Peanut and peanut products: A food safety perspective

Alexandra S. Chang a, Aswathy Sreedharan b, Keith R. Schneider b,*

aLand O’Lakes, Inc. P.O. Box 64101, MS 0075, St. Paul, MN 55164-0101, USA
bFood Science and Human Nutrition Department, University of Florida, 359 FSHN Bldg., Newell Drive, Gainesville, FL 32611-0370, USA

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A B S T R A C T

Peanuts are a common food allergen. The increased prevalence of peanut food allergy in recent years has led food processors to be more proactive in their responsibility for preventing peanut contamination by implementing good manufacturing practices (GMPs) and allergen control programs. Further, safety of peanuts and peanut-derived products must be considered throughout production to minimize risk of foodborne disease. Peanuts can be a source of aflatoxin, produced by the mold Aspergillus spp., the cause of liver defects and cancer, especially in developing countries. Though historically not associated with foodborne disease outbreaks, recent events have put peanut products in the limelight. Perhaps the most well-known peanut-related food safety issue has been the peculiar relationship between peanut butter and Salmonella. Though there have only been a few outbreaks caused by Salmonella in peanut butter, they have been prominent and widespread. The costly multistate outbreak of 2009 involving a Georgia peanut manufacturer influenced recognition of the importance of corporate responsibility and GMPs. [Federal regulations to help prevent such outbreaks have also been developed including the proposed Food Safety Modernization Act of 2011.] Prevention of outbreaks is best accomplished through cooperation within the food industry community and with regulators to implement effective GMPs and other prevention-based food safety programs. These, and other peanut food safety issues are discussed in this review.

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1. Introduction

Peanuts (Arachis hypogaea) are an inexpensive, nutritionally powerful food source for people worldwide (Reese & Lehrer, 1999). Over one billion pounds of peanuts are produced annually in the US alone, most of which remains in the country for human consumption. Peanuts are consumed whole roasted and as a variety of products including peanut butter, peanut oil, and other forms as ingredients. Improved diet nutrient profiles and lower body mass index (BMI) have been linked to eating peanuts (Griel, Eisenstat, Juturu, Hsieh, & Kris-Etherton, 2004). Though they are energy-dense, peanuts provide satiety, low energy absorption, and increased energy expenditure after consumption so that they do not significantly contribute to increased body weight (Mattes, Kris-Etherton, & Foster, 2008). Given the health benefits and scope of peanut consumption worldwide, it is important that consumers are able to enjoy a safe peanut supply.

Peanut is one of the eight most common food allergens. The increased prevalence of peanut food allergy in recent years has led food processors to be more conscientious in their good manufacturing practices (GMPs) and allergen control programs to prevent peanut contamination. Further, safety of peanuts and peanut-derived products must be considered throughout production for foodborne disease risk. While they have not been associated with many foodborne disease outbreaks, peanuts can be a source of aflatoxin, produced by the mold, Aspergillus spp., which can cause liver defects and cancer, especially in developing countries (Wild & Gong, 2010). More recently, peanut butter has been in the spotlight as the source of foodborne disease outbreaks caused by Salmonella (CDC, 2007; 2009). In light of the 2009 multistate outbreak involving numerous peanut products from a Georgia peanut manufacturer, corporate responsibility and GMPs are being increasingly recognized and important (US FDA, 2009a). Federal regulations to prevent such outbreaks have also been developed. This review article will provide an overview of major safety concerns in peanut and peanut products production.

2. Peanuts

2.1. Production

Peanuts, also known as a goober, groundnut, earthnut, and pinder, among other names, originated in South America around 950 BC. Peanuts were brought to Africa by early explorers, who then transported them to North America in the colonial age. Peanuts were first used primarily as animal feed and human consumption began only in the late 19th century (Woodroof, 1983). Today, the average American consumes approximately 6.4 lbs of peanuts a year, about half of which is in the form of peanut butter (USDA 2010).

World production of peanuts is estimated at 40 billion pounds (Woodroof, 1983). The US comprises about 10% of world production, producing approximately 4.2 billion lbs in 2010 with a value of over $900 million (USDA, 2011a; b). Value increased over $100 million from 2009, but in previous years the peanut industry achieved over $1 billion in sales (USDA, 2011b). Georgia is the number one producer of peanuts, comprising almost half of US production and value. In total, over one million acres of peanuts are planted and harvested in the US each year (USDA, 2011a; b). Approximately 60% of total production is dedicated for human consumption and the remaining is used for seed, animal feed, and oil. Of the peanuts used for human consumption, approximately 63% is processed into peanut butter (Woodroof, 1983).

Peanuts are the fruit or pod of annual legumes, the plants belonging to the family Leguminosae. Legumes also include peas, beans, and soybeans (Reese & Lehrer, 1999). The central stem of the peanut plant produces flowers. Pods containing one to three seeds develop in branches and are pushed underground where they mature. Branch-type peanuts grow closely in clusters at the bottom of branches while runner-type peanuts grow pods throughout the branches (Woodroof, 1983).

Woodroof (1983) describes the following process for peanut production. Peanuts are harvested by mechanical inverting, digging, then picking. At digging, peanuts are at 35–50% moisture. Entire plants are removed from the ground and formed into windrows, which are dried in open air. After a specific time, peanuts are mechanically picked and brought to a processing facility where they are dried by fan at 35°C (95°F) to 10% moisture. Peanuts are allowed to mature as determined by the internal surface color of shell of a representative sample. During cleaning, dirt, stones, plant material, and other debris are removed by screens and blowers. Peanuts can also be stored under conditions that maintain moisture below 10%. Temperature should be maintained between 0 and 10°C (32–50°F). If shelled, peanuts must be stored at a lower relative humidity to maintain low moisture content. Shelled peanuts can be stored a maximum of 18 months compared to 24 months for in-shell peanuts.

The critical control point in peanut processing is roasting, which can be performed in two ways, dry and oil roasting. In dry roasting, peanuts reach 160°C in an oven of 400°C for 25–60 min (roasting times vary based on batch properties and final product characteristics desired). Peanuts are then cooled slightly and undergo a blanching process, where peanut skins, dust and other foreign materials are blown off. In oil roasting, peanuts are blanched and then fried at 140°C for 3–10 min. Roasted peanuts have approximately 1.25% moisture content (Woodroof, 1983).

2.2. Areas of concern

Consumption of peanuts and peanut products can induce potential life threatening anaphylactic reactions to individuals who are allergic to peanuts. Currently, since there are no effective curative treatments available, the most effective way to deal with peanut allergies is to strictly avoid peanut and peanut products. However, accidental exposures are frequent due to the possibility of cross contamination of other foods. Hence, it is imperative that the food processing industries follow strict guidelines to ensure that the food prepared is safe for allergic consumers, by following good manufacturing practices to reduce cross contamination, and by including proper advisory labeling on packages.

Peanuts do not pose a large risk for foodborne disease largely due to the roasting step where peanuts are reduced to the 1.25% moisture content and <0.75 water activity (a_w). Moisture is required for most microorganisms to survive. The low a_w inhibits growth of most bacteria and many molds. Since peanuts are rarely eaten raw, roasting not only improves peanut aroma, flavor, and texture, but also destroys contaminating microorganisms (Woodroof, 1983).

Very few outbreaks of foodborne diseases have been attributed to whole peanuts and any outbreaks that have occurred were likely due to poor handling practices after a thermal treatment step, particularly roasting. Proper peanut processing and handling post-harvest should ensure a safe product for consumers. Instead, from the foodborne diseases perspective, areas of concern for peanuts revolve primarily around contamination from aflatoxin, a mycotoxin produced by the mold Aspergillus, and cross contamination from sources that introduce pathogens to peanuts after processing.

2.2.1. Allergens in food processing and product formulation

Peanut is one of eight major food allergens of humans, which also include milk, egg, soy, wheat, tree nuts, fish, and shellfish.
Estimated prevalence of food allergies among adults in North America is 3–4% (Sicherer & Sampson, 2010). In 2007, the Centers for Disease Control and Prevention (CDC) reported approximately 3.9% (3 million) children in the US had food allergies (Branum & Lukacs, 2008). From 1997 to 2007, prevalence of food allergy increased 18% among children. Additionally, hospitalizations related to food allergies have increased, and children with food allergy were more likely to report having asthma. A more recent study reported a higher prevalence of food allergies, at 8.0% of children in the US, 25.2% of which were allergic to peanut (Gupta et al., 2011). Kotz, Simpson, and Sheikh (2011) reported prevalence of peanut allergy in children in the US to be 0.4–1.9%.

A food allergy is generally described as an adverse reaction to food (Bischoff & Sellge, 2003). Immunoglobulin E (IgE) mediated food allergy, also known as type I hypersensitivity, is considered a true food allergy as it elicits an immune response after multiple exposures to an allergen. Food allergens are antigens or proteins with which antibodies in the human body bind. The initial exposure to an antigen is known as the sensitization phase in which no symptoms occur but specific IgE antibodies that can bind to the antigen are produced in larger quantities (Bischoff & Sellge, 2003). Subsequent exposure to the antigen causes mast cells, basophils, and eosinophils to degranulate, releasing prostaglandins, leukotrienes and other immune mediators. Immune reactions caused by immune mediators vary in severity, but can include a tingling sensation around the mouth, urticaria, rhinitis, asthma, anaphylactic shock, and death within an hour of exposure (Branum & Lukacs, 2008; Reese & Lehrer, 1999).

Peanut is often considered the most severe allergy, the major allergens being Ara h 1 and Ara h 2, of 63.5 and 17.0 kDa molecular weight, respectively (Reese & Lehrer, 1999). The threshold at which allergic individuals experience symptoms is measured by the Lowest Observed Adverse Effect Level (LOAEL). The range of LOAEL doses in clinical trials has ranged from 0.5 to 10,000 mg of whole peanut, indicating high variation in allergic response in a population. A dose predicted to provoke a reaction in 10% of the peanut allergic population was estimated to be 18.0 mg of whole peanut (Taylor, Crevel, Sheffield, Kabourek, & Baumert, 2009).

The increasing prevalence of peanut allergy has garnered scientific attention. One study noted the lower prevalence of peanut allergy in China compared to in the US, perhaps because peanuts are usually fried or boiled instead of roasted (Beyer et al., 2001). Researchers found fried and boiled peanuts to contain less Ara h 1 and elicit less IgE binding of Ara h 2 and other allergens compared to roasted peanuts. Newer technologies such as pulsed light, sanitation, sanitation validation, and effective packaging. Effective allergen control programs can reduce number of allergen-related recalls and incidences leading to hospitalization or death.

2.2.2. Mycotoxins
Mycotoxins are produced by various fungi and are considered poisonous contaminants in susceptible foods and feeds. Aflatoxin has been identified as the most toxic mycotoxin associated with peanuts, and hence the toxicity, and measures to manage and prevent contamination by aflatoxin has been discussed in detail in this review. In addition to aflatoxins, another commonly occurring natural contaminant of peanut is the mycotoxin cyclopiazonic acid, produced by several species of Penicillium and Aspergillus (Landsen & Davidson, 1983). Cyclopiazonic acid is a potent inhibitor of the reticular form of the Ca$^{2+}$ ATPase pump (Plenge-Tellechea, Soler, & Fernandez-Belda, 1997). The compound is not considered to be a potent toxin in humans due to low oral LD50 values, in the range of 30–70 mg/kg in rodents (Antony, Shukla, & Janardhanan, 2003; Nishie, Cole, & Dorner, 1987). On peanuts, the natural level of contamination by this toxin is only 6.5 ppm (Landsen & Davidson, 1983), thus, the compound is toxic to humans only when it is consumed at levels that exceed the natural level of intake of the toxin (Burdock & Flamm, 2000; Van Rensburg, 1984). Further, in most cases, both cyclopiazonic acid and aflatoxin, both produced by Aspergillus flavus, are present concurrently (Urano et al., 1992), and this effectively disguises the presence of cyclopiazonic acid. A study conducted by Van Rensburg (1984) concluded that in the presence of usual hygiene and aflatoxin control practices in peanuts, no further control or screening measures for cyclopiazonic acid is warranted.

Although to a much lesser extent, there are also reports of natural occurrence of other mycotoxins associated with peanuts, including zearealenone (El-Maghraby & El-Maraghy, 1987; Kishore, Pande, Manjula, Rao, & Thomas, 2002; Mehan & McDonald, 1982), and trichothecene-toxins (Bhanavishankar & Shantha, 2006; El-Maghraby & El-Maraghy, 1987), both produced by Fusarium sp.; citrinin (El-Maghraby & El-Maraghy, 1987; Subrahmanjan & Rao, 1974), produced by Penicillium sp., Aspergillus sp. and Monascus sp.; and ochratoxin A (Magnoli et al., 2007) produced by Aspergillus sp.
Aflatoxin is identified as a known human carcinogen by the International Agency for Research on Cancer (2002), p. 301. A. flavus and Aspergillus parasiticus (commonly found in the soil) are the primary species of mold that produce aflatoxin as a secondary metabolite. Originally discovered in the 1960s, aflatoxin is one of several mycotoxins that persist in peanuts, tree nuts, oilseeds, and cereal grains, especially in developing countries where these commodities are grown and consumed (Wild & Gong, 2010). Optimal conditions for A. flavus growth are 12–35°C moisture at 27–38 °C (80–100 °F) (Woodroof, 1983). Once infected, the mold can proliferate in improperly stored peanuts, particularly in tropical regions.

The types of aflatoxin are B1, B2, G1, and G2 (IARC, 2002, p. 301). Aflatoxin B1 is the most ubiquitous form and the most toxic (Kamika & Takoy, 2011; Wild & Gong, 2010). Major effects of aflatoxin are hepatocarcinoma, immunosuppression leading to increased susceptibility to infections, and growth impairment in children due to the ability of aflatoxin to cross the human placenta (IARC, 2002, p. 301). Incidentally, chronic hepatitis B and hepatitis C infection are risk factors for hepatocarcinoma caused by aflatoxin (Bhat, Rai, & Karim, 2010). It is hypothesized that the viral infections interfere with the ability of hepatocytes to metabolize aflatoxin. The toxin resides in the liver for a longer period, causing damage to tumor suppressor genes. An immediate consequence of aflatoxin exposure can be aflatoxicosis, or aflatoxin poisoning. Signs and symptoms include gastrointestinal problems and liver lesions (Bhat et al., 2010). Exposure to amounts less than 1000 ppb have been linked to aflatoxicosis. Consuming approximately 5000 ppb of aflatoxin can cause acute aflatoxicosis leading to death. The LD50 value of aflatoxin ranges from 0.3 to 10 mg/kg for most animal species, and from 0.54 to 1.62 mg/kg for human beings (Wild & Gong, 2010).

Aflatoxin content in peanuts is controlled. In the US, aflatoxin content in peanuts must be < 15 ppb to be certified edible quality grade (CFR, 2010a). The Food and Drug Administration (FDA) will pursue legal action if peanut and peanut products are found to contain at least 20 ppb aflatoxin (US FDA, 2000). It is a common difficulty to obtain a representative sample from a truckload of peanuts for testing, however (Doughtie, 1947). Representative sampling of peanuts is important because of non-uniform distribution of aflatoxin in a batch of peanuts. The level of contamination can also vary. Specific guidelines for acceptable levels of aflatoxin in food and feed, and sampling plans for sample acceptance or rejection have been established by different countries (Van Egmond, 1989; Whitaker, Springer, Defize, DeKoe, & Coker, 1995). In the US, the peanut marketing agreement established by the USDA, is administered by the Peanut Administrative Committee (PAC). According to the current aflatoxin sampling plan followed in the US, raw shelled peanuts are accepted or rejected based on a modified sequential plan where up to three 21.8 kg each representative test samples are drawn from a single lot (Whitaker & Dowell, 1995; USDA, 2004). Peanuts are finely ground into a composite since aflatoxin can exist on the order of parts per billion. Prevention of Aspergillus growth by effectively drying and storing peanuts at low relative humidity and temperature is the best way to prevent aflatoxin production. Genetically modified seeds resistant to Aspergillus have been developed but are expensive (Wild & Gong, 2010). If peanuts are contaminated, various treatments exist such as applying ammonia, hydrogen peroxide, and ozone, although the side effects of these treatments are not currently reported in detail (Bhat et al., 2010; Scott, 1998). Irradiation and high temperature roasting can also eliminate aflatoxin contamination (Bhat et al., 2010; Ogbadu, 1980; Staron, Thirouin, Perrin, & Frere, 1980). With ongoing research, many of these options may become more cost effective in the future.

2.2.3. Cross contamination and pathogen survival

Implementation and adherence to good agricultural practices (GAPs) and GMPs should prevent contamination and foodborne diseases caused by peanuts. What becomes a concern is the survival of pathogens after contamination occurs, post-harvest and/or post-processing. Cross contamination can occur anytime during peanut production and turn a low moisture, safe food into one that can cause foodborne disease. Pathogens can be transferred in several ways to food, such as through contaminated water and equipment, poor worker hygiene, and pests. If peanuts are contaminated, survival of pathogens becomes a major issue.

It is generally understood that low aW of a food prevents growth of many microorganisms. To verify the low association of pathogens and peanuts, Eglezos (2010) tested 343 samples of ready-to-eat peanuts and samples of other nuts produced at Australian facility over three years for Salmonella, other pathogens, and aerobic plate count (APC). No pathogens were found in any sample. In this study, APC tests revealed that 48% of the peanut samples show counts above the detection limit of 100 CFU/g. These positive samples had average plate counts of 2.7 log CFU/g. High APC counts do not necessarily indicate the presences of a foodborne pathogen, but instead, are a general indicator of sanitation and can assist peanut processors in identifying where sanitation can be improved.

Dry foods like peanuts do not often support microbial growth, but they may still be able to allow survival of pathogens. Komitopoulou and Penaloz (2009) found that inoculated Salmonella could survive for 3–4 weeks on dry, raw materials including crushed cocoa and hazelnut shells, cocoa beans, and almond kernels at both room temperature and 5 °C. Salmonella is typically inhibited by aW < 0.91, but the aW of these substances were likely much lower. It can be concluded that any heat treatment of food must be sufficiently high to destroy Salmonella while still providing a quality product. This is especially important for dry foods with long shelf life because if pathogens were to survive during processing, they would more likely reach the consumer and increase risk of disease.

2.2.4. Peanut outbreaks

Foodborne disease outbreaks are not typically associated with peanuts and very few peanut outbreaks have been documented. In one international outbreak, Salmonella Stanley, S. Newport and other strains were found in packages of a brand of roasted, in-shell peanuts imported from Asia from May to October 2001 (Kirk et al., 2004). There were 109 cases total in Australia, Canada, England, Wales, and Scotland. Positive samples were found to contain < 0.03–2 CFU/g Salmonella. The peanuts were manufactured in one unidentified Asian country and distributed by several other countries. The original source of contamination was undetermined (Kirk et al., 2004).

The first Salmonella outbreak in the US associated with boiled peanuts occurred in 2006 at the Pumpkin Festival in Pumkpintown, SC. Twenty three cases were reported. While boiling should destroy any vegetative microorganisms in peanuts, in this outbreak, contamination by S. Thompson occurred after boiling and salting. Poor handling, inadequate temperature control, and possible ladle contamination all could have been prevented by following proper food service procedures (Christian et al., 2007). Other documented outbreaks have been associated with peanut products, especially peanut butter, and are described in section 3.2.1 and 3.3.

3. Peanut products

3.1. Peanut oil

About two-thirds of the world peanut crop is used for oil. In the US, peanut oil is primarily used as cooking oil or salad oil.
Survive for 24 weeks in peanut butter at 20°C. Ported that another pathogen, L. monocytogenes, despite its low inoculation level of 4 log CFU/g. Although no illness was reported, 3 log CFU/g after 1 week at 5°C was detected in a rural farming community in China. The resulting crude oil undergoes solvent extraction, refining, water washes, and bleaching. The resulting peanut oil is often used for frying. Foods containing peanut protein placed in oil may be transferred to non-allergenic food fried in the same oil afterward.

Aflatoxins are primarily a problem for whole peanuts, not peanut oil. In one study, peanut oil extracted from peanuts contained aflatoxin with 5500 ppb aflatoxin. Aflatoxin was found in a final product with <1 ppb aflatoxin. Peanut oil produced and used in developing countries, however, may be of higher risk. For example, researchers studying the high incidence of hepatocellular carcinoma in a rural farming community in China detected aflatoxin in peanut oil. Of 30 samples, 20 had detectable levels of AFB1, ranging from 0.1 to 52.5 ppb. Peanut butter is processed in the same way as peanuts up to the roasting stage, at which time peanuts are finely ground into a paste and vacuum packaged. The standard of identity of peanut butter is similarly defined in the Code of Federal Regulations, which also allows for seasoning and stabilizers. Peanut butter has 1% moisture and aw of 0.2–0.33.

Similar to peanuts, Salmonella can survive in peanut butter despite its low aw. Burnett et al. (2000) found that with an initial inoculum of 5.68 log CFU/g, Salmonella was found at greater than 3 log CFU/g after 1 week at 5°C and 21°C. Even after 24 weeks, approximately 2 and 1 log CFU/g Salmonella was detected at 5°C and 21°C, respectively. A 3-strain cocktail of Salmonella tennessee inoculated in peanut butter survived for 2 weeks at 4 and 22°C. Further, Kenney and Beuchat (2004) reported that another pathogen, Listeria monocytogenes could also survive for 24 weeks in peanut butter at 20°C with an initial inoculation level of 4 log CFU/g. Although no illness was reported, potential contamination by L. monocytogenes also prompted nationwide recalls of certain brands of peanut butter, and peanut butter & jelly sandwiches in 2010 and 2011 respectively (US FDA, 2010; US FDA, 2011a). Results of these studies suggest that any pathogen that contaminates peanut butter after heat processing could remain in the product throughout its shelf life.

### 3.2. Salmonella

Despite being one of the leading causes of foodborne disease in the US, Salmonella has not been traditionally associated with peanut butter. The first documented peanut butter outbreak occurred in 1986 in Australia caused by S. mbandaka. However, the first peanut butter outbreak occurred in the US, and this multistate outbreak of S. tennessee was attributed to moisture from a leaky roof of a Sylvester, GA processing plant. In total, 628 people were infected in 47 states. Age of patients ranged from two months to 95 years and 20% of infected individuals were hospitalized (CDC, 2007). The manufacturer took steps to prevent future outbreaks by redesigning the plant, installing new equipment, and appointing a food safety advisory committee.

A complex, multistate outbreak in 2009 was caused by Salmonella typhimurium associated with peanut butter and peanut products produced by a Georgia peanut manufacturer. They manufactured peanut butter sold in bulk to institutions, food service and private label companies. The first suspected product that was the common food consumed by infected patients was a brand of peanut butter produced at the GA plant (CDC, 2009). Peanut butter and other peanut products were used as ingredients in numerous other products. In total, over 3900 products were recalled from over 200 companies. The outbreak caused 714 cases in 46 states and 1 in Canada, 171 hospitalizations and 9 deaths (CDC, 2009).

These high-profile outbreaks will associate Salmonella with peanut butter for years to come. Many peanut butter companies likely adopted Salmonella and other pathogen testing as part of their routine microbiological testing. In March 2011, the FDA issued a recall of a reduced fat peanut butter spread because of possible contamination with Salmonella (US FDA, 2011b). Salmonella was found in the finished product as part of the company’s routine testing program, though no illnesses had been reported.

#### 3.2.2. Control technologies

The peanut butter outbreaks have inspired adaptation of new processing techniques such as electron beam radiation to control pathogens in peanut butter (Hvizdzak, Beamer, Jaczynski, & Matak, 2010; Matak, Hvizdzak, Beamer, & Jaczynski, 2010). These technologies need to be further examined for scale-up feasibility and efficacy. For example, a study examining high-pressure processing (HPP; 600 MPa for 5 min at 45°C) against inoculated Salmonella in peanut butter found only <2 log unit reductions (Grasso, Somerville, Balasubramaniam, & Lee, 2010). Since a 6.7 log unit reduction by HPP was achieved in a mixture of peanut flour and water, the fat content in oil could have provided protection for pathogens.

Thermal treatment of peanut butter may not always be effective as well. Typically, Salmonella can be destroyed by heating food to 71°C for a few seconds. Yet, Salmonella has been shown to survive in peanut butter heated to 90°C (Shachar & Yaron, 2006). A possible explanation is that the high fat content provides local buffer regions that allow Salmonella to survive with increased heat resistance. Heat shock proteins can be produced by bacteria, allowing them to survive better in adverse environmental conditions. Furthermore, a study modeling Salmonella thermal inactivation curves showed outbreak strains of S. tennessee to have increased heat resistance compared to strains that caused sporadic cases (Ma et al., 2009). The minimum time necessary to reduce the outbreak strains by 7 log units at 90°C was approximately double the time required for other strains, at 120 min compared to 55–86 min, respectively (Ma et al., 2009). This finding suggests peanut butter and other high fat, low aw foods cannot simply be heated to destroy all potential Salmonella. Additionally, a sub-lethal heat process that would normally function as a critical kill step under the correct temperature requirements could instead induce new stress response mechanisms in bacteria (Sirsat et al., 2011).

The simplest peanut butter contamination control methods may be GMPs. GMPs address sanitary water use, sanitary facilities, personal hygiene, plant sanitation, proper transportation, and traceback. GMPs and other prevention-based food safety programs must be implemented to prevent any initial contamination to the product. It is possible that the majority of outbreaks can be prevented by following proper GMPs.
3.3. Peanut-derived products

Other peanut products including peanut flour, peanut granules, peanut meal, peanut paste, and peanut protein are used as ingredients. Peanuts in these various forms are used in a variety of food products including confectionery, bakery type desserts, savory snacks, pet food, etc. Each type of food uses a different processing method, so the control of peanut safety may be specific to each operation.

Peanut as an ingredient must also be considered for food safety risk due to the occurrence of recent outbreaks. An outbreak of disease associated with a kosher peanut snack imported from Israel occurred in 1994. Twenty seven cases of *Salmonella* agona infection, 26 of which were children, were reported in England and Wales. An outbreak of *S. agona* was already occurring in Israel at the time. Positive samples ranged from 2 to 45 CFU/packet (Killala et al., 1996).

The largest outbreak related to peanut derived products involved a Georgia peanut manufacturer in 2009. Besides peanut butter, they also manufactured roasted peanuts used in snack mixes and peanut paste used in cookies, crackers, cereal, candy, ice cream, pet treats, and other products. After epidemiological investigations pointed to a Georgia peanut manufacturer as the source of the outbreak, the FDA investigated their plant in January 2009. The FDA observed numerous poor manufacturing practices including the use of unclean equipment, storing raw peanuts next to roasted peanuts (i.e., finished product), using uncleanable materials on equipment, using the same sink for handwashing and cleaning, allowed drips inside the plant and onto product. Product was stored near cracks in floors and walls that were contaminated with *Salmonella*. While the manufacturer recognized roasting as a critical control point, they did not validate and properly document the oven temperature (US FDA, 2009b).

Perhaps the most egregious mistake by the Georgia manufacturer was disregarding their internal food safety program. The manufacturer sent samples to third party laboratories for microbiological analyses. When the samples were positive for *Salmonella*, they resubmitted samples possibly hoping for negative results that would negate the positives. Regardless of results, product was shipped to buyers including institutions with high-risk populations. The same incident occurred 12 times over two years before the 2009 outbreak. Even after receiving positive *Salmonella* results, the manufacturer continued to use equipment that had processed contaminated peanut paste without first cleaning it (US FDA, 2009b). Amazingly, this manufacturer remained in operation for years without incident, when any one of these poor manufacturing practices could have caused an outbreak.

In order to prevent such large outbreaks in the future, the FDA issued a guidance for industry for food operations that use peanut derived products as ingredients in food. This document lists GMPs specific to peanut derived products used as ingredients, with special considerations for controlling *Salmonella*. For example, the FDA recommends manufacturers use suppliers that have validated processes that reduce *Salmonella* in the peanut derived ingredient by 5 log units (US FDA, 2009c).

4. Conclusions

Prevention of contamination is the best way to prevent foodborne disease outbreaks and should be a goal of every food processor and handler. Peanuts pose a large risk for individuals with peanut allergy and hence, preventing peanut contamination of other foods by following GMPs and implementing allergen control programs is of utmost importance. While peanuts are not typically associated with foodborne illness as they have caused very few documented outbreaks, peanuts are at risk for aflatoxin contamination.

Peanut butter has recently been implicated in large, national outbreaks. Questions arise as to what other outbreaks could be caused by pathogens that have never been associated with specific foods. *Salmonella*, for example, is ubiquitous and has caused outbreaks in a wide range of products from fresh fruits and vegetables to chocolate. Yet another question is why only *Salmonella* has been the cause of peanut related outbreaks. The peanut butter outbreaks have inspired adaptation of new processing techniques to control pathogens in peanut butter. Outbreaks also inspire improved methods of pathogen detection and regulations to address consumer concerns. For example, in January 2011, the Food Safety Modernization Act (FSMA) was signed into law (Public Law, 2011). FSMA aims to improve the capacity of the FDA to detect and respond to potential problems and to improve the safety of imported food. All large manufacturing and processing facilities must register with the FDA and operate under a hazard analysis, prevention-based food safety program Hazard Analysis & Critical Control Points (HACCP; pronounced ‘ha-sip’). The Act also includes requirements for mandatory laboratory testing and increased frequency of inspections (Public Law, 2011). While food testing and inspections do not ensure safety, there is increased potential of detecting a problem and implementing corrective action.

Prevention of outbreaks is best accomplished through cooperation among all individuals within a food operation and with regulators to implement effective GAPs, GMPs, and other prevention-based food safety programs. Peanut operations can learn from past outbreaks to provide safe products for consumers.

References


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