Reduction of Microbial Load on Boneless, Skinless Chicken Breast Using Ultraviolet Radiation

Daniel E. Martin Jr.

Thesis submitted to the Faculty of the
Virginia Polytechnic Institute and State University
in partial fulfillment of the requirements for the degree

Master of Science
in
Food Science and Technology

Joseph D. Eifert Ph D., Chair
Joseph E. Marcy Ph D.
Susan E. Duncan Ph D.

October 2002
Blacksburg, Virginia

Keywords: UV, Chicken, Pathogen, Spoilage

Reduction of Microbial Load on Boneless, Skinless Chicken Breast
Using Ultraviolet Radiation

Daniel E. Martin Jr.

ABSTRACT

This study examined the effectiveness of UV radiation in reducing numbers of naturally occurring aerobic psychotrophic bacteria, lactic acid bacteria, *Campylobacter* and surface inoculated *E. coli* on split, boneless, skinless chicken breasts and the effects the UV treatments had on the taste of the chicken. The objective of the study was to determine the UV dosage that gave the largest amount of microbial kill without adversely affecting the taste of the chicken.

Two groups of 12 breasts were individually vacuum packaged. One group was surface inoculated with 1ml of a 2.0 X 10^6 CFU/ml culture of generic *E. coli*. The other group received no inoculation. Two breasts from each group were treated with one of six different UV radiation doses, 0 mW s/cm^2 (control-no exposure), 34mW s/cm^2, 101mW s/cm^2, 202mW s/cm^2, 504mW s/cm^2 and 1008mW s/cm^2. Within 24 hr of the treatments and again after seven days, one breast from each group and each treatment was enumerated for bacterial load. The results showed that bacterial load on the inoculated UV treated breasts were significantly reduced ($p \leq 0.05$) at every treatment level by an average of 1.5 logs compared to the inoculated controls. There were however, no significant differences ($p \geq 0.05$) between the inoculated breasts at any of the five different UV treatment dosages. The non-inoculated breasts showed no significant differences in the numbers of bacteria on the controls, as compared to the breasts treated with any of the five UV doses ($p \geq 0.05$).

Another set of 50 breasts were individually vacuum packaged and divided into six groups. Five groups contained five breasts each. Each group was treated with UV doses of 202mW s/cm^2, 504mW s/cm^2, 1008mW s/cm^2, 2016mW s/cm^2 and 3024mW s/cm^2 respectively. The control group (n=25) received no exposure. Within 48 hr, and again seven days after treatments, triangle tests for difference were conducted to see if the taste of the chicken had been affected by the treatments. A sensory panel detected a significant taste difference between the untreated chicken and chicken treated at 504mW s/cm^2 ($p \leq 0.05$) two days after treatment, and between the control and chicken treated at 2016mW s/cm^2 seven days after treatment ($p \leq 0.05$).

TABLE OF CONTENTS

LIST OF FIGURES

ACKNOWLEDGMENTS

First and foremost I thank my parents, without which I wouldn't be on earth. It is sad that my Mom's last years brought her such ill health and that she wasn't around to see me receive my Master's Degree. I miss you Mom. I love you Dad.

Then there is my wife. Her faith in me and my ability, and the knowledge that there was something better waiting for us down the road held us together for the duration of my Master's program…and beyond.

My three children are an integral part of my life. I hope that my determination to get ahead through education will set an example that they will follow.

The staff and professors at VA. Tech were there when I needed them. Dishing out the encouragement when I needed it and dishing out the harsh stuff too. Believe me, sometimes I deserved a harsh word or two (or three). Drs. Joseph Marcy and Joseph Eifert were instrumental in forming a project and getting me supplies. Dr. Susan Duncan was there to offer her help. Other instructors and staff helped in ways that they may never even know. Smiles, encouraging words and looks of understanding go a long way.

Lastly I would like to thank Perdue Inc., WLR Foods Inc. and Rocco Inc. for supplying the chicken and Cryovac Inc. for allowing me to use their UV equipment.

INTRODUCTION

The presence of pathogens such as *Campylobacter* (52, 59) and *Salmonella* (53) and spoilage organisms such as pseudomonads and lactic acid bacteria (51) on poultry has been well established by many researchers. Varying amounts of microbial contamination can be found on different parts of individual freshly dressed poultry carcasses (38, 58). With current processing methods, poultry carcasses are exposed to many common machine and work surfaces and are handled by many different workers making cross contamination of product an almost certainty (76, 82). Since the majority of chicken prepared for consumers is packaged raw and not thermally processed (31, 39), these organisms can replicate and cause both spoilage and food borne illness.

Microbial contamination from old, new and emerging foodborne pathogens eluded traditional food inspection methods that relied heavily on visual and organaleptic identification of food hazards (80). These bacterial hazards demanded new control and elimination strategies that minimize the likelihood of contamination. In July1997, new regulations to modernize the nation's meat and poultry inspection system (Hazard Analysis Critical Control Point (HACCP)) were announced and have since been instituted in slaughter plants across the United States (103).

The food industry has a responsibility to use and/or help develop safe and effective technology to reduce the numbers of spoilage and pathogenic bacteria thus enhancing the safety and shelf life of the food supply. Food safety experts believe that we can prevent many infections and deaths annually by expanding our preventative efforts and employing new technologies.

According to Corry et al. (22), future methods of microbial reduction /elimination should be economically feasible and convenient to apply. These methods should not change the appearance, smell, taste or nutritional properties of the treated foods. They also shouldn't leave a residue, or pose a threat to the environment. In this article, it is stated "Salmonellas, E. coli and campylobacters are all relatively sensitive to UV, although little has been published to investigate this in detail."

UV could be a weapon in the fight against pathogens and spoilage microorganisms in poultry processing. This study was conducted to determine the usefulness of UV treatment for reduction of bacterial load on chicken.

CHAPTER 1 - Review of Literature

POULTRY

General Information

Poultry is part of the livelihood of many people in the United States. Farmers, feed suppliers, warehouse workers, production line workers, supervisors, managers, distributors, truck drivers, supermarkets and restaurateurs are but some of those involved in poultry production and distribution. Consumers and connoisseurs of poultry are the ultimate beneficiaries of the work done by these groups of people.

Methods for the production of poultry meat have changed since the days of single-family farming. Vertical integration in the poultry industry is the current standard and is best explained as two or more of the steps in production, processing and marketing, (to include hatcheries, feed mills, farms and slaughter facilities) being controlled by a single entity, usually a large poultry company (3, 31, 39, 82).

Broilers are raised in total confinement in large floor pens with many chickens in each pen. Supplemental heat, natural ventilation, and evaporative cooling control the environment. Many flocks are raised in each pen every year. Food for the birds is based on corn or soybean meal supplemented with animal or fish protein. In addition, the birds are given nutrients and subtheraputic doses of antibiotics to improve feed efficiency and to reduce illness and death among flocks. This close contact and communal growth makes the spread of microbes easier between birds within a flock (79).

Birds are normally caught in the flock houses by human hands and put into cages that are placed onto large trucks for transport to the slaughter facility. Many birds are crowded into each cage, and the slaughter facility can be far from the farms where the birds are raised. This situation can cause stress and excess shedding of microorganisms in the bird feces that can lead to greater cross contamination by microbial pathogens (78).

Consumption and Economics of Poultry

Total poultry and red meat per capita consumption set new records almost every year between 1960 and 1992. Per capita consumption of poultry nearly tripled during this time period (100). Since1993, chicken has shown a higher per capita consumption in the US on a retail weight basis than beef, pork, turkey or veal (108).

Data from 1980 shows that consumption of broilers was 45.8 lb., consumption of beef was 76.6 lb., consumption of pork was 57.3 lb., consumption of turkey was 10.3 lb. and consumption of veal was 1.5 lb. per person for a total of 195.1 lb. of meat consumed. Data from 1993 (the year broilers became the number one consumed meat) shows that consumption of broilers was 68.5 lb., consumption of beef was 65.1 lb., consumption of pork was 52.3 lb., consumption of turkey was 17.7 lb. and consumption of veal was 0.9 lb. per person. In 1998, consumption of broilers was 73.9 lb., consumption of beef was 68.1 lb., consumption of pork was 52.5 lb., consumption of turkey was 18.1 lb. and consumption of veal was 0.8 lb. per person (108).

Increased consumption of poultry has been attributed to perceived health benefits. Poultry is regarded as having less fat and lower cholesterol than red meat. However, levels of these meat components depend on method of preparation (100).

The production and value of broilers has shown a steady increase in the US from 1980-1997. In 1980, there were 3,963,211,000 head produced at a value of $4,302,818,000 (104). In 1990, there were 5,864,150,000 head produced at a value of $8,365,704,000 (105). In 1997, the last year of available data, 7, 764, 200, 00 head were produced at a value of $14,158,926,000 (106).

Poultry is one of Virginia's most valuable agricultural commodities. The poultry industry contributed over $2 billion in gross income to the States' economy between the years of 1993 and 1997. Virginia's 1994 commercial broiler production was 252,700,000 head, which totaled 1,187,700,000 lb. and contributed nearly $398 million dollars in gross income. In 1997, 259,400,000 broilers were produced for a total of 1,219,200,000 lb. and contributed over $445 million dollars in gross income to Virginia’s economy (107).

Poultry Microflora

Campylobacter

Campylobacter jejuni is a gram-negative, slender, curved and motile rod. It has a single polar flagellum at one or both ends and moves with a typical corkscrew type action (7). It is also a microaerophilic organism, meaning that it requires reduced levels of oxygen for optimum growth. An environment with 3 to 5% oxygen and 2 to 10% carbon dioxide provides for this optimal growth (109), and thus, media for recovery and isolation of *Campylobacter* should include sodium bisulfite acting as an antioxidant to reduce toxic derivatives from oxygen and thus protect *Campylobacter* cells (29). It is an oxidase positive organism and unlike most bacteria, uses amino acids or tricarboxylic acids intermediates instead of carbohydrates as an energy source (7). The development of selective media has allowed for isolation and growth in the laboratory setting (89).

*Campylobacte*r spp. are theromphilic and the temperature range for *C. jejuni* growth is between 30^{o} C and 47° C with the optimum being 42° C (85). Due to this thermophilic nature, it was thought that *Campylobacter* spp. would not grow at less than room temperature (11); however *C. jejuni* has been shown to grow at 4° C and can survive at 4° C on parts of poultry carcasses (41). *C. jejuni* has also been shown to survive long term storage on chicken skin at -70° C and -20° C under various packaging atmospheres (63).

Campylobacter is relatively fragile, and susceptible to environmental stresses such as drying, heating, disinfectants and acidic conditions. In addition, it cannot survive in salt levels above 2%, and grows in the narrow pH range of 6.5-7.5 (113). It is also destroyed by thorough cooking and by being processed through typical water treatment systems. Freezing cannot be relied on to destroy the bacteria (34).

As with a number of other bacteria that are medically important, *Campylobacter* is becoming more and more resistant to drugs that have been used to treat diseases in animals and humans largely because of the widespread use of antibiotics within the animal reservoir (30).

Within the last twenty years, *Campylobacter jejuni/coli* has risen from anonymity as a veterinary pathogen. *Campylobacter* was once considered a rare opportunistic bloodstream infection until veterinary diagnostic procedures used on human stool samples showed that it was a common cause of diarrheal illness in many industrialized nations (24, 99). Recent surveys have shown that *C. jejuni* is the leading cause of bacterial diarrheal illness in the United States (15, 16, 17). The illness caused by *C. jejuni* is known as campylobacteriosis, campylobacter enteritis or

gastroenteritis. The pathogenic mechanisms of *C. jejuni* are still being studied; therefore it is difficult to differentiate pathogenic from nonpathogenic strains. The pathogenic mechanisms of *C. jejuni* are not completely understood, but it does produce a heat-labile toxin that may cause diarrhea. In addition, *C. jejuni* may be an invasive organism (109).

C. jejuni infection causes diarrhea, which may be watery or sticky and can contain blood and leukocytes. Other symptoms include fever, abdominal pain, headache, nausea and muscle pain. The illness usually occurs within two to five days after ingestion of the organism. Most victims of campylobacteriosis recover completely within two to five days but relapses are not uncommon (about 25% of cases). In some cases recovery can take up to 10 days (7, 18, 27).

Most infections run their course and are not treated with antibiotics. Victims should drink plenty of fluids as long as the diarrhea lasts. In more severe cases, antibiotics such as erythromycin or flouroquinolone can be used to shorten the duration of symptoms if the illness is caught in early stages (18). The infective dose of *C. jejuni* is considered to be small; human feeding studies suggest that about 400-500 bacteria may cause illness in some individuals. A volunteer human feeding study suggests that host susceptibility also determines infectious dose to some degree (109).

Complications are relatively rare, but infections have been associated with hemolytic uremic syndrome, and following septicemia, infections of nearly any organ (109). Persons with underdeveloped or weakened immune systems such as newborns or the elderly or with immune systems weakened by chronic illness such as AIDS, or on medical treatment (e.g., cancer patients on immunosuppressive therapy) are more susceptible to health complications from *Campylobacter* (34). Meningitis, recurrent colitis, acute cholecystitis and Guillain-Barre syndrome are rare complications. Reactive arthritis and Reiter's syndrome have also been reported as sequelae of *C. jejuni* enteritis (74, 92).

Campylobacter bacteria are commonly found in the intestinal tracts of cats, dogs, poultry, cattle, swine, rodents, monkeys, wild birds, and some humans. The bacteria pass through feces to cycle through the environment and are also in untreated water (34, 71, 95). Results of numerous studies indicate that vertical transmission of *Campylobacte*r spp. from parent flocks to hatchlings via eggs is not likely to happen and that instead, flocks become infected from the environment (26, 55, 83, 87, 110). According to Welbourn (113), *C. jejuni/coli* contaminate between 80% and 90% of fresh chicken carcasses. Properly cooking chicken, pasteurizing milk, and chlorinating

drinking water will kill the bacteria (11).

In a case-control study of 275 men with HIV infection, the risk of acquiring campylobacteriosis was strongly associated with eating undercooked chicken or runny eggs and with eating or drinking while preparing raw chicken (1). In other case control studies, consumption of poultry was strongly associated with *Campylobacter* infections (23, 43). In 1996, an outbreak of *Campylobacter* Enteritis was traced by the Oklahoma State Department of Health to foods that were cross-contaminated with raw chicken (75).

Salmonella

The genus *Salmonella* is made up of over 2000 serologically distinct bacterial serovars (60). *Salmonella* Enteritidis and *Salmonella* Typhimurium account for about half of all human infections (35). They are motile bacteria (nonmotile exceptions *S. gallinarum* and *S. pullorum*), and are nonsporeforming gram-negative rods (7, 109). They can grow both aerobically and anaerobically between 7° C and 48° C, at a pH of between four and eight, and at water activities (Aw) above 0.93. Heat and acid kill them, however, they are resistant to both freezing and drying (5).

Surveys have shown that *Salmonella* is the second leading cause of bacterial diarrheal illness in the United States. Salmonellosis is the name of the illness caused by *Salmonella* spp. It is estimated that from two to four million cases of salmonellosis occur in the U.S. annually (15, 16, 17). *Salmonella* organisms invade through penetration and passage from the gut lumen into the epithelium of the small intestine where inflammation occurs. There is evidence that an enterotoxin may be produced. All age groups are susceptible, but symptoms are most severe in the elderly, infants, and persons with impaired immune systems (109).

S. typhi and *S. paratyphi* A, B, and C normally cause septicemia and produce typhoid-like fever in humans (109). Other forms of salmonellosis generally produce milder symptoms. Acute symptoms include nausea, vomiting, abdominal cramps, diarrhea, fever, and headache. The onset time is usually between 6 and 48 hr after ingestion. Symptoms last for one to two days or may be prolonged, depending on host factors, ingested dose, and strain characteristics (35, 73). The infective dose can be as few as 50 cells and as many as 10^9, depending on individual tolerances or susceptibility of the host, and virulence differences among strains (28).

Arthritic symptoms may follow three to four weeks after onset of acute symptoms. Septic arthritis, subsequent or coincident with septicemia, also occurs and can be difficult to treat. Reactive arthritis may occur with a frequency of about 2% of culture-proven cases. Various organs may be infected, leading to lesions (109). Joint pain, eye irritation and painful urination (Reiter's syndrome) have also been reported to occur in a small number of infected people (35).

Environmental sources of the organism include water, soil, flies, rodents, sewage, factory surfaces, and kitchen surfaces (5). There is a widespread occurrence in animals, especially in poultry and swine (109). Raw foods of animal origin including meats, poultry, eggs, milk, dairy products, fish and shrimp are some of the more common food sources (35).

Approximately one-third of reported foodborne outbreaks of salmonellosis for which a vehicle was identified have been linked to poultry (28). Eating chicken and undercooked eggs was associated with sporadic *Salmonella* Enteritidis (SE) and sporadic *Salmonella* Heidelburg infections (16).

Foodborne Illness

According to the Council for Agricultural Science and Technology (14) as many as 9,000 deaths and 6.5 to 33 million illnesses in the United States each year are food-related. Five million illnesses and 4,000 deaths annually are attributed to meat and poultry products. Most foodborne illnesses or infections go undiagnosed and are thus underreported.

The United States Department of Agriculture Economic Research Service estimates that human illness costs for six bacterial pathogens (*Salmonella*, *Campylobacter*, *E. coli* 0157:H7, *Listeria*, *S. aureus* and *C. perfringens*) range between $9.3 and $12.9 billion annually. Of these estimated costs, $2.9-$6.7 billion are attributed to foodborne bacteria, with $1.8-$4.8 billion attributable to meat and poultry. The total costs for all foodborne illnesses are likely to be much higher due to the fact that these estimates do not include the total burden placed on society by the chronic long-term illness caused by some foodborne pathogens (101). More research is needed regarding foods defined as sources of foodborne pathogens in order to develop better control/elimination strategies.

The Center for Disease Control (CDC) in cooperation with state and other federal agencies directs the Foodborne Diseases Active Surveillance Network, which goes by the

acronym of FoodNet. FoodNet is a major effort in attempting to enumerate the confirmable cases of foodborne illness in the United States that can lead to possible prevention strategies. Foodnet surveillance is being used to document the effectiveness of new food safety control measures or technology in decreasing the number of cases of major foodborne diseases in the United States each year (15, 16, 17). One of these new measures is the Hazard Analysis Critical Control Point (HACCP) system instituted by the United States Department of Agriculture- Food Safety and Inspection Service (USDA-FSIS). As of January 26, 2000, HACCP is a mandatory requirement for all federally inspected meat and poultry plants. A *Samonella* standard has been set by USDA-FSIS that must be met by all poultry producers and they are testing products to ensure those standards are met (103). FSIS is encouraging poultry and meat producers to use existing/developing technology to help meet the standard.

In 1997, FoodNet tracked infections caused by seven bacterial pathogens. Among the pathogens under surveillance, *Campylobacter* was the most frequently diagnosed, even though outbreaks are rare. Overall incidence rates were also highest for *Campylobacter* infections (19.7/100,000 population) with *Salmonella* (13.9/100,000) being second (16). A study of *Campylobacter* infections that began in 1998 will help identify control points and direct future prevention strategies. Based on these surveillance findings, the United States Department of Agriculture Research Service is encouraging research into possible prevention strategies for *Campylobacter* (15).

Spoilage Organisms

Poultry is a highly perishable food and the time between slaughter and deterioration varies between 4 and 10 days, even with refrigeration (72). It is an ideal medium for bacterial growth and is subject to rapid spoilage unless preventative measures are taken to retard or prevent microbial growth and by-product production.

Poultry is described as spoiled if organoleptic changes make the meat unacceptable to consumers. Changes associated with spoilage may include, but are not limited to, color, texture, odor, development of off flavors, and slime. Enzymatic activity within tissues contribute to changes during storage, but generally, the above mentioned changes occur due to decomposition and formation of metabolites resulting from microorganism growth (50).

Since spoilage of foods leads to economic losses and is often detrimental to the product manufacturer's reputation with the consumer, food producers should be vigilant in trying to rid themselves of spoilage organisms. Meat preservation involves measures to delay or prevent microbiological, chemical or physical changes that make the product unsuitable as food or downgrades some of it's quality (111).

Studies on the naturally occurring microflora of fresh poultry have shown the presence of over 25 genera. There are a number of organisms on a poultry carcass that can cause spoilage. The atmospheric gases (or lack thereof) inside of the package and the temperature during storage and distribution determine the predominate types of organisms that cause spoilage. At the time of spoilage, the primary organisms on eviscerated poultry undergoing low-temperature spoilage in air are pseudomonads (37, 51). The rate of bacterial growth under anaerobic conditions is considerably less than the growth rate under aerobic conditions. Lactic acid bacteria dominate the spoilage microflora of muscle tissue foods when oxygen is not present in the packaging environment (50).

CURRENT / RESEARCHED BACTERIAL REDUCTION METHODS

One of the top priorities of regulatory agencies, the poultry industry and consumer groups must be to reduce the incidence and/or level of spoilage organisms and enteropathogens on processed poultry. How to achieve this goal is a subject of much research and ongoing improvement of current technology. Physical, thermal, chemical and mechanical methods have been studied, researched or implemented to improve food safety and quality.

Farming

Many food safety policymakers and scientists believe that there should be a farm to table approach in reducing foodborne illness. A farm to table approach involves analyzing every aspect of meat or poultry production from birth/hatching to meal preparation, so that steps can be taken to reduce/eliminate the occurrence of foodborne illness (102). USDA-FSIS is currently advocating a farm to table approach in the poultry foodchain.

Farming practices are being scrutinized and some suggestions in changes are:

1. Cleaning/sanitizing poultry houses after every flock has been shipped to the

slaughterhouse.

2. Old litter should be changed after every flock.
3. Treat drinking water with appropriate chemicals/antimicrobials.
4. Keep other farm animals, domestic pets, rodents, insects, birds, and wild animals segregated from flocks by the best means possible.
5. Make sure that farm workers have the proper knowledge and know methods of cross-contamination, so as to avoid it.
6. Keep all farming equipment properly cleaned and maintained.
7. Animals should be sampled before slaughter and pathogen free flocks run through production lines before infected flocks to reduce the incidence of cross contamination.
8. Transport cages should be cleaned and disinfected after every use. (56, 110).

A study by Van De Giessen et al. (110) found that application of hygiene measures significantly reduced *Campylobacter* infections on broiler farms where some of the afore mentioned farming practices were in place. In the article they state "…introduction of the control measures indicates that this intervention strategy may reduce but can not totally exclude the risk of *Campylobacter* infections in broiler flocks. It must be realized that strict maintenance of the hygiene measures on the farm level is difficult."

Competitive exclusion (CE) is another farm level attempt at pathogen control. Over the last 20 years, there have been numerous studies on the efficacy of CE for the control of *Salmonella* and *Campylobacter* (10, 45, 93, 96). CE is the use of indigenous intestinal microflora from older, pathogen free chickens to inoculate young chicks, through drinking water, spraying (10) or oral administration (81). This microflora plays a role in protecting birds against pathogen colonization because the organisms contained within a culture are antagonistic to said pathogens (96). Chicks are usually exposed to these protective bacteria by contact with eggs from the hatchery, shipping containers and through contact with the fecal droppings from adult chickens (8). Because of the replacement of sitting hens with commercial hatcheries and the rearing of chickens in cleaned and disinfected broiler houses, the prevalence of such protective microflora has been reduced and it's development is slower to occur (93).

Undefined preparations of cultured fecal or cecal microflora in general reduce the numbers of infected chicks after challenge with a standard dose of *Salmonella* under laboratory

conditions, but in contrast, results under field conditions are more variable (93). Results obtained in a series of trials found that standard CE treatment is not consistently effective against chicken colonization by *C. jejuni* (96).

Slaughter Facilities

Slaughter and/or production facilities and equipment can be designed and processing lines/equipment layed out so that cross-contamination is minimized on poultry carcasses in processing areas. They can also be designed so that cleaning and sanitation periods, after the production day has ended, can be as easy and effective as possible. Proper training of staff and employees and strict adherence to established GMP's can also help reduce contamination as evidenced by the success of HACCP and Standard Sanitation Operating Procedures (SSOP's) instituted by USDA-FSIS in slaughter plants across the United States (15, 16, 17)

Bacterial contamination can come from either the outside or the inside surfaces of the bird carcass. The first bacterial control measure implemented inside the gates of the slaughter plant is usually scalding, which also helps to prepare feathers for removal (76).

Slavik et al. (91) showed that changing the temperature of the water in scalding tanks had a slight (<1 $\log_{10}$) effect on numbers of *Campylobacter* and *Salmonella* on chicken carcasses. They stressed that their results and the results of other skin model studies suggest the importance of choosing a proper scalding temperature that result in the least numbers of bacteria attached to chicken skin during processing.

Many different food grade chemicals generally recognized as safe (GRAS) by the United States Food and Drug Administration (FDA) and/or approved for use in poultry production by USDA have been researched and/or used in poultry processing facilities. Treatment with organic acids (25, 49, 86, 117) hydrogen peroxide (33) and other chemicals (46, 47, 57, 67) have had varying success rate against different pathogenic and spoilage bacteria. Factors such as cost, adverse sensory affects on the final product and damage of equipment have prevented their widespread usage.

In 1992, trisodium phosphate (TSP) was approved by USDA for use in poultry processing and has been shown affective against *Campylobacter*, *Salmonella*, Enterobacteriaceae, Pseudomonas and total plate counts (21, 90). Principal disadvantages of TSP

food decontamination are the requirement for a high concentration of the compound which can lead to altered organaleptic properties, increased wear of industrial equipment and environmental damage of effluents (13).

Ideally, the poultry processing industry needs a rinse/spraying system that can be incorporated into an existing production line, doesn't require a large amount of space, is not corroded or destroyed by the chemical it dispenses, and is easy to clean. Further, system optimization is needed to minimize spraying time while achieving a desired level of bacterial reduction by studying chemical, physical and mechanical parameters such as: temperature, rinsing time, nozzle dispensing patterns and spraying distance (67).

Hydrogen peroxide at 6,600 ppm or greater in poultry chiller water was shown to reduce the populations of aerobic organisms by as much as 99.5% (68). However, this chemical causes discoloration and swelling of poultry carcasses.

Lillard (69, 70) found that the combination of ultrasound waves and chlorine in chill tank water caused a reduction of *Salmonella* attached to broiler skin while sonication or chlorine alone showed no significant decrease of attached *Salmonella*. Other authors cited in Lillard (70) had varying successes/failures using different chicken parts, chemicals, temperatures and contact times in chiller water. The feasibility of using sonication in a poultry chiller depends on the development of appropriate equipment and an evaluation of the organoleptic effect of the process on the finished product. In 1996, USDA-FSIS allowed the use of chlorine dioxide to help control the microbial population in poultry chiller water. 21 CFR173.300 allows use at no greater than 3 ppm. It is used widely in the poultry industry due to it's GRAS status by USDA and because it is relatively cheap and easy to use.

Another attempt in manipulating the chilling process was to add pulsed electrical energy in combination with a salt to the chiller water. *C. jejuni* was effectively destroyed in chiller water with either sodium chloride or TSP at concentrations from 0.1% to 0.3%. The death rate was dependent on salt concentration, pH and treatment time. The feasibility of this process has yet to be tested in a production setting (66).

In 1992 the USDA approved the use of radiation on poultry and processors had a new weapon against microbes at their disposal. There are, however, some undesirable aspects of this technology, including some changes in organoleptic properties of irradiated poultry (20, 64, 65) fears of consumer rejection of irradiated foods (36, 42) and vocal opposition from well-organized

nuclear activists (2). Costs of a radiation plant with equipment are estimated at $10 million (40). The cost of leasing an irradiator is estimated to be $20,000-$25,000 a month for a facility that could irradiate 8,000 pounds an hour, 24 hr per day (9). This makes irradiating meats too expensive for most processors.

ULTRAVIOLET RADIATION (UV)

General Information

The region of the electromagnetic spectrum that includes UV is the wavelengths between 15 and 450 nm. The region of greatest antimicrobial activity lies between 220 and 300 nm (88). For most microbial species, the greatest bactericidal effect is seen in the range of 250-260 nm. UV inactivation of organisms is due to the effects of the radiation on nucleic acids. The nucleic acids strongly absorb UV energy with peak absorption of around 260nm (77). When UV energy is absorbed by nucleic acids, it causes photochemical changes that can cause mutations leading to cell injury or death (51). UV inactivation or destruction of organisms depends on the length of exposure time and the intensity of the UV (88). The dosage necessary to achieve a certain level of inactivation, measured in mW*s/cm2 can be expressed as applied intensity (milliwatts per square centimeter (mW/cm2)) multiplied by exposure time (seconds). UV radiation treatments, in comparison to ionizing radiation treatments have an advantage, in that they need no complicated, expensive ray-proofing measures or specially constructed areas for use (77).

Effect on Microorganisms

Overall, viruses and molds are more resistant to UV than are bacteria (98). The susceptibility of bacteria varies among different species and can vary between different strains of a particular species. Broadly speaking, gram-negative rods are the most susceptible to UV radiation. Gram-positive bacteria require approximately five times the dosage and bacterial spores about ten times more (48). The growth stage and the presence of spores are also factors in susceptibility (88). Generally, pigmented bacteria are more resistant than bacteria that form colorless colonies (32).

Previous research has shown up to a 7-log reduction of nalidixic acid resistant *Salmonella*

typhimurium were achieved on agar plates using doses of 1.56-9.36 mW s/cm^2 (97). Butler et al. (12) reported a 3 log (99.9%) inactivation dose for *C. jejuni, Y. enterocolitica* and *E. coli* of 1.8, 2.7, and 5.0 mWs/cm^2 respectively. Chang et al. (19) found that *E. coli, Salmonella typhi, Shigella sonnei* and *Staphylococcus aureus* grown in a broth culture, irradiated and then spread plated on nutrient agar, required about the same 7mW s/cm^2 for a 3 log reduction. Yousef and Marth (116) found that a dose of 3.4 mW s/cm^2 inactivated 90% of 24-hr old culture of *Listeria* spread plated on TA medium. Stermer et al. (94) found that a 2 mW s/cm^2 caused a 99.9% reduction of total plate counts (no attempt at identifying species) on pour plates of bacteria obtained by swabbing spoiled meat known to have high counts of bacteria.

In addition to continuous wave UV light, modulated UV light (6) and pulsed UV light (84) have been shown in studies to be effective against a number of food related microorganisms.

Uses in the Food and Beverage Industry

Germicidal UV lamps produce energy with a focal point of 253.7 nm, which can be used safely in many food industry applications (77). UV has been used for some time for the treatment of water supplies (115) and has applications in packaging and bottling material sterilization (4). Research has shown that a continuous dose of UV radiation of between 0.2 and 24□W/cm^2 on psychrophilic micro organisms on chilled beef slices at 0^0C resulted in an extension of the lag phase of *Pseudomonas* sp. and of the molds *Thamnidium* sp. and *Penicillium* sp. (54). A UV dose of 150mW s/cm^2 on smooth surface beef plate reduced bacteria about 2 log cycles (94). Huang and Toledo (44) successfully reduced the number of bacteria on fresh mackerel fish. Numbers of *Salmonella typhimurium* have been reduced by between 68.72% and 85.01% utilizing doses of between 1,□□□□W s/cm^2 and 9,780□W s/cm^2 on poultry skin (97). Bacterial levels of *Salmonella senftenberg* and *Escherichia coli* have also been reduced on pork muscle by 2.0 and 1.9 logs respectively using a 1000□W/cm^2 dose of UV radiation (114). Shell eggs treated with varying dose of UV radiation showed reductions in populations of *S. typhimurium,* aerobes, molds and yeasts (61, 62).

REFERENCES

1. Altekruse, S. F., F. Hyman, K. Klontz, B. Timbo, and L. Tollefson. 1994. Foodborne bacterial infection in individuals with the human immunodeficiency virus. South. Med. Jour. 87:169-173.
2. Anon. 1998. Radiation for food safety. Health News 4:1-2.
3. Austic, R.E. and M. C. Nesheim. 1990. Poultry Production. Lea and Febiger, Philadelphia, PA.
4. Bachman, R. 1975. Sterilization by intense ultraviolet radiation. Brown Boveri Rev. 62:206-209.
5. Baird-Parker, A. C. 1991. Foodborne salmonellosis. p. 53-61. *In* A Lancet Review Foodborne Illness. M. W. Waites, and J. P. Arbuthnott (ed.) Edward Arnold, Great Britain.
6. Bank, H. L., J. John, M. K. Schmehl, and R. J. Dratch. 1990. Bactericidal effectiveness of modulated UV light. Appl. Environ. Microbiol. 56:3888-3889.
7. Banwart, G. J. 1989. Basic Food Microbiology 2nd edition. Van Nostrand Reinhold, New York, New York.
8. Barnes, E. M., C. S. Impey, and B. J. H. Stevens. 1979. Factors affecting the incidence and anti-salmonella activity of the anaerobic caecal flora of the young chick. J. Hyg. Camb. 82:263-283.
9. Bjerklie, S. December 1999. Is irradiation in your future? Meat Processing: North American Edition: 38-43.
10. Blankenship, L. C., J. S. Bailey, N. A. Cox, N. J. Stern, R. Brewer, and O. Williams. 1993. Two-step mucosal competitive exclusion flora treatment to diminish *Salmonellae* in commercial broiler chickens. Poultry Sci. 72:1667-1672.
11. Bryan, F. L., and M. P Doyle. 1995. Health consequences of *Salmonella* and *Campylobacter jejuni* in raw poultry. J. Food Prot. 58:326-344.
12. Butler, R. C., V. Lund, and D. A. Carlson. 1987. Susceptibility of *Campylobacter jejuni* and *Yersinia enterocolitica* to UV radiation. Appl. Environ. Microbiol. 53:375-378.
13. Carneiro De Milo, A. M. S., C. A. Cassar, and R. J. Miles. 1997. Trisodium phosphate increases sensitivity of gram-negative bacteria to lysozyme and nisin. J. Food Prot. 61(7):839-844.

14. Council of Agricultural Science and Technology. 1994. Foodborne pathogens: Risks and consequences. Task Force report no. 122. Council of Agricultural Science and Technology. Iowa State University, Ames, Iowa.
15. Center for Disease Control. 1997. Foodnet CDC/USDA/FDA Foodborne Diseases Active Surveillance Network, CDC's Emerging Infections Program, 1997 Surveillance Results. National Center for Infectious Diseases, Center for Disease Control and Prevention Atlanta, GA.
16. Center for Disease Control. 1998. Foodnet CDC/USDA/FDA Foodborne Diseases Active Surveillance Network, CDC's Emerging Infections Program, 1998 Surveillance Results. National Center for Infectious Diseases, Center for Disease Control and Prevention Atlanta, GA.
17. Center for Disease Control. 1999. Foodnet CDC/USDA/FDA Foodborne Diseases Active Surveillance Network, CDC's Emerging Infections Program, 1999 Surveillance Results. National Center for Infectious Diseases, Centers for Disease Control and Prevention Atlanta, GA.
18. Center for Disease Control-National Center for Infectious Diseases. 1998. Campylobacter FAQ's. National Center for Infectious Diseases, Centers for Disease Control and Prevention Atlanta, GA.
19. Chang, J. C. H., S. F. Ossoff, D. C. Lobe, M. H. Dorfman, C. M. Dumais, R. G. Qualls, and J. D. Johnson. 1985. UV inactivation of pathogenic and indicator microorganisms. Appl. Environ. Microbiol. 49:1361-1365.
20. Coleby, B., M. Ingram, and H. J. Shepard. 1960. Treatment of meats with ionizing radiations. J. Sci. Food Agric. 11:61-71.
21. Coppen, P., S. Fenner, and G. Salvat. 1998. Antimicrobial efficacy of AvGard^{R} carcass wash under industrial processing conditions. British Poult. Sci. 39:229-234.
22. Corry, J. E. L., C. James, S. J. James, and M. Hinton. 1995. *Salmonella, Campylobacter* and *Escherichia coli* 0157:H7 decontamination techniques for the future. Int. Jour. Food Micro. 28:187-196.
23. Deming, M. S., R. V. Tauxe, P. A. Blake, S. E. Dixon, B. S. Fowler, and T. S. Jones. 1987. *Campylobacter* enteritis at a university: transmission from eating chicken and from cats. Amer. J. of Epidem. 126:526-534.

24. Dekeyser, P. J., M. Gossin-Detrain, J. P. Butzler, and J. Stenon. 1972. Acute enteritis due to related *Vibrio*; first positives stool cultures. J. Infect. Dis. 125:390-392.
25. Dickens, J. A., B. G. Lyon, A. D. Whittemore, and C. E. Lyon. 1994. The effect of an acetic acid dip on carcass appearance, microbiological quality, and cooked meat texture and flavor. Poult. Sci.73:576-581.
26. Doyle, M. P. 1984. Association of *Campylobacter jejuni* with laying hens and eggs. Appl. Environ. Microbiol. 47:533-536.
27. Doyle, M. P. 1990. *Campylobacter jejuni*, p. 217-222. *In* Foodborne diseases. D. O. Cliver (ed.), Academic Press, Inc., San Diego, California.
28. Doyle, M. P., and D. O. Cliver. 1990. *Salmonella*, p.185-204. *In* Foodborne diseases. D. O. Cliver (ed.), Academic Press, Inc., San Diego, California.
29. Doyle, M. P. and D. J. Roman. 1982. Sensitivity of *Campylobacter jejuni* to drying. J. Food Prot. 45 (6):507-510.
30. Endtz, H. P., G. H. Ruijs, B. van Klingeren, W. H. Jansen, T. ven der Reyden, and T. P. Mouton. 1991. Quinolone resistance in *Campylobacter* isolated from man and poultry following the introduction of fluorquinolones in veterinary medicine. J. Antimicrobial Chem. 27:199-208.
31. Ensminger, M. E. 1992. Poultry Science. Interstate Publishers, Inc. Danville, IL.
32. Farkas, J. 1997. Physical methods of food preservation. p. 497-519. *In* M. P. Doyle, L. R. Beuchat, and T. J. Montville (ed.), Food microbiology: fundamentals and frontiers. ASM Press, Washington, D.C.
33. Fletcher, D. L., S. M. Russell, J. M. Walker, and J. S. Bailey. 1993. An evaluation of a rinse procedure using sodium bicarbonate and hydrogen peroxide on the recovery of bacteria from broiler carcasses. Poult. Sci. 72:2152-2156.
34. Food Safety and Inspection Service. November 1997. Backgrounders: *Campylobacter* questions and answers. Food Safety and Inspection Service-United States Department of Agriculture, Washington, D.C.
35. Food Safety and Inspection Service. February 1998. Backgrounders: *Salmonella* questions and answers. Food Safety and Inspection Service-United States Department of Agriculture, Washington, D.C.

36. Frezen, P., J. Buzby, A. Majchrowicz, E. DeBess, K. Hechemy, H. Kassenborg, M. Kennedy, K. Gibbs-McCombs, A McNess, and the EIP Foodnet Working Group. July 2000. Consumer acceptance of irradiated meat and poultry in the foodnet sites. 2nd International Conference on Emerging Infectious Diseases. Atlanta, Ga.
37. Garcia-Lopez, M. L., M. Prieto, and A. Otero. 1998. The physiological attributes of gram-negative bacteria associated with spoilage of meat and meat products, p.1-28. *In* A. Davies and R. Board (ed.), The Microbiology of meat and poultry. Blackie Academic and Professional, New York, New York.
38. Georgnaras, I., A. E. deJesus, E. vanZyl, and A. vonHoly. 1997. Bacterial populations of different sample types from carcasses in the dirty area of a South African poultry abattoir. J. Food Prot. 60(5):551-554.
39. Gillespie, J. R. 1997. Modern livestock and poultry production 5th Edition. Delmar Publishers, Albany, New York.
40. Groves, M. February 13, 1999. USDA approves irradiation of meat to kill contaminants. Agriculture industry is unsure about consumer acceptance. Treated products would carry labels. The Los Angeles Times.
41. Grant, I. H., N. J. Richardson, and V. D. Bokkenhouser. 1980. Broiler chickens as a potential source of *Campylobacte*r infections in humans. J. Clin. Micro. 11(5):508-510.
42. Hanis, T., P. Jelen, P. Klir, J. Mnukova, B. Perez, and M. Pesek. 1989. Poultry meat irradiation-effect of temperature on chemical changes and inactivation of microorganisms. J. Food Prot. 32(1):26-29.
43. Harris, N. V., N. S. Weiss, and C. M. Nolan. 1986. The role of poultry and meats in the etiology of *Campylobacter jejuni/coli* enteritis. Amer. J. Public Health 76:407-411.
44. Huang, Y., and R. Toledo. 1982. Effect of high doses of high and low intensity ultraviolet irradiation on the surface microbiological counts and storage life of fish. J. Food Sci. 47:1667-1669, 1731.
45. Hume, M. E., D. E. Corrier, D. J. Nisbet and J. R. DeLoach. 1996. Reduction of *Salmonella* crop and cecal colonization by a characterized competitive exclusion culture in broilers during grow-out. J. Food Prot. 59(7):688-693.
46. Hwang, C., and L. R. Beauchat. 1994. Efficacy of selected chemicals for killing pathogenic and spoilage microorganisms on chicken skin. J. Food Prot. 58(1):19-23.

47. Hwang, C., and L. R. Beauchat. 1995. Efficacy of a lactic acid/sodium benzoate wash solution in reducing bacterial contamination of raw poultry. Int. J. Food Microbiol. 27:91-98.
48. International Commission on Microbiological Specifications for Foods. 1980. Microbial ecology of foods, Vol 1 factors affecting life and death of microorganisms. Academic Press, Inc., New York, New York.
49. Izat, A. L., M. Colberg, M. H. Adams, M. A. Reiber, and P. W. Waldroup. 1989. Production and processing studies to reduce the incidence of *Salmonella* on commercial broilers. J. Food Prot.52:670-673.
50. Jackson, T. C., G. R. Acuff, and J. S. Dickson.1997. Meat, Poultry and Seafood, p. 83-100. *In* M. P. Doyle, L. R. Beuchat, and T. J. Montville (ed.), Food microbiology: fundamentals and frontiers. ASM Press, Washington, D.C.
51. Jay, J. M. 1996. Modern food microbiology. 5th ed. Chapman and Hall, New York, New York.
52. Jones, F. T., R. C. Axtell, D. V. Rives, S. E. Scheideler, F. R. Tarver Jr., R. L. Walker, and M. J. Wineland. 1991a. A survey of *Campylobacter jejuni* contamination in modern broiler production and processing systems. J. Food Prot. 54(4): 259-262.
53. Jones, F. T., R. C. Axtell, D. V. Rives, S. E. Scheideler, F. R. Tarver Jr., R. L. Walker, and M. J. Wineland. 1991b. A survey of *Salmonella* contamination in modern broiler production. J. Food Prot. 54(7): 502-507.
54. Kaess, G., and J. F. Weidermann. 1973. Effects of ultraviolet radiation on the growth of microorganisms on chilled beef slices. J. Food Technol. 8:59-69.
55. Kazwala, R. R., J. D. Collins, J. Hannon, R. A. P. Crinion, and H. O'Mahony. 1990. Factors responsible for the introduction and spread of *Campylobacter jejuni* infection in commercial poultry production. Vet. Rec. 126:305-306.
56. Kapperud, G., E. Skjerve, and L. Vik. 1993. Epidemiological investigation of risk factors for *Campylobacter* colonization in Norwegian broiler flocks. Epidemiol. Infect. 111:245-255.
57. Kim, J. W., M. F. Slavik, and Y. Li. 1996. Cetylpyridinium chloride (CPC) treatment on poultry skin to reduce attached *Salmonella*. J. Food Prot. 59:322-326.

58. Kotula, K. L., and Y. Pandya. 1995. Bacterial contamination of broiler chickens before scalding. J. Food Prot. 58(12):1326-1329.
59. Kramer, J. M., J. A. Frost, F. J. Bolton, and D. R. A. Wareing. 2000. Campylobacter contamination of raw meat and poultry at retail sale: identification of multiple types and comparison with isolates from human infection. J. Food Prot. 63(12):1654-1659.
60. Krieg, N. R., and J. G. Holt. 1984. p.247. *In* Bergey's Manual of Systematic Bacteriology. Volume 1. Williams and Wilkins, Baltimore, MD.
61. Kuo, F. L., J. B. Carey, and S. C. Ricke. 1997. UV irradiation of shell eggs on populations of aerobes, molds, and inoculated *Salmonella typhimurium*. J. Food Prot. 60:639-643.
62. Kuo, F. L., S. C. Ricke, and J. B. Carey. 1997. Shell egg sanitation: UV radiation and egg rotation to effectively reduce populations of aerobes, yeasts and molds. J. Food Prot. 60:694-697.
63. Lee, A., S. C. Smith, and P. J. Coloe. 1998. Survival and growth of *Campylobacter jejuni* after inoculation onto chicken skin as a function of temperature and packaging conditions. J. Food Prot. 6 (12):1609-1614.
64. Lee, M., J. G. Sebranek, D. G., Olson, and J. S. Dickson. 1995. Irradiation and packaging of fresh meat and poultry. J. Food Prot. 59:62-72.
65. Lescano, G., P. Narvaiz, E. Kairiyama, and N. Kaupert. 1990. Effect of chicken breast irradiation on microbiological, chemical and organoleptic quality. Lebensm. Wiss. Technol. 24:130-134.
66. Li, Y., J. T. Walker, M. F. Slavik, and H. Wang. 1995. Electrical treatment of poultry chiller water to destroy *Campylobacter jejuni*. J. Food Prot. 58(12):1330-1334.
67. Li, Y., M. F. Slavik, J. T. Walker, and H. Xiong. 1997. Pre-chill spray of chicken carcasses to reduce *Salmonella typhimurium*. J. Food Sci. 62(3):605-607.
68. Lillard, H. S., and J. E. Thompson. 1983. Efficacy of hydrogen peroxide as a bactericide in poultry chiller water. J. Food Sci.48:125-126.
69. Lillard, H. S. 1993. Bactericidal effect of chlorine on attached *Salmonella* with and without sonication. J. Food Prot. 56(8):716-717.
70. Lillard, H. S. 1994. Decontamination of poultry skin by sonication. Food Technol. 72-73.
71. Litton, D. 1996. Old and new campylobacters. PHLS Microbiology Digest 13:10-15.
72. Marenzi, C. 1986. Proper meat storage prevents spoilage. Poultry 6:12-15.

73. McLean, D. M. 1987. Environmental aspects of *Salmonella* in humans. National Research Council of Canada, Associate Committee. Ottawa, Canada.
74. Mishu, M. D., A. A. Ilyas, C. L. Koski, F. Vriesendorp, S. D. Cook, F. A. Mithen and M. J. Blaser. 1993. Serologic evidence of previous *Campylobacter jejuni* infection in patients with the Guillian-Barre syndrome. Annals of Internal Medicine 118:947-953.
75. Morbidity and Mortality Weekly Review. 1998. Outbreak of *Campylobacter* enteritis associated with cross-contamination of food—Oklahoma, 1996. Morbidity and Mortality weekly Review 47:129-131.
76. Moreng, R. E., and J.S. Avens. 1985. Poultry science and production. Waveland Press, Inc. Prospect Heights, Illinois.
77. Morgan, R. 1989. UV 'green' light disinfection. Dairy Ind. Int. 54:33-35.
78. Mulder, R. W. A. W. 1995. Impact of transport and related stresses on the incidence and extent of human pathogens in pig meat and poultry. Int. J. Food Safety 15:239-246.
79. National Academy of Sciences. 1985. Meat and Poultry inspection: the scientific basis of the nation's program. National Academy Press, Washington, D.C.
80. National Academy of Sciences. 1990. Cattle inspection. Committee on evaluation of USDA streamlined inspection system for cattle (SIS-C). National Academy Press, Washington, D.C.
81. Nurmi, E., and M. Rantala. 1973. New aspects of *Salmonella* infection in broiler production. Nature 241:210-211.
82. Parkhurst, C. R., and G. J. Mountney. 1988. Poultry meat and egg production. Van Nostrand Reinhold Company, New York, New York.
83. Pearson, A. D., M. Greenwood, and T. D. Healing. 1993. Colonization of broiler chickens by waterborne *Campylobacter jejuni* in poultry broiler flocks. Appl. Environ. Micro. 59:987-996.
84. Rowan, N. J., S. J. MacGregor, J. G. Anderson, R. A. Touracre, L. McIlvaney, and O. Farish. 1999. Pulsed-light inactivation of food-related organisms. Appl. Env. Micro. 65(3):1312-1315.
85. Ryser, E. T., and E. H. Marth. 1989. New foodborne pathogens of public health significance. Journal of the American Dietetic Association. 89:948-953.

86. Sawaya, W. N., A. S. Elnawawy, S. Al-Zenki, J. Al-Otaibi, H. Al- Omirah, and H. Al-Amiri. 1995. Storage stability of chicken as affected by MAP and lactic acid treatment. J. Food Sci.60(3):611-614.

87. Shanker, S., A. Lee, and T. C. Sorrell. 1986. *Campylobacter jejuni* in broilers: the role of vertical transmission. J. Hyg. 96:153-159.

88. Shechmeister, I. L. 1991. Sterilization by ultraviolet radiation p. 553-565 *In* S. S. Block (ed.) Disinfection, sterilization, and preservation, 4th edition. Lea and Febiger, Malvern, Pennsylvania.

89. Skirrow, M.B., and J. Benjamin. 1980. 1001 *Campylobacters*: Cultural characteristics of intestinal *Campylobacters* from man and animal. J. Hyg. 85:427-442.

90. Slavik, M. F., J. W. Kim, M. D. Pharr, D. P. Raben, S. Tsai, and C. M. Lobsinger. 1994. Effect of trisodium phosphate on *Campylobacter* attached to post-chill chicken carcasses. J. Food Prot.57(4):324-326.

91. Slavik, M. F., J. W. Kim, and J. T. Walker. 1995. Reduction of *Salmonella* and *Campylobacter* on chicken carcasses by changing scalding temperatures. J. Food Prot. 58(6):689-691.

92. Smith, J. L. 1995. Arthritis, Guillain-Barre syndrome, and other sequelae of *Campylobacter jejuni* enteritis. J. Food Prot. 58:1153-1170.

93. Stavric, S., and J. Y. D'Aoust. 1993. Undefined and defined bacterial preparations for the competitive exclusion of *Salmonella* in poultry- a review. J. Food Prot. 56(2):173-180.

94. Stermer, R. A., M. Lasater-Smith and C. F. Brasington. 1987. Ultraviolet radiation- an effective bactericide for fresh meat. J. Food Prot. 50:108-111.

95. Stern, N. J. 1992. Reservoirs for *Campylobacter jejuni* and approaches for intervention in poultry, p. 49-60. *In* I. Nachamkin, M. J. Blaser, and L. S. Tompkins (ed.), *Campylobacter jejuni* current status and future trends. American Society for Microbiology, Washington, DC.

96. Stern. N.J. 1994. Mucosal competitive exclusion to diminish colonization of chickens by *Campylobacter jejuni*. Poultry Sci. 73:402-407.

97. Sumner, S. S., E. A. Wallner-Pendleton, G. W. Froning and L. V. E. Stetson. 1995. Inhibition of *Salmonella typhimurium* on agar medium and poultry skin by ultraviolet energy. J. Food Prot. 59:319-321,336.

98. Tartera, C., A. Bosch, and J. Jofre. 1998. The inactivation of bacteriophages infecting *Bacteroides fragilis* by chlorine treatment and UV-irradiation. FEMS Microbiol. Lett. 56:313-316.

99. Tauxe, R. V. 1992. Epidemiology of *Campylobacter jejuni* infections in the United States and other industrialized nations. *In* I. Nachamkin, M. J. Blaser, and L. S. Tompkins (eds.), *Campylobacter jejuni* current status and future trends. American Society for Microbiology. Washington, D.C.

100. United States Department of Agriculture Economic Research Service. 1993. U.S. poultry and red meat consumption, prices, spreads, and margins. Agricultural Information Bulletin Number 684. United States Department of Agriculture, Washington, D.C.

101. United States Department of Agriculture Economic Research Service. 1995. Bacterial foodborne disease: medical costs and productivity losses. Agricultural Economic Report No. 741. United States Department of Agriculture Washington, D.C.

102. United States Department of Agriculture Economic Research Service. 1995. Tracking foodborne pathogens from farm to table. Agricultural Economic Report No. 1532. United States Department of Agriculture Washington, D.C.

103. United States Department of Agriculture-Food Safety and Inspection Service. 1996. Pathogen reduction; hazard analysis and critical control points (HACCP) system; final rule. Federal Register Vol. 61, No. 144.

104. United States Department of Agriculture- National Agricultural Statistics Service. 1985. Final estimates for 1980-83. Statistical Bulletin No. 725. United States Department of Agriculture, Washington, D.C.

105. United States Department of Agriculture- National Agricultural Statistics Service. 1995. Final estimates for 1988-93. Statistical Bulletin No. 910. United States Department of Agriculture, Washington, D.C.

106. United States Department of Agriculture- National Agricultural Statistics Service. 1999. Final estimates for 1994-97. Statistical Bulletin No. 958. United States Department of Agriculture, Washington, D.C.

107. United States Department of Agriculture-National Agricultural Statistical Service-Virginia Agricultural Statistical Service. 1997.
[Internet, WWW], ADDRESS: http://www.nass.usda.gov/va/pg076.htm.

108. United States Department of Agriculture-National Agricultural Statistical Service. 1999.[Internet,WWW],ADDRESS:http://www.usda.gov/nass/pubs/stathigh/1999/ec-mc.htm
109. United States Food and Drug Administration-Center For Science And Nutrition. 1998. Foodborne pathogenic microorganisms and natural toxins handbook. United States Food and Drug Administration, Washington, D.C.
110. Van De Giessen, A. W., J. J. H. C. Tilburg, W. S. Ritmeester, and J. Van Der Plas. 1998. Reduction of *Campylobacter* infections in broiler flocks by application of hygiene measures. Epidemiol. Infect. 121:57-66.
111. Van Laack, R. L. J. M. 1994. Spoilage and preservation of muscle foods. p. 378-405. *In* Muscle foods: meat, poultry, and seafood technology. D. M. Kinsman, A. W. Kotula, and B. C. Breidstein (eds), Chapman and Hall, Inc., New York, N.Y.
112. Wallner-Pendleton, E. A., S. S. Sumner, G. W. Froning, and L. V. E. Stetson. 1994. The use of ultraviolet radiation to reduce *Salmonella* and psychrotrophic bacterial contamination on poultry carcasses. Poultry Sci. 73:1327-1333.
113. Welbourn, J. L. 1998. Campylobacter: No longer the "quiet pathogen". Food Testing and Analysis 4:20-22.
114. Wong, E., R. H. Linton, and D. E. Gerrard. 1998. Reduction of *Escherichia coli* and *Salmonella senftenburg* on pork skin and pork muscle using ultraviolet light. Food Micro. 15:415-423.
115. Yip, R. W., and D. E. Konasewich.1972. June:14-18, Ultraviolet sterilization of water- its potential and limitations. Water pollution Control (Canada).
116. Yousef, A. E., and E. H. Marth. 1988. Inactivation of *Listeria monocytogenes* by ultraviolet energy. J. Food Sci. 53 (2):571-573.
117. Zeitoun, A. A. M., and J. M. Debevere. 1990. The effect of treatment with buffered lactic acid on microbial decontamination and on shelf-life of poultry. Int. J. Food Microbiol. 11:305-312.

Reduction of Microbial Load on Boneless, Skinless Chicken Breasts Using Ultraviolet Radiation

Daniel E. Martin Jr, Joseph D. Eifert*, Joseph E. Marcy, and
Susan E. Duncan

Department of Food Science and Technology, Virginia Tech,
Blacksburg, VA 24061

Keywords: UV, chicken, spoilage, pathogen

*Corresponding author:
Mailing Address: Department of Food Science and Technology,
Virginia Tech, Blacksburg, VA 24061.
Phone: 540-231-3658
Fax: 540-231-9293.
Electronic mail address: jeifert@vt.edu

CHAPTER 2

ABSTRACT

This study examined the effectiveness of UV radiation in reducing numbers of naturally occurring aerobic psychotrophic bacteria, lactic acid bacteria, *Campylobacter* and surface inoculated *E. coli* on split, boneless, skinless chicken breasts and the effects the UV treatments had on the taste of the chicken. The objective of the study was to determine the UV dosage that gave the largest amount of microbial kill without adversely affecting the taste of the chicken.

Two groups of 12 breasts were individually vacuum packaged. One group was surface inoculated with 1ml of a 2.0 X 10^6 CFU/ml culture of generic *E. coli*. The other group received no inoculation. Two breasts from each group were treated with one of six different UV radiation doses, 0 mW s/cm^2 (control-no exposure), 34mW s/cm^2, 101mW s/cm^2, 202mW s/cm^2, 504mW s/cm^2 and 1008mW s/cm^2. Within 24 hours of the treatments and again after seven days, one breast from each group and each treatment was enumerated for bacterial load. The results showed that bacterial load on the inoculated UV treated breasts were significantly reduced ($p \leq 0.05$) at every treatment level by an average of 1.5 logs compared to the inoculated controls. There were however, no significant differences ($p \geq 0.05$) between the inoculated breasts at any of the five different UV treatment dosages. The non-inoculated breasts showed no significant differences in the numbers of bacteria on the controls, as compared to the breasts treated with any of the five UV doses ($p \geq 0.05$).

Another set of 50 breasts were individually vacuum packaged and divided into six groups. Five groups contained five breasts each. Each group was treated with UV doses of 202mW s/cm^2, 504mW s/cm^2, 1008mW s/cm^2, 2016mW s/cm^2 and 3024mW s/cm^2 respectively. The control group (n=25) had no exposure. Within 48 hours, and again seven days after treatments, triangle tests for difference were conducted to see if the taste of the chicken had been affected by the treatments. A sensory panel detected a significant taste difference between the untreated chicken and chicken treated at 504mW s/cm^2 ($p \leq 0.05$) two days after treatment, and between the control and chicken treated at 2016mW s/cm^2 seven days after treatment ($p \leq 0.05$).

INTRODUCTION

The presence of pathogens such as *Campylobacter* and *Salmonella,* and spoilage organisms such as pseudomonads and lactic acid bacteria on poultry has been well established. Five million illnesses and 4,000 deaths annually are attributed to meat and poultry products. Most foodborne illnesses or infections go undiagnosed and are thus underreported (6).

Poultry is a highly perishable food. The time between slaughter and deterioration varies between four and 10 days, even with refrigeration (13). Poultry is an ideal medium for bacterial growth and is subject to rapid spoilage unless preventative measures are taken to retard or prevent microbial growth and by-product production. Meat preservation involves measures to delay or prevent microbiological, chemical or physical changes that make the product unsuitable as food or downgrades some of its quality (20).

In 1992 the USDA approved the use of radiation on poultry (19). There are, however, some undesirable aspects of this technology, including some changes in organoleptic properties of irradiated poultry, fears of consumer rejection of irradiated foods and vocal opposition from well-organized nuclear activists. Costs of a radiation plant with equipment are estimated at $10 million (7). The cost of leasing an irradiator is estimated to be $20,000-$25,000 a month for a facility that could irradiate 8,000 pounds an hour, 24 hours per day (3). This makes irradiating meats too expensive for most processors. Ultraviolet (UV) radiation treatments, in comparison to ionizing radiation treatments, have an advantage in that they need no complicated, expensive ray-proofing measures or specially constructed areas for use (15).

The region of the electromagnetic spectrum that includes UV is the wavelengths between 15 and 450 nm. For most microbial species, the greatest bactericidal effect is seen in the range of 250-260 nm. UV inactivation of organisms is due to the effects of the radiation on nucleic acids. The nucleic acids strongly absorb UV energy with peak absorption of around 260nm (15). When UV energy is absorbed by nucleic acids, it causes photochemical changes that can cause mutations leading to cell injury or death (9). UV inactivation or destruction of organisms depends on the length of exposure time and the intensity of the UV (16). The dosage necessary to achieve a certain level of inactivation can be expressed as applied intensity (milliwatts per square centimeter (mW/cm^2) multiplied by exposure time (seconds) = $mW*s/cm^2$.

Previous research has shown up to a 7-log reduction of nalidixic acid resistant *Salmonella*

typhimurium were achieved on agar plates using doses of 1.56-9.36 mW s/cm^2 (18). Butler et al. (4) reported a 3 log (99.9%) inactivation dose for *C. jejuni, Y. enterocolitica* and *E. coli* of 1.8, 2.7, and 5.0 mWs/cm^2 respectively. Chang et al. (5) found that *E. coli, Salmonella typhi, Shigella sonnei* and *Staphylococcus aureus* grown in a broth culture, irradiated and then spread plated on nutrient agar, required about the same 7mW s/cm^2 for a 3 log reduction. Yousef and Marth (23) found that a dose of 3.4 mW s/cm^2 inactivated 90% of 24-hr old culture of *Listeria* spread plated on TA medium. Stermer et al. (17) found that a 2 mW s/cm^2 caused a 99.9% reduction of total plate counts (no attempt at identifying species) on pour plates of bacteria obtained by swabbing spoiled meat known to have high counts of bacteria.

Germicidal UV lamps produce energy with a focal point of 253.7 nm, which can be used safely in many food industry applications (15). Research has shown that a continuous dose of UV radiation between 0.2 and 24 mW/cm^2 on psychrophilic microorganisms on chilled beef slices at 0°C resulted in an extension of the lag phase of *Pseudomonas* sp. and of the molds *Thamnidium* sp. and *Penicillium* sp. (10). A UV dose of 150mW s/cm^2 on smooth surface beef plate reduced bacteria about 2 log cycles (17). Huang and Toledo (8) successfully reduced the number of bacteria on fresh mackerel fish. Numbers of Salmonella *typhimurium* were reduced 68.72% - 85.01% utilizing doses between 1.63mW s/cm^2 and 9.78mW s/cm^2 on poultry skin (18). Bacterial levels of *Salmonella senftenberg* and *Escherichia coli* have also been reduced on pork muscle by 2.0 and 1.9 logs respectively using a 1.0mW/cm^2 dose of UV radiation (22). Shell eggs treated with varying doses of UV radiation showed reductions in populations of *S. typhimurium,* aerobes, molds and yeasts (11, 12).

UV could be a weapon in the fight against pathogens and spoilage microorganisms in poultry processing. This study was conducted to determine the usefulness of UV treatment for reduction of bacterial load on chicken.

MATERIALS AND METHODS

UV Treatment of Inoculated Agar Plates

Stock cultures of *E. coli* (ATTC Number: 25922) were grown to 5.5 X 10^8CFU/ml. Serial dilutions from 10^1 to 10^8 of the *E. coli* inoculum were spread plated on Standard Methods Agar (SMA) (Difco Laboratories, Inc., Detroit, Michigan) plates at the Food Science and Technology building, Virginia Polytechnic Institute and State University (VPI and SU) in Blacksburg, Virginia. After inoculation, they were placed in insulated containers with ice gel packs and transported to Cryovac, Inc. (Duncan, South Carolina) for UV treatment.

In preparation for sample treatments, measurements of UV radiation intensity were obtained on the UV machine (PureLight™ low-pressure system Model PL-L4-1065, PureLight Technologies, LLC, San Jose, California). A UV meter (Spectronics Corporation model DM254N, Westbury, N.Y) was inserted into both ends of the UV machine chamber 10 times. The intensity of the UV radiation, as measured by the Spectronics meter ranged between 20.1 and 21.9mW/cm^2, for an average intensity of 21mW/cm^2. The actual intensity varied, according to the position under the source bulbs in the chamber. Since dose =intensity X time, the following doses were calculated for use on the agar plates:

D0 = 0mW/cm^2 X 0 s = 0mW s/cm^2 (control-no exposure)

D1 = 21mW/cm^2 X 12 s = 252mW s/cm^2

D2 = 21mW/cm^2 X 30 s = 630mW s/cm^2

D3 = 21mW/cm^2 X 60 s = 1260mW s/cm^2

D4 = 21mW/cm^2 X 120 s = 2520mW s/cm^2

D5 = 21mW/cm^2 X 180 s = 3780mW s/cm^2

Lids were removed from the plates and groups of three plates were exposed to D0, D1, D2, D3, D4, and D5. The plates were then placed back into the containers and transported to VPI and SU. Upon return to VPI and SU, plates were removed from the insulated containers and incubated at 35° C for 48 hr.

After treatment, incubation and enumeration of the *E. coli* inoculated agar plates had been completed, inoculation levels and UV dosages were selected for chicken samples based on

logarithmic kills on plates and other research results.

Chicken Breasts

Fresh whole chicken breasts from similar production lots were obtained from three different Virginia poultry processing facilities over a six-week period. They were transported within 24 hr of slaughter in insulated containers with ice gel packs to VPI and SU. At VPI and SU, the whole chicken breasts were handled aseptically, and halved into split, boneless, skinless chicken breasts and then separated into three groups. Groups one and two consisted of twelve chicken breasts each used for microbial testing and group three contained 50 chicken breasts used for sensory testing. These groupings occurred in each of the three replications of the project.

All chicken breasts (groups one, two and three = 74 total per rep) were placed in multilayered, coextruded polyolefin E-bags (Cryovac Inc., Duncan, South Carolina). Breasts from group one were surface inoculated with 1ml of *E. coli* (ATTC Number: 25922) grown in a liquid culture medium and serially diluted to approximately 2.0 X 10^6 CFU/ml. All breasts were vacuum packaged (Koch Industries model X180, Kansas City, Missouri) and returned to insulated containers with ice gel packs for transport and UV treatment at Cryovac, Inc.Vacuum packaging flattened the chicken breasts to where there were essentially only two sides to treat; i.e. no edges.

The transmission rate of the E-bags was measured and ranged between 79.5% and 80.5% of the unshielded, full dose for an average transmission rate of 80% (0.80). Since dose = (intensity X transmission rate of packaging material) X time, the following doses were calculated (rounded to nearest whole number) for chicken breasts:

T0 = 0mW s/cm^2 (control-no exposure)

T1 = (21mW/cm^2 X 0.80) X 2 s = 34mW s/cm^2

T2 = (21mW/cm^2 X 0.80) X 6 s = 101mW s/cm^2

T3 = (21mW/cm^2 X 0.80) X 12 s = 202mW s/cm^2

T4 = (21mW/cm^2 X 0.80) X 30 s = 504mW s/cm^2

T5 = (21mW/cm^2 X 0.80) X 60 s = 1008mW s/cm^2

T6 = (21mW/cm^2 X 0.80) X 120 s = 2016mW s/cm^2

T7 = ($21 mW/cm^2$ X 0.80) X 180 s = $3024 mW\ s/cm^2$

Within eight hr of vacuum packaging, two chicken breasts from groups one and two were given treatments T0, T1, T2, T3, T4 and T5 respectively. Five chicken breasts from group three were given treatments T3, T4, T5, T6 and T7 respectively and 25 breasts were given treatment T0.

The vacuum packaged chicken breasts were placed flat on a plastic tray. Transit time on a steady speed conveyor belt, which traversed the center of the chamber, was 12 sec from the entrance to the exit of the chamber. The samples with < 12 sec exposure time were placed into the chamber on the tray with UV resistant gloved hands and removed after correct exposure time. Samples with 12 sec exposure were run straight through the chamber. Chicken breasts with >12 sec exposure were conveyed to the middle of the chamber where the belt was stopped and the tray of packages left under the UV source bulbs until the time had passed for the proper UV dosage to have been administered. The belt was then restarted and the tray of chicken breasts removed from the belt after it had exited the chamber. The chicken breasts were then flipped over 180° and sent through a second time using the same procedure so that both surfaces received an equal dose. The treated packages were then placed back into the insulated containers with ice gel packs and transported back to VPI and SU. Packages were removed from the containers and stored in a refrigerator maintained at $\leq$ 4° C until preparation for microbial or sensory testing.

One Day Microbial Sample Set

Within 24 hr of the UV treatments, one chicken breast from groups one and two, at each UV treatment level, were unpackaged and aseptically placed in separate sterile plastic stomacher bags (Seward, Ltd., London, U.K.). One hundred ml of sterile buffered peptone water (Difco Laboratories, Inc., Detroit, Michigan) were then added to the rinse bags and the rinse bags were vigorously shaken for 1 min. The rinse solution or serial dilutions of the rinse solution were spread plated on duplicate plates of appropriate media for enumeration of aerobic psychotrophs, lactic acid bacteria, *Campylobacter* and *E.coli* as described below.

Aerobic Psychrotrophs

Plates for aerobic psychrotrophic counts were prepared by spread plating 0.1ml of the poultry breast rinse and 0.1ml serial dilutions of the rinse on duplicate plates of SMA per standard spread plating procedure (2). Plates were incubated at 7° C +/- 1° C for 10 days. After incubation, plate counts were taken and raw numbers were transformed to $\log_{10}$ CFU/ml and reported as aerobic psychrotrophic CFU/ml.

Lactic Acid Bacteria

Plates for lactic acid bacteria (LAB) counts were prepared by spread plating 0.1ml of the poultry breast rinse and 0.1 ml serial dilutions of the rinse on duplicate plates of de-Man Rogosa Sharp (MRS) agar (Difco Laboratories, Inc., Detroit, Michigan) per standard spread plate procedure(2). The plates were then overlaid with an additional 12-15 ml of MRS agar to create an anaerobic environment conducive to LAB growth. Plates were incubated at 7° C +/- 1° C for 10 days. After incubation, plate counts were taken and raw numbers were transformed to $\log_{10}$ CFU/ml and reported as LAB CFU/ml.

Campylobacter

Plates for *Campylobacter* counts were prepared by spread plating 1ml of the poultry breast rinse on four plates (0.25ml per plate) of Campy-CEFEX agar (1). The plates were then placed upside down in an anaerobe jar. A gas-generating pouch (BBL GasPak Plus *Campylobacter* pouch, Becton Dickinson Co., Cockeysville, MD.) was activated and sealed in the anaerobe jar to create the microaerobic growth conditions (85% nitrogen, 10% carbon dioxide, 5% oxygen) conducive to *Campylobacter* optimum growth. The plates were incubated at 42° C +/- 1° C for 48 hr. After incubation, plate counts were taken on the four plates and added together. Raw numbers were reported as *Campylobacter* CFU/ml.

Ten ml of the poultry breast rinse were added to 30 ml of double strength Bolton's enrichment broth (1) in a plastic bag (Whirl-pak, Fort Atkinson, Wisconsin). Sample bags were then flushed with a mixture of 85% nitrogen, 10% carbon dioxide and 5% oxygen and heat-sealed. The bags were placed in an agitating incubator (Innova model 4230, New Brunswick

Scientific, Edison, New Jersey) and agitated at 100 RPM and 42° C for 24 hr. After incubation, the bags were removed from the incubator and 0.1ml of the solution was spread plated onto a Campy-CEFEX agar plate. The plates were then placed upside down in anaerobe jars and a gas-generating pouch was activated and sealed in the anaerobe jar to create the microaerobic growth conditions conducive to *Campylobacter* optimum growth. The plates were incubated at 42° C for 48 hr. After incubation, plates were checked for presence or absence of *Campylobacter*. Tests were conducted to confirm suspected colonies using a latex agglutination test for identification of *Campylobacter* spp. (Oxoid Limited, Basingstoke, England). Data were reported as +/- for presence of *Campylobacter*.

E. coli

One ml of the poultry breast rinse was pipetted directly onto 3M™ Petrifilm™ *E. Coli*/Coliform (EC) plates in duplicate (3M Company, St.Paul, Minnesota) or serially diluted and 1 ml of this solution was plated. Petrifilm™ EC plates were incubated at 35° C +/- 1° C for 48hr. After incubation, plate counts were taken and raw numbers were reported as *E. coli* CFU/ml.

Seven Day Microbial Sample Set

After seven days of storage at $\leq$ 4° C, the second set of group one and group two chicken breasts, were unpackaged, rinsed and plated as described above. Plates were prepared and reported as above for aerobic psychrotrophs, LAB and *E. coli*.

All microbiological data, in the form of raw counts, were analyzed by running proc GLM on SAS software (SAS Institute Inc. Cary, NC.).

Two and Seven Day Sensory Sample Sets

Two days after UV treatments, and again seven days after treatments, triangle tests for difference were conducted on the chicken breasts to evaluate possible organaleptic changes in the chicken. The tests were conducted in the sensory lab of the Food Science and Technology building at VPI and SU. Samples were presented in the order as represented by Table in

appendix (A1)

On the day of each test, the chicken breasts were removed from the packages and cooked in a still air gas oven (Magic Chef model31001SAW, Cleveland Cooking Products, Cleveland, TN.) at 325° F for 25 min (internal temperature of 175°-180° F). After the chicken breasts cooled, they were cut into approximately 1" cubes. Each sample was placed in a zippered plastic sandwich bag and labeled with a three-digit code. Codes corresponded to either untreated chicken breast (T0), or chicken breast treated with one of the five different doses of UV radiation (T3, T4, T5, T6 and T7). Each individually cooked, bagged and labeled sample was stored in an ice gel packed container until final preparation and presentation for sensory testing. Approximately 20 min before testing, the bagged samples were placed in a warm water bath (approximately 55° C) for presentation to a sensory taste panel at simulated serving temperature.

The panel for each test consisted of between twenty and thirty members. Panelists were seated in individual booths. Red lighting was used to mask any differences in color of samples due to cooking. Panelists tasted one set of samples from each of the five UV dose level-VS-control groups (15 total samples).

Bagged samples were placed on a plastic tray and delivered through a pass-through hatch. Included on the tray were a triangle test scorecard (appendix A2) and an informed consent form that was signed and dated by the participant (appendix A3).

Panelists were instructed to taste the samples in order as presented on the tray. They were told that two of the samples were identical, that one was different and that in the case where no difference could be discerned, a guess was required. Each 2 day/ 7 day set of chicken breasts were tested in triplicate for a total of six triangle tests for difference on weekdays between the hours of 9 am and 3 pm over a six-week period.

Statistical analysis was conducted using a triangle test for difference and the corresponding triangle test for difference: critical number (minimum) of correct answers chart as described in Meilgaard et al. (14).

RESULTS AND DISCUSSION

Agar Plates

There was no growth on any of the *E coli* inoculated plates treated with 630mW s/cm^2, 1260mW s/cm^2, 2520mW s/cm^2 and 3780mW s/cm^2 doses of UV. On the 10^6, 10^7, and 10^8 plates treated with a 252mW s/cm^2 dose, there were colonies that had grown together between ¼" and ½"inch on the edge of the agar plates. It was impossible to get a microbial count because colonies could not be divided into distinct individual units. Tests were run on the UV transmission rate of the plastic petri dishes and were found to be 0 mW s/cm^2. It was hypothesized that the UV treatment had killed all of the colonies on all of the plates except those in high CFU/ml concentration that possibly had shielded each other or had been shielded/shaded by the edge of the non-transmissive plastic petri dish. In summary, 252mW s/cm^2 dose of UV radiation from the UV machine had reduced bacteria counts of *E. coli* (ATTC Number: 25922) on agar plates by 8 logs.

Poultry Microflora

Data analysis for *E. coli* inoculated breasts showed that there was a statistical difference ($p \leq 0.05$) in control-VS- treated chicken breast bacterial counts at all treatment levels. The analysis also showed that there was no statistical difference among bacterial counts of any of the samples treated at any of the five UV levels ($p \geq 0.05$). This means that there was an initial kill on all treated samples, but beyond a point, extra exposure time (larger dose) resulted in little if any additional kill.

These results are in agreement with previous research on inoculated samples of other meats/foods. Wong et al. (22) found that larger doses of UV radiation did not significantly increase bacterial kills on pork skin and pork muscle inoculated with *Salmonella senftenberg*. Kuo et al. (11) found that bacterial levels of *Salmonella typhimurium* inoculated onto the surface of eggshells and treated with doses of between 0mW s/cm^2 and 37mW s/cm^2 showed significant kill, but that additional doses of between 37mW s/cm^2 and 260mW s/cm^2 showed no significant additional kill. Also, Stermer et al. (17) found that an increase of a 16mW s/cm^2 to a128mW

s/cm^2 dose caused a meager 2% additional reduction on beef plate meat. They attributed this lack of beneficial effect of higher doses to the belief that a portion of the bacteria were shielded from the UV irradiation.

Data analysis for aerobic psychrotrophs, showed that there was no statistical difference ($p \geq 0.05$) in control-VS- treated chicken bacterial counts in any of the three repetitions of the experiment. This is in agreement with findings by Wallner-Pendleton et al. (21). They UV treated halved poultry carcasses and found no statistical differences in numbers of naturally occurring psychrotrophic bacteria on day 0 or day 3 after treatment and that psychrotrophic bacterial populations were not appreciably altered by UV treatments when their numbers were compared with bacterial counts obtained from untreated chicken halves held for 10 days at 7° C.

Analysis for lactic acid bacteria also showed that there was no statistical difference ($p \geq 0.05$) in control-VS- treated chicken bacterial counts in any of the three repetitions of the experiment.

Natural microflora reduction results were in agreement with results that had been obtained from informal testing done on different cuts of fresh meats tested by the staff at Cryovac. In running these tests, they had not achieved natural bacterial flora reduction on any product at a level greater than 1 log cycle, which they had preset as their desired level of significance (Personal Conversation, Carolyn Henry, Cryovac Inc. 1999).

Campylobacter growth was not observed on any of the unenriched *Campy-Cefex* agar plates at either 10^0 or 10^{-1} dilution factors for any of the treatments or from the controls. To determine if *Campylobacter* was present at a concentration of < 1/100ml, sample rinses were enriched in double strength Bolton's broth and plated onto Campy-Cefex agar plates. Two of the plates showed *Campylobacter* growth. Those were plates from treatment # 2 (101mW s/cm^2) from repetition # 2 and treatment #4 (504mW s/cm^2) from repetition # 3. Suspect colonies from both plates tested positive for *Campylobacter* in the Oxiod^R latex agglutination test. The UV treatments did not appear to completely inactivate the low levels of *Campylobacter* that were naturally present. *Campylobacter* is an organism that is sensitive to many environmental and experimental stresses and is not as easy to isolate and identify as some other foodborne bacteria. Lack of confirmable isolates could be due to many factors, not just the presumption that it was absent or at detectable levels.

Sensory

Upon unpackaging and preparation, the aroma of the chicken was evaluated subjectively before cooking and after cooking when the oven doors were first opened. No off odors were encountered at any level of UV treatment. Informal visual observation of both raw and cooked chicken showed no detectable difference in color or appearance between the treated and untreated chicken. This is in agreement with research by Wallner-Pendleton et al (21) who reported no difference in Hunter Lab color values, a measure of color difference (Hunter Associates Laboratory, Inc., Reston, VA) in chicken breasts removed from UV-treated halved chicken carcasses.

A total of 71 panelists participated in the 2 day sample set testing and a total of 81 panelists participated in the 7 day sample set testing. Chicken exposed to 504mW s/cm^2 was the only one of the five different UV treatments on the second day post-treatment test that showed significant difference from control ($p \leq 0.05$) using a Triangle Test for Difference test table (14). Treatment #4 (2016mW s/cm^2) was the only one in the 7 day set that showed significant difference from control ($p \geq 0.05$).

Demographic questions asked at the end of the test form showed that the panel was comprised of 50.6% females and 49.4% males. Ages ranged from 18-52, with 29.6% being 18-24, 29.6% were 25-31, 14.5% were 32-38, 13.8% were 39-45, and 10.5% were 46-52. Eighty nine percent of respondents indicated that they purchased chicken from a grocery store at least several times per month.

REFERENCES

1. Agricultural Research Service. 1998. Method for the enumeration of *Campylobacter* jejuni/coli from poultry rinses. Agricultural Research Service, Athens, Georgia.
2. American Public Health Association. 2001. Compendium of Methods for the Microbiological Examination of Foods 4th edition. Washington, D.C.
3. Bjerklie, S. December 1999. Is irradiation in your future? Meat Processing: North American Edition: 38, 40, 42-43.
4. Butler, R. C., V. Lund, and D. A. Carlson. 1987. Susceptibility of *Campylobacter jejuni* and *Yersinia enterocolitica* to UV radiation. Appl. Environ. Microbiol. 53:375-378.
5. Chang, J. C. H., S. F. Ossoff, D. C. Lobe, M. H. Dorfman, C. M. Dumais, R. G. Qualls, and J. D. Johnson. 1985. UV inactivation of pathogenic and indicator microorganisms. Appl. Environ. Microbiol. 49:1361-1365.
6. Council of Agricultural Science & Technology. 1994. Foodborne pathogens: Risks and consequences. Task Force report no. 122. Council of Agricultural Science & Technology. Iowa State University, Ames, Iowa.
7. Groves, M. February 13, 1999. USDA approves irradiation of meat to kill contaminants. Agriculture industry is unsure about consumer acceptance. Treated products would carry labels. The Los Angeles Times.
8. Huang, Y., and R. Toledo. 1982. Effect of high doses of high and low intensity ultraviolet irradiation on the surface microbiological counts and storage life of fish. J. Food Sci. 47:1667-1669, 1731.
9. Jay, J. M. 1996. Modern food microbiology. 5th ed. Chapman & Hall, New York, New York.
10. Kaess, G., and J. F. Weidermann. 1973. Effects of ultraviolet radiation on the growth of microorganisms on chilled beef slices. J. Food Technol. 8:59-69.
11. Kuo, F. L., J. B. Carey, and S. C. Ricke. 1997. UV irradiation of shell eggs on populations of aerobes, molds, and inoculated *Salmonella typhimurium*. J. Food Prot. 60:639-643.
12. Kuo, F. L., S. C. Ricke, and J. B. Carey. 1997. Shell egg sanitation: UV radiation and egg

rotation to effectively reduce populations of aerobes, yeasts and molds. J. Food Prot. 60:694-697.

13. Marenzi, C. 1986. Proper meat storage prevents spoilage. Poultry 6:12-15.
14. Meilgard M., G. V. Civille and B. T. Carr. 1991. Sensory evaluation techniques 2nd edition. CRC Press, Boca Raton, Florida.
15. Morgan, R. 1989. UV 'green' light disinfection. Dairy Ind. Int. 54:33-35.
16. Shechmeister, I. L. 1991. Sterilization by ultraviolet radiation p. 553-565 *In* S. S. Block (ed.) Disinfection, sterilization, and preservation, 4th edition. Lea & Febiger, Malvern, Pennsylvania.
17. Stermer, R. A., M. Lasater-Smith and C. F. Brasington. 1987. Ultraviolet radiation- an effective bactericide for fresh meat. J. Food Prot. 50:108-111.
18. Sumner, S. S., E. A. Wallner-Pendleton, G. W. Froning and L. V. E. Stetson. 1995. Inhibition of *Salmonella typhimurium* on agar medium and poultry skin by ultraviolet energy. J. Food Prot. 59:319-321,336.
19. United States Department of Agriculture-Food Safety and Inspection Service. 1999. Backgrounders: USDA Issues final rule on meat and poultry irradiation. Food Safety and Inspection Service-United States Department of Agriculture, Washington, D.C.
20. Van Laack, R. L. J. M. 1994. Spoilage and preservation of muscle foods. p. 378-405. *In* Muscle foods: meat, poultry, and seafood technology. D. M. Kinsman, A. W. Kotula, and B. C. Breidstein (eds), Chapman and Hall, Inc., New York, N.Y.
21. Wallner-Pendleton, E. A., S. S. Sumner, G. W. Froning, and L. V. E. Stetson. 1994. The use of ultraviolet radiation to reduce *Salmonella* and psychrotrophic bacterial contamination on poultry carcasses. Poultry Sci. 73:1327-1333.
22. Wong, E., R. H. Linton, and D. E. Gerrard. 1998. Reduction of *Escherichia coli* and *Salmonella senftenburg* on pork skin and pork muscle using ultraviolet light. Food Micro. 15:415-423.
23. Yousef, A. E., and E. H. Marth. 1988. Inactivation of Listeria monocytogenes by ultraviolet energy. J. Food Sci. 53 (2):571-573.

CONCLUSIONS

UV treatment of *E. coli* inoculated petri plates with a 252mW s/cm^2 dose, showed a reduction of 8 logs. If the surface is smooth and there is no place for the microorganisms to "hide" (i.e. pores, shaded areas), UV radiation is a very effective bactericide.

It was concluded from the results of microbiological testing that a steady exposure of UV radiation at the tested intensities and times of exposures would not significantly benefit the keeping quality or the shelf life of split, boneless, skinless chicken breasts. The reason for this is probably because of the bacteria hiding in pores, shaded areas or on the edges of the chicken breasts. UV irradiation has limited penetrating ability.

It was concluded from the results of the triangle tests for difference and the written comments offered by panelists on the questionnaires, that UV treatments do not significantly affect the taste of exposed chicken breast. Furthermore, since the average consumer adds some sort of spice or seasoning, or bakes chicken in combination with another dish or a seasoned coating to fried chicken, the chances of consumer detection of UV treated chicken seem even more remote.

FIGURE 1.

E. coli #25922 survival on vacuum packaged UV treated boneless skinless chicken breasts

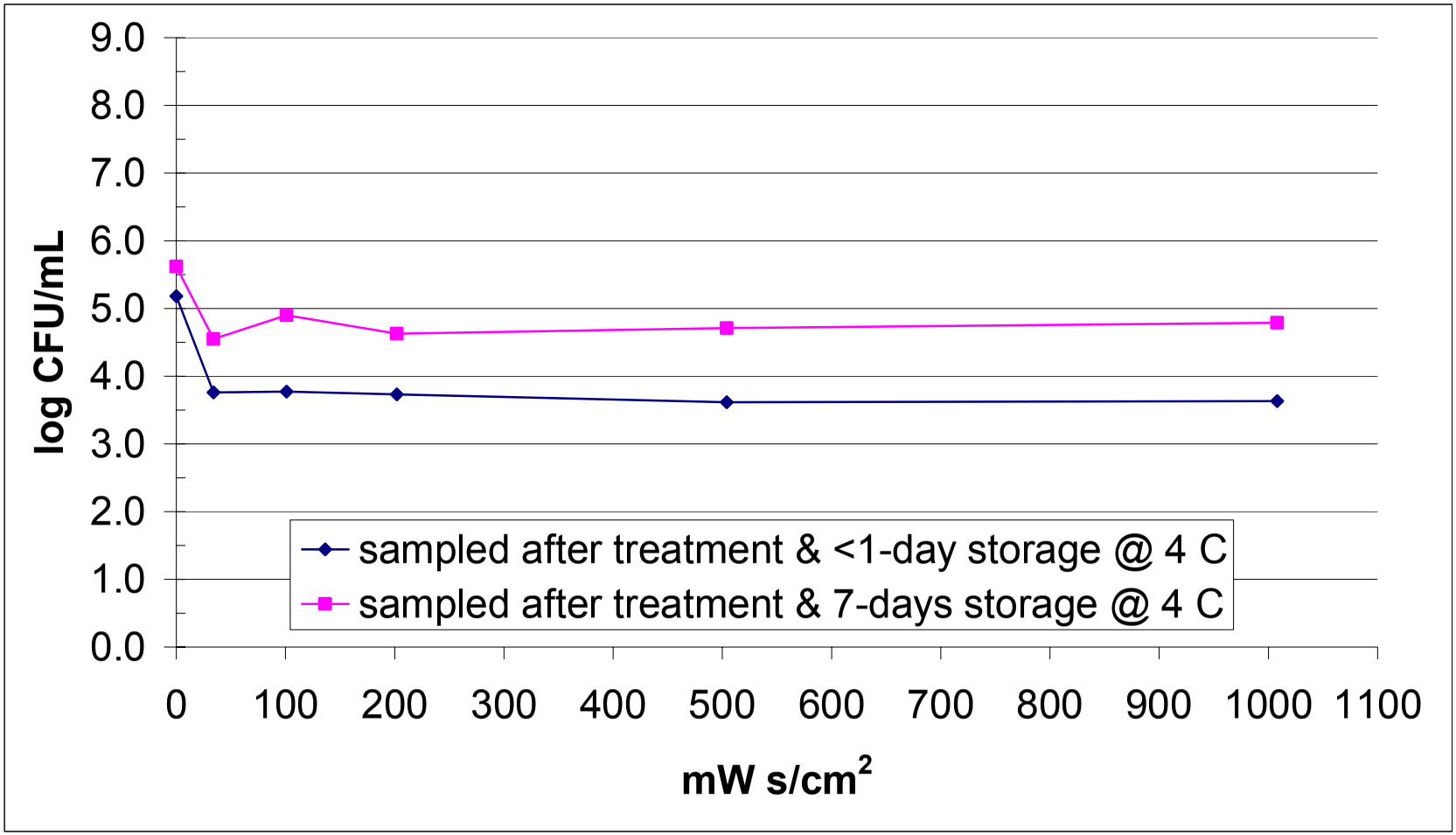


FIGURE 2.

Aerobic bacteria survival on vacuum packaged

UV treated boneless skinless chicken breasts

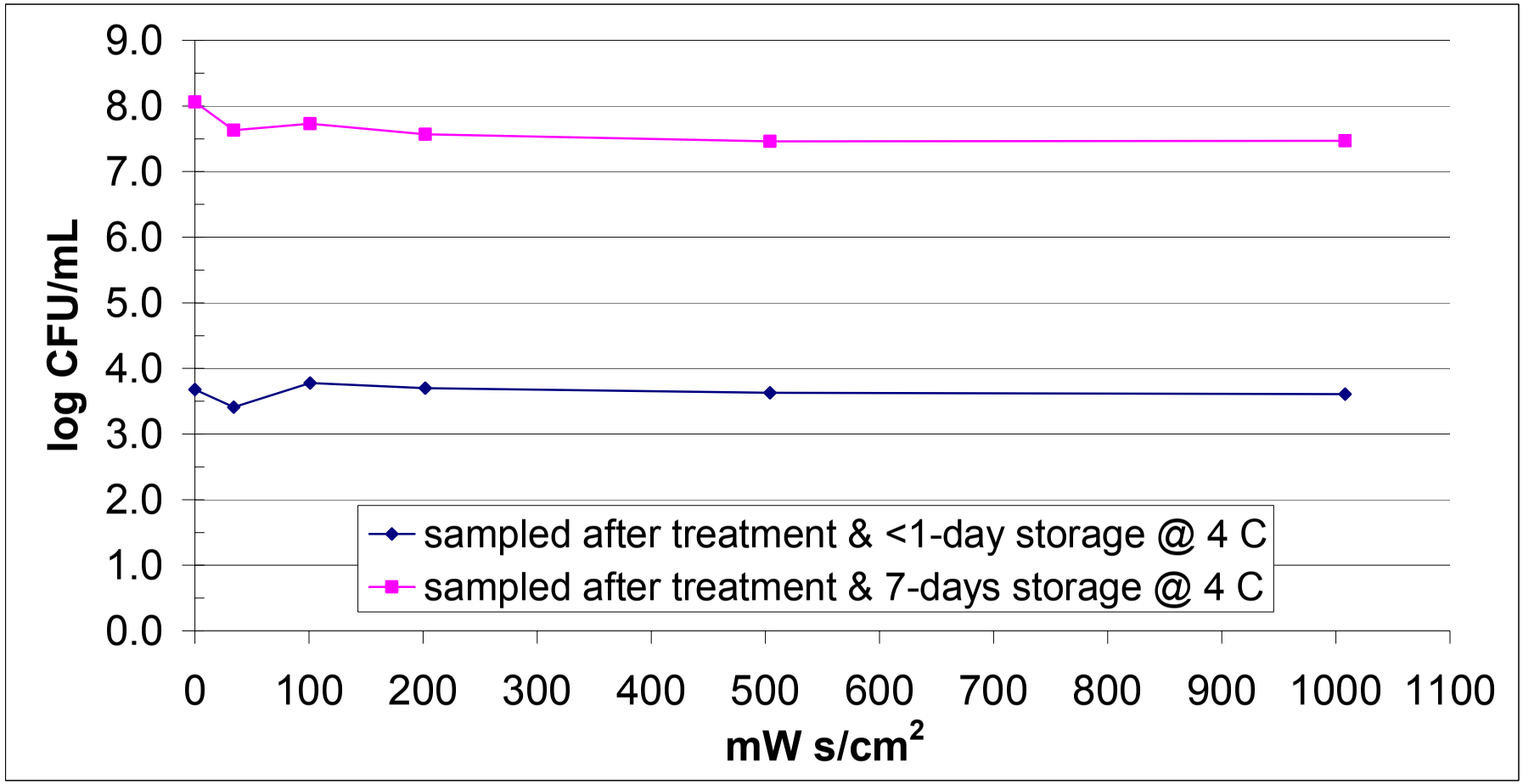
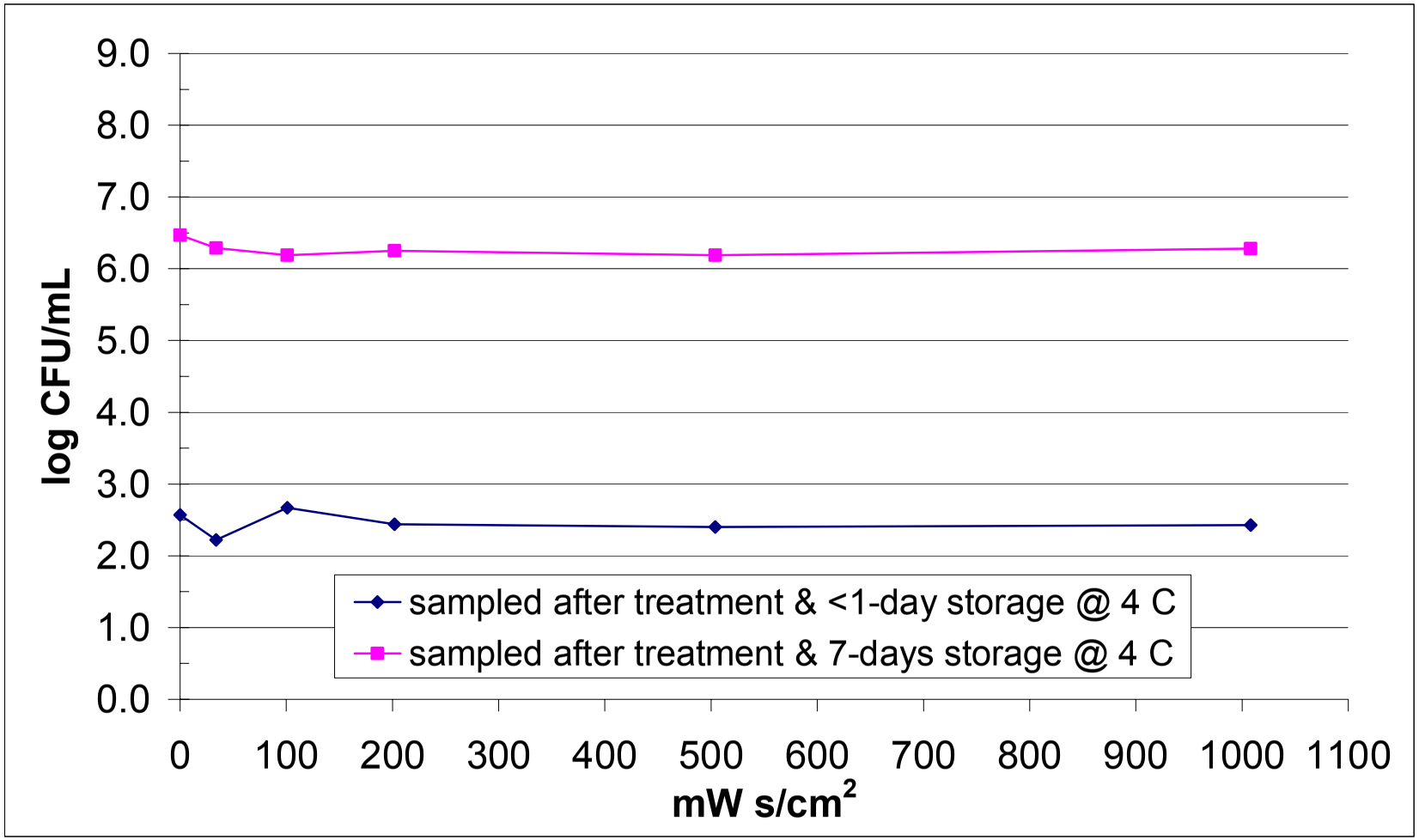


FIGURE 3.

Lactic acid bacteria survival on vacuum packaged UV treated boneless skinless chicken breasts



APPENDIX

A1

Presentation Order Left To Right

Panelist	T 3	T 4	T 5	T 6	T 7
1	T/U/U	U/T/T	T/T/U	U/U/T	T/U/T
2	U/T/T	T/T/U	U/U/T	T/U/T	U/T/U
3	T/T/U	U/U/T	T/U/T	U/T/U	T/U/U
4	U/U/T	T/U/T	U/T/U	T/U/U	U/T/T
5	T/U/T	U/T/U	T/U/U	U/T/T	T/T/U
6	U/T/U	T/U/U	U/T/T	T/T/U	U/U/T
7	T/U/U	U/T/T	T/T/U	U/U/T	T/U/T
8	U/T/T	T/T/U	U/U/T	T/U/T	U/T/U
9	T/T/U	U/U/T	T/U/T	U/T/U	T/U/U
10	U/U/T	T/U/T	U/T/U	T/U/U	U/T/T
11	T/U/T	U/T/U	T/U/U	U/T/T	T/T/U
12	U/T/U	T/U/U	U/T/T	T/T/U	U/U/T
13	T/U/U	U/T/T	T/T/U	U/U/T	T/U/T
14	U/T/T	T/T/U	U/U/T	T/U/T	U/T/U
15	T/T/U	U/U/T	T/U/T	U/T/U	T/U/U
16	U/U/T	T/U/T	U/T/U	T/U/U	U/T/T
17	T/U/T	U/T/U	T/U/U	U/T/T	T/T/U
18	U/T/U	T/U/U	U/T/T	T/T/U	U/U/T
19	T/U/U	U/T/T	T/T/U	U/U/T	T/U/T
20	U/T/T	T/T/U	U/U/T	T/U/T	U/T/U
21	T/T/U	U/U/T	T/U/T	U/T/U	T/U/U
22	U/U/T	T/U/T	U/T/U	T/U/U	U/T/T
23	T/U/T	U/T/U	T/U/U	U/T/T	T/T/U
24	U/T/U	T/U/U	U/T/T	T/T/U	U/U/T
25	T/U/U	U/T/T	T/T/U	U/U/T	T/U/T
26	U/T/T	T/T/U	U/U/T	T/U/T	U/T/U

27	T/T/U	U/U/T	T/U/T	U/T/U	T/U/U
28	U/U/T	T/U/T	U/T/U	T/U/U	U/T/T
29	T/U/T	U/T/U	T/U/U	U/T/T	T/T/U
30	U/T/U	T/U/U	U/T/T	T/T/U	U/U/T

T=Treated, U= Untreated

A2

TRIANGLE TEST

Judge Number_____ Date_____

INSTRUCTIONS

Please taste samples on the tray in order from left to right.

In each separate test, two of the samples are identical, one is different.

Determine which sample is different.

If no difference is apparent, you must guess.

Rinse mouth with water between samples.

Circle the number of the sample that is different.				Comments
Test #1	_____	_____	_____	_____
Test #2	_____	_____	_____	_____
Test #3	_____	_____	_____	_____
Test #4	_____	_____	_____	_____
Test #5	_____	_____	_____	_____

Questions:

1.) Gender: Female_____ Male_____ (check one)

2.) Age:

18-24_____

25-31_____

32-38_____

39-45_____

46-52_____

53-59_____

60-66_____

>67 _____

3.) How often do you purchase chicken from the grocery store

Never_____

Rarely (3 per year)_____

Sometimes (several times a month)_____

Often (weekly)_____

Alot (Daily)_____

THANK YOU VERY MUCH FOR PARTICIPATING IN THIS SENSORY TEST.

A3

Virginia Polytechnic Institute and State University

Informed Consent for Participation in Sensory Evaluation

Title of Project: Reduction of microbial load on boneless, skinless chicken breast lobes using UV light.

Principal Investigator: Daniel Martin

I. THE PURPOSE OF THIS PROJECT

You are invited to participate on a sensory evaluation test to determine if chicken samples exposed to UV light taste the same or taste different than samples not exposed to UV. Your input may help determine whether poultry producers will use UV light as a processing aid.

II. PROCEDURES

There will be 6 sessions over a period of 7 weeks involving about 15 minutes at each session. You will be presented with 5 comparisons at each session. Should you find a sample unpalatable or offensive, you may choose to spit it out and continue to other samples.

III. BENEFITS/RISKS OF THE PROJECT

You may receive the results or summary of the panel when the project is completed. Some risk may be involved if you have an unknown food allergy. Certain individuals are sensitive to some foods such as milk, eggs, wheat gluten, strawberries, chocolate, legumes (nuts), etc. If you are aware of any food or drug allergies, list them in the following space._____

_____

IV. EXTENT OF AN0NYMITY AND CONFIDENTIALITY

The results of your performance as a panelist will be kept strictly confidential. Individual panelists will be referred to by code for analyses and in any publication of the results.

V. COMPENSATION

For participation in the project, you will receive candy for each session completed.

Course Credit: You may not receive extra credit for any classes in which you are enrolled.

VI. FREEDOM TO WITHDRAW

It is essential to sensory evaluation projects that you complete the session in so far as possible. However, there may be conditions preventing your completion of this session. If after reading and becoming familiar with the sensory project, you decide not to participate as a panelist, you may withdraw at any time without penalty.

VII. APPROVAL OF RESEARCH

This research project has been approved by the Institutional Review Board for projects involving human subjects at Virginia Polytechnic Institute and State University and by the human subjects review of the Department of Food Science and Technology.

VIII. SUBJECT'S RESPONSIBILITIES and PERMISSION

I have read the information about the conditions of this sensory evaluation project and give my voluntary consent for participation in this project. I know of no reason I cannot participate in this study:

_____

Signature/Date

Please provide address and phone number so investigator may reach you in case of emergency.

Address _____

Phone _____

Should I have any questions about this research or its conduct, I should contact:

Daniel Martin MS Candidate (540) 231-8796
Department of Food Science and Technology

Joseph Marcy Ph.D. (540) 231-7850
Department of Food Science and Technology

Susan Duncan, Ph.D. (540) 231-6805
Department of Food Science and Technology

Joseph Eifert Ph.D. (540) 231-3658
Department of Food Science and Technology

Tom Hurd (540) 231-5281
Director, Sponsored Programs

Detach This Page and Take It With You.

VITAE

Daniel E. Martin Jr. was born to Daniel E. Martin Sr. and Flora Catherine Sizemore Martin at 10:25 am in Logan General Hospital, located in Logan West Virginia on July 29, 1959. The family moved to Roanoke, Virginia in 1965 and have lived within 25 miles of Roanoke ever since.

Daniel Jr. was married to Julie Lynn Meyers in May of 1987. They have three children, Alissa Ann, 14, Brandi Lea, 12, and Bryan Christopher, 8. The family currently resides in Shawsville, approximately 13 miles from the Virginia Tech campus.