

ECOLOGY OF PATHOGENS

FORMATION OF BIOFILM AT DIFFERENT NUTRIENT LEVELS BY VARIOUS GENOTYPES OF *LISTERIA MONOCYTOGENES*

(J.P. Folsom, G.R. Siragusa, and J.F. Frank)

Strains of *Listeria monocytogenes* exhibit a range of ability to form biofilms. The objectives of this study were to determine if genetically related strains exhibit similar biofilm-forming capacity, and the effect nutrient concentration has on the ability of different strains to produce biofilm. Biofilms of 30 strains of *L. monocytogenes*, obtained from a variety of sources, were grown on stainless steel in tryptic soy broth [TSB] or a 1:10 dilution of TSB [DTSB] for 24 hours at 32°C. The amount of biofilm formed was determined using image analysis after staining the cells with bisBenzimide H 33258 (Hoechst 33258). The strains were genetically subtyped by repetitive element sequence-based PCR (rep-PCR) using the primer sets rep-PRO_{D1} and rep-PRO_{G5}. Data were analyzed by using ANOVA and Duncan's multiple range test. Eleven strains produced the same amount of biofilm in the two media. Fourteen strains produced more biofilm in TSB than DTSB. Five strains produced more biofilm in DTSB than TSB. Serotype 4b strains produced more biofilm accumulation in TSB than serotype 1/2a strains, while serotype 1/2a strains produced more in DTSB than did serotype 4b strains. Growth in DTSB resulted in decreased biofilm accumulation for serotype 4b strains. There was no correlation between genetic subtype and the amount of biofilm accumulation. These results indicate that serotype 1/2a and serotype 4b strains differ in the regulation of their biofilm phenotype. The poor biofilm accumulation of serotype 4b isolates when grown in DTSB could be a factor in the predominance of serogroup 1/2 strains in food processing plants, where nutrients may be limited.

CHARACTERIZATION OF A CHLORINE TOLERANT MUTANT OF *LISTERIA MONOCYTOGENES*

(J.F. Frank and J.P. Folsom)

Rough colony variants of *Listeria monocytogenes* ScottA (SA) were isolated from chlorine-treated cells that were tolerant to hypochlorous acid and produced increased amounts of biofilm. A derivative of one of these variants was smooth, produced even more biofilm and exhibited greater chlorine resistance biofilm when grown as a biofilm. The objective of this research was to compare the protein expression of the chlorine tolerance cultural variant to the wild type, to identify proteins that may be associated with biofilm production and chlorine tolerance. Suspension chlorine tolerance for several cultural variants (SAR, SAR5, and SBS) was determined by exposure to 60-120 ppm hypochlorous acid for five minutes. Hypochlorous acid tolerance of biofilms was determined after growing biofilms on stainless steel followed by exposure to 200 ppm hypochlorous acid for 5 min. All cultural variants were able to survive 120 ppm of hypochlorous acid in suspension. There was little difference in the hypochlorous acid tolerance of the cultural variant planktonic cells. The cultural variants produced greater amounts of biofilm than the S-form, and were more hypochlorous acid tolerant. The SBS variant was selected for proteomic comparison because it was the variant that produced the most biofilm and was most tolerant of hypochlorous acid when grown as a biofilm. Protein expression of planktonic and biofilm cells of SBS was compared to SA by using two dimensional difference gel electrophoresis. The 50s ribosomal protein, L10 was down regulated in biofilm SBS. Other proteins down regulated in planktonic SBS were the peroxide resistance protein (Dpr), and a sugar binding protein (LMO0181). This sugar binding protein was also up regulated in biofilm SBS. One protein spot down regulated in planktonic SBS contained both 50s ribosomal protein L7/L12 and an unknown protein (LMO1888). The frequency at which chlorine tolerant variants of *L. monocytogenes* arise in food processing environments should be a topic for further investigation.

CROSS-CONTAMINATION OF *LISTERIA MONOCYTOGENES* BETWEEN PROCESSING EQUIPMENT AND DELI MEATS

(L. Ma, G. Zhang, C-M. Lin, and M.P. Doyle)

Contamination of ready-to-eat meats by *Listeria monocytogenes* has resulted in outbreaks of listeriosis and major product recalls. Food processing equipment such as slicers can serve as a potential contamination source. This study was conducted to determine (i) the dynamics of cross-contamination of *Listeria monocytogenes* from slicing equipment to two different types of turkey meat, cured and non-cured, (ii) the role of the conveyor belt in

the transfer event, (iii) the fate of *L. monocytogenes* on contaminated samples during storage at 4°C for up to 90 days, and (iv) the efficacy of the BAX-PCR and USDA conventional enrichment culture assays in detecting *L. monocytogenes* on turkey meats. A five-strain mixture of *L. monocytogenes* was inoculated at ca. 500 CFU onto the blade of a commercial slicer. Five consecutive meat slices were packed per package, vacuum sealed, stored at 4°C, and sampled (entire package) at 1, 30, 60, and 90 days postslicing. Of the two types of deli meats, a larger number of *L. monocytogenes*-positive samples were obtained from non-cured turkey meat, 48 of 800 samples compared to 14 of 800 of cured turkey meat. Most of the *L. monocytogenes*-positive samples in cured turkey meat were detected at 30 days postslicing, whereas the largest number of *L. monocytogenes*-positive samples for non-cured turkey meat was recovered at 90 days postslicing. Slightly more (48 vs. 43 of 800 samples) *L. monocytogenes*-positive meat samples were obtained when the conveyor belt was used and the positive meat samples obtained at the middle or near the end of sliding were likely from contamination of the conveyor belt. For cured turkey meat, *L. monocytogenes* was detected in 5 meat samples by both the enrichment culture and BAX-PCR assays and in 9 samples only by enrichment culture assay. For non-cured turkey meat, *L. monocytogenes* was detected in 23 samples by both assays in 13 samples only by the enrichment assay, and in 10 samples only by the BAX-PCR assay. *L. monocytogenes* cell numbers were generally very low when detected on either type of turkey meat contaminated during processing. The results indicate that *L. monocytogenes* can be transferred from a contaminated slicer onto deli meats and survive storage at 4°C. The enrichment culture and BAX-PCR assays are complementary to each other in the detection of *L. monocytogenes*, especially for non-cured turkey meat.

SURVIVAL OF *ENTEROBACTER SAKAZAKII* IN POWDERED INFANT FORMULA AS AFFECTED BY COMPOSITION, WATER ACTIVITY, AND TEMPERATURE

(J. B. Gurtler and L. R. Beuchat)

Infant *Enterobacter sakazakii* infection was linked to cross-contamination from a blender used to prepare reconstituted powdered infant formula, but the pathogen was not isolated from the powdered formula. Although the blender was washed in a commercial dish-washing machine daily, the authors surmised that *E. sakazakii* may have adhered to, undergone desiccation, and survived on surfaces of the blender. In an outbreak of *E. sakazakii* meningitis, the pathogen was isolated from a stirring spoon and a dish brush but the powdered infant formula tested negative. Other reports suggest that reconstituted infant formula that has subsequently dried on abiotic surfaces may provide a suitable harbor whereby *E. sakazakii* survives and poses a risk of cross-contamination. Conditions to which powdered infant formulas are exposed, whether in the container in which they are manufactured or in open containers under environments with fluctuating relative humidity and temperature, may affect the viability of *E. sakazakii*. However, the interacting effects of composition of formulas, a_w , and temperature on survival of *E. sakazakii* are unknown. We did a study to determine the ability of *E. sakazakii* to survive in six commercially manufactured milk-based and soy-based powdered infant formulas. A ten-strain mixture of *E. sakazakii* was inoculated into the six infant formulas at three a_w ranges (a_w 0.25 – 0.30, 0.31 – 0.33, and 0.43 – 0.50) to give low (0.80 log CFU/g) and high (4.66 – 4.86 log CFU/g) populations. At an initial population of 0.80 log CFU/g, *E. sakazakii* was detected by enrichment in 6 of 6, 4 of 6, and 1 of 6 formulas stored for 12 months at 4, 21, and 30°C, respectively. In 4 of 6 formulas at a_w 0.25 – 0.30, initially high populations decreased significantly ($p \leq 0.05$), although by less than 1 log CFU/g, within 6 months at 4°C. Populations decreased significantly in all formulas at a_w 0.25 – 0.50 during storage for 1 month at 21 or 30°C, and again between 1 and 6 months in most formulas. Significant reductions occurred between 6 and 12 months in some formulas. At all storage temperatures, reductions in populations tended to be greater in formulas at a_w 0.43 – 0.50 than in formulas at a_w 0.25 – 0.30. The rate of inactivation of *E. sakazakii* in formulas was not markedly influenced by formula composition. Cells from mucoid and non-mucoid colonies formed by two strains on violet red bile glucose agar supplemented with pyruvate were inoculated into a milk-based powdered infant formula and a soybean-based powdered infant formula at a_w 0.43 – 0.86 and stored at 4, 21, and 30°C for up to 36 weeks. With few exceptions, populations of both strains decreased significantly in both formulas within 2 weeks at all temperatures; rates of death increased with increased a_w and storage temperature. The presence of mucoidal extracellular materials on the surface of *E. sakazakii* cells was not associated with protection against death. This study shows that the retention of viability of *E. sakazakii* in powdered infant formula is affected by a_w and temperature. Increases in both parameters cause an increase in the rate of death.

GROWTH OF *ENTEROBACTER SAKAZAKII* IN RECONSTITUTED INFANT FORMULA AS AFFECTED BY COMPOSITION AND TEMPERATURE

(J. B. Gurtler and L. R. Beuchat)

The Food and Agricultural Organization and the World Health Organization lists *E. sakazakii* as one of only two category-A pathogens in powdered infant formula, based on its ability to cause infections in infants and because powdered infant formula has been confirmed to be a vehicle of infection. At least 76 cases of *E. sakazakii* infections and 19 deaths of infants and children have been documented. The first outbreak of *E. sakazakii* infection linked to powdered infant formula obtained from a previously unopened can occurred in 2001. In another outbreak, powdered formula tested negative for *E. sakazakii* but the blender used to prepare the reconstituted formula was positive for the pathogen. This suggests that contamination of the blender could have resulted from contact with powdered, reconstituted infant formula, or some other source containing the pathogen. Because powdered infant formula contains an abundance of nutrients to potentially support the growth of *E. sakazakii*, appropriate temperature control of reconstituted formula is critical to inhibiting multiplication and minimizing the risk of illness. It is not known if differences in infant formula composition affect the rate of growth at various storage temperatures.

We undertook a study to determine survival and growth characteristics of *E. sakazakii*, inoculated at populations of 0.02 and 0.53 CFU/ml (ca. 13 CFU/log and 40% CFU/100 g of powdered formula, respectively), as affected by composition of six powdered infant formulas reconstituted with water. Reconstituted formulas were stored at 4, 12, 21, and 30°C and populations were monitored up to 72 h. *E. sakazakii* did not grow in formulas stored at 4°C, although it was detected by enrichment of all formulas 72 h after reconstitution. Initially at a population of 0.02 CFU/ml, *E. sakazakii* grew to populations ≥ 1 log CFU/ml of reconstituted formulas held at 12, 21, and 30°C for 48, 12, and 8 h, respectively. At an initial population of 0.53 CFU/ml, the pathogen grew to populations ≥ 1 log CFU/ml in reconstituted infant formula held at 12 and 21°C for 24 and 8 h, respectively, and to populations > 3 log CFU/ml when held at 30°C for 8 h. Populations initially at 0.02 and 0.53 CFU/ml of reconstituted formula increased to ≤ 0.25 and 0.40 log CFU/ml, respectively, when formulas were held at 30°C for 4 h. Growth was not greatly influenced by the composition of formulas. Results of our study support the U.S. Food and Drug Administration recommendation that the hang time for reconstituted infant formula in neonatal intensive care units should be no longer than 4 h. Portions of reconstituted infant formula not fed to infants should be stored at $\leq 4^\circ\text{C}$, a temperature at which *E. sakazakii* will not grow.

SURVIVAL OF *ENTEROBACTER SAKAZAKII* IN INFANT CEREAL AS AFFECTED BY COMPOSITION, WATER ACTIVITY, AND TEMPERATURE

(L.-C. Lin, and L. R. Beuchat)

Outbreaks of infections have implicated powdered milk substitute infant formulas as vehicles of *E. sakazakii*. Studies have shown that *E. sakazakii* is more thermotolerant than some Enterobacteriaceae in milk products, e.g., in infant milk formula; however, standard pasteurization practices are thought to be effective for inactivation of the bacterium. Post-pasteurization contamination of powdered infant formulas before packaging may occur in some commercial operations, as evidenced by its isolation from previously unopened cans of formula. *E. sakazakii* has been isolated from various dry foods, dry food processing plants, and the environment. The pathogen has been detected in rice seed, rice starch and flour, brown rice, and dry infant cereals. Survival characteristics of *E. sakazakii* in infant cereals as affected by the type of grain component, a_w , and storage temperatures under which they are held during distribution or in hospital, day-care center, and home settings have not been reported. We did a study to determine the survival characteristics of *E. sakazakii* initially at populations of 0.31 and 5.03 log CFU/g of infant rice cereal (a_w 0.30, 0.45 - 0.46, and 0.68 - 0.69). Cereal was stored at 4, 21, and 30°C and populations were monitored for up to 12 months. Survival of the pathogen in infant rice, barley, oatmeal, and mixed grain cereals (a_w 0.63 - 0.66, 0.76, or 0.82 - 0.83) initially containing a population of 4.93 - 5.64 log CFU/g and held at 4, 21, and 30°C up to 24 weeks was determined. Populations decreased significantly ($p \leq 0.05$) in all cereals stored at 21 and 30°C, regardless of a_w . Increases in a_w or storage temperature accelerated the rate of death of *E. sakazakii* in dry infant cereals. However, at an initial population of 0.31 log CFU/g, *E. sakazakii* survived in rice cereal (a_w 0.30 - 0.69) for up to 12 months at all storage temperatures. Survival of *E. sakazakii* was not affected by the composition of dry infant rice, barley, mixed grain, and oatmeal cereals (initial a_w 0.63 - 0.83) stored for up to 24 weeks at 4, 21, or 30°C. This study reveals that *E. sakazakii* can survive for up to 12 months in infant cereals

having a wide range of a_w when storage is at temperatures simulating those to which they may be exposed during distribution, at retail, and in the home.

SURVIVAL AND GROWTH OF *ENTEROBACTER SAKAZAKII* IN INFANT CEREAL AS AFFECTED BY COMPOSITION, RECONSTITUTION LIQUID, AND STORAGE TEMPERATURE

(L.-C. Lin and L. R. Beuchat)

During the period of 1958 - 2005, documented *Enterobacter sakazakii* infections were associated largely with neonates and infants less than 2 months of age. Infections in children as old as 4 years and in adults also have been reported. *E. sakazakii* has been shown to have a high tolerance to desiccation. It has been isolated from various dry foods, dry food processing plants, and the environment. The pathogen has been detected in rice starch and flour, dried infant cereals, dried infant foods, and environmental samples from 8 of 9 food factories, including a cereal factory. Studies have shown that some bacterial pathogens can survive and grow in reconstituted infant cereal. However, little is known about the behavior of *E. sakazakii* in infant cereals as affected by composition of cereal, composition of reconstitution liquid, and temperature at which they may be prepared and stored in hospital, day-care center, and home settings. We did a study to determine the survival and growth characteristics of *E. sakazakii* in infant cereals reconstituted with various liquids as influenced by storage temperature. Survival and growth characteristics of *E. sakazakii* initially at populations of 0.005 and 0.52 CFU/ml of infant rice cereal, oatmeal cereal, or rice with mixed fruit cereal reconstituted with water, milk, or apple juice were determined. Reconstituted cereals were stored at 4, 12, 21, and 30°C and populations were monitored for up to 72 h. Growth did not occur in reconstituted cereals stored at 4°C or in cereals reconstituted with apple juice and stored at 12°C. Populations (≥ 1 CFU/ml) were detected in cereals reconstituted with water or milk and stored at 12, 21, and 30°C for 24, 8, and 4 h, respectively. The composition of infant cereals did not markedly affect the survival or growth of *E. sakazakii* in reconstituted cereals. Populations of *E. sakazakii* in reconstituted cereal decreased with increases in populations of mesophilic aerobic microflora up to 8 - 9 log CFU/ml, which was concurrent with decreases in pH. *E. sakazakii*, initially at 2.62 log CFU/ml of rice cereal reconstituted with apple juice (pH 4.32), survived at 4°C for at least 14 days. The pathogen was not detected (< 1 CFU/10 ml) in cereal stored at 21°C for 5 days or 30°C for 4 days. Initially at 7.32 log CFU/ml, *E. sakazakii* was detected in rice cereal stored at 4°C for 50 days. It is recommended that reconstituted infant cereals stored at 21°C or 30°C be discarded within 4 h after preparation or stored at $\leq 4^\circ\text{C}$, temperatures at which it will not grow.

ATTACHMENT AND BIOFILM FORMATION BY *ENTEROBACTER SAKAZAKII* ON STAINLESS STEEL AND ENTERAL FEEDING TUBES

(H. Kim, J.-H. Ryu, and L. R. Beuchat)

Enterobacter sakazakii is a foodborne pathogen capable of causing meningitis, sepsis, bacteremia, and necrotizing enterocolitis in preterm neonates and immunocompromised adults. Powdered infant formula and milk powder have been implicated as vehicles in outbreaks of *E. sakazakii* infections. However, the pathogen also has been isolated from various clinical sources, food processing plants, the environment, lettuce, alfalfa sprouts, tomatoes, and other vegetables, cheese, minced beef, and sausage. Its presence in fresh produce raises the possibility of this food group serving as a vehicle of the pathogen for infections in immunocompromised adults, particularly patients in hospitals and elderly adult assisted-care facilities. *E. sakazakii* has been reported to be able to attach to and form biofilms on silicon, latex, polycarbonate, stainless steel, glass, and polyvinyl chloride. Foods such as powdered infant formula and fresh produce represent potential vehicles of *E. sakazakii* infections in infants and immunocompromised adults, respectively. Contact of these and other foods containing the pathogen with abiotic or biotic surfaces could result in attachment and biofilm formation. Removal or inactivation of pathogens on inert surfaces in infant formula preparation areas and produce processing environments by washing with water or treating with disinfectants or sanitizers is not always achieved, possibly because cells are enmeshed in biofilms or otherwise protected against exposure to antimicrobials. Attachment and biofilm formation by *E. sakazakii* as affected by temperature and nutrient availability have been given only meager research attention.

We did a study to determine the effects of temperature and nutrient availability on attachment and biofilm formation by *E. sakazakii* on stainless steel and enteral feeding tubes. Five strains grown to stationary phase in tryptic soy broth (TSB), infant formula broth (IFB), and lettuce juice broth (LJB) at 12°C and 25°C were examined for the extent to which they attach to these materials. Higher populations attached at 25°C than at 12°C. Stainless steel coupons and enteral feeding tubes were immersed for 24 h at 4°C in phosphate-buffered saline suspensions (7

log CFU/ml) to facilitate attachment of 5.33 - 5.51 and 5.03 - 5.12 log CFU/cm², respectively, before immersing in TSB, IFB, or LJB and incubating at 12°C or 25°C for up to 10 days. Biofilms were not produced at 12°C. The number of cells of test strains increased by 1.42 - 1.67 log CFU/cm² and 1.16 - 1.31 log CFU/cm² in biofilms formed on stainless steel and feeding tubes, respectively, immersed in IFB at 25°C; biofilms were not formed on TSB and LJB at 25°C, indicating that nutrient availability plays a major role in processes leading to the accumulation of biometrics on the surfaces of these inert materials. These observations emphasize the importance of temperature control in reconstituted infant formula preparation and storage areas in preventing attachment and biofilm formation by *E. sakazakii*.

INACTIVATION OF ZOOBOTIC PATHOGENS DURING STATIC COMPOSTING OF CHICKEN LITTER AND PEANUT HULLS

(M.C. Erickson, C. Smith, J. Liao, G.E. Boyhan, M.P. Doyle, L. Ma, and X. Jiang)

During aerobic composting, the primary factor responsible for inactivation of fecal pathogens is heat generated from the metabolic activity of thermophilic microorganisms. Moreover, to ensure inactivation of pathogens at the surface of static compost piles, it is recommended that compost be turned periodically during the first weeks of composting. This safeguard practice, however, is not often implemented in situations where labor and resources are limited. To develop alternative management strategies for these situations, baseline data is needed to determine inactivation profiles of zoonotic pathogens at surface and interior sites of static piles. The fate of zoonotic pathogens [gfp-labeled *Escherichia coli* O157:H7 (Shiga toxin-negative) and *Listeria innocua* and rifampicin-resistant *Salmonella* Typhimurium (vaccine strain)] in the field was monitored at both interior and surface sites of static composting piles composed of chicken litter and peanut hulls. Zoonotic pathogen populations declined by 4-8 log CFU/g within 4 days of composting but were still detectable by enrichment culture. Despite exposures to elevated temperatures, *Salmonella* continued to be detected in interior samples by enrichment for up to 14 days after composting was initiated. In surface samples, the fate of pathogens was dependent on the season and ambient temperature conditions in which composting was conducted. During the summer, *S. Typhimurium*, *E. coli* O157:H7 and *L. innocua* were detected by enrichment only in 3-day, 3-day, and 7-day compost surface samples, respectively. In contrast, 28, 56, and 56 days of composting in the late fall/early winter were required to reduce *S. Typhimurium*, *E. coli* O157:H7, and *L. innocua* populations, respectively, to levels detectable only by enrichment. In conclusion, zoonotic pathogens survived on the surface of unturned static composting piles containing chicken litter for up to 2 months.

INACTIVATION OF *ESCHERICHIA COLI* O157:H7 AND *LISTERIA MONOCYTOGENES* IN COW MANURE COMPOSTING SYSTEMS

(M.C. Erickson, J. Liao, L. Ma, M.P. Doyle, and X. Jiang)

Aerobic composting may be applied to manure whereby microbial metabolite degradation of organic matter generates heat for inactivation of pathogens. When equipment and manpower are not available to turn the compost mass and expose all the material to sufficient levels of heat, other management guidelines are needed to assure that pathogen inactivation of surface compost has been achieved. Towards that end, research has been addressing the potential for the initial carbon:nitrogen (C:N) ratio of the compost mixture to affect pathogen inactivation. Using a cow manure, straw, and cottonseed mixture in a laboratory-scale bioreactor, C:N ratio did not significantly affect the time to inactivation of *Listeria monocytogenes*. In contrast, *Escherichia coli* O157:H7 survived for significantly longer periods of time in 40:1 C:N systems than in 30:1 or 20:1 systems despite the fact that the cumulative heat exposure of the former system was much greater than the exposure encountered in the two latter systems. In addition, an escalation in pH to values between 8 and 9 occurred initially for 40:1 C:N systems whereas 20:1 and 30:1 systems experienced an initial decline in pH to values between 5.5 and 6 before climbing to alkaline values (8-9) after 2 days of composting. It is hypothesized that organic acids generated in the acidic stage of 20:1 and 30:1 systems may act in concert with heat to inactivate *E. coli* O157:H7. Such situations may be beneficial to the inactivation of pathogens on the surface of compost piles where temperatures are found to increase only slightly above ambient.

PRESENCE OF *NEOSPORA CANINUM* SPECIFIC ANTIBODIES ISOLATED FROM DAIRY FARMS IN GEORGIA AND TEXAS

(Y.R. Ortega, M.P. Torres, and K.D. Mena)

Bovine neosporosis is a parasitic disease produced by *Neospora caninum* which induces abortion in cows, and consequently has a negative impact on the herd's reproductive efficiency. This study demonstrated the presence of specific IgG to *Neospora* in milk and serum samples obtained from three dairy farms in Georgia and two in Texas. Using a western blot assay, samples from four hundred fourteen dairy cows were examined of which 362 were milk and 87 were serum. Samples with antibodies to *Neospora* were identified in 32.1% (105/327) of the examined animals in Georgia, whereas in Texas it was identified in 10.3% (9/87). Positive Georgia samples were found in 24% from farm A (28/115), 21.6% from farm B (30/139), and 64.4% from farm C (47/73). In Texas, 13.5% (7/52) of animals in farm D and 5.71% (2/35) from farm E also had specific antibodies to *Neospora*. The number of animals from Georgia dairy farms with antibodies to *Neospora* was significantly higher than the Texas dairy farms. This may be related to the age of the animals examined in this study (more than 2 years old). Antibodies present in sera had excellent agreement with the antibodies present in milk. Collection of milk samples for serological testing is easier and less invasive than obtaining bovine sera, therefore offering an alternative for testing of animals.

**DETECTION OF *CYCLOSPORA* IN VEGETABLES
AND POTENTIAL CONTAMINATION SOURCES IN ENDEMIC AREAS**

(Y.R. Ortega, A. Robertson, V.A. Cama, A. Mann, L. Cabrera, C. Taquiri, L. Xiao, and R.H. Gilman)

Cyclospora cayetanensis causes gastrointestinal illness and it is thought that people get infected via the food- or water-borne routes. In this study, we investigated the presence of *Cyclospora* in vegetables and hands of produce vendors in Pampas de San Juan in Lima Peru, a community where *Cyclospora* is endemic. We also surveyed other potential contamination factors, such as river irrigation water and soil from three agricultural fields. Vegetables and hand washes were collected from 20 vendors from 4 markets at 3 survey dates in 2004. All samples were analyzed by microscopy and PCR-RFLP for the presence of *Cyclospora*. It was identified in vegetables in the March and June surveys, but not in December. Hand washes were positive only in the March survey, one with *Cyclospora* and two with *Eimeria* spp. Irrigation water with *Cyclospora* was identified in March in 2 of 3 rivers, and 2 of 3 soil samples and, 1 of 3 rivers in August. Our findings show a seasonal trend in the detection of *Cyclospora*, which mimics the presence of cyclosporiasis in the community. Detecting *Cyclospora* in vegetables, hands of vendors, irrigation water, and agricultural soil demonstrate the food and water-borne potential of this parasite and give new information to understand the dynamics of produce contamination.

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