

#### **Review Article**

# A Brief History of Pet Foods, the Pathogenic Organisms of Concern, and the Potential Harboring Capacity of Animal Derived Fats

#### Kelsey Lamb<sup>\*</sup>, Melissa Morgan and Roberta Dwyer

Department of Animal and Food Sciences, Food Microbiology, College of Agriculture, Food and Environment, University of Kentucky, Lexington, Kentucky, USA

\*Corresponding author: Kelsey Lamb, Department of Animal and Food Sciences, Food Microbiology, College of Agriculture, Food and Environment, University of Kentucky, Lexington, Kentucky, USA, Tel: +1 8592573855; E-mail: kela226@uky.edu

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## Abstract

The extrusion process is acclaimed as one of the most efficient and safest manufacturing processes for developing dry food items intended to be shelf-stable with an extensive shelf life. Similarly, the fat rendering process results in a safe and sterile product that can be utilized for flavorings or nutrient enrichments. Both methods have been widely accepted for the production of safe shelf-stable dry pet foods and pet food related products. Despite the safety associated with these products and their processing methods, the Food and Drug Administration (FDA) has issued 235 recalls from January 1, 2010 to April 1, 2018 concerning pet foods, livestock feeds, pet treats, and other animal specialty items. Of the 235 total recalls, possible contamination due to *Salmonella* was responsible for 124 recalls and recall expansions. Possible contamination due to *Listeria* was responsible for 19 recalls and recall expansions; nine of these cases overlapped with possible *Salmonella* contamination recalls. One Shiga-toxin producing *E. coli* (*STEC*), *E. coli* O128, was implicated in a raw pet food recalled March 26, 2018. It is suggested that the addition of fats and other nutrient compounds, following the primary cooking process of pet foods, could contribute to the introduction of pathogenic organisms in the final product. It has been shown that bacteria are more likely to survive in heated environments when lipids are present. Animal derived fats, specifically beef tallow, pig lard, and duck fat, have not been associated with having antimicrobial properties. The potential pathogenic harboring capacity of animal fats should be further studied.

Keywords: Pet food; Extrusion; Fat rendering; *Salmonella*; STEC; *Listeria* 

### Introduction

#### **Pet Foods**

Commercial pet foods can be described as nutritionally complete or nutritionally complementary [1]. The majority of products, labeled as pet foods, are nutritionally complete meals which contain all of the necessary nutrients to support or maintain growth, depending upon the stage of life, without the need for additional supplementation [1,2]. Treats and other supplemental foods are considered complementary items to be used in conjunction with nutritionally complete meals or other complementary components to form a well-balanced diet [1]. Complete diets and complementary treats comprise the two types of pet foods that can be subdivided into various categories.

Currently, researchers and the pet food industry appear to be divided over how to categorize pet foods based upon the characteristics acquired during processing. In the industry, pet foods can be subdivided based on what processing technique was utilized to develop the final product. This can include canned/retorted, baked, extruded, frozen, freeze-dried, air-dried, and refrigerated products [2,3]. The issue with this division is that there is often overlap within many of the product characteristics such as baked and extruded products which both produce dry pet foods. Zicker presented a categorization of commercial pet foods based on processing characteristics. In his topical review, pet foods were divided into three types based on water content: moist, semi-moist/soft, and dry [4]. The largest of these three categories is dry pet food, which accounts for the largest weight and monetary value within the pet food industry [5,6]. Moist pet foods contained a range of 60-87% water, semi-moist/soft pet foods contained a range of 25-35% water, and dry pet foods generally contained 11% water or less [4]. These ranges have also been verified in other works with some variation [7-10]. Treats are sometimes typed as an additional category; however, they are generally included into these three overarching types.

During the 1860's, pet foods became more commercialized with the development of baked pet food biscuits [3,11]. The first canned pet foods appeared in the consumer market in the 1920's in hermetically sealed retorted containers [3]. It was not until the 1950's that an extruder was used to produce food based items with the development of the Single screw extruder and the Twin screw extruder [3]. In the mid 1950's, the first pet foods were produced using extrusion technology and rapidly grew to the largest share of the pet food market [3-6].

All pet foods are regulated by the Food and Drug Administration (FDA) through the Federal Food, Drug and Cosmetic Act (FDCA), with some overlap with the United States Department of Agriculture (USDA) and Environmental Protection Agency (EPA) [4,12,13]. The Association of American Feed Control Official (AAFCO) holds no regulatory authority, but works closely with the FDA and other state governments to provide the necessary safety and nutritional information concerning pet food products [13]. AAFCO formed the Canine Nutrition Expert Subcommittee in 1990-1991 followed by the Feline Nutrition Expert Subcommittee in 1991-1992 to address various nutrient requirements to be met by pet food label claims of complete diets [12]. Within the dry pet food category, Spears and Fahey reported

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that extrusion was the production method utilized for 95% of the pet food diets [14]. Semi-moist/soft foods and treats may have also been included in the final percentages since many of these products undergo the same initial extrusion process, but negate the final drying step [4,6].

Extrusion is defined as a high temperature, short time (HTST) bioreactor process [6,15,16]. Under these processing conditions, physical and chemical changes affecting the nutrition, structure, and palatability occur as the product is cooked and subsequently extruded [6]. Temperature conditions range from 80-200 °C (typically 110-150  $^\circ C$  ) with an average 300g/kg moisture content and 34-37 atm pressure for a relatively short time of 10-270 seconds followed by a varied drying time at 120-160 °C [4,6,16,17]. Raw ingredients are ground and mixed together to form a viscous homogenous dough [4,18]. This dough can then be conditioned with water or it can be added directly into the extruder barrel which cooks the product under heat from steam and friction caused by the increased pressure experienced by the spiral screws in the chamber [6,16,19,20]. Once the food product has been rendered sterile, due to the temperature, pressure, and added steam combination within the barrel, it is forced through a die, at 3-6 MPa pressure, which forms the ultimate shape of the product [2,4,15,21]. A knife is used to cut the extruded pet food product to the desired length prior to drying to a moisture content of 6-8% and subsequent cooling at ambient temperature [4,6,19]. The extrusion process ultimately results in a sterile product free of microorganisms and their related toxins [2,4,6,15].

Despite the numerous safety, nutritional, and production benefits of the extrusion process, the oxidation of various fat components, which may lead to rancidity, continues to be a concern within the final products [20,22]. The loss of heat labile nutrients, specifically vitamins and minerals, is also of concern within the industry, especially for products that are formulated to be complete meals [6,23,24]. Many pet food companies adjust the fat and heat labile nutrients within their products to compensate for losses during extrusion. Fats and heat sensitive nutrients can be added in excess at the beginning of the extrusion process to compensate for the loss of some during the processing [6,23,24]. This method of nutrient compensation can be extremely costly if the final nutrient balance deviates from the estimated loss to a point of deficiency or excess outside of the accepted limits. The addition of excessive fats to the initial dough mixture before extrusion can ultimately influence the cooking process of the extruded product by decreasing the friction within the screw chamber [6,22]. Excessive fat levels at the beginning of production can also result in a product that lacks the proper expansion and structural properties necessary for extruded products [6,22]. Due to these undesirable characteristics, fats and heat sensitive nutrients are typically added as flavors and nutrient enhancements during the drying and cooling phases of pet food production [16]. This can be accomplished by several different methods of spray drying, drop coating, drum drying, and other coating styles with various antioxidant mixes such as tocopherols (Vitamin E) [2,16]. The addition of these fats and heat labile nutrients after the primary cooking step and the initial drying process helps to ensure their structural survival during production. It does, however, increase the likelihood of microbial contamination within the final product.

# Pathogens of Concern

## Salmonella various strains

From the beginning of January 1, 2010 to April 1, 2018, the FDA issued 235 individual recalls for livestock and domestic pet related products [25]. The largest number of these recalls was attributed to suspected or laboratory confirmed bacterial contamination, specifically Salmonella spp. [25]. The following categorization of the FDA recall cases was summarized by the writer for use in this review. Salmonella spp. have been implicated in the contamination of 42 dry pet food products, 29 moist or refrigerated/frozen pet food products, 45 pet treat products, three animal medications, and five specialty pet foods or added ingredients [25]. Two of the dry pet food recalls included associated treat recalls and 16 of the listed 45 pet treat recalls were of animal origins, such as pig ears and cow hooves [25]. The majority of these recalls were precautionary with no formally reported pet or human illness despite some food samples having confirmed laboratory evidence [25]. Minimally processed pet food products, such as raw diets and pet treats of animal origin, are known to increase the risk of human exposure to Salmonella via handling contaminated food items or direct animal contact [26-29]{Centers, 2006, Human salmonellosis associated with animal-derived pet treats--United States and Canada`, 2005;PHAC: Public Health Agency of Canada, 2000, Preliminary Report - Human Health Risk from Exposure to Natural Dog Treats -CCDR Volume 26-06 - Health Canada; Pitout, 2003, Association between handling of pet treats and infection with Salmonella enterica serotype Newport expressing the AmpC β-lactamase', CMY-2;Sato, 2000, Salmonella virchow infection in an infant transmitted by household dogs]. White et al. examined the frequency of Salmonella spp. in animal derived pet treats available in stores across the United States. Of the 158 sampled products, 65 (41%) were contaminated with at least one strain of *Salmonella*, with several of the isolates displaying resistance to one (36%) or multiple (13%) antimicrobials [30]. Nemser et al. reported the analysis of 480 dry and semi-moist pet products yielded a positive Salmonella recovery in one dry cat food. In the analysis of 576 raw pet foods, exotic feeds, and jerky-style treats, 15 positive Salmonella spp. were recovered from raw pet food samples [31].

Although the recovery of Salmonella is less frequent from dry pet foods, there have been two major outbreaks associated with these products. From January 1, 2006 to December 31, 2007 the Centers for Disease Control and Prevention (CDC) and FDA traced an outbreak of Salmonella enterica ser. Schwarzengrund in 70 patients across 19 states with the majority being reported in the northeastern United States [32]. The outbreak was later expanded from December 31, 2007, to September 18, 2008, due to nine associated cases resulting in a total of 79 patients, with 30 cases in children under the age of two, from 21 states [33]. In both reports, some patients experienced bloody diarrhea and were hospitalized, but no deaths were reported [32,33]. The cause of the outbreak was traced to two brands of dry dog food produced by Mars Petcare US at their Everson, Pennsylvania, location [32,33]. The manufacturing plant was shut down for cleaning and renovations in July of 2007, after the outbreak strain was identified by the FDA in unopened packages of the final products [32]. The plant was later reopened in November of 2007, but with the addition of more outbreak cases in 2008, continuing the outbreak for three years, the Everson plant issued a nationwide recall of all dry cat and dog foods produced at the plant over a five month time frame and officially closed in October 2008 [32,33]. This was the first reported human Salmonella

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outbreak associated with dry pet food [32]. Although the source of the bacterial contamination was never definitively identified, it was hypothesized that contamination may have occurred during the flavoring and enhancement of the products after extrusion [32].

From February 1, 2012, to July 18, 2012, the CDC reported an outbreak of *Salmonella Infantis* in 47 patients from 20 states and two patients from Canada [34,35]. Upon recovery of *Salmonella* from a routine retail test, conducted by the Michigan Department of Agriculture and Rural Development on April 2, 2012, Diamond Pet Foods recalled 17 brands of dry dog and cat food from its manufacturing plant in Gaston, South Carolina. This particular *Salmonella Infantis*, in addition to a second strain, was linked to the outbreak of human illness attributed to contact with the contaminated pet food or animals that had access to the food [34]. This was the second reported human *Salmonella* outbreak to be associated with dry pet food [34,35]. The source of the contamination was never confirmed; however, the FDA report suggested post-processing contamination of the product due to poor employee hygiene and a lack of microbial analysis for incoming animal fats [36].

Salmonella are generally described as motile rod shaped Gramnegative bacteria with peritrichous flagella (some non-motile), can produce hydrogen sulfide (varied), and are facultative anaerobes that do not form spores [37,38]. All known serotypes, over 2500, are pathogenic to humans and may be pathogenic in other animals [37-39]. The number of bacteria in an infective dose varies with the strain of Salmonella. Although most records indicate illness at 5.0-log to 10.0-log cfu/g among human participants, as few as 3.0-log cfu/g to a few hundred cells have been thought to cause Salmonella outbreaks among humans [38,40]. Salmonella infection from ingestion of contaminated food items or contact with animal carriers can result in fever, diarrhea, and abdominal cramping within 12-72 hours after exposure with symptoms persisting for four to seven days [41]. In the CDC Foodborne Illness Fact Sheet (2000-2008), Salmonella was reported as the most common pathogenic bacteria to cause the largest number of food-borne related illnesses (1,000,000 people), hospitalizations (19,000 people), and deaths (380 people) per year [42,43].

The first human isolate of *Salmonella*, specifically *Salmonella enterica* subsp. *enterica* ser. Typhi, was attributed to Georg Gaffky in 1884 [39]. In 1885, veterinarian Daniel Salmon and microbiologist Theobald Smith isolated *Salmonella*, specifically *Salmonella enterica* ser. Choleraesuis, from swine [38,39]. Salmon was credited with the discovery and the bacteria were subsequently named in his honor [38]. Around 1983, DNA-DNA hybridization revealed only two separate species of *Salmonella*, *S. enterica* and *S. bongori*, therefore, resulting in the categorization of *S. enterica* into six subspecies [38,39]. *Salmonella* are also categorized by serotypes, or serovars, based on surface structures including flagella (H) and somatic (O) antigens [38]. These serotypes are described in the White-Kauffmann-Le Minor (WKL) scheme and recent supplemental listings [39,44-46].

# Shiga-Toxin Producing Escherichia coli (STECs)

Only one Shiga-Toxin Producing *E. coli, E. coli* O128, has been associated with any pet food related recalls from January 1, 2010 to April 1, 2018. In 2014, Nemser et al. reported that no STEC strains were isolated in the 480 dry and semi-moist pet foods assayed. There was also no detection of *STECs* in any of the exotic pet foods or jerky-type treats. There was, however, recovery of non-O157 *STECs* in 10 of the 196 raw pet food diets [31]. This indicated that raw pet food diets

have the potential to harbor STECs and that there is the possibility for human exposure to the organisms, if proper hygiene is neglected [31]. This is reflected in the recent raw pet food recall associated with E. coli O128 which is a non-O157 STEC. Despite the lack of formal recalls associated with these organisms in pet products, there have been recalls due to E. coli and STEC contamination in several human food grade ingredients such as: meats, nut-spreads, flours, cheeses, and leafy greens [47]. All of these products have the potential to be used for manufacturing pet foods. Outbreaks of various STECs have been documented in several products ranging from raw and unpasteurized products to cured meats and packaged cookie dough [48-52]. Outbreaks have also been reported from contact with livestock and with infected persons [53,54]. STECs have likewise been isolated from numerous mammalian species including: cattle, pigs, sheep, goats, cats, and dogs [55-57]. Although it has been proposed that companion animals may be a source of potential contact with STECs, the actual source of the organisms to humans remains unclear [55,58]. E. coli, specifically STECs, remain a prominent concern within the pet food industry due to the severity of the disease and the potential transfer to humans.

The identification of *E. coli* was credited to Theodor Escherich in 1885 [37,59,60]. The German pediatrician isolated *Bacterium coli commune* from the human colon, which was later renamed *Escherichia coli* in his honor [59]. Despite *E. coli* belonging to the *Enterobacteriaceae* family with other exclusive pathogens, such as *Salmonella*, it is considered to be an opportunistic pathogen [37,59]. *E. coli* are generally described as rod shaped Gram-negative non-spore forming facultative anaerobes with variable motility and produce acid and gas from both glucose and lactose [37,61]. The structures, including flagella (H) and somatic (O) and encapsulation (K) antigens, help to differentiate the serotypes of *E. coli* from one another [62]. These serotypes are described in the World Health Organization reference collection for *E. coli*, similar to the White-Kauffmann-Le Minor (WKL) scheme used for *Salmonella* [63,64].

There are six different pathotypes of E. coli including: Diffuselyadherent E. coli (DAEC), Enteroaggregative E. coli (EAEC), Enterohemorrhagic E. coli (EHEC), Enteroinvasive E. coli (EIEC), and Enterotoxigenic E. coli (ETEC) [61,62,65]. The Enterohemorrhagic E. coli (EHEC) pathotype are categorized within the larger Shiga-toxin producing E. coli (STEC) group [66]. Konowalchuk et al. discovered that certain E. coli produced toxins that were cytotoxic to vero cells, thus describing them as Vero-toxins or Vero-toxigenic E. coli [67,68]. O'Brien et al. discovered that a strain of E. coli linked to a hemorrhagic colitis outbreak, E. coli O157:H7, produced a Shiga-like toxin [69,70]. The research of the E. coli Vero-toxins, Shiga-toxins, and Shiga-like toxins met in the study conducted by Karmali that showed a significant correlation between patients displaying hemolytic uremic syndrome (HUS) and the recovery of Vero-toxin producing E. coli from their fecal samples [69,71]. Here, the terms of Shiga-toxin (Stx), Shiga-like toxin (SLTx), and Vero-toxin (VT) became synonymous in describing various strains of E. coli which produced toxins that caused severe illness among human patients including HUS and thrombic thrombocytopenic purpura (TTP) [68,69,71]. The Shiga-toxin is an A1B5 toxin [15,72]. The toxin causes death to various eukaryotic cell types, namely vero cells, by binding to the cell surface with the five B subunits and inserting the active A subunit into the cell [15]. The A subunit disrupts normal protein synthesis within the cell and ultimately leads to cell death [15,72]. General symptoms of infection include watery diarrhea, vomiting, muscle cramps, nausea, variable mild fever, variable blood in stool, and possible progression to the

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development of HUS or TTP [68,73,74]. The illness typically lasts five to seven days, but can cause permanent damage or death, especially when the individual is immunocompromised [73,74].

## Listeria monocytogenes

Listeria spp. have also been associated with several contaminated pet related foods, but to a lesser extent than Salmonella spp. The following categorization of the FDA recall cases was determined by the writer for use in this review. Listeria spp. were implicated in 19 of the 235 recalls, with the majority being in refrigerated/frozen meals and raw pet food diets [25]. A freeze-dried pet food and a fermented airdried pet food were also listed among the recalled items due to potential Listeria contamination [25]. Nine of the Listeria recalls were also associated with possible Salmonella contamination in the same products [25]. Nemser et al. reported the analysis of 480 dry and semimoist pet products that yielded a single positive Listeria in one dry cat food, separate from the dry cat food that recovered Salmonella. In the same study, 32 positive LM samples were exclusively isolated from raw pet food samples [31]. An additional 34 positive non-monocytogenes Listeria were isolated from other raw pet food samples and a single jerky-style treat [31].

Unlike Salmonella and E. coli serotypes, which can reside within certain host animals without causing illness, Listeria does not have a defined asymptomatic animal host [75-77]. Silage, milk bulk tanks, and ill cattle on dairy farms have been implicated as potential sources for Listerial biofilms [78,79]. Several Listeria strains have also been shown to survive in various adverse environments and food items for prolonged periods of time. Liao and Shollenberge showed that 27 Listeria strains were recoverable up to three years from storage in sterile phosphate buffer (SPB). LM, specifically, survived in SPB until the conclusion of a four week and a 30 week study [80]. Listeria has also been shown to survive in spray dried milk with a moisture content of 3.6-6.4% and pork rinds and cracklings at a water activity of 0.27 [81,82]. Due to the death of Listeria during pasteurization and other processing treatments, it is thought that most contamination occurs post processing [78,79,83]. Although there are no reported outbreaks directly linking human illness from Listeria spp. contamination in pet foods, the industry remains concerned due to the severity of the illness and the recent emergence of the organism in refrigerated/frozen meals and raw pet food diets [25].

The identification of Listeria, initially Bacterium monocytogenes, was attributed to Dr. Everitt Murray and colleagues who observed the bacteria upon the seemingly spontaneous deaths of several laboratory rabbits and guinea pigs in 1924 [84,85]. Listeria were not implicated in human disease until 1929, in Denmark, despite earlier possible isolations from human patients in Germany, 1893, and from France, 1891 and 1921 [85,86]. In 1927, Pirie observed the same bacteria in the liver of the African jumping mouse which he named Listerella hepatolytica [86]. The genus of the bacteria was later changed to Listeria in honor of the English surgeon and bacteriologist Joseph Lister [37,87]. Listeria are generally characterized as an invasive bacteria observed as small rounded rods [37]. Listeria are generally described as non-spore forming Gram-positive rods which are microaerophillic, motile via peritrichous flagella, psychotrophic (2.5 °C -25 °C ), are salt (NaCl) tolerant, and produce acid without gas from glucose [37,85,88]. Listeria, specifically LM, can cause a wide array of symptoms from gastrointestinal distress to septicemia, meningitis, spontaneous abortions, and death [88]. Laboratory research and recovery from outbreak samples have suggested a large

infective dose (>6.0-log cfu/g) is necessary to induce the typical nausea, vomiting, diarrhea, muscle cramps, and fever associated with gastrointestinal related febrile gastroenteritis [85,88-90]. It has also been suggested that outbreaks have been caused by 2.0-log to 4.0-log cfu/g and that precautions should be taken by immunocompromised individuals, especially pregnant women. LM are categorized into 14 serotypes with 1/2a, 1/2b, and 4b attributing to most of the human illness [91].

# **Animal Based Fats**

Contrary to the plethora of recalls and outbreaks related to pet food products, the extrusion process of pet foods has been deemed one of the most efficient and safest processing methods for dry shelf stable food with a prolonged shelf-life [2,6,21]. With the high temperature short time (HTST) process, resulting in a pathogen free product, it is thought that the majority of bacterial contamination results from additional industrial processes [2,6,16,92,93]. Similar to the thermal treatment of the extrusion process, the rendering process of animal fats uses processing temperatures intended to completely reduce the bacterial load within the final products [3,94-96]. The National Renderers Association recognizes treatment of animal fats ranging from 115°C to 146°C for an average of 40 min or more is a sufficient rendering process [95,97]. Since the thermal treatment experienced by the animal fats during the rendering process will kill any pathogenic bacteria of concern, the source of contamination must come postprocessing. These contaminated fats may subsequently contaminate dry pet foods when added as flavors and nutrient enhancements after the primary cooking and drying steps in the pet food production process.

It is suggested that various fats and oils can act as vehicles for microbial contamination through buffering capacities experienced during heat treatments [98-100]. Burnett, Gehm, Weissinger, and Beuchat reported that Salmonella spp. were capable of surviving in various peanut butters, reduced sugar and reduced sodium peanut butters, and reduced fat spreads for 24 weeks at 5°C and 21°C storage [101]. Juneja and Eblen displayed an increase in the lag time of Salmonella spp. within ground beef as the fat content increased, contributing to an overall increase in bacterial survival during heat treatment. This prolonged survival was contrasted with the rapid linear decline of Salmonella spp. populations within heated chicken broth (3% fat) [102]. Holliday et al. reported on the survival of Salmonella spp, E. coli O157:H7, and LM in various butters and yellow spreads over three weeks at 4.4 °C and 21 °C storage. Overall, the three genus of bacteria were reported to have higher surviving counts within the higher fat (>61%) products compared to the lower fat products [103]. The survival of the different bacterial populations was ultimately compromised due to the confounding influence of pH and preservatives within several of the inoculated products [103]. The phenomenon of increased heat resistance and prolonged survival rates in fatty products have been attributed to possible encapsulating and buffering capacities of the fatty acids within the lipid medium, as well as the uneven dissipation of heat through the lipid medium due to reduced water activity [98-100,102,104].

A review of the literature yielded no natural antimicrobial abilities associated with beef tallow, pig lard, or duck fat resulting in a reduction of *Salmonella, E. coli,* or *Listeria* populations. Beef tallow, pig lard, and poultry fat (namely duck) fatty acid composition varied within a wide range based on the sex, age, breed, geographic location, and diet of the animal [3,94,105,106]. Edible tallow is exclusively

derived from beef fat and bone sources that had been approved by USDA, or country specific, inspectors for human consumption [3,94]. Beef tallow can be refined, polished, and deodorized to make it more appealing for human consumption; it can also be left in a crude state for animal consumption [3,94]. Lard, also referred to as edible grease, is derived from the rendered adipose tissue of pigs that had been approved by the USDA, or country specific, inspector for human consumption [3,94,107]. The pet food industry is said to utilize anywhere from 10-20% of the entire annual yield of rendered poultry fat [3]. Duck fat would be a specific category of rendered poultry fat and would have duck as the sole source of fat.

The lack of natural antimicrobial activity within animal fats and their proposed ability to act as buffers during heat treatment processes supports the idea that contaminated post-rendered fats may be a source of contamination within the dry pet food industry. If the animal fats, contaminated post-rendering, are not the initial source of the contamination they may provide enough lipid buffering capacity to allow for the survival of pathogenic organisms from other sources during the coating process. Good hygiene, physical cleaning, and stringent sanitation procedures should be stressed throughout the pet food production process, especially the flavoring and enrichment areas. These areas should be considered a critical control point to prevent the introduction of food borne pathogens into the food products.

# Conclusion

The complete reduction of pathogenic microbial populations associated with the high temperature processing methods of both extrusion and fat rendering should ensure the safety of dry pet foods and pet related products. Two of the presented cases associated with human illness from dry pet foods cite poor hygiene and a lack of microbial testing associated with the flavoring and enrichment areas within the manufacturing plants. It is therefore necessary to consider the introduction of pathogenic organisms to dry pet foods through contaminated post-rendered fat sources and the potential ability of the fats to act as insulating lipid buffers, allowing the survival of pathogens from other sources. Further study into the ability of animal fats to harbor various pathogenic organisms is recommended.

# References

- 1. PFMA (2015) Types Of Dog Food.
- 2. Thompson A (2008) Ingredients: where pet food starts. Top Companion Anim Med 23: 127-132.
- 3. Aldrich G (2006) Rendered products in pet food. Essential rendering. All about the animal by-product industry. Virginia: National Renderers Association, p. 159-177.
- 4. Zicker SC (2008) Evaluating pet foods: how confident are you when you recommend a commercial pet food? Top Companion Anim Med 23: 121-126.
- 5. Laxhuber S (1997) Drying and cooling in production of extrudates. Kraftfutter 11: 460-466.
- 6. Tran Q (2008) Extrusion processing: effects on dry canine diets, in Wageningen Institute of Animal Sciences. Wageningen University and Research Center: Wageningen, The Neatherlands. p144.
- 7. Bone DP, Shannon EL (1977) Process for making a dry pet food having a hard component and a soft component, in US Google Patents.
- 8. Bren L (2011) Pet food: The lowdown on labels. FDA Veterinarian Newsletter.
- Erickson L (1982) Recent developments in intermediate moisture foods. J Food Prot 45: 484-491.

- 10. Miller RC (1985) Low moisture extrusion: Effects of cooking moisture on product characteristics. J Food Sci 50: 249-253.
- 11. Corbin J (2003) The history of petfood. Petfood technology, ed. JL Kvamme and TD Phillips. Mt. Morris, IL: Watt Publishing Company.
- 12. Dzanis DA (1994) The Association of American Feed Control Officials dog and cat food nutrient profiles: substantiation of nutritional adequacy of complete and balanced pet foods in the United States. J nutr 124: 2535-2539.
- 13. FDA (2016) Animal and Veterinary, FDA's Regulation of Pet Food.
- 14. Spears JK, Fahey GC Jr (2004) Resistant starch as related to companion animal nutrition. J AOAC Int 87: 787-791.
- 15. Harper JM, Clark JP (1979), Food extrusion. Crit Rev Food Sci Nutr 11: 155-215.
- Mościcki L, van Zuilichem DJ (2011) Extrusion Cooking and Related Technique. Extrusion-Cooking Techniques: Applications, Theory and Sustainability, ed. L Mościcki. Weinheim, Germany: Whiley-VCH Verlag and Co.
- 17. Dziezak J (1989) Single-screw and Twin-screw Extruders in Food Processing. Food Technol 43: 164-174.
- Serrano X, Agroturia S (1996) The extrusion-cooking process in animal feeding: Nutritional implications. Feed manufacturing in Southern Europe: New challenges Zaragoza 26. p114.
- Muthukumarappan K (2012) Advances in food extrusion technology. Chapter 9: Extrusion of Pet Foods and Aquatic Feeds. Boca Raton: Taylor & Francis Group: CRC Press.
- Rokey Ga (1995) Process description: Pet food production. Wenger Mfg, Inc.: Sabetha, KS, USA. Pp. 1-18.
- Kazemzadeh M (2012) Advances in food extrusion technology. Chapter 1: Introduction to Extrusion Technology. Advances in food extrusion technology, ed M Maskan and A. Altan. Boca Raton: Taylor & Francis Group: CRC Press.
- 22. Lin S, Hsieh F, Huff H (1998) Effects of lipids and processing conditions on lipid oxidation of extruded dry pet food during storage. Anim Feed Sci Technol 71: 283-294.
- 23. Engelen GMA, Poel AFB (1999) Post-pelleting application of liquid additives.
- 24. Killeit U (1994) Vitamin retention in extrusion cooking. Food Chem 49: 149-155.
- 25. FDA (2018) Animal and Veterinary. Recalls and Withdrawals.
- CDC (2006) Human salmonellosis associated with animal-derived pet treats--United States and Canada. Morbidity and mortality weekly report 55: 702.
- 27. PHAC (2000) Preliminary Report-Human Health Risk from Exposure to Natural Dog Treats - CCDR Volume 26-06 - Health Canada. Canada Communicable Disease Report (CCDR) 26: 41.
- Pitout JD, Reisbig MD, Mulvey M, Chui L, Louie M, et al. (2003) Association between handling of pet treats and infection with *Salmonella enterica* serotype Newport expressing the AmpC β-lactamase, CMY-2. J Clin Microbiol 41: 4578-4582.
- 29. Sato Y, Mori T, Koyama T, Nagase H (2000) *Salmonella virchow* infection in an infant transmitted by household dogs. J Vet Med Sci 62: 767-769.
- 30. White DG, Datta A, McDermott P, Friedman S, Qaiyumi S, et al. (2003) Antimicrobial susceptibility and genetic relatedness of Salmonella serovars isolated from animal-derived dog treats in the USA. J Antimicrob Chemother 52: 860-863.
- Nemser SM, Doran T, Grabenstein M, McConnell T, McGrath T, et al. (2014) Investigation of Listeria, Salmonella, and toxigenic Escherichia coli in various pet foods. Foodborne Pathog Dis 11: 706-709.
- 32. CDC (2008) Multistate outbreak of human Salmonella infections caused by contaminated dry dog food--United States, Morbidity and mortality weekly report 57: 521.
- 33. CDC (2008) Update: recall of dry dog and cat food products associated with human Salmonella Schwarzengrund infections--United States, Morbidity and mortality weekly report 57: 1200.

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- 34. CDC (2012) Multistate outbreak of human Salmonella Infantis infections linked to dry dog food (final update). Morbidity and mortality weekly report.
- 35. CDC (2012) Notes from the field: Human Salmonella infantis infections linked to dry dog food--United States and Canada, Morbidity and mortality weekly report, 61: 436.
- 36. FDA (2012) US Department of Health and Human Services. Food and Drug Administration. Inspectional Observations Atlanta USA.
- 37. Breed R (1957) Bergey's manual of determinative bacteriology. 7th ed Mt. Royal and Guilford Aves. Baltimore USA: The Williams & Wilkins Company.
- Pegues DA, Miller SI (2014) Salmonella Species, in Mandell, Douglas, and Bennett's principles and practice of infectious diseases, JE Bennett, Dolin R, Blaser MJ, Editor. Elsevier Inc pp. 2559-2568.
- Gossner CM, Hello SL, Jong B, Rolfhamre P, Faensen D, et al. (2016) Around the world in 1,475 Salmonella geo-serotypes. Emerg Infect Dis 22: 1298.
- Blaser MJ, Newman LS (1982) A review of human salmonellosis: I. Infective dose. Rev Infect Dis 4: 1096-1106.
- 41. CDC (2016) Salmonella Homepage.
- 42. CDC (2012) Pathogens causing US foodborne illnesses, hospitalizations, and deaths, 2000–2008. Fact Sheet.
- Scallan, E, Hoekstra RM, Angulo FJ, Tauxe RV, Widdowson MA, et al. (2011) Foodborne illness acquired in the United States — major pathogens. Emerg Infect Dis 17: 7-15.
- 44. Grimont PA, Weill FX (2007) Antigenic formulae of the Salmonella serovars. WHO collaborating centre for reference and research on Salmonella 9: 1-161.
- 45. Issenhuth-Jeanjean S, Roggentin P, Mikoleit M, Guibourdenche M, de Pinna E, et al. (2014) Supplement 2008–2010 (no. 48) to the White-Kauffmann-Le Minor scheme. Res microbiol 165: 526-530.
- 46. Popoff MY1, Bockemühl J, Gheesling LL (2004) Supplement 2002 (no. 46) to the Kauffmann–White scheme. Res microbial 155: 568-570.
- 47. FDA (2017) Safety. Archive for Recalls, Market Withdrawals & Safety Alerts.
- Cody SH, Glynn MK, Farrar JA, Cairns KL, Griffin PM, et al. (1999) An outbreak of Escherichia coli O157: H7 infection from unpasteurized commercial apple juice. Ann Intern Med 130: 202-209.
- Denny J, Bhat M, Eckmann K (2008) Outbreak of Escherichia coli O157: H7 associated with raw milk consumption in the Pacific Northwest. Foodborne Pathog Dis 5: 321-328.
- 50. Neil KP, Biggerstaff G, MacDonald JK, Trees E, Medus C, et al. (2011) A novel vehicle for transmission of Escherichia coli O157: H7 to humans: multistate outbreak of E. coli O157: H7 infections associated with consumption of ready-to-bake commercial prepackaged cookie dough—United States, 2009. Clin Infect Dis 54: 511-518.
- Riley LW, Remis RS, Helgerson SD, McGee HB, Wells JG, et al. (1983) Hemorrhagic colitis associated with a rare Escherichia coli serotype. N Engl J Med 308: 681-685.
- 52. Schimmer B, Nygard K, Eriksen HM, Lassen J, Lindstedt BA, et al. (2008) Outbreak of haemolytic uraemic syndrome in Norway caused by stx 2positive Escherichia coli O103: H25 traced to cured mutton sausages. BMC Infect Dis 8: 41.
- 53. MacDonald E, Dalane PK, Aavitsland P, Brandal LT, Wester AL, et al. (2014) Implications of screening and childcare exclusion policies for children with Shiga-toxin producing Escherichia coli infections: lessons learned from an outbreak in a daycare centre, Norway, 2012. BMC infect dis 14: 673.
- 54. Smith KE, Stenzel SA, Bender JB, Wagstrom E, Soderlund D, et al. (2004) Outbreaks of enteric infections caused by multiple pathogens associated with calves at a farm day camp. Pediatr Infect Dis 23: 1098-1104.
- 55. Kudva IT1, Hatfield PG, Hovde CJ (1997) Characterization of Escherichia coli O157: H7 and other Shiga toxin-producing E. coli serotypes isolated from sheep. J Clin Microbiol 35: 892-899.

- Wells JG, Shipman LD, Greene KD, Sowers EG, Green JH, et al. (1991) Isolation of Escherichia coli serotype O157: H7 and other Shiga-liketoxin-producing E. coli from dairy cattle. J ClinMicrobiol 29: 985-989.
- Beutin L, Geier D, Zimmermann S, Karch H (1995) Virulence markers of Shiga-like toxin-producing Escherichia coli strains originating from healthy domestic animals of different species. J Clin Microbiol 33: 631-635.
- Beutin L, Geier D, Steinrück H, Zimmermann S, Scheutz F (1993) Prevalence and some properties of verotoxin (Shiga-like toxin)-producing Escherichia coli in seven different species of healthy domestic animals. J Clin Microbiol 31: 2483-2488.
- 59. FDA (2002) BAM: Enumeration of Escherichia coli and the Coliform bacteria. Bacteriological Analytical Manual: Chapter 4: Enumeration of Escherichia coli and the Coliform bacteria.
- Shulman ST, Friedmann HC, Sims RH (2007) Theodor Escherich: the first pediatric infectious diseases physician? Clin infect dis 45: 1025-1029.
- Donnenberg M (2015) Enterobacteriaceae, in Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases, JE Bennett, Dolin R, Blaser MJ Editor Elsevier Inc.
- 62. Nataro JP, Kaper JB (1998) Diarrheagenic escherichia coli. Clin microbiol rev 11: 142-201.
- Prager R, Strutz U, Fruth A, Tschäpe H (2003) Subtyping of pathogenic Escherichia coli strains using flagellar (H)-antigens: serotyping versus fliC polymorphisms. Int J Med Microbiol 292: 477-486.
- 64. WHO (2016) WHO Collaborating Centre for Reference and Research on Escherichia and Klebsiella.
- Kaper JB, Nataro JP, Mobley HL (2004) Pathogenic escherichia coli. Nat Rev Microbiol 2: 123-140.
- Donnenberg MS, Whittam TS (2001) Pathogenesis and evolution of virulence in enteropathogenic and enterohemorrhagic Escherichia coli. J Clin Invest107: 539-548.
- 67. Konowalchuk J, Speirs JI, Stavric S (1977) Vero response to a cytotoxin of Escherichia coli. Infect immun 18: 775-779.
- Tarr PI, Gordon CA, Chandler WL (2005) Shiga-toxin-producing Escherichia coli and haemolytic uraemic syndrome. Lancet 365: 1073-1086.
- 69. Kaper JB, O'Brien AD (2014) Overview and historical perspectives. Microbiol spectr 2: 1.
- 70. O'Brien AO, Lively TA, Chen ME, Rothman SW, Formal SB (1983) Escherichia coli 0157: H7 strains associated with haemorrhagic colitis in the United States produce a Shigella dysenteriae 1 (Shiga) like cytotoxin. Lancet 321: 702.
- 71. Karmali MA, Petric M, Lim C, Fleming PC, Arbus GS, et al. (1985) The association between idiopathic hemolytic uremic syndrome and infection by verotoxin-producing Escherichia coll. J Infect Dis 151: 775-782.
- 72. Melton-Celsa AR (2014) Shiga toxin (Stx) classification, structure, and function. Microbiol spectr 2: 1.
- 73. CDC (2017) E.coli (Escherichia coli).
- 74. Thorpe CM (2004) Shiga toxin—producing Escherichia coli infection. Clin infect dis 38: 1298-1303.
- 75. Ferens WA, Hovde CJ (2011) Escherichia coli O157: H7: animal reservoir and sources of human infection. Foodborne pathog dis 8: 465-487.
- Kourany M, Myers CW, Schneider CR (1970) Panamanian amphibians and reptiles as carriers of Salmonella. Am J Trop Med Hyg 19: 632-638.
- Winfield MD, Groisman EA (2003) Role of nonhost environments in the lifestyles of Salmonella and Escherichia coli. Appl environ microbial 69: 3687-3694.
- Borucki MK, Reynolds J, Gay CC, McElwain KL, Kim SH et al. (2004) Dairy farm reservoir of Listeria monocytogenes sporadic and epidemic strains. J food prot 67: 2496-2499.
- Muraoka W, Gay C, Knowles D, Borucki M (2003) Prevalence of Listeria monocytogenes subtypes in bulk milk of the Pacific Northwest. J food prot 66: 1413-1419.

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- 80. Liao CH, Shollenberger LM (2003) Survivability and long term preservation of bacteria in water and in phosphate buffered saline. Lett appl microbial 37: 45-50.
- Doyle MP, Meske LM, Marth EH (1985) Survival of Listeria monocytogenes during the manufacture and storage of nonfat dry milk. J Food Prot 48: 740-742.
- 82. Ingham SC, Buege DR, Dropp BK, Losinski JA (2004) Survival of Listeria monocytogenes during storage of ready-to-eat meat products processed by drying, fermentation, and/or smoking. J food prot 67: 2698-2702.
- Tompkin R (2002) Control of Listeria monocytogenes in the foodprocessing environment. J food prot 65: 709-725.
- Murray E, Webb RA, Swann M (1926) A disease of rabbits characterized by a large mononuclear leucocytosis, caused by a hitherto undescribed bacillus Bacterium monocytogenes (n. sp.). J Pathol Bacteriol 29: 407-439.
- 85. Vázquez-Boland JA, Kuhn M, Berche P, Chakraborty T, Domínguez-Bernal G, et al. (2001) Listeria pathogenesis and molecular virulence determinants. Clin microbiol rev 14: 584-640.
- Gray ML, Killinger A (1966) Listeria monocytogenes and listeric infections. Bacteriol rev 30: 309-382.
- 87. Pirie JH (1940) Listeria: Change of Name for a Genus of Bacteria. Nature p145.
- Lorber B (2015) Listeria monocytogenes, in Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases, JE Bennett, Dolin R, Blaser MJ, Elsevier Inc.
- Dalton CB, Austin CC, Sobel J, Hayes PS, Bibb WF, et al. (1997) An outbreak of gastroenteritis and fever due to Listeria monocytogenes in milk. N Engl J Med 336: 100-106.
- 90. Farber JM, Daley E, Coates F, Beausoleil N, Fournier J (1991) Feeding trials of Listeria monocytogenes with a nonhuman primate model. J Clin Microbiol 29: 2606-2608.
- 91. Borucki MK, Call DR (2003) Listeria monocytogenes serotype identification by PCR. J Clin Microbiol 41: 5537-5540.
- Kukanich KS (2011) Update on Salmonella spp contamination of pet food, treats, and nutritional products and safe feeding recommendations. J Am Vet Med Assoc 238: 1430-1434.
- 93. Podolak R, Enache E, Stone W, Black DG, Elliott PH (2010) Sources and risk factors for contamination, survival, persistence, and heat resistance of Salmonella in low-moisture foods. J food prot 73: 1919-1936.

- 94. Alm M (2013) The Production of Triglyceride Oils: Animal Fats, in Edible Oil Processing, AJ Dijkstra USA.
- 95. Meeker DL, Meisinger JL (2015) Companion Animals Symposium: Rendered ingredients significantly influence sustainability, quality, and safety of pet food. J anim sci 93: 835-847.
- 96. OIE Ad hoc Group (2002) OIE Ad hoc Group on Carcass Disposal. OIE ad hoc Group, Paris.
- 97. Meeker DL, Hamilton C (2006) Essential Rendering: All about the Animal By-Products Industry. An overview of the rendering industry ed. DL Meeker. Arlington, Virginia: National Renderers Association.
- Molin N, Snygg BG (1967) Effect of lipid materials on heat resistance of bacterial spores. Appl microbiol 15: 1422-1426.
- 99. Senhaji A, Loncin M (1977) The protective effect of fat on the heat resistance of bacteria (I). Int J Food Sci Technol 12: 203-216.
- 100. Zuccaro JB, Powers JJ, Morse RE, Mills WC (1951) Thermal death times of yeast in oil and movement of the yeast between the oil and water phases of French dressing. J Food Res 16: 30-38.
- 101. Burnett SL, Gehm ER, Weissinger WR, Beuchat LR (2000) Survival of Salmonella in peanut butter and peanut butter spread. J Appl Microbiol 89: 472-477.
- 102. Juneja VK, Eblen BS (2000) Heat inactivation of Salmonella typhimurium DT104 in beef as affected by fat content. Lett Appl Microbiol 30: 461-467.
- 103. Holliday SL, Adler BB, Beuchat LR (2003) Viability of Salmonella, Escherichia coli O157: H7, and Listeria monocytogenes in butter, yellow fat spreads, and margarine as affected by temperature and physical abuse. Food microbiol. 2: 159-168.
- 104. Senhaji A (1977) The protective effect of fat on the heat resistance of bacteria (II). Int J Food Sci Technol 12: 217-230.
- 105. Gunstone F (1996) Typical fatty-acid composition of some common fats. Fatty Acid and Lipid Chemistry.
- 106. Witak B (2008) Tissue composition of carcass, meat quality and fatty acid content of ducks of a commercial breeding line at different age. Archiv fur Tierzucht 51: 266-275.
- 107. Rohman A, Triyana K, Erwanto Y (2012) Differentiation of lard and other animal fats based on triacylglycerols composition and principal component analysis. Int Food Res J 19:1.