Risk analysis for biological hazards in meat and poultry production – 2

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As we saw in the previous article, the fact that different people can perceive risks in vastly different ways creates the need for an agreed scientific approach based on authoritative sources of information to provide a transparent evidence based food safety system.

Table 1 describes the salient differences between biological and chemical hazards.

**Biological hazards**

- **Bacterial infections**
  Live organisms invade and multiply to cause disease in the host. To initiate a foodborne infection, sufficient viable organisms (viruses, bacteria or parasites) must be ingested in the food.
  Foodborne infections that are confined to the gastro-intestinal tract mostly present as diarrhoeal disease together with vomiting and abdominal pain. Examples include:
  - Salmonella.
  - Campylobacter.
  - Escherichia coli (VTEC).
  - Yersinia enterocolitica.
  - Clostridium perfringens.
  Some pathogens cause disease by spreading outside the gastrointestinal tract (extra-intestinal infections) into the blood or other organs in the body. Bacterial examples include:
  - Listeria monocytogenes.
  - Brucella.
  - Mycobacterium.

- **Bacterial intoxications**
  Intoxications are diseases caused by the consumption of preformed toxins in the food. They are generally formed as a result of growth of the organisms in processed foods during inappropriate storage of the food, generally involving some degree of temperature abuse.
  - Neurotoxins of Clostridium botulinum.
  - Enterotoxins of Staphylococcus aureus.
  - Emetic toxins of Bacillus cereus.

- **Parasites and waterborne infections**
  Live organisms invade and multiply to cause disease in the host. To initiate a foodborne infection, sufficient viable organisms must be ingested in the food. These are fully destroyed by cooking so are generally associated with meats eaten raw.
  Control options include inspection to remove diseased animals from the food chain, cooking and freezing.
  Examples include:
  - Toxoplasma, Trichinella, Taenia (tape-worm).
  - Cryptosporidium.
  - Sarcocystis.

- **Prion proteins**
  Transmissible Spongiform Encephalopathies (TSEs) are a family of untreatable fatal diseases caused by the build-up of abnormal prion proteins in the brain and nervous system. In humans the TSE is Creutzfeldt-Jakob Disease (CJD and vCJD), in cattle it is called Bovine Spongiform Encephalopathy (BSE), or Mad Cow disease.
  A similar TSE in sheep, scrapie, is not thought to be harmful to humans. There is another variant in sheep and goats, atypical scrapie, but there is no scientific evidence that it is of any risk to humans.
  Prion proteins are particularly stable in chemical terms and so are resistant to denaturation by chemical and physical agents, making their destruction and disposal difficult.
  Control is by the removal of infected ani-

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<thead>
<tr>
<th>Biological hazard</th>
<th>Chemical hazard</th>
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<td>Hazards can enter foods at many points from production to consumption</td>
<td>Hazards usually enter foods in the raw food or ingredients, or through certain processing</td>
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<td>The prevalence and concentration of hazard changes markedly at different points along the food production chain</td>
<td>The level of hazard present in a food after the point of introduction often does not significantly change</td>
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<td>Health risks are usually acute and result from a single edible portion of food</td>
<td>Health risks may be acute but are generally chronic</td>
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<td>Individuals show a wide variability in health response to different levels of hazard</td>
<td>Types of toxic effects are generally similar from person to person, but individual sensitivity may differ</td>
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The pandemic (H1N1) 2009 virus has not been shown to be transmissible to people through eating properly handled and prepared pork (pig meat) or other products derived from pigs. The pandemic influenza virus is killed by cooking temperatures of 70°C, corresponding to the general guidance for the preparation of pork and other meat.

Hazard characterisation
Severe illness or death in three age-based populations were considered: prenatal/perinatal; the elderly; and an intermediate age population.

Dose-response relationships were estimated by using contamination and growth data to predict levels of L. monocytogenes at the time of consumption for all ready-to-eat foods. These data were combined with epidemiology data to derive a dose-response model for each population group.

Risk characterisation
Individual food category data and the dose-response model were used to estimate the number of cases of illness per serving and per year for each food category and each population group. The ability of a food to support growth of L. monocytogenes to high numbers and the opportunity for growth is a key risk factor in foodborne listeriosis.

Key findings
The model indicates that it is the few servings with very high levels of contamination that are responsible for most of the illnesses and deaths. The vast majority of cases of listeriosis are associated with the consumption of foods that do not meet the current standards for L. monocytogenes in foods, whether that standard is zero tolerance or 100 cfu/g.

Details of authoritative sources of information are available from the author on request.

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