Linking Illness to Food:  
Summary of a Workshop on Food Attribution

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Abstract:

To identify and prioritize effective food safety interventions, it is critical not only to identify the pathogens responsible for illness, but also to attribute cases of foodborne disease to the specific food vehicle responsible. A wide variety of such “food attribution” approaches and data are used around the world, including the analysis of and extrapolation from outbreak and other surveillance data, case-control studies, microbial subtyping and source-tracking methods, and expert judgment, among others. The Food Safety Research Consortium sponsored the Food Attribution Data Workshop in October 2003 to discuss the virtues and limitations of these approaches and to identify future options for the collection of food attribution data in the United States. This discussion paper summarizes workshop discussions and identifies challenges that affect progress in this critical component of a risk-based approach to improving food safety.

Key Words: foodborne illness, food attribution, outbreaks, case-control studies, microbial fingerprinting, microbial subtyping, FoodNet
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Introduction

The Food Safety Research Consortium (FSRC) sponsored the Food Attribution Data Workshop on October 31, 2003, in Atlanta, Georgia, to discuss the virtues and limitations of the many methods used worldwide to attribute cases of foodborne disease to causal food vehicles.

Food safety is a difficult and dynamic issue. With changing demographics and eating patterns, microbiological hazards have come to the fore as an important food safety challenge, responsible for as many as 76 million foodborne illnesses each year (Mead et al. 1999). The federal food safety agencies, the National Academy of Sciences, and other expert bodies share the goal of a science- and risk-based food safety system (IOM 1998, 2003; GAO 2001). Such a system requires that risk managers prioritize food safety hazards and preventive interventions using the best available data on the distribution of risk and on how risk can be reduced most effectively and efficiently. This includes understanding the many factors that can cause or prevent foodborne illness,

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from the point of production to the point of consumption, and systematically targeting efforts—including research, regulation, education, and private initiatives—in ways that contribute most effectively to risk reduction.

The FoodNet active surveillance system administered by the Centers for Disease Control and Prevention (CDC) is producing increasingly robust, quantitative data on the incidence of illness due to specific enteric pathogens. However, there is no such active surveillance system to categorize these illnesses by pathogen source—whether waterborne, environmental, or foodborne—or to identify and distinguish specific food vehicles. To design and prioritize interventions, it is essential that we know which foods are responsible for specific illnesses (“food attribution”) and how these foods contribute to the total disease burden associated with foodborne pathogens.

This reflects the reality that interventions (and regulations) are almost always food specific (or process specific) and involve, for example, procedures to limit *Escherichia coli* O157:H7 in ground beef or *Campylobacter* in broiler chickens. Although we may have increasingly accurate estimates of the total number of patients with symptomatic *E. coli* O157:H7 disease, development of a rational system of interventions requires that these estimates be matched with comparably accurate data defining the source from which humans are acquiring these pathogens. Such food attribution data are of particular importance for U.S. government agencies that regulate food and food animals, including the Food Safety Inspection Service (FSIS) of the U.S. Department of Agriculture (USDA), the Center for Food Safety and Applied Nutrition (CFSAN), and the Center for Veterinary Medicine (CVM) of the Food and Drug Administration (FDA).

Foodborne illnesses can be attributed to various foods using a variety of data sources and analytic approaches. It is important to understand the differences and limitations of these approaches and to recognize that different approaches may best answer different questions about foodborne illness. Does the question pertain to a single pathogen or a single food, or is the question about the range of foodborne pathogens? Should we attribute illnesses to broad categories of foods (e.g., “pork”) or is
it necessary to identify specific food items responsible (e.g., “ready-to-eat pork frankfurters”)? Do we seek to attribute illnesses to the infected foods when they are consumed (“point-of-consumption attribution”) or to the on-farm origin or reservoir of infection (“reservoir attribution”) before processing, distribution, preparation, and possible cross-contamination? Are we interested in identifying illnesses that are not directly due to food consumption but may be associated with food production, such as transmission from live farm animals to humans or transmission through drinking water contaminated by farm animal waste? Do we wish to use collected data to track small changes from year to year, or are we primarily concerned with attribution over a longer time frame? Is our purpose to identify broad policy needs, or do we seek to use collected data to evaluate the success of specific interventions after they are implemented?

The Food Attribution Data Workshop was organized to explore these questions and the many approaches of food attribution in detail. Attendees included representatives from CDC, FSIS, CFSAN, CVM, the Environmental Protection Agency (EPA), consumer advocacy organizations, and member institutions of FSRC, including the University of Maryland at Baltimore, University of Georgia, Iowa State University, University of California at Davis, and Resources for the Future. The presenters and attendees who constitute the Food Attribution Working Group are listed in Appendix A, while the workshop agenda is attached as Appendix B.

**Food Categorization**

Before illnesses can be attributed to specific food risks, it must be decided how to categorize these pathways. Seafood, for instance, could be one large category, or it could be subdivided in hundreds of species of animals. To consider categories that fit with common marketing and consumer expectation and yet are specific enough to represent distinct industries, it may be useful to categorize seafood into three groups of animals: fish, mollusks, and crustaceans. Using similar logic, a list of major food
commodities might be structured to comprise 12 categories in all: poultry, pork, beef, meat from other farm mammals (such as lamb and mutton), eggs, dairy, fish, mollusks, crustaceans, wild game, row crops (such as lettuce and corn), and tree crops (such as apples and oranges). Each of these commodities could be divided further, leading to such subcategories as broiler chickens and raw oysters.

Developing a food categorization scheme is not straightforward, however, since foods might be classified in many additional ways, including level of processing (raw, fresh-cut, canned, frozen), origin (domestic, imported), and location of preparation (home, food processor, food service). These classifications become important for identifying possible intervention strategies. For interventions close to consumption, it may be useful to distinguish between preparation behaviors (raw eggs versus cooked eggs), whereas farm-level interventions may need to distinguish only among animal sources.

Furthermore, illnesses are often linked to foods with multiple ingredients—such as soups, salads, and casseroles—in which it is impossible to identify the specific ingredient responsible. In such situations, one option might be to restrict analysis to single-food vehicles (e.g., ground beef but not meatloaf); another option might be to categorize foods by their “essential” ingredients (e.g., egg salad would be categorized as egg). One could also partition such foods into all of their ingredients and attempt to model the attribution from the frequency of ingredients found in implicated foods. To maximize information, it is best to include as many foods as possible. To retain accuracy, however, it is best to limit analysis to the contaminated ingredient.

Development of a common food categorization scheme is essential if different sources of data are to be combined or compared. Since different approaches may obviously yield different results, it would be useful to compare, for example, the results of risk assessments with outbreak data. A barrier to making such comparisons is likely to be incompatible food categorization schemes used between studies. Because of this lack of agreement in categorization, data from CDC, state health departments, and FDA
and USDA and their constituent agencies are often not directly comparable. It may not be possible to reconcile all food categories across all approaches, but without standardized food categories, comparison between approaches may be difficult, if not impossible. As a necessary first step in any approach to food attribution, food categories need to be standardized across government agencies, using a scheme acceptable to industry, academia, and consumer groups.

**Current Approaches to Food Attribution**

There are numerous approaches and data sources currently utilized by researchers and regulators worldwide to attribute foodborne illnesses or risks of foodborne illness to specific pathogens in specific foods. These approaches to food attribution can generally be grouped into two broad categories, loosely designated as “epidemiological” and “microbiological.” Epidemiological information, whether from data series of reported foodborne outbreaks or from case-control studies of sporadic cases, focuses on the final foods as consumed and may serve to link a broad array of pathogens and foods (as in the outbreak series) or a single pathogen with a limited array of foods. Microbiological information includes data on microbes collected from humans and from animals and foods at various stages in the food production process. Microbial fingerprinting techniques, which use markers to group similar pathogen subtypes, can be used to compare microbes from different sources and to link pathogen sources to contaminated foods or to specific cases of illness.

These methods provide focused information about single pathogens and about the range of reservoirs or foods that are included in comparative samples. In Denmark, the Salmonella Account uses this approach successfully to attribute cases of human salmonellosis to several animal reservoirs and thus to target and evaluate reservoir-specific control measures on a year-to-year basis. In addition, risk assessments of foodborne illness utilize predictive microbiological models, along with microbiological data on pathogen prevalence in sampled foods, to estimate illnesses due
to specific pathogen-food pathways. These and other approaches for food attribution point to possible future directions of and improvements to the food safety system of the United States.

**Danish Experiences**

Denmark is regarded as an international leader in food safety, with responsibilities unified in a formal network of agencies and an extensive science-based program. In Denmark’s integrated system, all data from public health surveillance and from pathogen monitoring on foods and animals are routinely collected, collated, analyzed, and reported by a single coordinating body, the Danish Zoonosis Center. Cultures collected from infected people, animals, and retail food sources are subtyped, allowing for the direct comparison of surveillance and monitoring data and the identification of public health outcomes by food source.

Denmark has three sources of foodborne illness surveillance data: notifications by doctors and hospitals for all suspected foodborne infections, reports by clinical microbiology laboratories of identified gastrointestinal pathogens, and individual accounts and outbreak investigations of persons who report food poisoning to the authorities (MFAF 2002). Because health care is free in Denmark, there is no financial barrier preventing consumers from seeking medical attention, and reporting of illnesses is therefore generally high.

The regular monitoring of food sources is performed in Denmark along the farm-to-fork pathway—on farms, at slaughter, and on retail foods—though the emphasis is on primary production facilities and on *Salmonella* (MFAF 2002; Wegener et al. 2003). Every flock of egg-laying chickens (layers) is regularly tested for *Salmonella* by a combination of serological and bacteriological methods. Additional testing is performed in flocks testing positive for verification of infection. All flocks of broiler chickens, turkeys, and ducks are tested by bacteriological examination approximately three weeks prior to slaughter. Finishing-pig herds producing more than 200 finishers—pigs
ages 12 to 26 weeks—per year are continuously tested by serology, and herds exceeding a predetermined proportion of seroreactors receive a follow-up bacteriologic examination. Dairy herds are monitored serologically and categorized according to levels of antibodies. Batches of broiler chickens and turkeys, and carcasses of pork and beef are examined bacteriologically after slaughter. Imported products of poultry, pork, and beef are also monitored. Isolates from wild animals, birds, and pets are also monitored and subtyped (MFAF 2002). At the retail level, surveys are performed: raw meat, pork, and poultry are monitored, as are fruits, vegetables, and shell eggs.

The critical linkage between public health surveillance data and animal and food monitoring data is achieved through the extensive use of subtyping of isolated pathogens. Microbial subtyping, or microbial fingerprinting, is an umbrella term for the numerous methods used to distinguish bacterial and viral isolates from one another. The subtyping of foodborne pathogens is useful for two reasons. First, it can aid epidemiological investigations of outbreaks by identifying and tracking bacterial isolates, grouping illnesses by isolate, and thus positively identifying the food responsible. Second, certain pathogen subtypes can be associated with particular foods or animal sources, thus enabling illnesses from those subtypes to be similarly associated.

Denmark, the United Kingdom, and the United States all use microbial subtyping to aid with outbreak investigations, but only Denmark subtypes with enough regularity in humans, animals, and food to attribute sporadic pathogen illnesses to specific food and animal sources. The subtyping methods used in Denmark include serotyping, phage typing, and pulsed-field gel electrophoresis.

When *Salmonella* sero- and phage types isolated from animals and food with isolates from humans are compared in a quantitative manner, the attribution (impact) of the major animal reservoirs to human disease incidence can be assessed indirectly. A prerequisite of the model is predominance of at least one *Salmonella* subtype in each of the main reservoirs. It is assumed that all human infections with these “distinctive”
types originate only from that particular source. Human infections caused by *Salmonella* types found in multiple reservoirs are then distributed proportionally to the occurrence of the distinctive types. The application of this rather simple model for more than a decade has allowed for the evaluation of trends and dynamics in the major sources of human salmonellosis and provided decision-support for risk managers by documenting either the need for or the effect of surveillance programs. During this time, dramatic changes in the prevalence of specific subtypes associated with major food sources were reflected in similar changes in the number of human cases with these types.

The principle of *Salmonella* case attribution has generated annual estimates for the impact of major animal-food sources in Denmark from 1988 to 2002 (MFAF 2002, 2003). During this period, several independent observations have supported the outcome of the case attribution model. These include results from case-control studies, outbreak reports, time-series analysis, and risk assessments (Mølbak 2003; Mølbak and Neimann 2002). The observation that changes in prevalence in both animals and food are reflected in similar changes in (the nonprevalence-based) food attribution estimates strengthens confidence in the method.

It should be noted that although Denmark’s method of attributing cases of salmonellosis to animal sources provides an excellent general assessment of reservoirs of infection, it does not necessarily identify the causal vehicles implicated in individual cases of illness. The method relies on matching subtyped isolates from illnesses to monitored animal sources, but many such subtypes are not unique to a particular animal, and illnesses are therefore attributed proportionally. Other sources capable of causing human illness (e.g., vegetables, fish, pets, water) are not represented directly by the extensive subtyping of isolates. Some human cases associated with these sources may be allotted to the “unknown” category of the assessment, whereas others may be referred to one of the major sources. In some cases, this approach may be acceptable, since contamination at some point in time originates from an animal reservoir.
Danish food attribution modeling is currently focused solely on *Salmonella*. Though extensive subtyping also is performed on *Campylobacter* isolates from a variety of sources, the homogeneous distribution of subtypes across reservoirs does not allow similar food attribution modeling. The application of the Danish food attribution method is limited to those pathogens that fulfill the prerequisites of the model (i.e., heterogeneous distribution of subtypes across reservoirs, the availability of representative distributions, and if need be, the ability to discard outbreak- and travel-associated cases). Consequently, it cannot be used in its present form for a great number of foodborne pathogens.

If the purpose of a food attribution assessment is to identify various critical control points along the farm-to-fork continuum, and to simulate the effect of control strategies at these points, the Danish model does not suffice. Nor does the model suffice to identify responsible foods at the point of consumption. Nonetheless, if the purpose of performing food attribution is to identify reservoirs of infection in animal populations, the Danish approach is appropriate, comprehensive, and scientifically rigorous.

**United Kingdom Experiences**

The United Kingdom has long been an active leader in food safety, a role furthered in 2000 with the creation of the Food Standards Agency (FSA). As one of only a handful of countries to have consolidated responsibilities into a single government office, the United Kingdom utilizes an integrated systems approach to food safety that includes both epidemiological and microbiological methods.

In recent years, the U.K. public health and food safety agencies, headed by the Department for Environment, Food, and Rural Affairs, have produced annual reports on zoonoses (MAFF 2000, 2001; DEFRA 2002, 2003). These integrated reports combine the reporting and interpretation of public health surveillance data with monitoring for pathogens in live animals, carcasses at slaughter, and retail foods.
The United Kingdom has three surveillance systems in place for foodborne illness: statutory reporting by doctors of suspected cases of food poisoning, national surveillance of laboratory-confirmed infections, and national surveillance of general outbreaks of infectious intestinal disease (Wall et al. 1996). Outbreak surveillance data have been used in etiological analyses of such outbreaks associated with specific foods, such as fish and shellfish, poultry, and red meat (Gillespie et al. 2001; Kessel et al. 2001; Smerdon et al. 2001). These analyses detail illnesses by pathogen and food source, seasonality, severity of illness, and additional factors, such as where the food was prepared (home, catering service, restaurant).

U.K. agencies also perform regular monitoring of animals and retail food, though the program is not as comprehensive or consistent as that of Denmark. Salmonella is given the most attention, with regular testing of poultry in breeder, boiler, and laying hen flocks. Reported incidences from cattle, sheep, and pigs are also collected for Salmonella, and the pathogen strains from animal infections are subtyped and analyzed (DEFRA 2003). Shellfish are also monitored quite heavily (FSA 2003). In addition to such monitoring, FSA performs prevalence studies on various animals and food products to determine contamination levels. Two such studies on animals include a two-year survey for E. coli O157:H7 in cattle, sheep, and pigs and a survey of cows’ milk for Mycobacterium bovis (DEFRA 2002). Studies are also performed on retail food products, including fresh and frozen whole and portioned chicken (DEFRA 2003); salad vegetables (DEFRA 2003); halal butchery products (Little et al. 1999); and various ready-to-eat products, such as hamburgers, sliced deli meats, and quiche (Little et al. 2001; Gillespie et al. 2000, 2001). These monitoring data are useful for isolating and quantifying known risks, for identifying emerging risks, and for discerning trends over time. They are not enough, however, to adequately attribute illnesses to foods. Unlike Denmark, the United Kingdom does not regularly subtype isolates taken from infected humans, so the critical link between illnesses and foods is unknown.
The food safety agencies of the United Kingdom have also performed some risk assessments, including those for *Listeria*, *E. coli* O157:H7, fluoroquinolone-resistant *Campylobacter*, and *Norovirus*, though these studies have not been used explicitly for food attribution (O’Brien 2003).

**U.S. Outbreak Data**

Of the numerous foodborne illness surveillance systems in place at CDC, of particular interest for food attribution are data on outbreaks. CDC conducts ongoing surveillance for the entire United States and territories, and foodborne outbreaks are investigated by every health department. For many outbreaks, the likely food vehicle responsible for infection has been identified through epidemiological investigations and, when possible, verified through laboratory testing. As such, the outbreak data constitute the only national surveillance system that explicitly links cases of foodborne illness with food vehicles and therefore the only source of information readily available for point-of-consumption food attribution.

Outbreak data are observed at the public health endpoint and are therefore a direct measure of attribution. For many pathogens, outbreak data provide the only conclusive indication of which foods cause specific cases of illness. Outbreaks have been caused, to varying degrees, by nearly all important foodborne pathogens, including some that remain undiagnosed. In addition, outbreaks have implicated a wide range of food vehicles, including those not originally expected to be the source of illness (e.g., *E. coli* O157 in sprouts, *Salmonella* Newport in tomatoes, *Cyclospora* in raspberries). When collected routinely and consistently, outbreak data can be systematically analyzed for temporal and geographic changes.

These data are limited, however, in that outbreaks generally reflect unusual occurrences and breakdowns of standard control practices, as well as standard transmission patterns, and therefore may misrepresent sporadic incidence. Additionally, some pathogens have sparse coverage, and inference from too few cases
may be misleading. Responsibility for investigating foodborne disease outbreaks resides with local and state health departments, which then report these data to CDC. Because of differences in local capabilities and reporting, regional and temporal coverage is uneven and could therefore provide a biased estimate of the proportion of outbreaks due to specific food vehicles. The magnitude of this bias could be assessed with appropriate epidemiologic analyses.

CDC has estimated food attribution from outbreak data (Bean et al. 1996) and is currently working toward a more comprehensive estimate. Challenges faced in completing the estimate include the need to merge data from three outbreak reporting systems. The foodborne outbreak reporting system from 1973 to 1997 was limited to 38 food vehicles. A revised system, beginning in 1998, included more than 1,300 vehicles. In 2002, Internet reporting was introduced to reduce delays caused by entering paper-based reports, but resulted in a different coding and database structure.

Attempts to model attribution from outbreak data need to consider several issues. One of the central problems with using outbreak data to estimate the attribution of foodborne illness is that the number of outbreaks due to a given etiology is typically not proportional to the percentage of sporadic illnesses caused by the same etiology. A dramatic example comes from the disproportional number of outbreaks due to ciguatera poisoning (typically caused by consumption of contaminated sport-caught reef fish) that are reported to CDC. There are almost as many reported ciguatera outbreaks as shiga toxin–producing *E. coli* (STEC) outbreaks, but there may be 100 cases of STEC in the United States for every case of ciguatera. Even among common bacterial causes of foodborne illness, outbreak reports are not proportional to the estimated number of illnesses per year. It is estimated that STEC and *Shigella* spp. each cause approximately 90,000 foodborne illnesses per year, and yet twice as many reported foodborne outbreaks are due to STEC compared with *Shigella* spp. As a result, a ranking of the food vehicles associated with foodborne outbreaks is likely to overrepresent foods associated with commonly reported pathogens.
One method to adjust for the unrepresentative nature of outbreaks would be to stratify the data by etiology and weight the strata by the incidence of that etiology (i.e., the burden of illness). As an example, outbreaks of ciguatera poisonings would first be used to estimate the attribution for ciguatera poisoning, presumably 100% finfish. Then, the result would be weighted by the contribution of ciguatera to all foodborne illness. Assuming that finfish were not involved in other causes of foodborne illnesses, the overall attribution due to finfish would be small, even though it is large for ciguatera, because ciguatera illnesses are a small proportion of foodborne illness. This approach is analogous to other study designs that control for confounding factors; in this case it is necessary to control for etiology.

To create an unbiased estimate of the vehicles of foodborne illness, a reliable model for foodborne attribution could be constructed using outbreaks to define the proportion of illness attributable to a food commodity for each etiology. In practice, this means stratifying outbreak data by etiology and then creating a weighted average, weighting by the estimated burden of foodborne illness caused by each pathogen. The output could be displayed in a pie chart to represent the proportion of foodborne illness caused by each food commodity. Other weights could be employed, such as the number of hospitalizations or deaths for each etiology. Using this model, one could estimate not only the foods responsible for foodborne illness but also the foods responsible for severe illness.

Another issue with analyzing outbreak data is whether to count outbreaks or individual cases of illness to determine the attribution of each food commodity. An advantage to using the number of ill people is that vehicles implicated in large outbreaks may be commonly consumed and therefore a more frequent cause of illness than a vehicle that causes only a few cases. A disadvantage to this approach is that one large outbreak may be due to a vehicle that rarely causes sporadic illness but just happened to cause a very large outbreak. For example, a single *Salmonella* Typhimurium outbreak due to milk that sickened 17,000 people (or 90% of all outbreak
cases) would skew the analysis. One remedy would be to reduce the influence of extreme numbers through a statistical procedure. For etiologies that rarely cause outbreaks, detecting more outbreaks would be critical. Approximately a dozen *Campylobacter* outbreaks are reported each year, and about half have been due to unpasteurized milk, but this vehicle is probably much less important in sporadic cases. Increasing the sensitivity of outbreak detection may change the spectrum of identified vehicles and perhaps improve the correlation between vehicles of outbreaks and vehicles of sporadic illness. Prior to the development of PulseNet, a molecular subtyping network used by CDC for early detection and timely investigation of outbreaks, most recognized outbreaks of listeriosis were caused by unpasteurized milk and milk products; PulseNet has since detected a series of multistate outbreaks that indicate the importance of ready-to-eat meats.

Improvements in modeling attribution will come with improved accuracy of outbreak investigations. It is important to use standardized definitions for confirming the microbial etiology of an outbreak, such as those used in the CDC outbreak surveillance system. Interpretation of outbreak investigations depends on the ability of state, local, and regulatory investigators to conduct a thorough “traceback.” It would be extremely beneficial to strengthen the capacity to conduct analytic investigations and conduct complete source tracing. Through improved coding of source of contamination (farm, factory, kitchen) and thorough investigations, outbreak data could estimate the attribution of illness resulting from contamination at each stage of food production (farm, factory, kitchen). Restricting analyses to multistate and multilocation outbreaks, for example, would remove from the data those illnesses caused by improper preparation in the kitchen, thus focusing on those illnesses due to upstream contamination.

National outbreak data have been collected and analyzed by the Center for Science in the Public Interest (CSPI). These data are based primarily on unpublished CDC data obtained through Freedom of Information Act requests but also include
additional outbreaks investigated by CSPI. Of the 2,472 outbreaks listed in the September 2002 Outbreak Alert, 300 (12.1%) were not from CDC sources (DeWaal and Barlow 2002). In this report, for each of 13 broad food categories (e.g., seafood) and 38 subcategories (e.g., finfish), outbreaks are listed by pathogen etiology, state, date, number of cases, and specific food vehicle (e.g., salmon). CSPI’s summary of these data presents the number of outbreaks and cases by food subcategory and shows, for example, that 21% (539 of 2,472) of outbreaks and 8% (6,781 of 90,355) of cases in the outbreak data are due to consumption of seafood, and that of these, 65% (351 of 539) of outbreaks and 30% (2,035 of 6,781) of cases are due to finfish. These numbers are not reported by pathogen, however.

FSRC relies on the CSPI data in the draft version of the Foodborne Illness Risk Ranking Model (FIRRM), an analytic tool that allows for the comparison of the public health burden of various pathogen-food combinations (Batz et al. 2004; FSRC 2003, 2004). After pathogens not incorporated into FIRRM were excluded, the data set contained entries for 1,977 outbreaks, representing 83,619 individual cases of foodborne illness. Coverage was not adequate across all pathogens; the data included zero outbreaks for two pathogens (Astrovirus and Brucella) and included fewer than five outbreaks for six pathogens (Cryptosporidium, Giardia lamblia, Rotavirus, Streptococcus, Toxoplasma, and Vibrio vulnificus). FSRC widened the CSPI food categorization scheme to include 47 subcategories within the same 13 major categories. The food vehicles associated with each outbreak were then reclassified into the FSRC food categorization scheme. For each pathogen, the FSRC counted the total number of illnesses, and of these, the number of illnesses due to each food category, to compute the number of illnesses due to each pathogen-food combination. From these, FSRC computed the percentage attribution of illnesses for each pathogen.

Estimates of national annual incidence of each pathogen, computed separately and based on different data, were multiplied by the food attribution percentages to obtain estimates of national annual incidence by pathogen-food combination. That is,
for some hypothetical Pathogen A, there may have been 1,000 cases of illness in the CSPI outbreak data, of which 600, or 60%, were traced to produce and the remaining 400, or 40%, were traced to chicken. If the model assumes a national annual incidence of Pathogen A to be 15,000 cases, it would then estimate that 60% of these cases, or 9,000, were due to produce and the remaining 6,000 were due to chicken. Food attribution percentages are also applied to estimates of the number of hospitalizations and deaths due to each pathogen, to obtain estimates for each pathogen-food combination. Approximations of the annual number of cases, hospitalizations, and deaths are estimated for each food category by summing pathogen-food combinations over all pathogens. Thus, FIRRM produces estimates of incidence by pathogen, by pathogen-food combination, and by food.

Although this model provides useful information, it also highlights some of the problems that arise when using outbreak data as the sole source for food attribution. As one example, in the model “produce” emerges as the most common vehicle for *Campylobacter* infections, yet community-based studies suggest that poultry is the most common source of sporadic campylobacteriosis (Friedman et al. 2004).

**FoodNet Sporadic Case-Control Studies**

The Foodborne Diseases Active Surveillance Network, or FoodNet, is an active surveillance program centered at CDC that tracks foodborne illnesses from nine pathogens in 10 well-defined target populations. It is a collaborative program with the Department of Agriculture, the Food and Drug Administration, and 10 state and local health departments. Although follow-up investigations for food sources are not included in regular FoodNet surveillance, CDC has investigated food attribution of FoodNet illnesses through numerous case-control and epidemiological studies. In these

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1 This section is drawn largely from Hardnett et al. (2004).
studies, patients reporting through FoodNet are contacted for follow-up interviews and administered questionnaires to ascertain the proportion of illnesses associated with specific foods, food preparation and handling practices, and other behavior, such as pet ownership, farm visits, or international travel. Thus far, CDC has performed case-control studies on *Salmonella* (Kimura et al. 2004; Hennessy et al. 2004; Glynn et al. 1998; Moore 2004; Mermin 2004), *E. coli* O157:H7 (Kassenborg et al. 2004a; Kennedy et al. 2002), *Campylobacter* (Friedman et al. 2004; Kassenborg et al. 2004b), *Cryptosporidium* (CDC 2003), and *Listeria monocytogenes* (Varma 2004).

FoodNet case-control studies have a particular advantage for determining food attribution of sporadic illness because they are population based; they involve the administration of follow-up surveys to all patients with laboratory-confirmed infections occurring within the active surveillance area during the study period. In population-based case-control studies, the impacts of selection bias are easier to predict because the base population (the population from which cases are drawn) is well defined. Furthermore, FoodNet case-control studies use incidence sampling design rather than cumulative incidence sampling, so direct estimates of disease rate ratios are obtained for the measured exposures. Furthermore, Hardnett et al. (2004) note,

> Because the diseases under investigation in the FoodNet population-based case-control studies are rare in all population subgroups, rate ratios closely approximate risk ratios. Along with case exposure percentages, these risk ratio estimates are used to calculate the population-attributable fraction. The population-attributable fraction is defined as the proportion of new cases occurring during a given period in a particular population at risk that was attributable to the effect of one or more exposures. In other words, the population-attributable fraction is the proportion of cases that might not have occurred during the study period if everyone in the population had been unexposed (or had been exposed at reference levels). Because FoodNet case-control studies are population-based, these studies directly estimate the relative risk and population-attributable fraction. An additional important advance in the FoodNet case-control studies is the calculation of precise confidence intervals around the population-attributable fraction. In the 6
FoodNet case-control studies included in this supplement, we computed confidence interval around the point estimates using a “jackknife” procedure to estimate variance. The jackknifing procedure estimates the variance of the estimated population-attributable fraction by using the observed data (rather than statistical assumptions) to approximate the population-attributable fraction distribution.

Case-control studies can also identify risk factors, such as diet, that may be associated with a lower risk. In one study, eating a diverse diet (i.e., consuming more than the median number of different food items during the exposure period) was found to be protective against infection with sporadic *Salmonella enteriditis* (Kimura et al. 2004). Also, in several FoodNet case-control studies, there was higher consumption of fruits and vegetables in controls than in case patients. Further research is needed to determine whether diverse diets or the consumption of fruits and vegetables are protective against infection, or whether there is another explanation.

Nonetheless, FoodNet case controls do have some shortcomings, namely recall bias, long exposure windows, and durable population immunity. Recall biases arise because there are natural limits on what patients and controls can remember and report and because of limitations of the interview format itself. In FoodNet case-control studies, the time period during which exposures are established for patients and controls tend to be long. The exposure windows were predominantly seven days in the *Campylobacter* case study and mostly five days in *Salmonella* case-control studies (Hardnett et al. 2004). Such long exposure periods are problematic when inspecting common exposures, since high exposure frequencies make it difficult to detect differences in exposures between patients and controls. A recent Danish study found, for example, that reducing the exposure window from five days to one day for a casestudy on *Salmonella enteritidis* resulted in identifying an increased risk from eating eggs, a common exposure among patients and controls (Molbak and Neimann 2002). Furthermore, if a relatively common infection conveys durable and protective immunity in the population, such as may be the case for *Campylobacter* (Scott and
Tribble 2000), a significant component of the population may not be susceptible to infection. If this occurs, it can be difficult to demonstrate an association between exposure and an increased risk of infection, since some immune controls may have high exposures to a risky food. Further research is necessary to further understand the impacts of exposure window duration and population immunity to the findings of FoodNet case-control studies.

**Microbial Subtyping and Microbial Source Tracking**

Numerous U.S. food safety and public health agencies have been involved with microbial subtyping. As discussed above in the section on Denmark, microbial subtyping is an overall approach to identify specific bacterial and viral isolates and distinguish them from one another. In Denmark, isolates taken from human, animal, and food sources are subtyped and compared; illnesses are attributed by subtype to matching animal sources. In the United States, subtyping is used primarily to aid outbreak investigations through PulseNet, though research is under way to investigate the use of subtyping for microbial source tracking (MST).

CDC’s PulseNet program, which developed concurrently with FoodNet in the 1990s, is a network of public health laboratories that subtype bacteria using DNA fingerprinting and submit the results to an electronic database (Swaminathan et al. 2001). Bacterial strains can be compared quickly in the PulseNet database, which thus provides an early warning system of emerging outbreaks when related strains emerge. The method of DNA fingerprinting used by PulseNet is pulsed-field gel electrophoresis (PFGE), and all participating labs follow standardized protocol and use the same equipment. PulseNet currently includes five bacteria: *Escherichia coli*, *Salmonella*, *Shigella*, *Listeria*, and *Campylobacter*.

PulseNet is used primarily to aid epidemiological investigations of foodborne outbreaks. It cannot be relied on for food attribution, and it does not include active surveillance of sporadic cases or isolates routinely drawn from food or animal sources.
Questions may also be raised about the relative discriminatory power of the PFGE technique used by PulseNet. For example, what does it mean if two isolates (from, say, California and Maryland) have an identical pattern, in the absence of any epidemiological linkage?

Microbial source tracking refers to a specific application of microbial subtyping, in which markers from an isolate can be used to trace that isolate back to a specific animal source. The basic idea is that even though the same pathogen might be found in different animal species, these species might be host to unique populations of subtypes of these pathogens. An isolate drawn from an infected person, for example, could be subtyped, and the results might indicate that the isolate originated in chicken as opposed to cows or some other animal. MST research, however, is in relatively early stages.

Though discussed here in regard to foodborne illness, MST methods were originally developed specifically for identifying and tracking sources of microbial pollution in natural waters—such as lakes, rivers, and streams—failing to meet regulatory standards. The U.S. Environmental Protection Agency has therefore had extensive experience with MST even though EPA is not directly involved with food safety. In February 2002, EPA held a workshop on MST methods, at which the major approaches were discussed and compared (EPA 2002).

Because microbial pollution in natural waters is generally due to human or animal waste, nearly all MST methods strive to distinguish sources by subtyping fecal bacteria. Most MST subtyping methods are based on genetic or phenotypic fingerprinting methods, though some approaches use chemical markers, biomarkers, viruses, and bacteriophages as indicators of animal source (Simpson et al. 2002). Though their titles may be cryptic to nonbiologists, some of the major approaches of subtyping include serotyping, pulsed-field gel electrophoresis, antimicrobial resistance analysis, ribotyping, species-specific genetic biomarkers, terminal restriction fragment
length polymorphisms, repetitive polymerase chain reaction, amplified fragment length polymorphism, and multilocus sequence typing.

The sheer number of microbial subtyping methods indicates both the breadth of possible approaches and the lack of one clear strategy that meets all needs. Some approaches require libraries of fingerprints, some do not; some are expensive but highly reproducible, others are cheaper but less robust. Some are better for larger-scale surveys than others. It is unclear which subtyping methods hold the most promise for MST or, more specifically, for food attribution, though data that can be compared across geographic and temporal boundaries are likely to be the most useful for large-scale attribution. The use of antimicrobial resistance analysis, for example, may not be ideal for cumulative or comparative analyses over time, since antimicrobial traits change rapidly.

Though MST has been researched extensively for microbial pollution in water, studies into its efficacy for sourcing foodborne illnesses to specific foods are less common. The Center for Veterinary Medicine (CVM) within FDA has undertaken some research on a multitude of genotypic and phenotypic methodologies to determine the food animal origins of Salmonella and Campylobacter. CVM has investigated the capabilities of serotyping, fatty acid profiling, PFGE, repetitive polymerase chain reaction, protein profiling, antimicrobial susceptibility profiling, and multilocus sequence typing to distinguish isolates from pigs, cattle, turkeys, and chickens (Singh 2003). These studies have thus far concluded that MST methods hold promise for isolating sources of foodborne illness but are only initial steps toward conclusively attributing illnesses to food animals.

The Agricultural Research Service (ARS), within USDA, has also undertaken research into microbial subtyping techniques, though not specifically for MST purposes. ARS has researched comparative genomics, ribotyping, and serotyping and has started to develop databases and libraries of the results of their microbial fingerprinting studies. These types of research into subtyping methods complement CVM research.
Risk Assessments

Risk assessments utilize food contamination data, food storage and consumption patterns, risk behavior, and dose-response functions to predict risks of illness from specific pathogens found in specific foods. Risk assessments are complex and resource-intensive and require knowledgeable modelers to estimate population or per capita risks of illness, which may differ by age, gender, or other variables. Because risk assessments are so time-consuming and complicated, they have only been undertaken for a limited number of pathogen-food combinations.

The only pathogen for which risk assessments have been performed on a reasonably comprehensive set of food vehicles is *Listeria monocytogenes*, undertaken through the combined resources of the FDA Center for Food Safety and Applied Nutrition (CFSAN), the USDA Food Safety and Inspection Service (FSIS), and CDC. In a project initiated in 1999 and completed in 2003, the agencies performed individual risk assessments on 23 ready-to-eat foods, including seafood, produce, dairy, and deli meats, to compare their relative risks (CFSAN 2003). Excluded were foods generally cooked before consumption, such as most seafood and meats, and low-risk foods for *Listeria*, such as grains, eggs, and soft drinks.

In addition, FSIS has published its own risk assessment results from this study (Gallagher 2003). FSIS has also performed two other major risk assessments, on *E. coli* O157:H7 in ground beef (FSIS 2001) and on *Salmonella enteritidis* in shell eggs and egg products (FSIS 1998). Although these are of limited benefit for food attribution without comparable risk assessments of other foods for both pathogens, they can be used to estimate the rough percentage of total *E. coli* O157:H7 foodborne illnesses due to ground beef and total *Salmonella* foodborne illnesses caused by eggs. Also of note is a
CFSAN risk assessment on *Vibrio parahaemolyticus* in molluscan shellfish, currently in draft form (CFSAN 2000).

For risk assessments to be useful for estimating the percentage of total illnesses of a particular pathogen due to specific foods, risk assessments must be performed on all relevant food items. Considering the duration and complication of the *Listeria* risk assessment project, even though it was limited to a subset of food items associated with *Listeria*, performing full risk assessments on all relevant pathogen-food combinations would be a colossal if not impossible task.

Aside from the large resource requirements, the major limitation of using risk assessments to measure the relative burden of illnesses due to particular foods is that they are inherently predictive. They do not measure the actual public health impacts of illness, as do surveillance data, but rather estimate illnesses given assumptions of risk factors, dose-response functions, food storage and consumption patterns, and consumer behavior. These assumptions, though derived from scientific research, are difficult to validate, especially in a dynamic and changing system. In particular, dose-response functions, which estimate the quantitative impacts on human health due to increasing doses of pathogens, are often defined with large uncertainties that necessarily propagate through the entirety of the risk assessment. Dose-response functions are occasionally determined through expert opinion, an inherently uncertain and less scientific approach than laboratory testing. Also, risk assessments are ill-suited for temporal analyses, since most are not regularly or routinely updated with new information about pathogen behavior, food consumption patterns, or food contamination levels.

Because of these limitations, it is likely that risk assessments are most useful in conjunction with other estimates, such as those based on outbreak data or case-control studies. Estimates based on public health surveillance data are often hampered by cross-contamination, recall bias, long exposure windows, and other limitations that do
not affect risk assessments. Likewise, risk assessments may predict sporadic cases not captured through outbreak or passive reporting systems.

**U.S. Food Monitoring**

The testing of food and animal sources for prevalence of pathogens is performed by various U.S. food safety agencies, both through routine monitoring and through case studies of specific food items. The Food Safety and Inspection Service (FSIS), within USDA, routinely tests raw meat and poultry products for *Salmonella* contamination (FSIS 2003), ground beef for *E. coli* O157:H7 (FSIS 2004a), and multiple ready-to-eat meat and poultry products for multiple pathogens (FSIS 2004b). The Animal and Plant Health Inspection Service (APHIS), also within USDA, performs bacterial testing on live animals. FSIS also performs national prevalence studies on the bacterial contamination of meat and poultry products. In addition, ARS has examined pathogen prevalence in commercial food products, as described in a recently published study on the prevalence of *Listeria* in frankfurters (Wallace 2003). These data are useful for quantifying known risks and for discerning trends over time but are insufficient for food attribution of illnesses. To best most useful for food attribution, monitoring data must be harmonized with public health surveillance data.

**Expert Elicitation**

When scientific or epidemiological data are lacking or sparse, or when there is significant uncertainty associated with interpreting such data, expert judgment may be a powerful means to fill gaps or combine conflicting estimates into a meaningful solution. Because proper handling and presentation of uncertainty are an important aspect of risk-based decisionmaking, expert judgments have been increasingly used and recommended for use in assessments of risk and health impacts of regulations (NRC 2002; OMB 2003, EPA 2004). Formal methodologies have been developed for eliciting expert judgments and for using them in analyses (Morgan et al. 1990; Cooke 1991).
Through surveys, experts may be asked to estimate a value for an unknown variable, or they may be given numerous values stemming from different data sources or studies and asked to estimate probabilities of plausibility for these values.

As noted previously, the FSRC Foodborne Illness Risk Ranking Model incorporates outbreak data for the purposes of food attribution (FSRC 2003). Because of the limitations of these data, however, FSRC administered an expert elicitation of researchers, public health scientists, and authorities with extensive food safety experience. Produced specifically for FIRRM using a standardized, vetted methodology, the survey had respondents give their best, low, and high estimates for the percentage of 11 pathogens caused by each listed food category. The survey included measures of respondent uncertainty, as well as additional variables to capture respondent biases and the sources of data upon which they relied. Although full analyses have yet to be completed, FIRRM incorporated the average of expert best estimates. Although for many pathogens, expert judgments closely match percentages calculated from outbreak data, there are significant differences. For example, outbreak data suggests that only 16% of foodborne *Campylobacter* illnesses are due to poultry, but experts estimated this number to be closer to 70%, a result much more in line with previously described FoodNet case-control studies.

Expert elicitations are not without limitations: they are based on perception, not on observable data. Results may be circular if experts rely on the same sources or deceptive if experts are similarly misinformed or biased. For these reasons, expert judgments are not an ideal source of food attribution data, but they likely hold great utility when data are few or inconsistent and uncertainty is substantial.

**Additional Relevant Studies**

Although the USDA Economic Research Service (ERS) has not explicitly done any research on food attribution, they have used data from other sources in their studies of the economic costs of foodborne illness. In one such study, ERS researchers
estimated the percentage of foodborne *Salmonella* due to eggs by dividing the estimated annual salmonellosis cases from the FSIS risk assessment by the estimated total annual foodborne salmonellosis cases estimated by Mead et al. (1999) using FoodNet data (Frenzen et al. 1999). In the same study, to estimate the percentage of illnesses due to pork, ERS utilized outbreak data from CDC studies and from CSPI. The study is significant because it uses a combination of approaches—risk assessment results and outbreak data. This combination of approaches shows that novel ways of food attribution can be performed by making use of the best available data, but it raises methodological questions about combining incompatible datasets. For its final regulatory impact assessment for the hazard analysis and critical control point (HACCP) final rule, FSIS estimated the percentage of foodborne illnesses from *Campylobacter, Salmonella, E. coli* O157:H7, and *Listeria* due to meat and poultry (FSIS 1996). In this analysis, FSIS relied on CDC outbreak data, epidemiological and community-based studies, and expert opinion. In a subsequent study, ERS estimated the cost of foodborne illnesses due to meat and poultry by multiplying these FSIS attribution percentages by their total cost-of-illness estimates (Crutchfield et al. 1997).

Research and data on antimicrobial resistance may yield data useful for food attribution. Three agencies—CVM, ARS, and CDC—are involved with the National Antibiotic Resistance Monitoring System, which regularly tests bacteria from animals, food, and human sources for susceptibility to 17 antimicrobial drugs. CVM has also performed risk assessments on the human health impacts of the antimicrobial resistance of bacteria, specifically for *Campylobacter* in chicken (CVM 2001).

**Future Options**

Table 1 summarizes the primary methods of food attribution: none of the current data sources for food attribution are sufficient on its own because of methodological limitations or gaps in available information. Furthermore, in the United States, activities and data sources are spread over a wide range of agencies and researchers, resulting in
a myriad of individual studies covering different aspects of the food attribution
problem without any comprehensive plan or way to unify them. These issues with
current approaches make it impossible to accurately and dependably attribute national
public health measures of foodborne illness, such as annual number of cases or deaths,
to the foods that are the vehicles for these pathogens.

It is critical, therefore, to step back and approach the problem of food attribution
under a unified framework that combines and enhances current science-based
approaches in an effective and efficient manner and that will ultimately reveal the
causal links between the consumption of contaminated foods and public health
outcomes. There are many directions for such a plan to take, and this section is included
to foster discussion and engender comparisons among possible approaches.

To start, we might consider the issue of food attribution in the context of the
recent National Academies of Science report, Scientific Criteria to Ensure Safe Food, which
argues first and foremost for “the development of a comprehensive national plan to
harmonize the foodborne disease surveillance that is conducted by public health
agencies with the monitoring of pathogens across the food production, processing, and
distribution continuum that is conducted by food safety regulatory agencies” (IOM
2003, 2). The report calls for enhancing FoodNet and PulseNet and increasing data
sharing between agencies. The report suggests that both pathogen monitoring and
foodborne illness surveillance be reevaluated and retooled so that more explicit links
between public health outcomes and food safety objectives can be met. Such an
approach would take advantage of the existing infrastructure of food safety and public
health data collection and concentrate efforts on the efficacy of these programs.

The report calls for connecting food monitoring to the surveillance of foodborne
illnesses, but another approach might be to explicitly add a food attribution surveillance
component to FoodNet itself, in which individual illnesses would be investigated and
sourced to the foods responsible. No small feat, such an approach would entail an
extremely large number of time- and resource-intensive epidemiological investigations
into individual cases of illness. The results of this labor would be very rich, however, since the best aggregate data on foodborne illness and public health outcomes would be directly and explicitly connected to active surveillance of food attribution of those illnesses. Over the past few years, FoodNet has begun adding new sites to increase its coverage of the U.S. population. If ongoing collection of food attribution data is to be included in FoodNet tasks, it may be necessary to focus data collection on a smaller number of sites—moving back to the original concept of getting in-depth, high-quality data from a small number of representative sites.

Similarly, PulseNet might be expanded beyond its role as an early warning system for outbreaks, to become the foundation for a national microbial source tracking network that would use microbial fingerprinting to source and track bacterial subtypes by food. Such a network could serve as a national electronic library of reference fingerprints and incorporate data on pathogens collected during microbial monitoring of food, cultures collected through FoodNet and other surveillance systems, and additional sources.

A different route toward aggregating food attribution data would be to expand the array of risk assessments from *Listeria* in ready-to-eat foods to include additional pathogens and food pathways. An extensive library of comparable risk assessments would increase the utility of individual risk assessments by making aggregation possible across food categories. Risk assessments have been performed by the food safety agencies on *E. coli* O157:H7, *Salmonella*, and *Campylobacter* and could be incorporated into a comprehensive array of risk assessments for four of the most important foodborne pathogens.

Several characteristics should be considered in the evaluation and comparison of food attribution methodologies, with their relative importance depending on the purpose for which the attribution data are sought. These include scientific accuracy and uncertainty, quality and breadth of data, computational consistency, practical feasibility, cost of implementation, flexibility and scalability, utility for targeting...
interventions, and congruency with other relevant data sources, such as surveillance of public health outcomes. This last point is arguably the most critical. As stated by the 2003 National Academy of Sciences report, it is absolutely necessary that data from food attribution methods be congruent with data from the surveillance of public health outcomes of foodborne illness (IOM 2003). Among the critical unresolved issues is how to balance such factors as scientific accuracy and practical feasibility to produce attribution data that will be both useful and affordable.

With so many institutions responsible for various aspects of the food safety system, collaboration is paramount, as is the explicit delineation of responsibilities and powers. One agency needs to assume the lead in building an appropriate system for the collection of food attribution data, but access to these data is a critical issue. Such data must be openly shared among agencies and with industry and academia, and privacy issues with individuals and industry participants have to be addressed.

On the grounds of strengths and weaknesses, the major sources of food attribution data described in this paper may again be briefly evaluated. Outbreak data are, by far, the most robust and useful data series in the United States for the attribution of a large number of pathogens to specific foods, but they are very limited for attributing sporadic illnesses. They are also inconsistent because of geographic reporting differences, changes in reporting procedures over time, and the lack of standardized categories of food vehicles. Limitations to the investigations required for such food sourcing include cross-contamination among foods and water sources, the contribution of person-to-person transmission, recall bias (particularly when there are delays in contacting persons after occurrence of illness), and disposal of origin food sources.

Incorporating the sourcing of food items into public-health, active-surveillance systems such as FoodNet is appealing because active-surveillance data on food attribution would then be directly connected to data on illness surveillance, but performing food sourcing investigations on every case of illness would be very costly.
And although it would capture sporadic cases not captured through outbreak data, it would be limited by the same issues that confront food-sourcing investigations.

Although FoodNet active surveillance does not include routine food sourcing, FoodNet has produced case-control studies to quantify risks, such as foods or behaviors, associated with laboratory-confirmed illnesses. These population-based studies are powerful but limited by recall biases, long exposure windows, and the fact that food vehicles identified as likely to be responsible cannot be verified through laboratory testing. Also, case-control studies are time-limited, as opposed to ongoing, and therefore provide snapshot, as opposed to dynamic, estimates of attribution.

The monitoring and prevalence testing of food items are useful for tracking food contamination along the farm-to-fork pathway and for identifying trends over time, but they are limited for the purposes of food attribution because they are disconnected from public health outcomes. To be useful for food attribution, food monitoring data must be combined with public health surveillance data, as called for in the National Academy of Sciences report (IOM 2003). To make food monitoring data congruent with surveillance data, it is likely that isolates from both sources would need to be comprehensively and regularly subtyped.

Although microbial subtyping methods hold great promise, they do not provide food attribution answers on their own, and there is no consensus on which methods are the most applicable. To be useful for food attribution, subtyping must be performed systematically on both food monitoring and public-health surveillance data, as is done in Denmark for Salmonella. In the United States, a far larger country, this effort would be particularly resource intensive and confront large institutional obstacles, since data collection activities are currently spread across multiple agencies within USDA, FDA, and CDC. Data ownership, management, and sharing are likely to be serious issues.

Risk assessments are very useful tools for risk management and science-based decisionmaking but are limited for the purposes of food attribution because they are inherently predictive of public health outcomes, rather than observational. Risk
assessments are implicitly removed from surveillance of public health outcomes. They are built upon microbial science methods that have their own issues, limitations, and uncertainties. They are also resource intensive and time consuming, and therefore ill suited to emerging pathogens and fast-changing food production systems.

Expert elicitation methods are useful for drawing inferences and quantifying uncertainty, especially for a field with so many different approaches and data sources, but rely on the least scientific of data—perceptions. Expert surveys may be most useful for identifying areas in which data-driven food attribution approaches deviate from expert opinion, because of either human bias in judgments or limitations and failures of data. The differences between these methods, and others not identified or yet conceived, need to be defined more clearly. It is likely, however, that none of these approaches will be sufficient on its own for the accurate attribution of foodborne illnesses to pathogens in specific food vehicles. The implicit conclusion, therefore, is that to attribute foodborne illnesses to specific foods scientifically and accurately, we must develop a comprehensive program that combines some or many of the discussed methods and data. It will not come about without significant resources and cooperation between food safety institutions.
Acknowledgements

The Food Attribution Data Workshop was sponsored by the Food Safety Research Consortium, a multidisciplinary collaboration to improve public health; members include the University of Maryland, Baltimore; University of Georgia; Iowa State University; University of Massachusetts; University of California, Davis; Michigan State University; and Resources for the Future. The workshop grew out of an FSRC project funded by the Robert Wood Johnson Foundation and received generous support from the Joint Institute for Food Safety and Applied Nutrition, the Center for Food Safety and Applied Nutrition and Center for Veterinary Medicine within the U.S. Food and Drug Administration, and the Office of Risk Assessment and Cost-Benefit Analysis and the Economic Research Service within the U.S. Department of Agriculture.
References


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### Table 1: Current approaches to food attribution (continues next page)

<table>
<thead>
<tr>
<th>Approach</th>
<th>Primary Virtues</th>
<th>Primary Limitations</th>
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<tbody>
<tr>
<td>Denmark Salmonella Accounts</td>
<td>- microbial subtyping provides direct link between public health endpoint and animal - high reporting of illnesses (social health care) - national, temporal coverage for both illnesses and animal/product monitoring</td>
<td>- difficult to expand to other pathogens; requires distinctive sub-types across reservoirs - focus on animals ignores non-animal sources - focus on reservoirs, not food products at point of consumption</td>
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<tr>
<td>U.K. outbreak data</td>
<td>- large dataset: national, temporal coverage - results correlate with local epidemiological findings</td>
<td>- misrepresents sporadic cases - not all pathogens well represented - dependence on general practitioners</td>
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<tr>
<td>U.S. outbreak data</td>
<td>- national and temporal coverage - large common dataset - straightforward, uses existing data - outbreaks and outbreak cases can be aggregated into food categories</td>
<td>- misrepresents sporadic cases - geographic and temporal inconsistencies (local reporting), and biases towards certain foods - environmental and cross-contamination - not all pathogens well represented - under-reporting; must use multipliers</td>
</tr>
<tr>
<td>FoodNet case-control studies</td>
<td>- population-based studies (ratios in samples closely approximate ratios in population) - captures risk factors not included in most surveillance data (travel, food preparation questions) - can implicate risks missed by laboratory testing</td>
<td>- survey format has recall bias and other limits - long exposure windows (problems with common exposures) - durable immunity in population can impede associating exposures with illnesses - no laboratory verification</td>
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<tr>
<td>Microbial subtyping</td>
<td>- subtyping of illnesses and foods can provide direct link between public health endpoint and source of infection - can be used to identify specific foods responsible (outbreak investigations) or animal reservoirs (source tracking by species) - many different techniques, growing fast</td>
<td>- for animal sourcing, sub-types must be distinctive across species (see Danish Salmonella Accounts) - may only be effective for limited pathogens - resource intensive; requires human surveillance, extensive monitoring of food and animals, plus laboratory testing, data storage, analysis</td>
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| Risk assessments | - can estimate cases not captured by surveillance methods (not limited by under-reporting or biases in epidemiological methods)  
- utilizes consumption and contamination data ignored by surveillance-based approaches | - predictive; can’t be verified  
- large uncertainties in dose-response models and exposure estimates  
- resource and time-intensive (each pathogen-food combination requires its own exhaustive study) |
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<tr>
<td>Food monitoring data</td>
<td>- captures upstream contamination (avoids environmental- and cross-contamination after purchase)</td>
<td>- not usable for food attribution unless made compatible (through subtyping or other means) with public health data</td>
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| Expert elicitation/ judgment | - useful when data is sparse or conflicting  
- formal methodologies increase utility | - respondents can be similarly biased  
- requires some level of consensus for reasonable error-bounds  
- based on perception, not data |
### Appendix A: The Food Attribution Working Group

<table>
<thead>
<tr>
<th>Name</th>
<th>Institution and Location</th>
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<tbody>
<tr>
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<td>Iowa State University, College of Agriculture, Ames, Iowa, USA</td>
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Appendix B: Agenda of the Food Attribution Data Workshop

Food Safety Research Consortium
Food Attribution Data Workshop

October 31, 2003
Holiday Inn Select
Atlanta, GA

AGENDA

8:00–8:20 a.m.    Continental Breakfast

8:20-8:40 a.m      Introductions and Goals of the Workshop - Mike Doyle

8:40–9:10 a.m    Defining the Importance of Associating Food Groups with Cases of Foodborne Illnesses - Glenn Morris, Bob Buchanan, and David Goldman

9:10-10:30 a.m    Current Approaches for Associating Food Groups with Cases of Foodborne Illnesses: Strengths and Weaknesses

• Danish experiences (30 minutes)
  Danilo Lo Fo Wong, Danish Veterinary Institute
  Techniques used to associate food groups with cases of foodborne illness
  Reporting procedures
• United Kingdom experiences (15 minutes)

  Sarah O’Brien, Health Protection Agency

• CDC experiences (75 minutes)

  Overview - Rob Tauxe
  Individual case data – John Painter/Rob Tauxe
  Expert opinion (Delphi approach) – Rob Tauxe
  Outbreak data – John Painter
  FoodNet case-control studies – Matt Moore
  Molecular epidemiology; genetic fingerprinting of pathogens isolated from humans and foods
  – Efrain Ribot/ Fred Angulo

• Public Interest experiences (15 minutes)

  CSPI outbreak database - Caroline Smith DeWaal,
  Center for Science in the Public Interest

• FDA-CVM experiences (40 minutes)

  Overview - Steve Sundlof
  Microbial source tracking of foodborne Salmonella and Campylobacter– Ruby Singh
  Classification and Regression Trees (CART) for Predictive Modeling of Food Animal Sources of Specific Bacteria – Gregg Claycamp

• FDA-CFSAN experiences (20 minutes)

  Risk assessment methods - Bob Buchanan

• EPA experiences (30 minutes)

  Overview- Jorge Santo Domingo
  Molecular tracking (rRNA); Bacterial source tracking to identify animal source of bacteria contaminating water/environment – Katherine Field
10:30–10:45 a.m.  Break

10:45–12:15 p.m.  Continue Current Approaches Discussion

12:15–1:00 p.m.  Lunch

1:00–2:30 p.m.  Continue Current Approaches Discussion/ Begin to Identify Best Approaches for a Food Attribution System

2:30–2:45 p.m.  Break

2:45–4:00 p.m.  Continue to Identify Best Approaches for a Food Attribution System

- Technical aspects
- Institutional roles
- Obstacles

4:00–4:30 p.m.  Wrap Up and Conclusions - *Mike Doyle*
MEMBER INSTITUTIONS

Center for Food Safety at the University of Georgia

Department of Epidemiology and Preventive Medicine, University of Maryland School of Medicine

Food Marketing Policy Center at the University of Massachusetts

Institute for Food Safety and Security at Iowa State University

The National Food Safety and Toxicology Center at Michigan State University

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