003
Campylobacter	Carcass	USA	39.3–48.7%	39.3% in air chilled, 48.7% in immersion chilled broilers	Sanchez et al., 2002
Campylobacter	Raw retail meat	Multiple (review)	0–100%	Review paper. Meat: avg 58% (8.1–100)%; By-products: avg 69.6% (23.1–100%); Ground meat: avg 17% (0–21.6%); Frozen: avg 47.2% (7.9–67.9%)	Suzuki and Yamamoto, 2009a
E. coli	Carcasses or portions at retail	Australia	59.6–100%	Whole birds: 85.7–100% (14–28 samples); chicken portions: 59.6–90.1% (141–252 samples). Avg 0.43–0.50 Log CFU/cm^2 in whole birds, 0.50–0.91 Log CFU/cm^2 in chicken pieces	Pointon et al., 2008
E. coli	Carcass	USA	N/A	Broilers from *Campylobacter*-positive flocks. 100% (48/48) for different age; 100% (72/72) for feed withdrawal; 100% (72/72) before chilling; 100% (72/72) after chilling	Northcutt et al., 2003
E. coli	Carcass	Austria	N/A (presumed 100%)	scalding: 5.91 log CFU/mL; defeathering: 5.16 log CFU/mL;	Smulders et al., 2011

(Continued on next page)

Table 2e Evidence of pathogen contamination in poultry carcasses and meat *(Continued)*

Pathogen	Product/ production stage	Country	Prevalence range	Result highlights	References
				evisceration: 5.63 log CFU/mL; refrigeration: 3.83 log CFU/mL; packaging: 4.05 log CFU/mL	
E. coli	Carcass	Czech Republic	N/A (presumed 100%)	After plucking: 3.5 Log CFU; after evisceration: 3.1 Log CFU; after washing: 2.7 Log CFU; after chilling: 1.8 Log CFU	Svobodova et al., 2012
E. coli	Raw retail products	Greece	22.3–25.9%	Conventional:25.9% (52/201); nonconventional: 22.3% (45/201)	Zhang et al., 2011
E. coli EHEC	Raw retail products	Multiple (review)	0.5%	At processing: 1.2%; raw products in retail: 0.5%	Mataragas et al., 2008
Enterococcus	Raw retail products	Greece	17.4–21.3%	Conventional: 17.4% (35/201); nonconventional: 21.3% (43/201)	Zhang et al., 2011
Listeria monocytogenes	Carcass at retail	Greece	N/A (presumed 100%)	100 samples conventional, 60 free-grazing. Means: non detected for conventional; 0.16 log CFU/mL for free grazing	Voidarou et al., 2011
Listeria monocytogenes	Raw retail meat	Italy	1.9%	24/1269 samples	Busani et al., 2005
Listeria monocytogenes	Raw retail products	Multiple (review)	14.5%	At processing: 14.9%; raw products at retail: 14.5%	Mataragas et al., 2008
Listeria spp.	Broiler carasses	Czech Republic	7.5%	12/160 samples	Svobodova et al., 2012
Salmonella	Carcasses or portions at retail	Australia	35.5–47.7%	262/549 (New South Wales), 110/310 (South Australia). Avg levels 250–380 CFU/cm^2 in whole bird meat, 0.04–0.95 MPN/cm^2 in chicken pieces	Pointon et al., 2008
Salmonella	Carcass	Canada	60.9%	408/670 samples, years 1983–86	Lammerding et al., 1988
Salmonella	Carcass at slaughtering	Czech Republic	3.75%	6/160 samples	Svobodova et al., 2012
Salmonella	Carcass at retail	Greece	N/A (presumed 100%)	100 samples conventional, 60 free-grazing. Mean levels: non detected for conventional; 0.29 log CFU/mL for free grazing	Voidarou et al., 2011
Salmonella	Raw retail products	Greece	1.5–5.0%	Conventional: 1.5% (3/201); nonconventional: 5.0% (10/201)	Zhang et al., 2011
Salmonella	Carcass at retail	Senegal	32.0%	96/300; no difference among fresh, refrigerated, and frozen	Cardinale et al., 2003
Salmonella	Raw retail products	Ireland	5.10%	26/510 samples	Madden et al., 2011
Salmonella	Raw retail meat	Ireland	0.9–5.1%	Years 2005–2009	Duggan et al., 2012
Salmonella	Raw retail meat	Italy	9.9%	291/2953 samples	Busani et al., 2005
Salmonella	Raw retail products	Multiple (review)	9.6%	At processing: 2.0%; raw products in retail: 9.6%	Mataragas et al., 2008
Salmonella	Raw retail meat	Spain	35.83%	71/198 samples	Dominguez et al., 2002
Salmonella	Raw retail products	UK	8.4%	62/739 samples fresh: 8% frozen: 9.7%; butchers:6.8% retailers: 9.1%	Meldrum et al., 2004
Salmonella	Raw retail meat	UK	5.6%	118/2104 samples	Little et al., 2008a
Salmonella	Raw whole chickens	UK	25%	60/241 samples	Jorgensen et al., 2002
Salmonella	Whole poultry at retail	UK	5.7%	42/736 samples; fresh: 4.4%, frozen: 9.4%	Meldrum et al., 2005
Salmonella	Raw retail meat	USA	8.3–12.9%	12.9% in large establishment; 8.3% in very small establishment	Rose et al., 2002
Salmonella	Carcass	USA	18.7–24.7%	18.7% in air chilled, 24.7% in immersion chilled broilers	Sanchez et al., 2002

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Table 2e Evidence of pathogen contamination in poultry carcasses and meat *(Continued)*

Pathogen	Product/ production stage	Country	Prevalence range	Result highlights	References
Salmonella Heidelberg	Raw retail meat	USA	36.9%	110/298 samples	Zhao et al., 2008
Salmonella	Raw retail products	New Zealand	0%	0/163 samples	Chrystal et al., 2008
Staphylococci	Carcass	Austria	N/A (presumed 100%)	scalding: 6.59 log CFU/mL; defeathering: 5.37 log CFU/mL; evisceration: 4.79 log CFU/mL; refrigeration: 4.20 log CFU/mL; packaging: 4.29 log CFU/mL	Smulders et al., 2011
S. aureus	Carcass at retail	Greece	N/A (presumed 100%)	100 samples conventional, 60 free-grazing. Mean levels: 0.69 log CFU/mL for conventional; 1.15 log CFU/mL for free grazing	Voidarou et al., 2011
Yersinia enterocolitica	Raw retail products	Multiple (review)	7.6%	At processing: 7.8%; raw products in retail: 7.6%	Mataragas et al., 2008

contamination in oils and oil seeds is available (Table 2g). The few published studies highlight *Salmonella* prevalences that are usually low, but can be up to 14%. In addition, contamination has been observed in oilmeal plants, pointing out the risk of cross-contamination for oil products (Morita et al., 2006). Most of the mentioned studies measured prevalence, but not concentrations, thus limiting the value of the collected information for risk assessment purposes.

PATHOGEN DYNAMICS DURING PET FOOD PRODUCTION

During the commercial production of pet food, ingredients and intermediate products are subjected to conditions that may affect microbial levels. For example, conditions involving high water activity, moderate heat, and neutral pH may foster bacterial growth, while high heat, extreme pH, and desiccation could cause a decline in bacterial levels. Since the process is often designed to minimize bacterial growth, it is unlikely that an entire batch of product is subjected to conditions favorable to growth. However, localized events such as the presence of condensate at the top of a tank, or moisture dripping over a small portion of dry ingredients or finished product could, if bacteria are present, significantly amplify cell numbers. A high number of cells, initially concentrated in a small mass of product, could then potentially be distributed to a larger batch during mixing. In the case of *Salmonella*, particular attention needs to be paid to the interaction between humidity and temperature, since there is evidence that *Salmonella* that underwent desiccation is more resistant to heat (Izurieta and Komitopoulou, 2012; Podolak et al., 2010; Doyle and Mazzotta, 2000). In the case of viruses or protozoa, no growth is possible outside of the host, and levels could only decline or remain constant. However, mixing, batching, and cross-contamination still play an important role in how widespread or concentrated the contamination can be.

The kinetics of pathogenic bacteria populations in pet food ingredients has important implications for the safety of the finished product. Generally bacteria such as *Salmonella* or *E. coli* do not grow at low moisture/water activity conditions, i.e., $a_w < 0.90$, though fungi can potentially grow at substantially lower ranges, i.e., $a_w > 0.6$–0.7, and *S. aureus* can grow aerobically at $a_w > 0.83$. In all studies carried out on dry foods pathogenic bacteria decline over time or remain at stable population levels, depending on environmental variables. A review of decline kinetics of common bacterial zoonotic pathogens on dry food matrices is presented in Table 3. For example, *Salmonella* on dry nuts declines slowly but steadily at ambient temperature (23–25°C) and above, but population levels can remain stable for more than a year at 4°C and −20°C (Blessington et al., 2012a; Kimber et al., 2012). Decline rate depends on bacterial species and strain, temperature, water activity, and the specific substrate. Observed decline rates range from −0.15 Log CFU/month on inshell pistachios at 25°C (Kimber et al., 2012), to an average of −3.9 Log CFU/month on chorizo (Escartin et al., 1999). When tests are carried out over several months or years, in most cases decline kinetics are markedly nonlinear: an initial phase of rapid and approximately linear decline is followed by a long phase of slow decline or persistence at constant low levels. This trend has important implications for estimating risk, as accounting only for the initial fast-decline phase would underestimate cell counts and thus exposure. Even when declining, *Salmonella* has been detected on dry food for several months or even years (Jones, 2011). Depending on inoculum levels, substrate, moisture, and strain, *Salmonella* was observed to survive for up to 10 months on dried milk (Ray et al., 1971), up to 12 months on dry pasta (Rayman et al., 1979), more than 9 months on milk chocolate at 20°C (Tamminga et al., 1976), up to 24 months in peanut butter at 5°C and 21°C (Burnett et al., 2000), and more than 8 months on paprika powder (Lehmacher et al., 1995). A review of survival and decline rates of *Salmonella* in low-moisture food was

Table 2f Evidence of pathogen contamination in grains, flours, and plant-derived meals

Pathogen	Product	Country	Prevalence	Result highlights	References
E. coli, coliforms	Brown rice	Korea	0%	0%	Kim et al., 2012
E. coli, coliforms	Wheat flour	Australia	0%; 58.3%	58.3% coliform positive (median concentration 1.4 CFU/g); no *E. coli* detected. Coliform levels up to 10^3 in bran, pollard, conditioned wheat	Eyles et al., 1989
E. coli	Flours	Australia	1–11%	1%, 4%, 11% in flour, bran, wheat germ	Berghofer et al., 2003
E. coli	Wheat and flour	Australia	0–89%	3.4–12% in red wheat/flour, 0% in wheat, 0% in wheat flour. Up to 89% in spring wheat flour	Eglezos, 2010
E. coli	Flours	USA	12.8%	12.8% of 3350 samples	Richter et al., 1993
E. coli	Plant-derived meals	USA	39.30%	46/201 samples (soybean, cottonseed, sunflower, linseed, canola, corn, hominy)	Ge et al., 2013
Salmonella	Cereals	EU	0.2–0.4%	summary of EU member states	EFSA, 2010
Salmonella	Cereals	EU	0.2–0.9%	By year: 27/3035 (0.9%) in 2010, 15/3633 (0.4%) in 2009, 11/5262 (0.2%) in 2008	EFSA and ECDC, 2012
Salmonella	Cereals (maize, barley, wheat, oats)	Poland	0–0.9%	Only positive was barley: 0–6.5% in 2007–2010	Kukier and Kwiatek, 2011
Salmonella	Flours	Australia	0%	0% in final product (2/412 mill samples)	Berghofer et al., 2003
Salmonella	Flours	USA	1.3%	40/3040 samples	Richter et al., 1993
Salmonella	Grains, soy	Greece	3.5–5.8%	Grains: 1/28, soy: 1/17	Zdragas et al., 2001
Salmonella	Sesame seeds	Germany	0.3%		Brockmann et al., 2004
Salmonella	Soy meal, cereals	NL	0%	0/52 soy meals, 0/80 cereal samples	Sauli et al., 2005
Salmonella	Soy meal, rapeseed meal	10–14.6%	10% rapeseed meal, 14.6% soy meal	Wierup et al., 2010	
Salmonella	Tapioca, maize for animal feeds	Holland	2–27%	tapioca: 2%; maize grits: 27%	Veldman et al., 1995
Salmonella	Plant-derived meals	USA	22.90%	46/201 samples (soybean, cottonseed, sunflower, linseed, canola, corn, hominy)	Ge et al., 2013

carried out by Podolak and collaborators (GMA, 2009b; Podolak et al., 2010).

A completely different situation could present itself if ingredients or the intermediate product were rewetted, for example through condensation or leaks in the facility, or if the product were recontaminated while still wet, for example by dust, after preconditioning and before complete drying. Studies on wetted or rehydrated products that are usually dry demonstrated that significant pathogen growth can occur, in particular at room temperature. Different food matrices can support pathogen growth to different extent and at different rates, depending on several variables including water activity,

Table 2g Evidence of pathogen contamination in oils, oil seeds, and oilmeals

Pathogen	Product	Country	Prevalence	Result highlights	References
Salmonella	Oil seeds and products	EU	1.3–1.8%	175/11683 (1.5%) in 2010, 139/10720 (1.3%) in 2009, 338/18786 (1.8%) in 2008	EFSA and ECDC, 2012
Salmonella	Oil seeds and products	EU	1.8–2.5%	NA	EFSA, 2010
Salmonella	Oilmeal	Japan	2%	NA	Sato, 2003
Salmonella	Rapeseed, sunflower, soy oils	Poland	2.3–9.7%	Years 2007–2010. Rape seed 2.0–4.5%, sunflower 0–1.14%, soy 2.3–9.7%, all oil seeds 1.4–2.5% (4026 samples)	Kukier and Kwiatek, 2011
Salmonella	Soybean, rapeseed, safflower oilmeals	Japan	5.9–52.4%	Soybean: 5.9%; rapeseed: 30.4%; safflower 52.4%	Kinoshita et al., 1988; Kobayashi et al., 1989

Table 3 Survival or decline kinetics of *Salmonella* on selected biological substrates

Microrganism	Food matrix	Experimental conditions	Duration of test	Decline rate (Log CFU/month) at room temperature [a]	Result highlights	References
Bacillus cereus	Infant formula	20°C	9 days	0.18	1.04 Log reduction in 9 days (semi-linear)	Jin et al., 2008
Cronobacter sakazakii	Infant formula	4°C, 21°C	15 days		At 21°C: 1.5 Log CFU growth by day 6 (capsulated strain) and by day 9 (noncapsulated), then declined 2–2.5 Log CFU in 6–9 days. Overall 1 Log CFU decline in 15 days. At 4°C, 0.5 (noncapsulated) to 1.5 (capsulated) Log CFU reduction in 15 days	Al-Holy et al., 2009
Cronobacter sakazakii	Infant formula	21°C, a_w:0.2, 0.31, 0.44, 0.52, 0.75, 0.81, 0.86	12 months	0.19 (a_w 0.2 and 0.31); 0.79 (a_w 0.44); 6.5–12.4 (a_w: 0.52, 0.75, 0.81, 0.86)	Review article. At a_w<0.5, faster decline in first month, then linear. At a_w>0.5, fastest decline in first 2 weeks, all nondetect at 4 weeks	Beuchat et al., 2009
Cronobacter sakazakii	Infant formula	Ambient temperature, 10 strains	30 months	0.05 to 0.20 (NL)	Faster decline in first 4–6 weeks	Caubilla-Barron et al., 2007
Cronobacter sakazakii	Infant formula	Ambient temperature	2 years	0.15	−2.4 Log CFU in first 5 months, additional −1 Log CFU in next 19 months	Edelson-Mammel et al., 2005
Cronobacter sakazakii	Infant formula	22°C	16 days	0.32 to 0.39 (NL)	Rate estimate based on $D = 78$ days (Weibull function, best fit) and $D = 95$ (Log-linear)	Kandhai et al., 2010
Cronobacter sakazakii	Infant formula	21°C. Infant rice, barley, mixed grain, and oatmeal cereals. a_w 0.30–0.83	24 weeks, 12 months	Over 24 weeks: 1.6 to 1.7 (a_w 0.83); 0.7 to 1.0 (a_w 0.63–0.66). Over 12 months: 0.21 (a_w 0.30), 0.42 (a_w 0.45 and 0.68)	Approximately linear decline at 21°C over 24 weeks on all 4 cereal types. Linear decline at a_w 0.30, nonlinear at a_w 0.45 and 0.68 over 12 months.	Lin and Beuchat, 2007a
Cronobacter sakazakii	Infant formula (milk-based, soy-based)	4, 21, 30°C	12 months	0.56 to 0.83 (a_w 0.49); 0.25 (a_w 0.26).	At 21°C significantly faster decline at aw 0.52–0.83: −5 to −10 Log CFU/mo. At 4°C: −0.04 to −0.13. At 30°C : −3.5 to −5 Log CFU in first month, and −5 Log in 3 months (a_w 0.26) or −5 Log in 12 months (a_w 0.44–0.49)	Gurlter et al., 2007
Cronobacter sakazakii (Enterobacter sakazakii)	Infant formula	20°C	9 days	0.18	−0.54 Log in 9 days (linear)	Jin et al., 2008
Cronobacter sakazakii	Infant formula ingredients	Ambient temperature. Multiple strains.	338 days	0.38 to 1.07 (lecithin); 0.27 to 0.98 (insulin)	Environmental strains more persistent than clinical ones	Walsh et al., 2011
E. coli	Wheat grains	17°C, RH: 72%, 2 wheat varieties	63 days	−1.7 to −2.2	Linear decline	Schwarz et al., 2013
E. coli ec0001	Infant formula	22°C	16 days	0.32 to 0.48 (NL)	Rate estimate based on $D = 64$ days (Weibull function, best fit) and $D = 119$ (Log-linear)	Kandhai et al., 2010

E. coli O157:H7	Almond, pistachio	24, 4, −19°C, inoculated then dried for 72 h.	12 months	0.60 almonds, 0.35 pistachios (NL)	Nonlinear, with tailing near detection limit at day 80–140 (almonds) and tailing above detection limit at day 265 (pistachios). 1–2 Log CFU reduction during air drying. At 4°C −0.09 Log CFU on almonds, no decline on pistachios. No decline at −19°C	Kimber et al., 2012
E. coli O157:H7	Walnut kernels	23, 4, −20°C. Inoculated, dried for 72 hours	1 year	0.21 to 0.86 (NL)	Initial concentrations 2–8 Log CFU. Best fit with Baranyi model. Experiment yielding an overall rate of −0.21 Log CFU/month better curve fit than that yielding −0.86	Blessington et al., 2012
Enterococcus faecalis	Poultry feed, cattle feed	28°C, 65% humidity	7 days	12.2, 12.5	Best fit with exponential decay function	Channaiah et al., 2010
Escherichia coli STEC (35 strains)	Chocolates, roasted peanuts, potato chips, dried squid chips, dried apples, sun-dried squids, dried sour plum pickles	4, 25, 35°C. On paper disks and on dry food. Dried for 24 hours before long-term storage	22–24 months at 4C; 35 to 70 days at 4,25,35 C	NA	3–4 Log CFU reduction after drying for 24 hours at 35°C. 89% STEC samples positive after 24 hours, but only 40% of nonpathogenic *E. coli*. At 4°C: detected at day 70. At 25°C: detected at day 35, not at 70. At 35°C: detected at day 15, not at 35	Hiramatsu et al., 2005
Listeria monocytogenes	Almond, pistachio	24, 4, −19°C, inoculated then dried for 72 hours.	12 months	0.71 (almonds), 0.86 (pistachios)	At 24°C marked nonlinear tailing from day 80 for both almonds and pistachios. Best fit with Baranyi curve. No decline at −19°C or 4°C	Kimber et al., 2012
Listeria monocytogenes	Walnut kernels	23, 4, −20°C. Inoculated, dried for 72 hours, stored.	1 year	1.1 to 1.3	At 23°C, best fitted with a Baranyi or linear function	Blessington et al., 2012
Salmonella spp.	Infant formula	22°C	16 days	0.35 to 0.65 (NL)	Rate estimate based on $D = 47$ days (Weibull function, best fit) and $D = 86$ (Log-linear)	Kandhai et al., 2010
Salmonella spp.	Dry milk products	Ambient temperature	1 year	0.18 to 0.50	Naturally contaminated. Still detected after 1 year	Ray et al., 1971
Salmonella Derby, *S.* Anatum, *S.* Infantis, *S.* Typhimurium, *S.* Brandenburg	Pork sausage (chorizo)	18–22°C	35 days	3.89	From 900 to <0.03 MPN/g in 35 days	Escartin et al., 1999
Salmonella Eastbourne, *Salmonella* Typhimurium	Chocolate	20°C	9 months	0.45 to 6.93 (NL)	Well fit by Weibull function	Tamminga et al., 1976
Salmonella enterica	Wheat grains	17°C, RH: 72%, 2 wheat varieties	63 days	31.2 to 1.4	Approximately linear. *D*-value: 10–12 days.	Schwarz et al., 2013

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Table 3 Survival or decline kinetics of *Salmonella* on selected biological substrates *(Continued)*

Microrganism	Food matrix	Experimental conditions	Duration of test	Decline rate (Log CFU/month) at room temperature [a]	Result highlights	References
Salmonella enterica (18 strains)	Chocolate, roasted peanuts, potato chips, dried squid chips, dried apples, sun-dried squids, dried sour plum pickles	4, 25, 35°C. On paper disks and on dry food.	22–24 months at 4°C; for 35 to 70 days at 4,25,35°C	NA	Dried at 35°C for 24 hours before storage. −3 to 4 Log CFU after drying at 35°C. No decline at 4°C on paper disks. Stored at 25°C: detectable at 35 days, not at 70 days. Stored at 35°C: detectable at 15 days, not at 35	Hiramatsu et al., 2005
Salmonella enterica (5 serotypes)	Peanut butter and spread	5, 21°C, a_w: 0.20–0.33	24 weeks	0.74, 0.81	At 5°C: −0.52, −0.78 Log CFU/month	Burnett et al., 2000
Salmonella Enteriditis, *S.* Anatum, *S.* Oranienburg, *S.* Tennessee	Black pepper	25°C	365 days	0.21 (NL)	Initial a_w 0.6 declining to < 0.2. Initial 3–4 Log decline in 56 days (−1.5 Log CFU/month at 25°C, −3.0 at 35°C), then stable for > 8 months at both 25°C and 35°C	Keller et al., 2013
Salmonella Enteriditis, *S.* Anatum, *S.* Oranienburg, *S.* Tennessee	Walnut kernels	−20, 4, 23°C	1 year	0.05 to 0.86 (NL)	Declined at 23°C; no decline at −20 or 4°C. At 23°C, best fitted with a Baranyi or linear function, when decline observed	Blessington et al., 2012
Salmonella Enteriditis, *S.* Anatum, *S.* Oranienburg, *S.* Tennessee, *S.* Montevideo	Almond, pistachio	24°C	12 months	0.2 (almond), 0.15 (pistachio)	a_w < 0.5. 2–2.5 Log CFU linear reduction over 350 days at 24°C, stable persistence at 4 and −20°C	Kimber et al., 2012
Salmonella Enteritidis	Almonds	23°C	171, 550 days	0.18 to 0.30	Linear decline. At 23°C over 161 days, range of 0.22–0.29 Log CFU/month for initial levels 1–8 CFU/almond; at 23°C over 550 days: 0.22 log CFU/month; at 4°C over 171 days and initial 7.1 CFU/almond: no reduction; at −20°C or 4°C over 550 days and initial 8.0 CFU/almond: no reduction; at 35°C over 171 days: 1.1 log CFU/almond from day 0 to 59, no further reduction from day 59 to 171	Uesugi et al., 2006
Salmonella Enteritidis	Cereal flakes	Ambient temperature	28 days	12.16	6 Log CFU in 15 days. Not detected by plate count at day 15, but still detectable by enrichment at day 28	Ui et al., 2009
Salmonella Enteritidis	Halva	6°C, 18–20°C	8 months	0.10, 0.14	Air-sealed or vacuum packed. At 6°C: −0.04 to 0.10 Log CFU/month	Kotzekidou, 1998

Salmonella Enteritidis PT30, *S.* Oranienburg	Cocoa beans	21°C	21–28 days	1.63 to 5.43	Slower decline for inoculum grown on lawn vs. broth. Marked difference across serovars	Komitoupoulou and Peñaloza 2009
Salmonella Enteritidis PT30, *S.* Oranienburg	Crushed cocoa shells	21°C	21–28 days	0 to 3.62	Slower decline for inoculum grown on lawn vs. broth. Marked difference across serovars	Komitoupoulou and Peñaloza 2009
Salmonella Enteritidis PT30, *S.* Oranienburg	Crushed hazelnut shells	21°C	21–28 days	0.72 to 2.90	Slower decline for inoculum grown on lawn vs. broth. Marked difference across serovars	Komitoupoulou and Peñaloza, 2009
Salmonella Enteritidis PT30, *S.* Oranienburg	Whole almonds	21°C	28 days	NA	Little or no visible decline	Komitoupoulou and Peñaloza 2009
Salmonella Enteritidis, *S.* Typhimurium, *S.* Mbandaka, *S.* Seftenberg	Poultry feed	Ambient temperature	34 months	0.53 (NL)	−5 Log in 10 months, but persisted at <43 MPN/100g for at least 26 months	Davies et al., 1996
Salmonella Enteritidis, *S.* Weltevreden, *S.* Senftenberg	Black and red pepper	Ambient temperature	28 days	6.5 to 26.1 (red pepper), 2.2 to 6.5 (black pepper)	In order of fastest decline: *S.* Enteriditis, *S.* Seftenberg, *S.* Weltevreden	Urabe et al., 2008
Salmonella Montevideo	Wheat	25°C, RH: 7–22%	28 weeks	0.31	At 7,11, 22% RH: −2 Log in 28 wks; at 33,43,53, 62% RH: −4.5 Log; at 75,84,92, 98% RH: > −6 Log decline, not detected at 28 weeks	Crumrine and Foltz, 1969
Salmonella Montevideo, *S.* Heidelberg	Cocoa powder	25°C, a_w 0.43, 0.52, 0.75	14 weeks	3.7 (a_w 0.43), 8.7 (a_w 0.52), 26.1 (a_w 0.75) (NL)	Fastest decline in first week. Nonlinear at all a_w	Juven et al., 1984
Salmonella Montevideo, *S.* Heidelberg	Dry milk	25°C, a_w 0.43, 0.52, 0.75	14 weeks	1.9 (all a_w)	Approximately linear	Juven et al., 1984
Salmonella Montevideo, *S.* Heidelberg	Meat and bone meal	25°C, a_w 0.43, 0.52, 0.75	14 weeks	1.9 (a_w 0.43, 0.52), 3.7 (a_w 0.75) (NL)	Nonlinear, fastest decline in first 3 weeks	Juven et al., 1984
Salmonella Montevideo, *S.* Heidelberg	Poultry feed	25°C, a_w 0.43, 0.52, 0.75	14 weeks	1.9 (NL)	At a_w 0.75 fast 5-LogCFU decline in first three weeks. More gradual decline at a_w 0.43 and 0.52	Juven et al., 1984
Salmonella Typhi	Infant formula	Ambient temperature	12 weeks	0.61 to 1.05	Higher decline in ambient air than nitrogen atmosphere	Day et al., 2011
Salmonella Typhi	Milk chocolate	20°C	19 months	0.11 to 0.38	Slower decline for *S.* Eastbourne (2 Log CFU in one month) than *S.* Typhimurium (~5 Log CFU in 13 mo.)	Tamminga et al., 1977
Salmonella Typhimurium	Egg powder	13°C, 37°C, a_w: 0.29–0.37; 0.51–0.61	8 weeks	1.40 to 2.30 (NL)	At a_w 0.3: overall −3 Log CFU/8 weeks at 13°C; −5 Log CFU/3 weeks at 37°C. At a_w 0.5–0.6: −2.5 Log CFU/8 weeks at 13°C; −5 Log CFU/8 weeks at 37°C	Jung et al., 1999

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Table 3 Survival or decline kinetics of *Salmonella* on selected biological substrates *(Continued)*

Microrganism	Food matrix	Experimental conditions	Duration of test	Decline rate (Log CFU/month) at room temperature [a]	Result highlights	References
Salmonella Typhimurium	Infant formula	20°C	9 days	0.18	−1.6 Log in 9 days (approximately linear)	Jin et al., 2008
Shigella (5 strains)	Chocolates, roasted peanuts, potato chips, dried squid chips, dried apples, sun-dried squids, dried sour plum pickles	4, 25, 35°C. On paper disks and on dry food. Dried for 24 hours before long-term storage.	22–24 months at 4C; for 35 to 70 days at 4,25,35° C	NA	No survival after 24 hours drying at 35°C. Stored at 4°C (after initial drying): detected at 70 days. At 25°C: detected at 35 days, not at 70. At 35°C: detected at 15 days, not at 35°C	Hiramatsu et al., 2005
Shigella dysenteriae	Infant formula	Ambient temperature. Ambient air or nitrogen atmosphere	12 weeks	0.15 to 0.29	Higher decline in ambient air than nitrogen atmosphere	Day et al., 2011
S. aureus	Cereal flakes (rice, wheat, corn)	Ambient temperature	28 days	7.1	3–3.5 Log CFU reduction in 15 days. 3–4 Log reduction by day 21. Nondetected by enrichment at day 28	Ui et al., 2009
S. aureus	Infant formula	22°C	16 days	0.18 to 0.26 (NL)	Rate estimate based on $D = 169$ days (Weibull function, best fit) and $D = 281$ (Log-linear)	Kandhai et al., 2010

[a]Approximate rates based on the overall reduction observed over the duration of the test. Nonlinearity of the decline function noted in "Result highlights."

temperature, and pH. For example, *Salmonella* and *S. aureus* can persist on cereal flakes (corn, rice, wheat) for more than three weeks, and grow rapidly when rewetted with milk (Ui et al., 2009). Among the many examples of bacterial growth on wet foods, *Salmonella* can also multiply on reconstituted infant formula (Deng et al., 1998), wet inshell pecans, and orchard soil (Beuchat and Mann, 2010). *Staphylococcus* can grow on ready-to-eat meat (Borneman et al., 2009), and *Listeria* on thawed salmon (Kang et al., 2012) and cooked meat (Legan et al., 2004).

PATHOGEN CONTROL DURING PET FOOD PRODUCTION

During the production of dry pet food, several measures are implemented to minimize the risk of contamination in the finished product (FEDIAF, 2010). At least one treatment step in the process is specifically designed to substantially reduce or eliminate pathogenic microrganisms. For kibble products, this step commonly involves extrusion at high temperatures above 90°C, in practice commonly above 100°C, at pressures of 34–37 atm (FEDIAF, 2010; Zicker 2008). In plants with HACCP (Hazard Analysis and Critical Control Points) programs in place, as recommended in the Codex Alimentarius best practices (Codex Alimentarius, 2003), the extrusion step is often the main critical control point for the control of microbial risk. In addition, the work flow, and plant layout are designed so that the product after the decontamination treatment step does not come in contact with equipment, ingredients, or product before the treatment step. Facility surfaces are routinely swabbed and tested for the presence of *Salmonella*. On a voluntary basis, pet food producers often choose to test the finished product for contaminants, in particular *Salmonella* and aflatoxins. In some cases, the product is held until the laboratory test results show no evidence of contamination. After the pathogen-abatement treatment, the product is usually dried to a low moisture level (approximately 10%) and sealed in bags or other retail container, without the addition of antimicrobials. However, recontamination of the product may occur between the treatment step and consumption, either at the plant or during household handling. While processing steps and consumer handling behavior may vary depending on the nature of the food, e.g., canned wet food or dry kibble, risk of recontamination at household exists for both types of products (Crane et al., 2010).

A variety of pathogen-abatement treatments have been developed by the food industry to minimize contamination in finished products. The degree of pathogen reduction provided by the treatments depends on target pathogen, food matrix and particle size, and treatment conditions. The range and effectiveness of treatments involving heat or steam for foods of similar nature to the ingredients used in dry pet food production are summarized in Table 4. In the production of wet canned pet food, the abatement step commonly involves cooking at high temperatures (Meeker and Hamilton, 2006). In the production of dry pet food, the main treatment step usually involves subjecting the ingredient mixture to high temperature under high-moisture conditions. Pet treat can undergo different treatments depending on the treat. Extruded treats usually undergo the same treatment as dry kibble, while treats such as pig ears and jerky are dried (possibly at higher than ambient temperature), but not treated at high temperature or by any other method intended to reduce pathogen loads.

Extrusion is one of the most common pathogen abatement steps used in the production process of dry kibble. This process involves injecting steam into a premixed and prewetted mixture of ingredients, and injecting the mixture at high pressure through a small opening, thus conferring a determined shape to the extruded product. Heat, pressure, shear forces, and expansion contribute to inactivating bacterial cells and other pathogens that may be present in the ingredients. Granulating and expanding also seems to contribute to bacterial reduction (Latala, 2000). Extrusion studies have observed a reduction of *Salmonella* Typhimurium in dry feed of at least 8 Log CFU at 83°C and 103°C (Okelo et al., 2008), a reduction of *E. coli* in dry feed of at least 6 Log CFU (Kelley and Walker, 2000), and a reduction of *Bacillus globigii* in a soy/corn mixture of 6.5 Log CFU with a final temperature between 100°C and 140°C (Likimani and Sofos, 1990). Guidelines by the American Feed Industry Association state that a short (1 sec) exposure to wet heat at 22% moisture and 77°C should guarantee complete inactivation of *Salmonella* (AFIA, 2010). Other studies yielded comparable results: reduction of *Salmonella* to nondetect levels was observed at 25–35% moisture at 93–177°C (199–351°F) for 45–60 sec during expansion extrusion (Institute of Food Technologists, 2001), while >5-Log reductions of *Enterococcus faecium* to nondetect levels were observed when >25% moisture material was processed at >75°C (Bianchini et al., 2012). In an industry validation study, extrusion at a temperature of 92°C consistently achieved a reduction in *Enterococcus faecium* concentrations in the order of 7 Log CFU (personal communication, proprietary information). Besides extrusion, a variety of wet heat methods are used in the food industry, including steaming, roasting, blanching, microwaving, and applying an external heat source to a wet product (Institute of Food Technologists, 2000). A review of the bactericidal performance of different extrusion methods, as well as other treatments involving heating a wet product, is presented in Table 4.

Pet food ingredients, before being delivered to a pet food production facility, could also undergo treatments intended to reduce pathogen contamination, besides being produced "under sanitary conditions.". Animal products, usually by-products of the meat industry, commonly undergo a rendering process aimed to separate fats from residual meat, organs, and bones. During rendering, discarded or by-product animal parts are ground, heated to temperatures of 121–138°C (250–280°F) that allow the separation of fats. Fats are filtered, and the mixture of meat and bones is dried into what is defined as "meal."

Table 4 Performance of heat treatments for the abatement of bacteria in various food matrices

Microrganism	Treatment	Temperature	Food matrix	Treatment duration	Bacterial count reduction	References
Bacillus globigii spores	Extrusion	zone 1: 80°C; zone 2: 100–140°C	Corn/soybean mixture (70/30%)	11.2 and 25.5 sec	At 11.2 sec residence time: 2.74 to 6.52 on TSA; 25.5 sec: 1.32 to 5.91 on TSA	Likimani and Sofos, 1990
Bacillus stearothermophilus	Extrusion	165°C, 182°C	Starch-protein-sucrose mix	Twin screw extruder at 78 rpm (40 kg/hr)	-3×10^5 CFU at 165C, -10^7 CFU at 182C. *D*-value at 121C: 3.9 minutes, *z*-value 18°C	Bouveresse et al., 1982
Coliforms	Extrusion	70, 80, 83, 90°C	Chicken feeds (pellets, ø 3.5 mm)	10 seconds	Coliform: prevalence from 0/5 to 0/5 (only EC at 70°C was detectable in 3/5 samples post-treatment)	Furuta et al., 1980
Enterococcus faecium	Extrusion	67.5 to 85°C	Carbohydrate-protein mix (moisture 25–31%); screw ø 1.9 mm	NA	Maximum reduction of 5 Log CFU at 81.1°C and 28.1% moisture. Good fit with response surface model as function of temperature and moisture	Bianchini et al., 2012
E. coli	Extrusion	110 to 135°C	Dry feed (swine waste, corn, soybean hulls); single screw ø 13.02 cm, die ø 0.79 cm	NA	From 5×10^6 Log CFU to nondetect	Kelley and Walker, 2000
E. coli O157:H7 (four strains)	Heat (water bath)	55, 57.5, 60°C	Ground beef (75% lean), 3-g packages, flattened	NA	*D*-values: 20.9, 7.8, 3.4 minutes at 55, 57.5, 60°C	Juneja, 2003
E. coli O157:H7	Heat	55 to 66°C	Ground beef (60-g portions)	NA	High fat (19.1%): *D*-value from 0.18 minutes at 63°C to 22.47 at 55°C. Low fat (4.8%): *D*-value from 0.16 minutes at 63°C to 20.08 55°C	Smith et al., 2001
E. coli O157:H7	Heat (water bath)	43.3, 46, 49°C	Lebanon bologna mix (50 cc in submerged tubes)	0, 10, 20 minutes	Reductions at 20 minutes: 2.5 Log CFU (43.3°C), 4 Log CFU (46°C), 3.5 Log CFU (at 49°C)	Ellajosyula et al., 1998
E. coli O157:H7	Heat (water bath)	55 to 65°C	Ground beef (90% lean) or chicken, 3-g samples, flattened to 1–2 mm thick	NA	*D*-values in beef: 21.13, 4.95, 3.17, 0.93 and .0.39 minutes at 55, 57.5, 60, 62.5, and 65°C (*z* values 4.9–9.2). Lower *D*-values in chicken: 11.8, 3.8, 1.6, 0.82, 0.36 minutes (*z* values: 5.8–6.8)	Juneja et al., 1997
E. coli O157:H7	Steam	100°C	Rice cakes (5 flour types), 50 g	30 minutes	From 6.5–6.7 to <0.78 Log CFU/g	Lee et al., 2006
Salmonella Typhimurium	Steam	100°C	Rice cakes (5 flour types), 50 g	30 minutes	From 6.6 to <0.78 Log CFU/g	Lee et al., 2006
Listeria monocytogenes	Steam	100°C	Rice cakes (5 flour types), 50 g	30 minutes	From 6.6–6.9 to <0.78 Log CFU/g	Lee et al., 2006
S. aureus	Steam	100°C	Rice cakes (5 flour types), 50 g	30 minutes	From 6.9–7.8 to <0.78 Log CFU/g	Lee et al., 2006
Bacillus cereus	Steam	100°C	Rice cakes (5 flour types), 50 g	30 minutes	1–2 Log CFU/g reduction	Lee et al., 2006

Fecal coliforms, Enterococci	Dry extrusion	110 to 135°C	Animal feed (food waste, corn, soybean hulls); single screw ø 13.02 cm, die ø 0.79 cm	NA	Fecal coliforms: from 4.4×10^3 to nondetect. Enterococci: from 1.9×10^3 to nondetect	Kelley and Walker, 1999
Listeria innocua	Heat (hot water cooker)	88°C	Chicken breast strips (13-mm wide, in 454-g and 227-g packages)	0–40 minutes	For a 7-Log reduction, 454-g packages needed 34 minutes; 227-g packages needed 20 minutes	Murphy and Berrang, 2002
Listeria innocua	Heat (water bath)	67.5°C, 70°C	Ground chicken breast (30, 15, 8.5 g)	0–2.5 minutes	0.85–1.04 Log CFU reduction. *D*-value: 0.39 (67.5°C), 0.13 (70°C)	Murphy et al., 1999
Listeria innocua	Steam	88°C	Chicken breast strips (454 g, vacuum-sealed pouches)	26 to 40 minutes in continuous process, 33 to 41min in batch process	Continuous process: *L. innocua* > -7 Log CFU in 34 minutes; batch process: > -7 Log CFU reduction in 42 minutes	Murphy et al., 2001
Listeria monocytogenes	Heat (water bath)	55, 57.5, 60°C	Ground beef (75% lean), 3-g packages, flattened	NA	*D*-values: 27.7, 9.6, 4.2 minutes at 55, 57.5, 60°C.,	Juneja, 2003
Listeria monocytogenes	Heat	50, 60, 70°C	Shrimp (1×1 cm carapace pieces, $1 \times 1 \times 0.5$ cm meat pieces)	50°C: 0–30 minutes; 60°C: 0–10 minutes; 70°C: 0–4 sec	*D*-values (minutes) for *L. monocytogenes* Scott A: 18.50–21.69 at 50°C; 0.98–1.97 at 60°C; 0.53 to 0.75 at 70°C; *L. monocytogenes* V7: 19.94–21.56 at 50°C; 1.63–2.50 at 60°C; 0.45–0.89 at 50°C. Attached and colonized cells more heat-resistant than planktonic	Wan Norhana et al., 2010
Microbacterium lacticum	Extrusion, rheometer	Extrusion: 50 to 73°C; rheometer: 60 to 75°C	Concentrated gelatin	Extrusion:49 to 58 s; rheometer: 4 minutes	Extrusion: 0.3 to 5.3 Log CFU reduction depending on maximum temperature and wall shear stress. Rheometer: 1.4 Log CFU reduction at 75°C and 2.8 kPa shear stress	Bulut et al., 1999
Salmonella	Heat	58 to 65°C	Ground chicken and turkey (1–12% fat, 5-g samples, flattened to 1–2 mm thick)	NA	*D*-values: 7.4–9.0 at 58°C; 0.41–0.59 at 65°C. Lag time up to 6 minutes	Juneja et al., 2001
Salmonella	Steam conditioning and pelleting	57°C; 72 to 76°C; 83 to 86°C	Poultry feed (extruder die ø 6.8 cm)	Until temperature reached	Broiler-starter feed: at low steam pressure (100 lb/hr) prevalence from 1/4 to 0/4; at medium pressure (200 lb/hr) from 3/4 to 0/4; at high pressure (300 lb/hr) from 1/4 to 0/4; layer breeder feed: low from 4/4 to 0/4; medium from 3/4 to 0/4; high from 0/4 to 0/4	Cox et al., 1986
Salmonella (8 strains)	Heat	50, 54, 58, 62, 66°C	Ground turkey thigh (5.1% fat, pH 6.4), turkey breast (0.5% fat, pH 6.0), pork (17% fat, pH 6.4). 1-g samples, flattened to 1 mm thick	NA	Nonlinear inactivation kinetics. Weibull parameters at 66°C: $b = 6.7$, $n = 0.43$ (turkey breast), $b = 12.7$, $n = 0.65$ (pork shoulder), $b = 22.5$, $n = 1.0$ (turkey thigh)	Takhar et al., 2009

(Continued on next page)

Table 4 Performance of heat treatments for the abatement of bacteria in various food matrices *(Continued)*

Microrganism	Treatment	Temperature	Food matrix	Treatment duration	Bacterial count reduction	References
Salmonella (8 serotypes)	Heat (water bath)	55, 57.5, 60°C	Ground beef (75% lean), 3-g packages, flattened	NA	*D*-values: 19.3, 9.3, 4.7 minutes at 55, 57.5, 60°C	Juneja, 2003
Salmonella (multi-drug resistant and non)	Heat (grill griddle)	55, 60, 65, 70°C	Ground beef (25% fat, 68.5 g patties, 1-cm thick, ø 9 cm)	55°C for 0–1,080 s; at 60°C for 0–60 s; at 65°C for 15–45 s; and at 70°C for 0–6 s	MDR strains: *D*-value from 136 to 287 sec at 55°C; 9 to 23 sec at 60°C; 4 to 6 sec at 65°C; 0.7 to 3 sec at 70°C; Non-MDR: *D*-value from 204 to 784 under 55°C; 13 to 148.1sec at 60 C; 5.0 to 20.2 at 65°C; 0.7 to 8.4 at 70°C	Stopforth et al., 2008
Salmonella Enteritidis PT 30, *Salmonella* Senftenberg 775W	Hot water immersion	60, 70, 80, 88°C	Almond kernels (40 g in wire-mesh baskets)	Up to 12 minutes	*S.* Enteritidis *D*-values: 2.6, 1.2, 0.75 and 0.39 minutes at 60, 70, 80 and 88°C. *S.* Senftenberg: 0.37 minutes at 88°C	Harris et al., 2012
Salmonella Enteritidis	Heat (water bath)	71.1 to 82.2°C	Poultry feed mash (5.8% fat, sample weight NA)	0–160 sec	Decline rate as % Log CFU at 15% moisture: −1.26 at 71. 1°C; −1.59 at 76.7°C; −1.91 at 82.2°C	Himathongkham et al., 1996
Salmonella Senftenberg, *Salmonella* Typhimurium DT 104	Heat (water bath)	55 to 66°C	Ground beef (60-g portions)	NA	*S.* Senftenberg: high fat (19.1%) *D*-values 0.16 minutes at 64°C to 21.98 at 55°C; low fat (4.8%). *S.* Typhimurium: *D*-values from 0.07 minutes at 64°C to 10.55 minutes at 55°C. : high fat *D*-value 0.2–22 minutes; low fat 0.1–10.5 minutes	Smith et al., 2001
Salmonella Senftenberg	Hot water cooker	88°C	Fully cooked chicken breast strips (13-mm wide, in 454-g and 227-g packages)	0–40 minutes	7 Log CFU reduction in 34 minutes (454-g packages), 20 minutes (227-g packages)	Murphy and Berrang, 2002
Salmonella Senftenberg	Steam pasteurization	88°C	Fully cooked chicken breast strips (454 g, vacuum-sealed pouches)	26–40 minutes in continuous process, 33–41 in batch process	Continuous processes: *Salmonella* Senftenberg >7 Log CFU reduction in 32 minutes. Batch process: >7 log reduction in 42 minutes	Murphy et al., 2001
Salmonella spp.	Heat (water bath)	67.5 and 70°C	Ground chicken breast (30, 15, 8.5 g)	0–2.5 minutes	Range: 1.1 to >6.3 Log CFU reduction depending on strain. *D*-value: 0.29 (67.5°C), 0.18 (70°C). Size- and shape-dependent	Murphy et al., 1999
Salmonella Typhimurium	Extrusion (single screw)	83°C	Surrogate feed matrix	7 sec	*S.* Typhimurium from 5×10^8 to nondetect, at 28.5% moisture	Okelo et al., 2006
Salmonella Typhimurium	Extrusion (single screw)	77 to 110°C	Dry feed (60% maize meal, 30% soy meal, 10% animal protein blend)	3–11 sec	5.6×10^8 to nondetect at 83°C; from 4.4–6.1×10^8 to nondetect at 103°C; moisture 24.5–34.5%, dry feed inoculums	Okelo et al., 2008
Salmonella Senftenberg, *S.* Typhimurium	Heat	50, 60, 70°C	Shrimp (1 × 1 cm carapace pieces, 1 × 1 × 0.5 cm meat pieces	50°C: 0–30 minutes; 60°C: 0–10 minutes; 70°C: 0–4 sec	*S.* Senftenberg 1734b: 18.4–20.8 at 50°C; 1.2–2.0 at 60°C; 0.4–0.6 at 70°C; *S.* Typhimurium ATCC 14028: 16.8–18.0 at 50°C; 0.8 to 1.7 at 60°C; 0.2–0.4 at 70°C. Attached and colonized cells more heat-resistant than planktonic	Wan Norhana et al., 2010

Both are sold as ingredients to other products (Meeker, 2006). Animal by-products received by 17 rendering plants in the United States were found to be contaminated by *Salmonella* at a prevalence of 84%, *Campylobacter* species at 30%, *Clostridium prefringens* at 71%, and *Listeria* species at 76%, with an 8% positive for *Listeria monocytogenes*. None of the samples of postrendering products was positive (Troutt et al., 2001). However, a survey has found approximately 25% of the tested samples positive for *Salmonella*, at concentrations ranging from 0.03 to 1,100 MPN/g (mean 16.3 MPN/g) (Franco, 2005). Other studies have detected *Salmonella* in 14% (Hofacre et al., 2001) and 8.7% (Kinley et al., 2010) of meat and bone meal samples. However, the *D*-values (time required to cause a 1-Log reduction in the levels of a specific pathogen, under a specific treatment process) for the *Salmonella* isolates detected in these studies imply that the rendering treatment should have deactivated the bacteria to nondetect levels, suggesting that crosscontamination after the heating step may be involved (Kinley et al., 2010).

The susceptibility of bacteria to heat depends on many factors such as strain, medium, moisture, pH, growth stage of the culture, and previous exposure to cell stress (O'Bryan et al., 2006). *Salmonella* strains, for instance, have a wide range of heat resistance, with *Salmonella* Seftenberg being the most resistant strain, and liquid eggs the most protective medium observed (Doyle and Mazzotta, 2000). The effectiveness of heat-based treatments can be impaired by increased heat resistance in the bacterial population. Such resistance could be caused by previous exposure to other stressors, such as desiccation or starvation. For example, *Salmonella* that survived a period of desiccation was observed to be less vulnerable to heat (Podolak et al., 2010; Spector and Kenyon, 2012), which is particularly important for low-moisture food processing.

While contaminated ingredients can directly be the cause of final product contamination, pathogens can also contaminate the food during processing, for example through contaminated equipment, dust, infected workers, or animals intruding into the facility could introduce pathogens into the unfinished product (Reij et al., 2004; Jones, 2011; Carrasco et al., 2012). Cross-contamination could happen during ingredients transport and receiving. For example, trucks transporting animal feed were found contaminated with *Salmonella* (Fedorka-Cray et al., 1997). In a shared receiving area, a high-risk ingredient could cross-contaminate a low-risk one. This is of particular concern if not all ingredients undergo the same pathogen abatement steps, which is the case in dry pet food. In the production facility, cross-contamination could also occur when pathogens present in a batch of contaminated ingredient or unfinished product spread to other product batches, for example through equipment surfaces, containers, or conveyor pipes. Several food outbreaks have been attributed to cross-contamination during production, including dry foods (Lyytikainen et al., 2000; Rowe et al., 1987; Lehmacher et al., 1995). Dust has been recognized as a major hazard for *Salmonella* cross-contamination in animal feed mills and other food production facilities. Dust, product particles, and debris accumulating on floors, while not directly in contact with the product, can be easily transported to sensitive plant locations by air flow, workers, and pests (Morita et al., 2006). Little information is available on pathogen contamination on surfaces and equipment in pet food production plants. While at very different conditions than pet food plants, pathogens have been detected on surfaces and dust in plants that process meat and other animal products, highlighting the potential role of noncontact surfaces in cross-contamination or as reservoirs. Pathogens observed in meat plants include *Salmonella* (Thorberg and Engvall, 2001; Fries, 2002; Schmidt et al., 2012), *Listeria* (Lawrence and Gilmour, 1995; Miettinen et al., 1999; Norton et al., 2001; Rhoades et al., 2009), and *Staphylococcus* (Isigidi et al., 1992; Granic et al., 2009). Quantitative risk models of cross-contamination processes in factories have started to be developed only recently (Yang et al., 2002). Although guidelines for cross-contamination control have been published for several food sectors (AFIA, 2010; USDA, 2010), this remains a major problem requiring attention.

PET FEEDING AND PET FOOD HANDLING IN THE HOUSEHOLD

Dog and cat food diets vary depending on breed and size of the animal, age, exercise level, and health conditions. Veterinary guidelines exist to determine nutritional requirements (Bartges et al., 2012; Dzanis, 1993; NRC, 2006; Baldwin et al., 2010), although the topic is still being debated (Phillips, 1998; Bermingham et al., 2010). Commercial pet food that can legally claim to provide "complete and balanced nutrition" is required to have feeding instructions printed on the food bag (AAFCO. Association of American Feed Control Officials, 2008). Also, several studies have evaluated the suitability of commercially produced food to meet pets' nutritional and health requisites (Streiff et al., 2002; Morris and Rogers, 1994). Specific diets are prescribed, for example, for aging pets (Laflamme and Library, 2012), or to manage the pet's weight (Laflamme et al., 1997, ; Heuberger and Wakshlag, 2011; Michel and Scherk, 2012). Therapeutic diets can be prescribed in case of acute or chronic diseases, although in a recent survey only 2.5% of dogs and cats were fed such diets (Freeman et al., 2006). Pet feeding habits are also dependent on the owner's own dietary culture and values (Joshi et al., 2007; Suarez et al., 2012). The owner's choice of diet for their pets, specifically the feeding of raw meat instead of cooked food, was observed to be based on affective, ideological, and empowerment factors, but not on risk of pathogen exposure (Michel, 2006; Lenz et al., 2009).

Dog feeding habits differ in different nations and cultures (Table 5). In a survey conducted in Sweden on one- to three-year-old dogs, 95% of dogs were fed dry food, and 6% of them were fed only dry food (Sallander, 2010). Dry dog food represented on average 69% of the dry matter intake, and a

linear relationship was observed between dry food intake and body weight, with intake ranging approximately from 100 to 500 g of dry matter per kg of body weight (the dry matter content of the food was 92.8%). One tenth of dogs consumed canned food, and only few dogs consumed frozen food. No relationship was observed between intake of canned or frozen food and body weight. The majority of dogs (79%) were also fed human—table—food, and 14% were fed homemade meals, but in small amounts (on average 6 g per kg of body weight per day). 62% of dogs were given commercial treats, in small amounts (average 4 g/day). On average, dogs were fed only three different commercial foods in their lifespan. Overall, in several countries the majority of dogs consume dry food (Slater et al., 1992, 1995; Thomson et al., 2008; Laflamme et al., 2008). In the majority of dogs fed dry food, this food make up for the substantial majority of the entire diet. In a study, dogs never fed table food obtained more than 90% of their daily calorie intake from commercial food, and the remainder from treats, while dogs fed table food (59% of all dogs) obtained 70–75% of their calories from commercial food, and 21% from table food (Heuberger and Wakshlag, 2011).

Feeding frequency and amounts vary by culture and place, as well as pet characteristics. In the Swedish study mentioned above (Sallander et al., 2010), 83% of the dogs were fed a restricted diet of one or more meals a day, while 13% were fed *ad libitum*, almost always dry food. Most dogs (66%) were fed twice a day, and a minority once or three times a day (9% and 8%, respectively, Table 5). When feeding a restricted diet, the amount of food per meal provided to the pet varies by pet size and owner's attitude. Adult cats require a mean maintenance intake of 56 kcal/Kg of body weight per day (range: 29–85 kcal/kg·day, depending on their activity level and size, which corresponds to a mean of 222 kcal/day, and range of 122–401 kcal/day (Bermingham et al., 2010). These values correspond to approximately half a cup (50 g) of a 4,500 kcal/kg dry food. Intake needs are higher for growing kittens and pregnant and lactating females. Cats fed *ad libitum* were observed to intake a median of 338 (range: 183–449) KJ/kg of body weight per day, spread over a median of 9 (range: 5–12) small meals over 24 hours (Martin and Rand, 1999). Other studies observed cats fed *ad libitum* eating 12–20 times in a 24-hour cycle (Kane et al., 1987; Peachey and Harper, 2002). For dogs, energy intake varies significantly with breed and size. In a study on kennel dogs, the average intake from dry food was 3,400 KJ/day (813 kcal/day) for beagles, 5,000 KJ/day (1,200 kcal/day) for huskies, and 5,600 KJ/day (1,340 kcal/day) for labradors (Finke, 1991). Caloric requirements were observed to decrease by 20% in senior dogs, but not in senior cats (Harper, 1998). Allometric equations of 655·body weight $^{0.69}$ (kg) and 643·body weight $^{0.73}$ (kg) have been proposed for low-activity and high-activity dogs, respectively (Burger and Johnson, 1991). The caloric content of popular brands of dry dog food available in the U.S. ranges from 2997 to 3875 kcal/kg, while dry cat food ranges from 3206 to 3980 kcal/kg (Hill's Pet Nutrition, 2013). The maximum caloric density recommended by AAFCO is 3100 and 3250 kcal/kg of dry food, for dog and cat food, respectively (Hill's Pet Nutrition, 2013).

Feeding patterns differ significantly between dogs and cats. Cats are more likely to be fed twice a day or ad libitum (approximately 38% each category), while approximately 15% are fed once a day (Laflamme et al., 2008). Conversely, most dogs were fed once or twice a day, and only about 13% were fed *ad libitum* (Table 5). Also, cats were more likely to be fed canned diets than dogs (29% and 11.4%, respectively, receiving at least half of their diet from canned food) (Laflamme et al., 2008). Daily food intake by cats also differs from dogs, and depends on several factors including feeding patters, age, and health status of the pet. For example, cats fed a dry diet *ad libitum* ate 86.3 ± 18g per day (67.5 ± 25 for a second high-magnesium diet), while cats whose food was available only for one hour a day ate 52.2 ± 11.5 (54.4 ± 13.4 for the second diet) (Finco et al., 1986). Water intake was also higher when provided continuously (176–179 mL/day) vs. for only one hour a day (125–128 mL/day) (Finco et al., 1986).

Table 5 Dog feeding practices

% fed once/day	% fed twice/day	% fed ≥3 times/day	% fed *ad libitum*	% fed dry food	% fed canned food	% fed treats	References
9	66	8	13	95	10	62	Sallander et al., 2010
21	50	9	20	57	29	79	Slater et al., 1992[a]
46.5	42.3	12.2	NA	NA	NA	78.6	Campbell et al., 1986
14.5–24.8	60.6–61.5	11.1–17.7 [b]	3.5–6.3	NA	NA	93–94	Thomson et al., 2008
37	45	4	13	83[c]	13[d]	63.6[e]	Laflamme et al., 2008[f]
NA	NA	NA	NA	55[g]	10[g]	NA	Becker et al., 2012

[a]Percents varied depending on the survey tool used: telephone interviews vs. a 7-day written record. Results in the table refer to the first telephone interview.
[b]Estimated by subtraction from 100%.
[c]Percentage of dogs receiving at least 50% of their diets from commercial dry food. For 50% of dogs surveyed 100% of the diet was dry food.
[d]Percentage of dogs receiving at least 50% of their diets from commercial canned food. For only 2% of dogs surveyed 100% of the diet was canned food.
[e]Dogs receiving commercial treats at least once a week. 45.4% received table food at least once per week.
[f]Values are approximate.
[g]Percent of dogs given only dry or wet commercial diets. 34% of dogs were reported as receiving a combination of dry and wet diets.

The way pet owners handle pet food varies with cultural and individual preferences. When feeding dry kibble to a pet, the product is usually poured or scooped from the food bag or other large container to the feeding bowl. Scooping tools range from bare hands to spoons and measuring cups. The size of spoon and bowl appears to affect the serving size (Murphy et al., 2012). Significant variability in serving sizes has been observed when using a measuring cup to determine food amounts (German et al., 2011). In terms of exposure to potential pathogens in pet food, the degree of contact between hands and food has never been measured, although it can be argued that when using a scooping tool a smaller hand area gets in contact with the food. These tools are usually dedicated to handling pet food, and not human food, although they may be washed together with other dishes and kitchen utensils. Owners following a restricted diet protocol for their pets are recommended using a dedicated scoop of predetermined capacity to better control the pet's food intake (Murphy et al., 2012). Dogs and cats are usually fed in a dedicated area that can include a food bowl, a food dispenser, water, and possibly toys and treats. Even when the pet is fed only once or twice a day, it can visit the feeding area several times a day for drinking, looking for food, or playing with treats and toys.

BACTERIAL ECOLOGY IN FINISHED PET FOOD AND ON HOUSEHOLD SURFACES

While pets are directly exposed to foodborne pathogens by ingesting their food, pet owners and household members can get in contact with such pathogens through direct contact with the feed, contact with the pet, contact with household surfaces, and ingestion of cross-contaminated human food. Hence, understanding the ecology of pet food pathogens in the household is of utmost importance to correctly estimate exposure, and to identify and modify behaviors that can increase exposure by favoring pathogen persistence or growth. General guidelines for safer pet food handling have been provided by the U.S. FDA (CVM, 2007), but no quantitative risk estimate is to date available. As reviewed in Section 6 (Pathogen dynamics during pet food production), several bacterial pathogens such as *Salmonella* and *E. coli* are able to grow in the environment at high moisture conditions (water activity $a_w > 0.9$), warm temperature, and the availability of a suitable substrate (Winfield and Groisman, 2003). *E. coli* has been observed to grow in water environments even at very low carbon concentrations (Vital et al., 2008). Moist food substrates kept at room temperatures are a known critical hazard for foodborne pathogen amplification. Even when not replicating, *Salmonella* and *E. coli* are able to persist at low levels for months or even years on dry food (Podolak et al., 2010) and in environmental media such as water and soil (Winfield and Groisman, 2003). For example, *Salmonella, E. coli*, and *Listeria* spp. inoculated on dry nuts have been observed to decrease exponentially, but still be detectable after one year (Blessington et al., 2012a; Kimber et al., 2012).

Dry pet food is a low-moisture food matrix of complex composition. The extent to which pathogenic bacteria such as *Salmonella, E. coli*, and *Listeria* spp. can grow or persist in dry dog food is not currently known. While the low water activity in these products (a_w of approximately 0.5, moisture of 10%) should ensure growth inhibition during dry storage, any water getting in contact with the rich substrate of the finished pet food at room temperature would create conditions favorable to bacterial growth. This situation could occur if the food is rewetted accidentally during storage (e.g., water on the garage floor), by the pet owner before feeding to increase the food's softness, or by the pet's drinking water or saliva. Studies carried out with dry foods (water activity $a_w < 0.84$) have demonstrated that pathogenic bacteria decline over time or remain at stable population levels, depending on environmental variables, most notably temperature (Podolak et al., 2010) (Table 3). Studies on wetted or rehydrated products that are usually dry demonstrated that significant pathogen growth can occur, in particular at room temperature (Baron et al., 1999; Wu et al., 2002; Abushelaibi et al., 2003; Richards et al., 2005; Lin and Beuchat, 2007b). To-date no data is available on the growth kinetics of bacterial pathogens on rewetted pet food. Preliminary data by our group have shown significant growth in 72 hours under a range of moisture levels, and as low as 30% (Oni and Buchanan, 2013).

The location of pet food storage and pet feeding areas in the household, in relation to sites of relevant human activity such as food preparation in the kitchen or high traffic through the entry door, can have a considerable impact on the likelihood of contamination spread and exposure. Little quantitative information is available on pet food handling habits. Anecdotally, bags of dry pet food are commonly stored either in the garage, or near the pet feeding area. When purchasing food in large bags, portions of the food could be transferred to smaller containers for easier lifting and handling. Such containers may be more likely to be stored near the feeding area. When feeding *ad libitum*, a portion of the food is also stored in the feeding bowl, or in a dispenser. If food is left in the bowl, it could become rewetted by the pet's saliva or its drinking water, and wet conditions at room temperature could support pathogen growth.

Bacterial pathogens have also been observed to persist for long times on surfaces and materials commonly present in households (Table 6). Persistence time varies depending on bacterial species and strain, surface properties, and environmental conditions. For example, after an initial fast reduction during drying, *Salmonella* has been documented to survive for months or even years in dry conditions. Viable bacteria have been detected on fabric after several months of dry storage. Synthetic fabric appears to support persistence better than natural fabrics such as cotton (Neely and Maley, 2000). Metals with bacteriostatic properties, such as copper and silver, do not support bacterial persistence for as long as other metals, such as stainless steel (Wilks et al., 2005, 2006). Rubber was

Table 6 Persistence and decline of selected bacterial pathogens on household surfaces

Pathogen	Surface	Experimental conditions	Population kinetics	Fitted decline model	Observed persistence time	References
Campylobacter jejuni	Formica laminate, ceramic, steel, cotton cloth	Room temperature (27°C), RH[a] 60–62%	7–8 Log CFU decline in 250 minutes in TBS. Best survival on formica, then steel, ceramic, and cloth (in PBS)	Steel: lag time 114 minutes, *D*-value 18.4 ± 2.8 minutes. Formica: lag 163 minutes, *D* 14.8 ± 2.7 minutes. Ceramic: lag 73 minutes, *D* 21.8± 3.8 minutes. Dishcloth: lag 63 minutes, *D* 18.1 ± 2.6 minutes	Detectable at 260 minutes except on cloth (max 180 minutes)	De Cesare et al., 2003
Campylobacter jejuni	Stainless steel	Room temperature (22–25°C), RH 40–45%, 3 inoculum levels: 10^7, 10^5, 10^3 CFU/100 cm^2	10^7 CFU/100 cm^2 inoculum: 4 Log CFU reduction in first 0.5 hours, then from 3 to 0.62 Log (DL) decline from hour 0.5 to 4. Medium ($\sim 10^5$) inoculum: 3 Log reduction in first 0.5 hr, then to 0.62 (DL) by hr 1, and until hr 4	NA	4 hours	Kusumaningrum et al., 2003
Campylobacter jejuni	Soil, sponges, kitchen surfaces with "real grease soil"	23°C, with and without washing	In soil: > −5 Log CFU in 24 hours	NA	24 hours (duration of trial)	Mattick, 2003
Cronobacter sakazakii	Stainless steel	4, 25, 37°C. RH: 43%	At 4°C: 1.5–2 Log CFU reduction in 60 days. At 25°C: 4 Logs in 60 days. At 37°C: 6.5–7 Logs in 60 days	NA	> 60 days	Kim et al., 2008
E. coli	Cutting boards	New or used, wet or dry	Reduction after 2 hours on wood: −1.7 Log CFU new and dry; −0.37 new and wet; −1.1 used and dry; −0.44 used and wet. After 24 hours: nondetect. On plastic:+0.06 after 24 hours (dubious)	NA	2 hours on all wood surfaces, nondetect at 24 hours	Abrishami et al., 1994
E. coli	Paper		−5.5 Log CFU in 7 days, most reduction in first 24 hours	NA	168 hours (duration of trial)	Huebner et al., 2011
E. coli	Soiled laminate to fingers or stainless steel		On fingers: 2.3–2.5 Log CFU reduction in 24 hours. On steel: 2.3–2.5 Log CFU reduction in 2 hours, nondetect at 24 hours	NA	24 hours on fingertips, 2 hours on steel	Scott and Bloomfield, 1990
E. coli	Steel, copper, copper alloys	4, 20°C	Steel at 20°C: 5 Log CFU reduction in first 2.5 days, then little decline; at 4°C 4 Log reduction in first 7 days, then little decline	NA	Steel: > 28 days at both 20°C and 4°C. Copper: nondetectable at	Wilks et al., 2005

					90 minutes at 20°C, at 270 minutes at 4°C. Copper nickel (73% copper): nondetectable at 120 minutes at 20°C, at 360 minutes at 4°C	
E. coli	Soil, sediment, water	Review article	Half life: 1 day in water, 1.5 days in sediment, 3 days in soil	NA		Winfield and Groisman, 2003
E. coli (STEC and non-STEC)	Stainless steel	12°C, 70% RH	−2–3.5 log in first day, −4.5–5.5 log after 7 days	NA	> 19 days. STEC survives better at 24 hours, no difference at 7, 14, 19 days	Moretro et al., 2010
E. coli O157	Soil, sponges, kitchen surfaces with "real grease soil"	22°C, with and without washing	In soil: −0.53/−1.0 Log CFU in 24 hours		24 hours (duration of trial)	Mattick, 2003
E. coli O157:H7	Stainless steel	3 inoculum media: saline solution, TSB, meat purge. 20°C, 50–60% RH		Log-linear: slope 0.005–0.011 Log CFU/hours. Weibull: a = 10.2–161.93, b = 0.34–1.2, Log (So) = 4.6–5.1	8 hours, 50 hours, >200 hours in saline, TSB, and meat purge	Perez-Rodriguez et al., 2013
Enterococci	Natural and synthetic fabrics	Various isolates. Cotton, terry, blended fabrics, Polyester, Polyethylene PE	NA	NA	*E. fecalis*: 11–33 days on cotton, 21–29 terry, 19–29 blended, >90 polyester and PE. E. faecium: 22–>90 days cotton, >80–>90 terry and blended, >80–>90 polyester and PE	Neely and Maley, 2000
Enterococci	Fingers, surfaces	Dried at room temperature	On fingers: E. faecium stable for 60 minutes after 6–9% recovered at drying. *E. fecalis* stable or < 1 Log reduction after 5–10% recovered after drying	NA	On finger >60 minutes, countertops *E. fecalis* 5 days, *E. faecium* 7 days, bedrails 24 hours without decline. Phone: 4% recovered after 60 minutes	Noskin et al., 1995
Enterococcus fecalis, reovirus	PVC	Room temperature, humidity NA.	*Enterococcus*: −3.5 Log CFU in 30 days.	NA	> 30 days	Howie et al., 2008

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Table 6 Persistence and decline of selected bacterial pathogens on household surfaces *(Continued)*

Pathogen	Surface	Experimental conditions	Population kinetics	Fitted decline model	Observed persistence time	References
Listeria monocytogenes	Steel, resin, fiberglass reinforced plastic (FRP)	4, 10°C; with or w/out condensate; with or w/out pork serum substrate.	Across temperatures and substrate presence (Log CFU/coupon): −3.39 on steel; −1.85 on resin; −1.79 on FRP. Faster decline on stainless steel	NA	15 days	Allan et al., 2004
Listeria monocytogenes	Steel, rubber	6, 25°C, RH 32.5% or 75.5%	On steel: best survival at 6°C and RH 75.5%, −1.5 Log first day, then −0.5 Log from day 1 to 10. Worst survival at 25°C and RH 32.5%: −4.5 Log in 5 days, with −4 Log in first 3 days. On Buna-n rubber: at 6°C and low RH −1 Log in 10 days, at 6°C and high RH −3 Log in 10 days, at 25°C and low RH −4.5 Log in 1 day to ND, at 25°C and high RH −4.5 Log to ND in 3 days	NA	> 10 days at 6°C and 75.5% RH on both steel and rubber. 5 days at 25°C and 32.5% RH on both steel and rubber	Helke and Wong, 1994
Listeria monocytogenes	Fingers	Inoculum of 10^2–10^4 CFU in saline buffer or milk. Other test at 35 CFU/finger.		NA	60 minutes in saline buffer inoculum, 45–210 minutes in milk	Snelling et al., 1991
Listeria monocytogenes	Steel, copper-based metal alloys	Room temperature (20 ± 1°C)	On steel at RT: lag of 100 minutes, then 2 Log decline from 100 to 270 minutes, detectable at 24 hours. On copper, brass, aluminum bronze, and silicon bronze: nondetectable at 60 minutes (5 Log reduction). On copper nickel, copper nickel zinc: nondetectable at 90 minutes	NA	Steel: 24 hours. Other metals: 60–90 minutes	Wilks et al., 2006
Listeria monocytogenes	Conveyor belt	10, 25, 37°C, with or without antimicrobial	Fast decline in first 6 hours, slower for hours 6–72. Faster decline at 37C (6 Log reduction to detection limit in 48 hours)	Belt with antimicrobials: 3.8 Log CFU reduction in hours 0–6, 1.3 Log in hours 6–72. Without antimicrobials: 2.3 Log in hours 0–6, 2.0 Log in hours 6–72	72 hours (duration of trial)	Chaitiemwong et al., 2010
Salmonella	Toilet, bathroom surfaces	After salmonellosis episodes, or inoculated		NA	After episodes: up to 4 weeks in toilet, nondetected on dry surfaces. After inoculation in toilet water: up to 50 days in toilet, in air, toilet seat and lid after flushing, immediately after inoculation	Barker and Bloomfield., 2000

Salmonella	Formica laminate, ceramic, steel, cotton cloth	Room temperature (27°C), RH 60–62%	Best survival on formica, then steel, ceramic, and cloth (in PBS)	Steel: lag time 120 minutes, *D*-value 300 ±94 minutes. Formica: lag 120 minutes, *D* 895 ± 244 minutes. Ceramic: lag 80 minutes, *D* 88 ± 22 minutes. Dishcloth: lag 70 minutes, *D* 33 ± 7 minutes (average of two media)	Detectable at 1920 minutes on formica, 1440 minutes on steel, ~350 minutes on ceramic, ~220 minutes on cloth	De Cesare et al., 2003
Salmonella	Soiled laminate to fingers or stainless steel		On fingers: 2.4–2.7 Log CFU reduction in 24 hours. On steel: ~2.4–2.7 Log CFU reduction in 2 hours, nondetect at 24 hours	NA	24 hours on fingertips, 2 hours on steel	Scott and Bloomfield, 1990
Salmonella	Steel, resin, fiberglass reinforced plastic (FRP)	4, 10°C; with or w/out condensate; with or w/out pork serum substrate.	Across temperatures and substrate presence (Log CFU/coupon): −2.60 on steel; −3.10 on resin; −1.02on FRP	NA	15 days	Allan et al., 2004
Salmonella Enteriditis	Stainless steel	Room temperature (22–25°C), RH 40–45%, 3 inoculum levels: 10^7, 10^5, 10^3 CFU/100 cm^2	10^7 CFU/100 cm^2 inoculum: 1.5 Log reduction in first 5 hours, 3.5 Log from hour 5 to 100. 10^5 inoculum: 2 Log reduction in first 5 hours, then 0.5 Log from hour 5 to 50. 10^3 inoculum: from 3 to 0.62 Log CFU/100 cm^2 (detection limit) in first 5 hours	NA	100 hours at 10^7 CFU/100 cm^2 inoculum	Kusumaningrum et al., 2003
Salmonella Typhimurium	Steel, rubber	6, 25°C, 32.5% or 75.5% RH	On steel: no decline at 6°C and RH 75.5%, at 6°C and 32.5% RH −4 Log to ND in 10 days, at 25°C and RH 32.5%: −4 Log to ND n 3 days, with −3 Log in first day, at 25°C and 75.5% RH −4 Log in 10 days. On Buna-n rubber: at 6°C and low RH −2 Log in 10 days, at 6°C and high RH −4 Log to ND in 10 days, at 25°C and low RH −4 Log in 1 day to ND, at 25°C and high RH −4 Log to ND in 3 days	NA	> 10 days at 6°C and 75.5% RH on both steel and rubber. At 25°C and 32.5% RH: 3 days on steel, 1 day on rubber	Helke and Wong, 1994
Salmonella Typhimurium	Soil, sponges, kitchen surfaces with "real grease soil"	21°C, with and without washing	In soil: −0.41 Log CFU in 24 hours	NA	24 hours	Mattick, 2003

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Table 6 Persistence and decline of selected bacterial pathogens on household surfaces *(Continued)*

Pathogen	Surface	Experimental conditions	Population kinetics	Fitted decline model	Observed persistence time	References
Salmonella Typhimurium	Stainless steel, formica, PPE, wood	with or without protein (tryptic soy broth with 5% horse serum), room temperature	3.5 Log reduction on formica in 6 hours; 4 Logs on other surfaces. Most reduction (2.5–3.5 Logs) in first hour	NA	6 hours	Moore et al., 2007
Salmonella Typhimurium	Fabrics	35% or 78% humidity; 3 inoculum methods (contact, aerosol, dust mixture)		NA	After direct contact, at 35% humidity: 24 weeks persistence on wool and cottons. At 78%: 6–12 weeks. Wool blanket and wash-and-wear cotton: 2–4 weeks at either 35% or 78% humidity. After aerosol exposure, all 1–4 weeks. After dust exposure, 6–14 weeks at 35%, 1–6 weeks at 78% humidity	Wilkoff et al., 1969
S. aureus	Paper	Wet inoculum (0.25 mL), air dried	Almost −5 Log CFU in 7 days; 2.5 Log reduction during drying	NA	168 hours	Huebner et al., 2011
S. aureus	Stainless steel	Room temperature (22–25°C), RH 40–45%, inoculum levels 10^7, 10^5, 10^3 CFU/100 cm^2	High inoculum (10^7 CFU/100 cm^2): 1 Log CFU decline in first 5 hours, then 2.5–3 Log CFU reduction from hour 5 to 100. Medium inoculum (10^5): 2 Log decline in first 5 hours, 1 Log from hour 5 to 100. Low inoculum (10^3): 2 Log reduction in first 5 hours, stable but detectable until hour 100	NA	100 hours at 10^7 and 10^5 CFU/100 cm^2 inoculum, 70 hours for 10^3 CFU/100 cm^2 inoculum	Kusumaningrum et al., 2003
S. aureus	Natural and synthetic fabrics	Various isolates. Cotton, terry, blended fabrics, Polyester, Polyethylene PE	NA	NA	4–24 days on cotton and terry, 1–21 on blended. 1–40 days polyester, 40–>51 days PE	Neely and Maley, 2000
S. aureus	Stainless steel	3 inoculum media: saline solution, TSB, meat purge. 20°C, 50–60% RH>		Log-linear: slope 0.015–0.425 Log CFU/hours. Weibull: a = 0.25–0.90, b = 0.19–0.30, Log (So) = 4.93–5.15	250 hours in saline or TSB, >800 hours in meat purge	Perez-Rodriguez et al., 2013
S. aureus (MRSA and MSSA)	Plastic, laminated table top, cloth	Room temperature	−1 Log CFU in first 1–3 days, stable at low levels afterwards	NA	9–11 days	Huang et al., 2006

S. aureus	Soiled laminate to fingers or stainless steel		On fingers: 2.3 Log CFU reduction in 24 hours. On steel: 2.2–2.3 Log CFU reduction in 2 hours, from nondetect to 2.3 Log CFU in 24 hours	NA	24 hours on fingertips and on steel	Scott and Bloomfield, 1990
Yersinia enterocolitica	Steel, resin, fiberglass reinforced plastic (FRP)	4, 10°C; with or w/out condensate; with or w/out pork serum substrate.	Across temperatures and substrate presence (Log CFU/coupon): −0.91 on steel; −1.43 on resin; −0.28 on FRP. Little change over 24 hours for all conditions and surfaces	NA	15 days	Allan et al., 2004
Multiple	Hospital surfaces	Review article		NA	Gram-positive (*Enterococcus, Staphylococcus, Streptococcus*): months. Gram-negative: most survive for months (*E. coli, Shigella, Klebsiella, Pseudomonas aeruginosa*), some only for days (*Vibrio cholera, Bordetella pertussis*)	Kramer et al., 2006

[a]RH: relative humidity
[b]The ">" sign indicates that microrganisms were still quantifiable at the end of the trial period

also observed to support *Salmonella* persistence for up to 10 days under high air relative humidity (Helke and Wong, 1994).

EXPOSURE THROUGH CONTACT WITH HOUSEHOLD SURFACES

Contact with surfaces in the household differs from public spaces, particularly because of the higher frequency of contact between the same people, pets, and surfaces. While contacts in public settings are commonly one-time and of short duration, contacts in the household are repeated on an hourly and daily basis, and can therefore sustain re-infection cycles even in the absence of new exposure (Davis et al., 2012). People in the U.S. and Canada report spending more than 60% of their time at home, and children below 11 years old more than 70% (Leech et al., 2002). Patterns of contact with surfaces depend on age and lifestyle. In particular, young children are more likely to come in contact with surfaces such as floors and toys, and more likely to directly touch objects with their mouth (Black et al., 2005; Auyeung et al., 2006; Morgan et al., 2007; Beamer et al., 2008; Beamer et al., 2012; Wilson et al., 2013).

An infected pet can disseminate pathogens in the household. An animal whose mouth, fur, paws, or perianal area are contaminated by pathogens can transfer them to household surfaces it comes in contact with. In particular, saliva, wet pet toys, or wet food particles can result in longer survival and possibly growth of bacterial pathogens. Between 50% and 60% of pet dogs and cats in the U.S. and Australia live mostly indoors, while a considerable portion (approximately 33% of dogs and 28% of cats) live both outdoors and indoors (Laflamme et al., 2008). Pets increasingly have access to household areas regularly used by the owner, such as bedrooms (APPA American Pet Product Association, 2013). Humans can also contribute to spreading pathogens within the household, for example, through shoes, hands, and clothes, including to surfaces not accessible to pets. Even without direct contact with a surface, both pets and humans can shed pathogens via aerosols, or by shedding hair and skin cells (Bloomfield and Scott, 1997; Mollema et al., 2010).

Microbes can be transferred to and from fomite surfaces at levels significant for public health (Table 7). Transfer efficiency have been observed to vary with pathogen species, moisture, contact time and pressure, residence time on the surface prior to contact, surface porosity, and microbial levels (Pérez-Rodríguez et al., 2008). In general, smooth surfaces such as metal or ceramic transfer microrganisms more efficiently than dry porous surfaces such as fabric (Rusin et al., 2002). Higher contact pressure can result in a significantly higher transfer ratio (Sattar et al., 2001; Kusumaningrum et al., 2003), although the relationship was unclear in another study (Mattick, 2003). Also, moist surfaces are more efficient at transferring bacteria than dry ones (Sattar et al., 2001). Conversely, bacteria that have been allowed to dry and age on a surface were less likely to be transferred to other surfaces or food matrices upon contact (Dawson et al., 2007), although in one study a short (15 minutes) drying time resulted in lower transfer rates from steel to chicken, but not from steel to cucumber (Kusumaningrum et al., 2003). Hand skin conditions can also affects transfer. For instance, recently washed hands were significantly less effective at transferring phages to glass (Julian et al., 2010).

Human exposure through direct contact with pets includes contact between hands and fur, pet mouth, nose, and paws, for example during feeding, grooming, petting, or play. Direct contact between a person's mouth and a pet is also common. A high percent of owners play with their dog (Adell-Bath et al., 1979; Messent, 1983; Stallones et al., 1988). A more recent survey of pet-owner interaction in the U.S. and Australia indicated that the majority of owners spend 0.5 to 3 hours a day interacting with their pet, with no significant difference between dogs and cats, while approximately 10–15% of owners spend less than 0.5 hours a day with the pet, and approximately 20% more than 3 hours (Laflamme et al., 2008). The most common activities shared by dogs and their owners include pet or cuddle (95%), talk to the pet (88%), play, walk, or run (62–79%), and groom (64%). For cats, the most common shared activities were more sedentary, and included pet or cuddle (96%), talk to the pet (91%), watch the pet play (72%), and sleep (61%) (Laflamme et al., 2008). Physical contact was observed to be a major form of interactions between children and dogs (Filiatre et al., 1986; Millot et al., 1988), and between older adults and dogs in a nursing home (Neer et al., 1987). Even people that do not own pets reported a significant level of contact with pets outside of their house, mostly at friends' houses or on farms (Stull et al., 2012). No study exists on microbial transfer from pets to hands or mouth. However, inferences can be made from data about other types of surfaces (Table 7).

Surface-mediated human exposure to pathogens includes contact between a person's mouth and a contaminated surface, or a two-step process where people first touch contaminated surfaces with their hands, and then touch their mouth with their hands. Both routes can lead to accidental nondietary ingestion. Direct surface-mouth contact can occur via dishes, silverware, towels, napkins, or food for adults. Infants and children often touch surfaces directly with their mouth. Surface-hands-mouth exposure can occur in a variety of situations. Contamination of household surfaces by pathogens and its link to outbreaks has been documented (Bloomfield and Scott, 1997; Josephson et al., 1997; Kagan et al., 2002; Ojima et al., 2002; Rice et al., 2003; Haysom and Sharp, 2005; Jackson et al., 2007), although most studies focused on the kitchen and secondarily the bathroom. While behavior leading to exposure to pathogens in the household is not well characterized, contact behavior has been studied in more details in regards to household exposure to pesticides (Auyeung et al., 2006; Beamer et al., 2012), and to some extent in regards to nosocomial infections (Neely and Maley, 2000; Ray, 2002).

Such studies highlight that patterns of contact with household surfaces vary significantly with age. For instance, children are considered more at risk of exposure to contaminants present on floors and in dust (Freeman et al., 2001; Auyeung et al., 2006; George and McCurdy, 2011). The type of activity also affects exposure risk, as highlighted by studies on hand surface contact (Lu and Fenske, 1999; AuYeung et al., 2008).

Pathogen transfer and exposure can also occur through human food and in the kitchen environment during food preparation (Fischer et al., 2007). After touching contaminated pet food and not sanitizing hands, a pet owner could cut vegetables for a salad on a cutting board or handle other ready-to-eat food. In contrast with other house surfaces (e.g., couch fabric, floor tiles), the humidity and organic matter content on kitchen and food preparation surfaces (e.g., sinks, sponges, cutting boards) and on food itself could support bacterial growth, acting as an amplifier step in the exposure chain. Bacterial contamination in domestic kitchens has been documented (Bloomfield and Scott, 1997; Gorman et al., 2002), as well as food preparation behaviors that can lead to contamination and cross-contamination (Jay et al., 1999; Redmond and Griffith, 2003; Gilbert et al., 2007). Higher moisture levels have also been observed to increase the effectiveness of transfer to and from surfaces (Sattar et al., 2001). Moreover, the development of biofilms can favor bacterial pathogen growth and persistence by maintaining high moisture levels and offering protection from sanitizing agents (Dourou et al., 2011, Bae et al., 2012).

Once hands are contaminated, pathogen transfer to the mouth can most likely occur while eating with hands or touching the face. In a study with *Enterobacter*, Rusin et al. (2002) observed transfer ratios from hands to lips in the 34–41% range. Nicas et al. (2008), in an observation study of ten college students (total of 30 person-hours), observed an average rate of hand-to-face contact of 15.7 episodes per hour (mean per subject of 47 per hour, standard deviation 34, range 3–104). Subjects who more frequently touched their lips were also more likely to touch their eyes and nostrils. A previous study of 89 person-hours of observation reported lower contact frequencies, namely 0.33 episodes/hour of nose picking and 0.37 episodes/hour of eye rubbing (Hendley et al., 1973). A meta-analysis of nine studies on hand-to-mouth behavior in U.S. children, observed in indoor and outdoor settings via video recording or direct observations, highlighted significant differences in contact frequency as a function of geographical location (state) and child age, but not gender or study (Xue et al., 2007). The average indoor hand-to-mouth contact frequency was 6.7–28.0 times per hour, and highest in the three to six-month-old age group. The average outdoor contact frequency was 2.9–14.5 times per hour, and highest in the 6–12 month olds. For each age group and study, the distribution of contact frequency was best fitted with a Weibull distribution (Xue et al., 2007).

Pets, besides spreading pathogens, can also become infected through contact with contaminated environments, such as sites heavily attended by other dogs. For example, dog and human outbreaks of salmonellosis have been attributed to visiting contaminated veterinary clinics (Ketaren et al., 1981; Cherry et al., 2004; Wright et al., 2005). Also, animal shelters have been observed to be contaminated (Steneroden et al., 2011) and cross-infection in such environment, possibly mediated by infected surfaces, has been documented (Yokoyama et al., 1991). In the household, dogs can become infected with methicillin-resistant *S. aureus* when living with infected humans (Damborg et al., 2004). When noninfected pets acquire pathogens from an already contaminated household surface, they can become infected, colonized, or simply harbor the pathogens on their fur and paws, thus becoming a reservoir and a vector within the household.

RISK MITIGATION MEASURES IN THE HOUSEHOLD

Pathogen control methods in the household fall in two main categories: (1) strategies to interrupt pathogen spread and prevent surfaces to become contaminated in the first place, and (2) methods to reduce or eliminate pathogens from surfaces. The first category includes storing potentially contaminated items away from sensitive areas and targets, e.g., storing pet food and in-use litter outside of the house or away from the kitchen, prevent cross-contamination, e.g., not washing the pet food bowl with other dishes and not putting dog toys on the kitchen table, or preventing pet contact, e.g., by not letting the pet into the kitchen area or not petting the cat while preparing food or eating. The latter category involves disinfecting surfaces, washing hands, and cooking food that may have come in contact with pathogens. In the U.S. no federal law addresses the risk of infection associated with contact with animals, and only few states regulate contact in public settings such as fairs and zoos (NASPHV and CDC, 2011). While recommendations have been developed to reduce such risk from animal contact in both public settings (NASPHV and CDC, 2011) and at home (CDC, 2013), such guidelines are less likely to be followed rigorously in the house, due to the high frequency of contact with pets and associated surfaces.

Precautions in handling pet food and managing pet feeding activities can act as a physical barrier and reduce the risk of pathogen cross-contamination and transfer to hands and household surfaces. Safer storage practices could include storing pet food in a dry area away from the kitchen or high-traffic household areas, and in containers where it cannot be accessed by children or wild animals. Safer pet food handling practices may include using a dedicated scooping tool that does not come in contact and is washed separately from dishes and eating utensils, washing hands after handling the food, and not leaving pet food accessible in the bowl for long times. Safer pet feeding practices can include not feeding pets in the kitchen, not allowing pets to spread food on the floors, and not feeding pets during cooking or eating time. Even more important than preventing direct contact between dry pet food and

Table 7 Transfer of pathogens between surfaces, hands, and food

Microrganism	Surfaces	Transfer ratio	Reference
Campylobacter	Raw chicken to kitchen surfaces	Ratio NA, concentration measured on hands, board, cloth, tap, knife, and door handle. Highest concentrations on board and cloth, lowest on door handle and knife. Most levels 1–10 CFU, but significant percents in 10–100 CFU range	Cogan, 2002
Campylobacter	Chicken, hands, cutting board, knife, food	Chicken legs to hands: mean 2.9% (SD 5.5%, range 0.2–19.7%). Chicken filets to hands: mean 3.8% (SD 5.9%, range 0.2–7.8%). Chicken to plate: mean 0.3% (SD 0.3%, range 0.1–0.7%). Plate to sausage: mean 27.5% (SD 23.7%, range 0–62.6%). Filets to board and knife: mean 1.1% (SD 0.7%, range 0.2–2.3%). Board and knife to cucumber: mean 10.3% (SD 9.6%, range 0–33.3%). Hands to bread: mean 2.9% (SD 3.8%, range 0.4–9.5%)	Luber et al., 2006
Campylobacter	Stainless steel to lettuce	31.5–38.4% for dry lettuce, 15.2–26.6% for wet lettuce in 40 minutes after inoculum, then drops to 16.3% and 7.2% at 60 minutes	Moore et al., 2003
Campylobacter jejuni	Stainless steel, sponges, cucumber, roasted chicken	Sponges to steel: 28–43%. Steel to cucumber: 177–185%. Steel to chicken: 66–101%	Kusumaningrum et al., 2003
Campylobacter spp.	Raw chicken, stainless steel, cucumber	Chicken to steel: fitted normal distribution (0.098.0.606). Steel to cucumber: normal (1.535, 0.320) as Log %	Kusumaningrum et al., 2004
E. coli	Damp cloth to hand	0.47%	Mackintosh and Hoffman, 1984
E. coli	Laminate, steel, fingers, cloth	Laminate to fingers; 18–28%. Laminate to steel: 18–27%. Cloth to fingers: 0.5–0.8%. Cloth to laminate: 3.2–3.8%	Scott and Bloomfield, 1990
E. coli	Paper	Hand-paper-hand: mean 0.009% (SD: 0.007%)	Huebner et al., 2011
E. coli O157	Dishes, sponges, kitchen surfaces, simulated food (solid agar)	Dish to sponge to surface: 0/12 and 5/6 positive surfaces at 10^3/dish with strains NCTC 12900 and ATCC 11229, 6/6 at 10^6. Dishes to agar: 0/4 positives with 5 minutes contact time, 0/4 (no pressure) and 2/4 (pressure) at 20 and 60 minutes, 0/4 (no pressure) and 1/4 (pressure) at 60 minutes	Mattick, 2003
E. coli O157:H7	Slicing machine blade to cooked meat	-2.7 to -2.8 as $100\times$Log(CFU per cm^2 slice/CFU on blade). Log-linear and Weibull models good fit	Perez-Rodriguez et al., 2007
E. coli, Bacillus atrophaeus	Brush, door handle, faucet	Hand to brush: 0.43%; hand to handle: 0.16%; hand to tap: 0.09%	Gerhardts et al., 2012
Enterobacter aerogenes	Chicken, cutting board, hands, gloved hands, spigot, lettuce	Chicken to board: 11.2%, board to lettuce: 6.2%, chicken to hand: 3.9%, hand to lettuce: 1.6%, spigot to hand: 1.4%, hand to spigot: 0.08%, gloved hand to lettuce: 0.05%, chicken to gloved hand: 0.001%	Montville and Schaffner, 2003
Enterobacter aerogenes	Chicken, hands, metal spigot, lettuce	Metal spigot to hands, mean (SD) as Log percent: 0.36 (SD: 0.90); hand to spigot: -0.80 (1.1); chicken to hand: 0.94 (0.68); board to lettuce: 0.90 (0.59); hand to lettuce: -0.12 (1.1). Best fitting distribution of Log percent transfer: beta, gamma, Weibull	Chen et al., 2001
Listeria monocytogenes	Commercial slicer to deli meats	Blade to meat: inoculum 10^8 CFU, approx. 10^7 in first slice, 2 Log CFU less (10^5) at 20 slices, 3 Log CFU less (10^4) at 30 slices	Vorst et al., 2006

(*continued on next page*)

Table 7 Transfer of pathogens between surfaces, hands, and food *(Continued)*

Microrganism	Surfaces	Transfer ratio	Reference
M. luteus; S. marcescens	Hands, paper towels, towel dispensers	Hand to dispenser: 0.03–0.04% (SD: 0.02–0.03%); dispenser to hands: 6.0–6.7% (SD: 0.22–0.25%)	Harrison et al., 2003
Micrococcus luteus, Serratia rubidea	Plastic, metal, cloth, carrots, meat patties, hands	Hard plastic and metal: 28–66% (faucet to hand 40% and 28% for the two organisms; phone to hand: 41.8% and 38.5%; carrot to hand 0.21% and 0.12%; hamburger to hand 0.06% and <0.01%); porous fomites: <0.01% (dishcloth to hand 0.04% and <0.01%; sponge to hand 0.03% and <0.01%; laundry from <0.01% to 0.13%)	Rusin et al., 2002
Salmonella	Egg, hands, formica	Hands positive after cracking eggs: 5% (10^3 CFU/egg) to 20% (10^6 CFU/egg). Countertop positive after mixing: 8% (2.7 Log CFU/mL) to 40% (3.7 Log CFU/mL) hand mixing; 30% (2.7 Log CFU/mL) to 100% (4.7 LogCFU/mL) electric mixer	Humphrey et al., 1994
Salmonella Enteriditis	Stainless steel, sponges, cucumber, roasted chicken	Sponges to steel: 21–29%. Steel to cucumber: 65–105%. Steel to chicken: 49–94%	Kusumaningrum et al., 2003
Salmonella Enteriditis PT4	Dry egg droplets to cooked beef and melon	Droplets to beef: 10–20% after 5 and 10 sec, 40–60% after 30 sec. Droplets to melon: 0–40% after 5 sec, 70–90% after 30 sec	Bradford et al., 1997
Salmonella spp.	Laminate, steel, fingers, cloth	Laminate to fingers: 11–16%. Laminate to steel: 13–18%	Scott and Bloomfield, 1990
Salmonella spp.	Raw chicken, stainless steel, cucumber	Chicken to steel: fitted normal distribution (0.171, 0.162). Steel to cucumber: normal (1.458, 0.198) as Log %	Kusumaningrum et al., 2004
Salmonella Typhimurium	Dishes, sponges, kitchen surfaces	Dish to sponge to surface: 0/9 positive surfaces at 10^3 CFU/dish, 3/3 at 10^4 and 10^5 CFU/dish	Mattick et al., 2003
Salmonella Typhimurium	Stainless steel to lettuce	36.3–66% for dry lettuce, 26.9–31.4% in 40 minutes after inoculum, then drop in transfer to dry lettuce	Moore et al., 2003
Salmonella Typhimurium	Stainless steel, formica, PPE, wood to cucumber	Steel: at inoculum 4.6% (PBS) and 0.4% (TSB); after 1 hour 76.4% (PBS) and 44.9% (TSB); after 6 hours 86.6% (PBS) and 46.8% (TSB). Formica: > 100% after inoculum; 62.2% (PBS) and 44% (TBS at 1 hour; 84% (PBS) and 100% (TSB) at 6 hours. PPE: 13.6% (PBS) and 23.9% (TSB) at inoculum; 66.7%(PBS) and 53% (TSB) at 1 hour; 100% (PBS and TSB) at 6 hours. Wood: 20.2% (PBS) and 19.6% (TSB) at inoculum; 70.8%(PBS) and 45.8% (TSB) at 1 hour, 100% (PBS) and 75% (TSB) at 6 hours	Moore et al., 2007
Salmonella Typhimurium	Wood, tile, carpet, sausage, bread	At zero residence time: wood to sausage 48%; tile to sausage 69%; carpet to sausage 0.1%; tile to bread 49%	Dawson et al., 2007
Salmonella	Raw chicken to kitchen surfaces	Ratio NA, concentration measured on hands, board, cloth, tap, knife, and door handle. Highest concentrations on board and cloth, lowest on door handle and knife. Most levels 1–10 CFU, but significant percents in 10–100 CFU range	Cogan et al., 2002
S. aureus	Fabric to hand or fabric	Dry fabric to hand: 0.043–0.046%, moist 0.13–1.21%; dry fabric to fabric: 0.003–0.013%, moist 0.15–0.17% (no friction)	Sattar et al., 2001
S. aureus	Laminate, steel, fingers, cloth	Laminate to fingers: 23–27%. Laminate to stainless steel: 18–22%. Cloth to fingers: 3.4–3.8%. Cloth to laminate: 1.0–1.2%	Scott and Bloomfield, 1990

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Table 7 Transfer of pathogens between surfaces, hands, and food *(Continued)*

Microrganism	Surfaces	Transfer ratio	Reference
S. aureus	Slicing machine blade to cooked meat	−1.25 to 1.48 as 100×Log(CFU per cm^2 slice/ CFU on blade). Log-linear and Weibull models good fit	Perez-Rodriguez et al., 2007
S. aureus	Stainless steel, sponges, cucumber, roasted chicken	Sponges to steel: 38–41%. Steel to cucumber: 95–117%. Steel to chicken: 62–76%	Kusumaningrum, 2003
Total viable counts	Sponges and dishcloth, cutting board	Cloth to board: 56%; sponge to board: 4.7%; rinsed cloth to board: 0.17%; rinsed sponge to board: 0.16%	Hilton and Austin, 2000

[a]Range observed.

sensitive surfaces, pet food should not come in contact with environments that can act as amplifiers or reservoirs of bacterial contamination, such as rewetted pet food itself, the pet's water, wet floors, wet food preparation surfaces, and human food. Moreover, the pet itself can act as a major pathogen amplifier. Hence, preventing contact between the pet and sensitive surfaces (e.g., kitchen, human dishes, sinks) or sanitizing such surfaces after pet contact and before sensitive activities (e.g., food handling, eating) could potentially provide a higher level of protection compared to barriers around pet food alone. Guidelines recommend that children younger than five years should not be allowed in pet feeding areas, should not touch pet food, and should be supervised when interacting with pets (CDC, 2013). While such recommendations follow common sense and stem from epidemiological evidence collected during outbreak investigations (CDC, 2000, 2003, 2008a, 2008b), the relative health benefits of different prevention practices have not been characterized.

Hand-washing is recommended after contact with animals, their food, or pet-associated objects (e.g., bowls, cages, toys) to reduce pathogen load on the hands, and therefore reducing the likelihood of accidental ingestion and transfer of pathogens to other surfaces (CDC, 2013). Hand-washing has been observed to effectively reduce bacterial counts on hands (Toshima et al., 2001). Also, a meta-analysis of hand washing effectiveness estimated a reduction in children diarrhea incidence of 39% in high-income countries, and of 32% in low-income countries (Ejemot et al., 2008). The effectiveness of hand sanitation practices, as well as the difficulties in enforcing and monitoring this practice, have also been extensively documented in health care workers (Haas and Larson, 2007; Sax et al., 2007; Bolon, 2011; Monistrol et al., 2012). Surveys in Europe, North America, and Oceania showed that while a high percent of the population is aware of the importance of washing hands before preparing meals to reduce the risk of food poisoning, up to 14–21% of the population could lack basic knowledge on this issue (Redmond and Griffith, 2003). However, independently from theoretical knowledge hand washing is usually not practiced as often and as rigorously as recommended. According to a recent survey, only 15% of dog owners and 8% of cat owners always wash their hands after direct contact with the pet (Overgaauw et al., 2009). In a different U.S. study 48% of the surveyed subjects reported not washing their hands after touching a pet, and 19% not washing hands before meals (Rusin et al., 2000). In a study in Japan, only 27–30% of men and 10–11% of women were observed to use soap when washing hands in public restrooms (compared to 9–46% for elementary school pupils), and even soap-users spent significantly less time lathering than the recommended 20–30 seconds (Toshima et al., 2001). Direct observations in Australian and U.S. homes found an avoidance of hand-washing, including before meal preparation, as well as other exposure-enhancing behaviors such as touching face and mouth, and allowing pets in the kitchen (Jay et al., 1999).

The effectiveness and feasibility of various cleaning and disinfection measures appropriate to household surfaces have been tested, and are reviewed elsewhere (Kagan et al., 2002). Common practices for cleaning dishes, bowls, and eating utensils involve washing with a detergent, by hand or in a dishwasher, followed by drying. Such methods have been shown to reduce bacterial levels on eating utensils by more than 5 Log CFU (Mattick, 2003; Lee et al., 2007). Kitchen surfaces such as countertops are usually wiped with a wet cloth or sponge, either with water, water and a detergent, or a disinfecting agent such as bleach (Rusin et al., 1998), vinegar, or other antibacterial agents. Both vinegar and baking soda were shown to reduce levels of enteric bacteria by less than 3 Log CFU, products containing ammonia by 4–6 Log CFU, and phenol or ethanol-based products by 4 Log CFU (Kagan et al., 2002). Hypochlorite cleaners have been shown to reduce fecal coliform concentrations on kitchen sink surfaces by 3.6 Log CFU (Rusin et al., 1998). Another study documented the effectiveness of bleach to inactivate *S. aureus, Salmonella* Typhi, and *E. coli* on surfaces, while other agents had varying success (Parnes, 1997; Kagan et al., 2002; Weber et al., 2003). However, several bacterial pathogens were still detected in dog food bowls after washing with soap, soaking in bleach, or washing in a dishwasher at 85°C (Weese and Rousseau, 2006). Several bacterial species could still be detected on dishes after washing with a detergent and rinsing, although rinsing was shown to play a crucial part in bacterial reduction (Cogan et al., 2002). In addition, cleaning agents are less effective on surfaces such as cutting boards (Miller et al., 1996). Moreover, sponges and dishcloths, often left wet at room temperature and

possibly soaked in food juices, have been shown to harbor high concentrations of bacteria (Speirs et al., 1995; Ojima et al., 2002; Rossi et al., 2013). Such bacteria could persist or grow on sponges (Nielsen et al., 2002; Lee, 2010; Bae et al., 2012), and could spread contamination to food and other kitchen surfaces (Hilton and Austin, 2000; Mattick, 2003; Rossi et al., 2013).

GOVERNMENT AND INDUSTRY INITIATIVES TO ENHANCE THE SAFETY OF PET FOODS

During the past decade, the association of pet foods with incidents of outbreaks of salmonellosis in humans and mycotoxicoses in dogs, as well as kidney failure in dogs and cats resulting from pet food ingredients intentionally contaminated with melamine, has led to a re-evaluation of pet food safety in the United States (Dzanis, 2008) and other countries (Commission of the European Communities, 2005; European Parliament, 2009) by both government and industry. The primary regulatory agency in the U.S. that is responsible for animal feed and pet foods is the U.S. Food and Drug Administration (U.S. FDA). Currently pet food sold in the U.S. does not require premarket approval from the FDA. However, under section 402(a)(1) of the U.S. "Food, Drug, and Cosmetic Act" enforced by the FDA pet food sold in the U.S. should be "safe to eat, produced under sanitary conditions, contain no harmful substances, and be truthfully labeled" (U.S. Code, 2006). Specifically, animal feed and ingredients found contaminated with *Salmonella* are considered adulterated (U.S. CFR, 2012a). Canned pet food also needs to comply with the good manufacturing practices for low-acid foods packaged in hermetically sealed containers (U.S. CFR, 2012b). Beginning with the passage of the "Public Health Security and Bioterrorism Preparedness and Response Act of 2002" (PHSBPRA), there has been an increase in food safety requirements encompassing both human food and animal feeds. This shift was reflected in the inclusion of animal feed facilities under the requirements for registration of food manufacturing facilities (U.S. FDA, 2006). This trend was expanded upon under the Food and Drug Administration Amendments Act of 2007 (FDAAA), which established the Reportable Food Registry, requiring both food and feed manufacturers to report to FDA within a specified time period if the manufacturer has information that a contaminated product has been introduced into commerce (U.S. FDA, 2013c). This effort to harmonize regulatory requirements has been further advanced by the passage of the Food Safety Modernization Act of 2011 (FSMA). In passing FSMA, Congress required FDA to publish new regulations that enhance the safety of human, pet foods, and animal feed (Taylor, 2012). Two of the key proposed regulations that are currently undergoing public comments are: (i) the requirement for manufacturers to develop food safety systems based on preventive controls, and (ii) requirements for the importation of foods into the United States. Again, these requirements will include human foods, pet foods, and animal feeds. In addition to new regulations, FDA has undertaken the publication of several new guidance documents and regulatory initiatives. The FDA Office of Regulatory Affairs has recently issued a new Compliance Policy Guide for *Salmonella* in Foods for Animals (U.S. FDA, 2013d). The FDA is also in the process of conducting a "field assignment" to acquire more information on the extent of *Salmonella* contamination in domestic dried pet foods, pet treats, and nutritional supplements for pets (U.S. FDA, 2013e). This information will presumably be used to help develop performance standards for the new preventive controls regulation of foods for animals.

The pet food industry and professional societies have also been active in developing strategies and guidance related to the control of *Salmonella* and other foodborne pathogens in pet food and animal feed. As part of a larger initiative to control *Salmonella* outbreaks associated with dry food products, the Grocery Manufacturers Association developed and disseminated guidelines for these products and ingredients, including pet food and treats (GMA 2009a, 2009b). The European Pet Food Industry Federation (FEDIAF), as well as the American Feed Industry Association (AFIA) have issued good manufacturing guidelines to minimize *Salmonella* contamination in animal feeds (AFIA, 2010; FEDIAF, 2010). In addition, in 2011 the AFIA has established the Pet Food Manufacturing Facility Certification Program and the Pet Food Ingredient Facility Certification Program, developed for the pet food and pet food ingredient industries respectively, to achieve an enhanced level of food safety protection within the pet food industry (AFIA, 2012). The American College of Veterinary Nutrition has provided information that veterinarians can share with pet owners on the proper handling of pet foods to reduce the risk of microbial hazards for both the pets and their owners (KuKanich, 2011). Both the pet food industry and the veterinary community are currently developing comments and recommendations related to the new proposed FSMA regulations so that the final regulations provide meaningful and practical approaches to enhancing the safety of pet foods for both animals and their owners.

CONCLUSIONS AND RESEARCH NEEDS

This review compiles and summarizes existing published information on the main variables affecting the risk of bacterial infection due to contact with pets and contaminated pet food, from ingredients to consumers. To the best of our knowledge this is also the first systematic review of pet-owner contact in the household and pet feeding habits from a public health perspective. This information can be used as input into quantitative risk assessment models describing production processes that involve dry ingredients, an extrusion or other heat step for pathogen abatement, and the hazard of rewetting. The information also supports the development of quantitative models describing bacterial pathogen ecology, transfer, and

exposure in the household. The review highlights several large data gaps in the risk profile of dry pet food. In particular, little quantitative information is available on pathogen levels in pet food ingredients, and on the potential for cross-contamination during production and transport. While food companies may collect data on the occurrence of *Salmonella, E. coli*, or other bacterial indicators in finished products, equipment, and facility surfaces, these data are usually not available due to confidentiality issues. Even in studies reporting prevalence, concentrations are most often not measured, which significantly impairs the ability to realistically estimate risk and the effectiveness of control strategies. While several studies have measured persistence and decline of bacterial pathogens on dry food, and growth on wet food matrices, no specific data are available for dry pet food or similar products. Only a handful of studies have surveyed pet owners' habits in regards to handling pet food in the household, pet feeding habits, and serving sizes among different pet breeds and sizes. While such studies provide useful information, they were usually not designed with risk assessment in mind, and therefore it is problematic to translate such information into even simple probabilistic distributions. No dose-response relationships are available to estimate the probability of infection, illness, or colonization and subsequent shedding in pets exposed to bacterial pathogens. In addition, the duration of pathogen shedding and pathogen concentrations in infected pets' feces or saliva is unknown. Therefore the role of the pet as reservoir, amplifier, and vector of foodborne pathogens can only be approximately estimated by making assumptions on these variables. Lastly, age-dependent contact behavior between family members, pets, and household surfaces is only partially documented in the literature. Rigorous quantification of these variables would significantly advance the development of risk-based prevention and mitigation strategies in the household (see also Bloomfield and Scott, 2003).

Quantitative microbial risk assessment models provide a powerful tool to estimate health outcomes as a result of contact with or ingestion of contaminated foods, and as a function of production conditions and product handling and consumption behavior. National and international organizations have been moving towards risk assessment as the framework of reference to determine the acceptability of industry practices and quantify control point performance in the food and water sectors. Such models can accommodate complex processes and a high number of stochastic variables, and can be applied to estimate the effects of changes in production practices or consumer behavior. However, the accuracy, precision, and overall usefulness of these models is highly dependent on the availability and quality of input data. In the absence of satisfactory data, caution should be used in drawing conclusions from modeling efforts. In future studies on microbial contamination of ingredients, facilities, and finished product, the measurement of concentration levels using an assay of appropriately low detection limit is warranted. Furthermore, due to insufficient data these models often omit important contamination routes, such as cross-contamination from contact with equipment or airborne microorganisms, thus falling for the fallacy of "looking for the keys where the light is." Lastly, while some aspects of cross-contamination and exposure in the household have been well documented (Kagan et al., 2002), and risk models developed for specific food preparation situations (Pouillot et al., 2012), no complete microbial risk models are available for household transmission of foodborne pathogens, and no partial model includes pets. Such models could strengthen the credibility of recommendations to pet owners.

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