Introduction

A meeting of a joint FAO/WHO Core Expert Group on VTEC/STEC was held in Geneva, Switzerland, from 19-22 July, 2016. The list of meeting participants is provided in Annex I. All participants were required to declare any relevant interests and based on the information provided, none of the experts were considered to present any potential conflict of interest. The meeting was chaired by Dr Peter Feng, Research Microbiologist, Food and Drug Administration, USA, and co-chaired by Dr Roger Cook, Ministry for Primary Industries, New Zealand.

Strains of *Escherichia coli* characterized by their ability to produce Shiga toxins are an important cause of foodborne disease and infections have been associated with a wide range of symptoms from mild intestinal discomfort, haemorrhagic colitis (HC), haemolytic uremic syndrome (HUS), end-stage renal disease (ESRD) and death. This pathogenic *E. coli* group is referred to as both verotoxin-producing *Escherichia coli* (VTEC) and Shiga toxin-producing *E. coli* (STEC). This variable terminology was discussed and it was agreed to use STEC throughout this work.¹

The Codex Committee on Food Hygiene (CCFH) has discussed the issue of STEC in foods since its 45th Session and at the 47th Session, November 2015, it was agreed that it was an important issue to address (REP 16/FH, 2015).² To facilitate this work, the CCFH requested FAO and WHO to develop a report compiling and synthesizing available relevant information, using existing reviews where possible, on the following aspects of STEC:

1. the global burden of disease attribution based on outbreak data, incorporating information from the WHO FERG³ as appropriate;
2. hazard identification and characterization of STEC, including information on genetic profiles and virulence factors; and
3. current monitoring and assurance programs including the status of the currently available methodology (commercially available and validated for regulatory purposes) for monitoring of STEC in food as a basis for management and control.

The CCFH noted that to facilitate this work a call for data would be required and feedback from countries would be critical. The CCFH also noted that the nature and content of the work to be undertaken by CCFH, including the commodities to be focused on, would be determined based on the outputs of the FAO/WHO consultation.

¹ See Section on Terminology for background to this decision.
³ FERG - Foodborne Disease Burden Epidemiology Reference Group
Process

While there is considerable knowledge of specific STEC, such as those belonging to serotype O157:H7, knowledge of foodborne STEC that can cause illness, and belong to other serotypes, is less well defined and the overall understanding of all STEC in relation to foodborne transmission and illness continues to emerge. Compiling global information relevant to the CCFH request is anticipated to progress over 2-3 years. A core group of multidisciplinary experts will develop the overall approach and a work plan and will provide oversight and input to the implementation of the work plan relevant to their expertise. Other experts will be invited to provide input as required.

This meeting was the starting point for the core group of experts in addressing the CCFH request on STEC. The objective of the meeting was to decide on the scope of the work and the approaches and the methodologies that may be used based on the experts' knowledge and experience, and to develop a forward work plan. The work was divided into the three main areas, indicated above, on which advice on STEC was requested by the CCFH, although it is recognized there will be some overlap between these.

This report summarizes the discussions and findings of first meeting. In the course of the work more detailed reports and updates will be produced on the key areas of work as defined in the work plan.
Meeting outcome

Terminology
This pathogenic group of *E. coli* has been referred to using multiple terms and acronyms. Some of these, e.g., verotoxin-producing and Shiga toxin-producing, are synonymous and refer to toxin producing capability. Others e.g. non-O157 STEC refers to the STEC group aside from serotype O157:H7. Misunderstanding and misinterpretation can arise if there is not a common understanding of terms, especially if these terms are used in food regulation and in international trade without appropriate explanation. To provide a harmonized approach for this work, the experts discussed the variations in terminology and provided some background information.

The Shiga toxins are AB5 bacterial protein toxins (Melton-Celsa, 2014) that are the definitive virulence factor of the class of *Escherichia coli* enteric pathogens known as Shiga toxin-producing *E. coli* (STEC). In this document the term Shiga toxin (Stx) is used to indicate the toxin, stx to indicate the toxin gene, and STEC to indicate the *E. coli* strains demonstrated to carry stx or produce Stx. However, more widely, the synonymous terms verotoxin, verocytotoxin, and Shiga-like toxin have also been used for the toxins, and the terms verotoxin-producing, verocytotoxin-producing and verotoxigenic (VTEC) and Shigatoxigenic *E. coli* have all been used for this class of pathogens.

These alternate terminologies originated in the history of the discovery of the toxins and the development of understanding of their relationship to other pathogenic *E. coli*. The discovery that *Shigella dysenteriae* type 1 produced a protein toxin was reported in 1903 in separate papers by Neisser and Shiga (Neisser and Shiga, 1903) and Conradi (Conradi, 1903); subsequent research culminated in the isolation and characterization of this toxin as Shiga toxin in the 1940’s (Melton-Celsa and O’Brien, 2000). In 1977, it was reported that *E. coli* isolated from persons with diarrhoea produced a toxin that had a characteristic cytotoxic effect on cultured Vero cells i.e. kidney cells from African green monkeys (Konowalchuck et al., 1977). Subsequent research determined that these toxins could be divided into two groups: Shiga toxin 1 which can be neutralized by antibodies to the Shiga toxin of *Shigella dysenteriae* and Shiga toxin 2 which cannot (O’Brien et al., 1983; Strockbine et al., 1986). During this period, two terminologies were developed independently for the same toxins: verotoxins 1 and 2, and Shiga-like toxins 1 and 2. The term Shiga-like toxin was later changed to Shiga toxin after the amino acid sequence of Shiga toxin 1 was determined to be nearly identical to the toxin of *Shigella dysenteriae* (O’Brien, Karmali, and Scotland, 1994). Since then, identification of numerous variations in the amino acid sequences has led to recognition of two major Stx families, Stx 1 and Stx2, both of which includes many subtypes and variants (Scheutz et al., 2012).

In 1987, Levine proposed the term enterohaemorrhagic *E. coli* (EHEC) to designate STEC that can cause an illness similar to that caused by STEC O157:H7 and had similar epidemiological and pathogenetic features (Levine 1987).

In this document and subsequent reports related to the CCFH request, the expert group agreed to only use the term STEC, as it includes EHEC and because the interaction between known and putative virulence factors of STEC and the pathogenic potential of individual strains is not fully resolved.

Call for data
A call for data relevant to the CCFH request on STEC was issued by FAO and WHO prior to the meeting. The request was sent through the national Codex contact points and other relevant channels. Seventeen (17) countries responded with the great majority of data coming from North America and Europe, with less data from Asia and the Pacific, Latin America and the Caribbean, and Africa. No
response was obtained from the Near East. Further information is desirable to obtain a global perspective on the key areas to be addressed. The Group agreed that revised templates for collection of information and data with more refined questions are needed. In addition, future request will target specific sources (experts/organizations) in different countries, including both those that responded to the initial call for data as well as new ones.

**The Global burden of STEC disease and source attribution**

The Group considered estimation of the STEC burden of disease and the proportion of STEC illnesses that can be attributed to both total foodborne transmission and to transmission by major food categories. The work completed by the WHO FERG on the global incidence of STEC infections and deaths, 1990 to 2012 was presented to the Group. The potential contribution of FERG results to this work, challenges and limitations, and the nature and potential value of further complimentary and updated work were discussed. Approaches to attribution were presented and the choice of the most appropriate approach, tailored to the risk management questions posed by the CCFH, was discussed including data availability and resources required. The following summarizes that discussion:

(1) Global burden of disease estimates for STEC

Estimation of the overall disease burden and identification of the most important food sources causing STEC illness requires collection, integration and analyses of data from public health, food and animal surveillance. It was noted that surveillance systems vary in their complexity and efficiency, as well as in their ability to collect different types of information, to monitor trends and provide evidence for intervention. Even in countries with well-established and efficient surveillance systems, available data typically do not provide a true picture of the burden of foodborne diseases in the population, or of which sources or foods are responsible.

*a. Current WHO estimates*

The WHO FERG estimated the global burden of foodborne disease for 31 microbiological and chemical agents. It estimated that more than 600 million people fell ill in 2010, resulting in 420,000 deaths and 33 million Disability-Adjusted Life Years (DALYs). Foodborne STEC caused more than 1 million illnesses (95% uncertainty interval [UI]: 754,000 to 2.5 million), 128 deaths (UI: 55 to 374), and nearly 13,000 DALYs (UI: 5951 to 33,664) (WHO, 2015). Evidence underpinning these estimates was obtained from a systematic review (Majowicz et al., 2014) incorporating all evidence on the incidence of human STEC infections available from 1990 to 2012.

Over 17,000 sources were screened, yielding data for 21 countries, in 10 of the 14 designated WHO Sub-Regions. These data were extrapolated globally via a variety of approaches, to generate global incidence estimates. The STEC estimates are subject to several limitations, including numerous modelling assumptions (such as how much surveillance data under-represent the true population incidence), as well as the lack of data from many countries and Sub-Regions. The 2014 systematic review attempted to address these limitations via a sensitivity analysis in which various scenarios were modelled, but improving the representativeness and precision of these estimates would require new data from countries beyond the 21 originally included.

*b. Opportunities and benefits of updating the current FERG estimates*

The incorporation of new data on the incidence of human STEC infections, either from peer-reviewed studies, or via national surveillance, would allow the above estimates to be:

- more globally representative (i.e., by including countries beyond the original 21); and

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4 WHO Sub-regions were regional areas identified based on child and adult mortality as described by Ezzati et al., 2002, Lancet, 360(9343): 1347-1360.
more precise (i.e., by narrowing the 95% UI).

For example, national surveillance data are now available from Argentina; incorporating these data would allow the WHO Sub-Region estimate for the Americas region with low child and very low adult mortality to be more accurate and representative, as the current FERG estimate is based on Chilean data alone.

c. Work required to update FERG global STEC burden of disease estimates

- Review of information, methods and assumptions used by FERG to identify further options for improvement;
- A more targeted data collection effort to identify new STEC incidence data that could improve and possibly update the FERG estimates.

(2) Source attribution

The main concepts and approaches to source attribution were considered. Source attribution for this purpose was defined as the partitioning of the human disease burden of foodborne STEC illnesses to specific sources, including reservoirs and vehicles. The point of attribution, which depends on the risk management needs, was considered (e.g., point of reservoir or exposure) when deciding what approach to use.

a) Approaches to source attribution

Approaches to source attribution considered by the Group to address the CCFH request are summarized below:

The **subtyping approach**, based on the characterization of the aetiological agents, is particularly useful to identify the most important pathogen reservoirs and can be used to attribute disease to the reservoir or to the point of processing (Guo et al., 2011; Little et al., 2010). However, weak associations between certain subtypes and sources can limit the usefulness of this method; for example, some subtypes spread and contaminate sources throughout the food production chain. The method also requires representative and complete surveillance data from both humans and either animal or food sources, which is unavailable in many countries or for many pathogens.

**Comparative exposure assessment** compares the relative importance of the known transmission routes by estimating the human exposure to the hazard via each route. Information is required for each known route on the prevalence and quantity of the hazard in the source, the changes in these throughout the transmission chain, and the frequency of human exposure by each route (e.g., consumption data). These estimates are used to partition the total number of illnesses caused by the specific hazard to each transmission route, proportionally to the total exposure from all routes. The estimates of exposure for each route can be subsequently combined with a dose-response model to predict the number of infections in the population from each route. The comparative exposure assessment approach is particularly useful for pathogens that can be transmitted to humans by several routes from the same reservoir, and can be applied at the points of reservoir and processing.

**Case-control studies of sporadic, laboratory-confirmed infections** are the most commonly used approach for determining the importance of possible risk factors for illness, including sources and predisposing behavioural or seasonal factors. Population attributable fractions (PAFs) from case-control studies are used to estimate the proportion of laboratory-confirmed illnesses in the target population attributable to each source. A systematic review (SR) of published case-control studies of a given hazard can provide an overview of the relevant exposures and risk factors for disease, as well as a summary of estimated PAFs generalized to a broader population. A PAF derived from a meta-analysis of several case-control studies can be combined with an estimate of the total number of
illnesses in a population caused by that hazard to estimate the number of illnesses attributable to each exposure. SRs of case-control studies attribute disease at the point of exposure, and are particularly useful for regional and global studies.

Analysis of data collected during outbreak investigations can be used to identify the most common foods involved in outbreaks and is useful for quantifying the relative contribution of different foods to outbreak illnesses, to estimate the total number of illnesses in the population attributable to different foods, and also to estimate the contaminated ingredients in "complex" foods. Analyses of outbreak data to attribute disease at the point of exposure are useful for pathogens that frequently cause outbreaks; this method has the advantage of using data that is widely available worldwide.

Expert elicitation are particularly useful to attribute human illness to the main routes of transmission, i.e., foodborne, environmental, and direct contact to humans or animals. FERG has conducted an expert elicitation for all foodborne diseases, including STEC (Hald et al., 2016; Havelaar et al., 2015) and the output of that work will be considered in this project.

b) Source attribution studies proposed
The Group agreed that two approaches to attribute regional and global burden of STEC infections to specific foods would provide the required estimates of STEC source attribution at the point of exposure:

i. analysis of data collected during outbreak investigations; and
ii. systematic review of case-control studies of sporadic, laboratory-confirmed infections.

In addition, the implementation of a comparative exposure assessment could be conducted in selected countries at a later stage in the project, if quality food-chain data become available. This approach would estimate source attribution at the reservoir, processing and/or exposure points. The Group noted that with data-driven approaches, the quality of the outcomes will depend on the availability and quality of the data. It was also noted that significantly more information will be available for STEC belonging to serogroup O157 than for other STEC serogroups.

For the purposes of source attribution, the expert group agreed to use the food categorization scheme produced by the United States’ Interagency Food Safety Analytics Collaboration (Available at http://www.cdc.gov/foodsafety/ifsac/projects/completed.html, cited 24/08/16), acknowledging that it may need to be modified to accommodate sub-categories that are common in the countries or regions of a particular study.

Hazard identification and characterization
STEC belonging to serogroup O157 and other serogroups were discussed. Considerable time was devoted to review and discussion of the evolving complexity of STEC, the scope of illness caused by STEC, and how STEC might be categorized to assist in the interpretation of the public health risk of STEC when detected in food and along the food chain. Other factors that can affect this risk-based approach were discussed. The main discussion points are summarized below.

(1) Criteria for categorizing E. coli on a risk basis and interpretation of the categories
STEC known to cause severe intestinal disease can attach to intestinal epithelial cells, therefore, adherence factors are critical factors for STEC pathogenicity. The principal adherence factor in STEC is the intimin protein coded by the eae gene. However, as shown by the enteroaggregative E. coli (EAEC) O104:H4 strain that caused the large outbreak in Germany in 2011, other means of attachment, such as the factors coded by the aggR regulatory gene coupled with the production of Stx2 can also have severe consequences (Beutin and Martin, 2012). But since aggR resides on plasmids, that can be
unstable, sometimes the chromosomal aaiC is also used to assess the likely ability of the EAEC strain to adhere to intestinal cells. As a result, the presence of stx1, stx2, eae, aggR and/or aaiC genes are often used as predictors that the strain has the capacity to cause severe diseases. It is important to note however, that each of these genes alone is insufficient to predict the likelihood to cause severe illness, so a combination of these genes has to be used to predict health risk. For example, stx2 and eae or stx2 and aggR/aaiC are reliable predictors of high risk.

But, STEC pathogenicity is highly complex. For example, there are 10 subtypes of stx1 and stx2 and some are rarely or have not been reported to be associated with human illness (Scheutz et al., 2012). Hence, determining the specific subtypes of stx can be useful in assessing the strain’s potential to cause severe illness. Similarly, there are over 470 serotypes of STEC, most of which have not been linked to human illness (Mora et al., 2011). So, identifying the STEC serotype can help predict health risk, partly from an analysis of the past illness-causing history of a strain of that serotype, and, also when virulence information is not available. Lastly, it is critical to recognize that many STEC virulence factors reside on mobile genetic elements that can be lost or transferred. As a result, strains with the same serotype may have different virulence profiles and therefore different health significance; hence, most often, health risk analysis for STEC can be done only on a case-by-case basis. At present, the criteria mentioned are those most often used; however, our knowledge of STEC virulence continues to evolve. Hence, if research reveals that additional genes are involved in STEC pathogenesis, these health risk criteria may have to be modified accordingly.

The Group decided that a set of criteria and/or a decision-tree based on current knowledge of factors known to be required in STEC pathogenesis and phenotypes historically linked with disease should be developed, to provide a harmonized risk-based approach to characterization of STEC isolated from a food or along the food chain. A database of strains and serotypes could be developed to facilitate application of the decision-tree. For example, the database could include information on strains that have certain patterns when assessed against the criteria used in the decision-tree and historically linked in different regions with different levels of health risk from severe to minimal, or if no known risk has been reported. This characterization together with other factors such as knowledge of the intrinsic nature of the food, further handling that may influence survival, the preparation of the food before consumption, and if the food is to be provided to known high risk consumer groups, could be used in determining the potential human health risk posed by a particular STEC found in the food chain.

(2) Geographical differences in STEC (serotype, virulence)

The Group recognised that there is geographical clustering and variation among countries, in the distribution of certain STEC serotypes, both in the environment and foods, and in human infections (Johnson et al., 2006). Noting geographic differences is important for identifying new putative virulence factors, strains, serotypes and combinations. It can also provide insight into how regions are responding to the characterisation schema and identification of STECs in their region.

Often times, the illness-causing strain is seldom found in foods, but in some cases, there is correlation between the STEC serotypes in food and human infections. These links form part of the significance of "history" in health risk decision-making described in the section above. It was noted further that the correlation between STEC isolation from food and human infections may not be specifically serotype-based per se but associated also with exposure from the region’s environment or food handling context. The experts believe that geographic clustering of serotypes becomes less important if the definition of STEC that pose a health risk is based primarily on the presence of genes for Stx and adherence factors. Although the presence of such genetic traits in STEC in a region may be dynamic,
This approach is more practical as the implications of concentrating on specific serotypes alone could have unintended serious trade issues without a risk basis.

(3) Dose-response assessment for STEC virulence types

Limited information is available on the dose-response of STEC. The risk of life threatening illness in humans and the absence of an animal model that replicates human pathology preclude experimental determination of STEC dose-response. Estimates of dose-response have been made for STEC O157:H7 based on food concentration of the pathogen and patient consumption data from outbreaks. It is thought that exposure to less than 100 cells of STEC O157:H7 may cause infection. Exposure estimates have been reported from three outbreaks where the concentration of STEC O157:H7 in the food at consumption could be determined; 2 to 45 cells in salami (Tilden et al., 1996), less than 700 cells in beef patties (Tuttle et al., 1999) and 31 to 35 cells in pumpkin salad with seafood sauce (Teunis, Takumi, & Shinagawa, 2004). These estimates are reinforced by reports of STEC O157:H7 concentration, expressed either as Colony Forming Units (CFU) or Most Probable Number (MPN), in a variety of foods involved in outbreaks e.g. in raw milk cheeses, 5-10 CFU/g (Strachan, Fenlon and Ogden 2001) and 0.0037 to 0.0095 MPN/g (Gill and Oudit, 2015) and in beef patties 1.45 MPN/g (Kudo and Takatori, 2011) and 0.022 MPN/g (Gill and Huszczynski, 2016). The probability of infection on exposure to a single viable cell has been estimated to be 26% for children and 17% for adults (Teunis, Takumi and Shinagawa, 2004). The frequency of transmission in child care centres and among family members suggests that the usual infective dose is very low.

It is unknown whether the dose-response of STEC that use intimin for attachment varies between strains belonging to other serogroups, although due to the known genetic and physiological variability of STEC it can be presumed to be significant. However, it is not currently possible to identify STEC strains that have a higher probability of causing infection than STEC O157:H7. An investigation of an STEC outbreak involving serotypes O145:H28 and O26:H11 in ice cream found concentrations of 2.4 MPN/g for O145 and 0.03 MPN/g for O26 (Buven et al., 2011). In an outbreak of STEC O111:H-associated with fermented sausage, the estimated exposure dose was 1 cell per 10g (Paton et al., 1996). This indicates that the probability of infection upon exposure to other STEC strains may approach that of STEC O157:H7.

In addition to STEC strain factors, host factors very likely affect dose-response relationships as well as disease outcome. Individuals with a weakened immune system, such as the frail, elderly, and individuals that lack acquired immunity, such as young children, have the highest rate of illness and HUS (Havelaar and Swart, 2014). This should be taken into account when extrapolating dose-response estimates to settings with different demographic compositions or epidemiological scenarios.

(4) Other factors that affect virulence characterization

It has long been established that the transfer of genetic material is an important feature of the lifestyle of pathogenic and non-pathogenic E. coli (Wirth, 2006). Mechanisms of genetic transfer involve homologous recombination (e.g., O antigen determinants), bacteriophage insertion (e.g. Shiga toxin) or transfer of whole mobile genetic elements including plasmids (e.g. multi-drug resistance and EAEC plasmids). This phenomenon facilitates the potential of combinations of mobile elements to arise in E.coli strains leading to the potential emergence of STEC with novel pathogenic abilities (e.g. EAEC/STEC O104:H4). For the incorporation of novel genetic material in the population, a relative fitness balance must be maintained. For established clones it is not uncommon to see intimate, stable relationships occur between a host and mobile elements.

Different human populations are known to have differential outcomes to STEC infections. This includes differing susceptibility to HUS across age groups and a range of observed symptoms across infected
family members. Host human genetic and epigenetic factors are assumed to contribute to this observation.

(5) Emerging issues - Antimicrobial resistance in STEC
It is beyond the scope of this Group’s tasks to comment on the antimicrobial management in patients presenting with STEC infection. Additionally, current burden estimates of STEC infections in humans have not incorporated data on the burden of antimicrobial resistance in STEC causing these infections. The Group noted that antimicrobials are sometimes used for management of STEC infections in humans, and are often administered before the aetiology is known to be STEC. There are discrepancies among different countries on whether antibiotics should be used and how they should be used. For example, antimicrobial treatment is generally not recommended for primary treatment of infections with STEC that have a high risk of HUS. Certain antimicrobials, for example ciprofloxacin can lyse the bacteria to release more Stx into the host and so may worsen the prognosis in STEC infection if used for treatment. Antimicrobials are however sometimes used to eradicate carriage of STEC, for instance to enable children to return to childcare centres or adults to return to food handling jobs. Moreover, some uncommon STEC strains may cause invasive infections that must be treated with antimicrobials. For these and other reasons, antimicrobial resistance (AMR) among STEC isolated from food is of clinical and public health interest.

Antimicrobial resistance occurs in a percentage of STEC isolates but does not constitute a virulence mechanism in the primary pathogenesis of severe disease due to STEC (HC, HUS) and thus would not specifically be a target for virulence or health risk analysis. Animals, particularly cattle, are the primary reservoir of STEC: AMR in STEC can occur as a result of antimicrobial use in animals. Acquisition of resistant STEC infection may provide an opportunity for such resistance mechanisms in STEC to be transferred within the human gut e.g. via mobile genetic elements such as plasmids.

Current monitoring and assurance programs (including methodology)
Selected country’s approaches were presented to illustrate the variability among approaches to risk-based monitoring and assurance programs for STEC in food, and in the laboratory methodologies used, including the different approaches taken for domestic and for export foods within a country.

A limited number of responses were received from the call for data on current monitoring and assurance programs for STEC in foods. In general, regulatory programs are in place in every country to ensure that food safety systems in food manufacturing establishments are functioning as intended. The Group noted differences in specific STEC monitoring approaches between countries, mostly driven by the purposes of the food control systems in place. The public health importance of STEC based on local human disease surveillance, if available, can vary among countries and regions and thus the evidence for the need for food monitoring and assurance programs varies. It was generally agreed that the need for STEC monitoring in foods should be developed for a valid purpose and should be commodity specific. Otherwise, other indicators should be used to monitor processing hygiene. Those food safety programs including specific sampling techniques and laboratory methods for screening and confirmation of STEC are often imposed for export or as market access requirements for foreign food manufacturing establishments. Some countries conduct exploratory testing programs performed at the point of consumption and establish national baselines for certain products for a variety of pathogens including O157 and non-O157 STEC.

The group noted some challenges and limitations with laboratory methods that are used in regulatory food testing, specifically their applicability to the variety of foods that are now implicated in STEC infections and the limited number of methods and the variability of methods that are available for
non-O157 STEC. The Group decided more data are required in order to develop a comprehensive compilation of currently available STEC monitoring programs.

Summary points and future work

1. Burden of STEC disease. The WHO FERG estimated the burden of STEC disease in 2010. The incorporation of new data on the incidence of human STEC infections, either from peer-reviewed studies, or via national surveillance, would make these estimates more globally representative and more precise. While the analysis undertaken by FERG will be collated in a manner that best meets the needs of the CCFH, no additional burden of disease estimate work will be undertaken at this point. It was agreed that priority will be given to source attribution studies.

2. Source attribution. Different source attribution methods were considered. Taking account of the request from CCFH and point of attribution, the Group decided to use two approaches to attribute regional and global burden of STEC infections to specific foods: analysis of data collected during outbreak investigations and case-control studies of sporadic, laboratory-confirmed infections. This is because the Group thought that data from a greater number of countries would be available to support these approaches compared with the sub-typing or comparative exposure assessment approaches. However, the Group agreed that a comparative exposure assessment in selected countries could also be considered depending on the availability of additional good quality food-chain data. It was also agreed that some form of expert consultation/elicitation would be useful in the validation of the source attribution results, particularly as they apply to different regions’ contexts. The food categorization scheme produced by the United States’ Interagency Food Safety Analytics Collaboration will be used, with some necessary modifications required for different countries or regions.

3. Hazard identification and characterization. There is no single trait of an STEC that can be used to assess the public health risk of its presence in the food chain; rather, a combination of criteria such as virulence and phenotypic properties and regional historical knowledge are required together with knowledge of the isolation context. A set of criteria and decision-tree approach will be developed to support interpretation of detection of an STEC in food in a harmonized and risk-based manner. A supporting historical database of strains and serotypes would facilitate this approach.

4. Monitoring and assurance programs. From the limited information obtained on country programs, the Group thinks that most programs, including specific monitoring and assurance requirements for STEC are often imposed as market access requirements for foreign food manufacturing establishments. It was agreed that monitoring for STEC should be commodity specific and require purpose for testing (e.g. market access, survey, baseline establishment). Otherwise other indicators should be considered to monitor overall hygiene control during processing. The template (annex II) could be useful in organizing data on country programs which will serve as a basis for further discussion. An overview of currently available methods will also be developed.

Lead authors have been identified for each of the papers/analysis identified in each of the above-mentioned areas. Milestones have been established for the next two years when it is expected the work will be completed. An interim report will be available after one year.

References


Annex I List of participants

Core Group Experts

Dr Nadia Boisen (Denmark) Research Scientist, International Escherichia and Klebsiella Centre, Statens Serum Institut (SSI), Copenhagen, Denmark.

Ms Isabel Chinen (Argentina) Biochemist, National Infectious Diseases Institute - ANLIS “Dr. Carlos G. Malbrán”, Buenos Aires, Argentina.

Dr Roger Lindsay Cook (New Zealand) Ministry for Primary Industries, Wellington, New Zealand.


Dr Brecht Devleesschauwer (Belgium) Scientific Institute of Public Health (WIV-ISP), Department of Public Health and Surveillance, Center for Burden and Risk Assessment, Brussels, Belgium.

Dr Peter Feng (USA) Research Microbiologist, Food and Drug Administration, College Park, MD. USA.

Dr Alex Gill (Canada), Research Scientist, Verotoxigenic Escherichia coli laboratory, Bureau of Microbial Hazards, Health Canada, Sir F.G. Banting Research Centre, Ottawa, Canada.

Dr Patricia Griffin (USA) Chief, Enteric Diseases Epidemiology Branch, Division of Foodborne, Waterborne, and Environmental Diseases, National Center for Emerging and Zoonotic Infectious Diseases, Centers for Disease Control and Prevention, Atlanta, United States of America.

Dr Karen Keddy (South Africa) Head, Centre for Enteric Diseases, National Institute for Communicable Diseases, National Health Laboratory Service, Johannesburg, South Africa.

Dr Shannon Majowicz (Canada) Assistant Professor, School of Public Health and Health Systems, University of Waterloo, Waterloo, Canada.

Dr Sara Monteiro Pires (Denmark) Senior Researcher, Risk Benefit Research Group, Division of Diet, Disease Prevention and Toxicology; National Food Institute, Technical University of Denmark, Copenhagen, Denmark.

Secretariat

Dr Sarah Cahill, Food Safety Officer, Food Safety and Quality Unit, Food and Agriculture Organization of the United Nations, Rome, Italy.

Mrs Verna Carolissen-Mackay Food Standards Officer, Joint FAO/WHO Food Standards Programme Food and Agriculture Organization of the UN, Rome, Italy.

Dr Patricia Desmarchelier, Director, Food Safety Principles, Brisbane, Australia.

Dr Rei Nakagawa, Technical Officer, World Health Organization, Department of Food Safety and Zoonoses, Geneva, Switzerland.

Dr Blaise Ouattara Chief, Risk Prioritization, Food Safety Division, Canadian Food Inspection Agency, Ottawa, Canada.
### ANNEX II Template for Monitoring Programs for STEC

<table>
<thead>
<tr>
<th>Country</th>
<th>Sampling plan</th>
<th>Purpose*</th>
<th>Commodity</th>
<th>Target organisms</th>
<th>Lab Method for screening/confirmation</th>
<th>Analytical Sample size (g or ml) (if available)</th>
<th>Number of samples /sampling period (if available)</th>
<th>Reference / Link</th>
<th>Observations**</th>
</tr>
</thead>
<tbody>
<tr>
<td>M201 domestic / M202 import</td>
<td>Regulatory testing Market access</td>
<td>Raw ground beef</td>
<td><em>E. coli</em> O157:H7</td>
<td>MFLP-76 or MFLP-30 (screening) MFHPB-10 (Confirmation)</td>
<td>325g (each sample is made up of five 65g subsamples)</td>
<td>844 per year</td>
<td>Methods: Refer to link in row 1 Sampling programme: Refer to link in row 1</td>
<td>Limited number of methods available for non-O157 STEC</td>
<td></td>
</tr>
<tr>
<td>M218 domestic / M219 import</td>
<td>Regulatory testing Market access</td>
<td>Beef trim (N60)</td>
<td><em>E. coli</em> O157:H7</td>
<td>MFLP-76 or MFLP-30 (screening) MFHPB-10 (Confirmation)</td>
<td>325g (each sample is made up of five 65g subsamples)</td>
<td>1007 per year</td>
<td>Methods: Refer to link in row 1 Sampling programme: Refer to link in row 1</td>
<td>Limited number of methods available for non-O157 STEC</td>
<td></td>
</tr>
</tbody>
</table>

* Options for the purpose: (e.g., regulatory testing, market access, exploratory testing, national baseline study etc.)

** Observation: Please indicate any changes or factors that may have a significant impact on the detection rate or constitute a limitation.

*** The information provided in these rows is provided as an example of the type of information required under each of the columns.