

Spring, 2007

VMD 413

VETERINARY FOOD SAFETY

(lectures [usually 12-1 p] & final, 1030 Valley Hall)

<<http://www.vetmed.ucdavis.edu/PHR/VMD413/vmd413.html>>

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Organization:

This is a 1.3-unit course, comprising 10 regular lectures, three “in-depth” lecture/discussions with practice quiz, and a final examination. Lectures will address the spectrum of topics significant to food safety; time will not permit detailed consideration of these, so the high spots will be outlined and an overview provided. You should read the material provided in the syllabus for the “in-depth” sessions and be prepared to be quizzed on this, as well as the material in the three most recent regular lectures. This is a change from previous years, when three 2-hour discussions were held with one-fourth of the class at a time. The Dean’s Office requested this change because of scheduling problems; we will make the best of it.

Purpose:

This course is intended to teach general principles of food safety, not to train food safety experts. It is a core course because every accredited veterinary school must have a required course in food safety. It is a useful course, because everybody eats. The course will attempt to describe the full scope of possible veterinary contributions to food safety. Some of the material presented is likely to be encountered again on board examinations.

Note that in many other of the world’s developed countries, and some that are less developed, veterinarians are the mainstay of food safety — not only the on-farm, preharvest aspect, but much of processing and distribution, even of products not of animal origin. In the U.S., veterinarians are active in food safety roles at all levels of government, not only to provide regulatory oversight, but as resources in pre- and postharvest food safety. Practitioners in food animal medicine are increasingly being looked to for measures that will reduce the incidence of human pathogens in the preharvest environment. Additionally, the urban public often looks to veterinarians (e.g., companion animal practitioners) for food safety counsel. In all of these situations, veterinarians can make an important contribution to the safety of the U.S. food supply.

VMD 413 will give you an introduction to the field of food safety. If you want to learn more, electives such as PHR 150 (Foodborne Infections and Intoxications) and PHR 452 (Veterinary Food Safety/Public Health) during spring quarter and PHR 450 (HACCP & Risk Assessment in Pre- and Post-Harvest Food Safety) during winter quarter may provide an opportunity to learn more about specific causes of foodborne disease and strategies for prevention, if they are offered in future years. There are also opportunities for summer jobs and for fourth-year externships in California government agencies, etc., to gain experience in veterinary activities in the many areas of food safety.

There is no textbook. A reading assignment in this syllabus addresses each in-depth topic. The accompanying 40-minute lecture/discussion will not address all of the points covered, which may appear on that day’s quiz or the final exam. Those seeking further information on any of the Food Safety topics may consult the following two books, which will be on reserve at the library.

Cliver, D. O., and H. P. Riemann, eds. 2002. *Foodborne Diseases*, 2d ed., Academic Press, London. xvii+407 pp.

Riemann, H. P., and D. O. Cliver, eds. 2006. *Foodborne Infections and Intoxications*, 3d ed. Academic Press (Elsevier), London, Amsterdam. xvii + 903 pp.

Grading:

Quizzes will not be graded; they are simply a study aid. Your grade will be based on the cumulative final examination. Last year's quizzes (which were graded) and final examination are in the course web site <<http://www.vetmed.ucdavis.edu/PHR/VMD413/vmd413.html>> for your guidance. A key to each quiz and the final examination will be posted on the web site as soon as possible after each has been completed. Deductions for answers that fall short of the ideal will be marked on the exam paper without explanation. Negotiation of these is discouraged.

Grades will not be awarded on either an arbitrary point-score scale nor on an arbitrary "curve" that assigns, say, "A" to a predetermined percentage of the class. Rather, the distribution of total points per person will be considered, with a view to identifying fortuitous groupings. Typically, there are gaps of as much as 1 point along the overall distribution that allow the modal total to be placed in the "B" range, with smaller, discrete groups receiving "A" and perhaps "B+," "A-," and "A+." Totals in discrete groups below the "B" range may earn a "C" or perhaps "B-". Grades of "D" or "F" are awarded only in truly exceptional circumstances. A consequence of separating grade ranges where gaps occur in the distribution is that students are generally assured that they did not miss the next higher grade by a fraction of a point on their total for the course.

Access:

I do not keep office hours as such. I spend 13 hours in the classroom during the run of VMD 413, plus teaching a 4-unit evening elective. When I am not in the classroom, I will either be in my office (1019 Haring Hall) or my laboratories (1117 and 1119 Surge I; 1010 Haring Hall). My telephone number is 754-9120 — if I'm not there, leave a voice mail. My e-mail, which is the surest way to reach me, is docliver@ucdavis.edu. If you need a one-on-one with me (about *anything*), we will find a way.

Objectives—you will learn:

- The types and causes of foodborne disease;
- The sources of the disease agents that occur in foods;
- The means available for controlling foodborne disease;
- The roles of the various participants in the food safety collaboration.

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Session 1. **Organization and safety problems of the food system**

Food typically passes through many hands on its way from the producer (farmer, fisherman, etc.) to the consumer. We will look at the organization of the food system in the U.S., and to a limited extent, in the world, noting how various functions are performed. We will also consider the record of reported and estimated foodborne disease, to see how problems are identified.

1.1 Why study food safety?

We all eat

Veterinarians have an important role to play

1.2 Why study food safety in the second year?

Electives available — PHR 250 (Foodborne Infections and Intoxications, 4 units) spring quarter and PHR 450 (HACCP and Risk Assessment in Pre- and Postharvest Food Safety, 3 units) winter quarter; both meet evenings

Job opportunities — paid internships in state agencies, etc.

Externships (fourth year) — California Depts. of Food and Agriculture, Health Services, other

Combined degree program — working toward an MPVM while earning a DVM

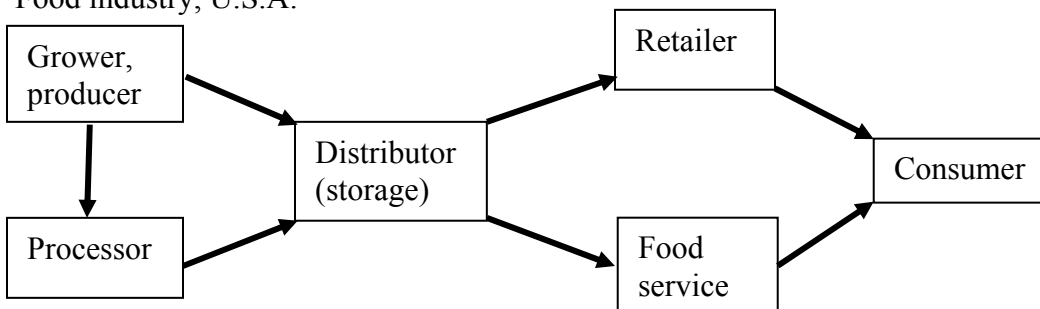
1.3 Organization of the course

Lectures — schedule provided

Discussion-quiz sections cover past three lectures, topic of the week, quiz (@ 20%)

Grading

1.4 Food industry, U.S.A.



Production

Farmers, ranchers, fishermen (~2.3% of U.S. population; 1.2% of population are officially employed in agriculture): Food is cheap in the U.S., compared to the rest of the world, but political power of producers can be diminished because of the small numbers of people engaged in food production.

Economies and challenges of scale — large-scale production of a single animal species or crop can greatly enhance economic efficiency, but can also make the operation vulnerable to introductions of disease agents.

Processing

Changes to raw material to increase usefulness (convenience, flavor, nutritional content, etc.): Every time someone handles food, they expect to be paid because they have somehow added value to the product (“value added” in 1992: \$120 billion).

Opportunities to improve safety come with many types of processing. The finished product should be at least as safe as, and typically safer than, the raw material from which it was made.

Storage, distribution

The “when” and “where” of food supply: Value is added to food by holding it until we want it and then delivering where we can get it conveniently.

Opportunities to degrade safety — or not: Improper storage or distribution can lead to hazards in food, but proper storage and distribution probably can never make foods safer.

Food service

Meals eaten away from “home” (2001: 46% of US food expenditures “in restaurants”; 2003 total \$426B)

Fast food vs other systems: There is a big difference in how the kitchen of a fast food establishment operates, compared to that of a regular, sit-down-and-be-served restaurant. Some of the larger fast food chains are well enough organized that they create less risks for the consumer than most sit-down restaurants. The biggest problems arise from rapid turnover of staff and lack of paid sick leave (whereby people who are ill are likely to continue to work with food because they need the money).

Institutional feeding: Foods served in dormitories, hospitals, nursing homes, military establishments, and prisons present special problems. All the same, if the help are paid well enough to minimize staff turnover, very safe operations are possible.

Retailing (2001: 54% of US food expenditures for foods “eaten at home”; 2002 total \$535B)

Costs, competition, discards: The grocery business is extremely competitive; costs are typically cut “to the bone” (average profit 0.95%), and the savings are passed on to the consumer. There is a fine line, in some instances, between economy and sacrifice of quality or safety. Large amounts of food are discarded (e.g., dented cans, slightly wilted produce, out-dated meat), rather than risk consumer illness or displeasure. Much of this food would be very acceptable in the poorer countries of the world.

Bar codes & product selection: The average supermarket offers 35,000 different food products. The bar code system makes it possible to identify each item at the checkout and charge the right price (assuming that the computer is working properly and fed with current information). It also helps trace food if a safety problem arises.

Preparation

What do “we” know how to do? Cooking skills have become increasingly rare, in the U.S. and elsewhere.

What are “we” willing to do at home? People no longer have the time to devote to long, involved cooking processes, so convenience foods are in demand.

Eating

Guilt & fear: The public is convinced that eating is a vice. *Food* is a four-letter word, and now “epidemic” obesity exacerbates the problem.

Enjoyment: We have forgotten that gastronomic pleasures are among the most enjoyable features of living. We can eat sensibly without losing all the fun. This entails emphasizing gastronomic quality over quantity.

1.5 Economic

Raw materials are largely produced on farms and ranches and are not ready-to-eat, nor even ready to sell to consumers.

Adding “value” — safety, convenience, time, and place are all things the food industry adds, by processing foods for added safety and convenience, storing them until we need them, and transporting them to where we have access to them. For example, although “farm-fresh” eggs may be the best, very few egg ranches would welcome consumers at their front gate, wanting to buy eggs. This would be a great challenge to biosecurity.

Cost to consumers — people in the U.S. spend less of their disposable income (i.e., income

left after taxes) on food than anywhere in the world. In the poorer parts of the world, incomes are so low that most of people's earnings go for food. In some of the richer countries, however, food costs more because people value food highly and are essentially paying extra for the things they like best. Americans tend to look for bargains, rather than for gourmet treats. Disposable income spent in U.S., 2003:

- 6.0% for food eaten at home
- 4.1% for food eaten away from home
- 10.1% total

Internationalism — food is one of the few trade “areas” in which the U.S. maintains a *favorable balance of payments* (i.e., we sell more than we buy, as measured in dollar amounts). About 22% of U.S. agricultural production is exported. International trade agreements may work to our disadvantage in some respects, but we seem to be holding our own in the food balance of trade.

U.S.	1999	2003
Food exports	\$48 billion	\$60 billion
Food imports	<u>38 billion</u>	<u>47 billion</u>
Balance	\$10 billion	\$13 billion

1.6 Safety

Sources of information, U.S. — Centers for Disease Control and Prevention (CDC), state epidemiologists (some city and county, as well), and non-government organizations.

Likelihood of foodborne illness

- Outbreak-associated illnesses/yr (CDC) 18,000
- Estimates in the 1994 *CAST Report* 6–12 million foodborne illnesses/yr
- *Emerging Infectious Diseases*, Sep.–Oct. 1999 — ~14 million illnesses, 5,000 deaths/yr

Incidence, rates, severity, sequelae, costs (CAST Rept.: ≥\$5 billion/yr)

Hunger in America

- 33.6 million in U.S. “food insecure” during 2001 (3.8 million households hungry at least some time during 2002), USDA-ERS
- 9 million in U.S. suffering from hunger in U.S. in 2001, USDA-ERS
- 4,000 deaths from malnutrition per year in U.S., CDC

“At risk” populations

- Age: infants and elderly people
- Pregnant women
- People with impaired immunity — cancer therapy, transplants, AIDS
- Genetic susceptibility — some agents require host-specific factors.
- Young adults are targeted by a few foodborne pathogens.

Who does what?

- Industry has the primary safety responsibility because most of what is done to food is under their control.
- Government serves a watchdog function, but can't be everywhere.
- Consumers need to be informed, have reasonable expectations, and handle food safely when it is in their possession.

HACCP (hazard analysis-critical control points) since 1996 in the USDA inspection scheme; “new” since the late 1960s; more about this in session 10.

Food Safety Initiative, 1997 — a broad federal attack on foodborne disease, now undergoing re-examination and re-interpretation

Other countries have their own systems. We tend not to respect the food safety systems, even of such groups as the European Union; they, in turn, are not too impressed with ours.

Looking ahead in this course

- Kinds and causes of foodborne disease
- In-depth: Bovine spongiform encephalopathy, TSEs
- How disease agents get into foods
- Pre-harvest food safety
- Life and death of pathogens in foods
- In-depth: *Escherichia coli* O157:H7
- Food preservation for safety
- HACCP and risk analysis for food safety
- Regulation of the food industry
- In-depth: Milk and dairy products — Risk analysis
- Summary and review for final

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Session 2. **Types of foodborne diseases and pathogens**

Foodborne infections and intoxications typically affect normal people, whereas allergies and intolerances are supposed to be effects of normal foods on abnormal people. The general pathogenesis of these conditions will be compared. Then we will begin a brief survey of the types of agents that cause foodborne diseases, beginning with bacteria as infectious agents.

2.1 Types of foodborne diseases (Food is a *vehicle*.)

- Infections — pathogens that are “alive” when ingested colonize the host’s body and multiply; may cause no illness, gastroenteritis (most often perceived as foodborne), or systemic illness. Ex.: salmonellosis, viral gastroenteritis.
- Intoxications — a poisonous substance is present in the food at the time that it is eaten, may be an intrinsic toxin, an extrinsic poisonous substance, or a toxin produced in the food by microorganisms. Ex.: shellfish poisoning, food botulism.
- Allergies — adverse immune (*antibody-mediated*) reactions to substances in food, or from other sources, that affect limited numbers of people. Most allergens in foods are wholesome and nutritious for the majority of people. Ex.: allergies to peanuts, soy, eggs.
- Intolerances — inability of some people's digestive systems to metabolize substances in food that are handled normally by others. In some contexts, people with these intolerances may be a majority, in which case the problem cannot properly be called an abnormality. Ex.: lactose intolerance, favism.
- Idiopathic illnesses — diseases that may or may not be foodborne, or whose mode of pathogenesis is unknown. Ex.: MSG-intolerance, sulfite-induced asthma.

2.2 Diagnosis (How do we know?)

Symptoms & lab findings — gastroenteritis (diarrhea, sometimes with nausea and vomiting) is often perceived as foodborne disease, sometimes incorrectly; association with food as a vehicle is best proven if the same pathogen is isolated from ill persons and from the food they ate in common.

Outbreaks, defined — the CDC defines an outbreak as two or more cases of the same illness, contracted from the same source (food vehicle).

Secondary cases — foodborne *infections* may be transmitted from the person who ate the contaminated food to others; no other kind of foodborne disease has secondary cases. Luckily, this mode of dissemination is generally self-limiting (i.e., secondary fewer than primary cases).

Sporadic cases, detection, “fingerprinting” — Sometimes only one person becomes ill with a foodborne disease, but in other cases there are multiple illnesses with no obvious connection; in such instances, it is sometimes possible to detect the outbreak by “fingerprinting” the pathogen isolated from ill people, and even from food. More outbreaks are being recorded in recent years as more laboratories are able to do fingerprinting, coordinated by CDC.

2.3 Foodborne pathogens (nothing further about allergies, intolerances, idiopathic illnesses)

Infectious agents — multiply in the host, some cause secondary infections

Bacteria
Viruses
Prions
Parasites

Intoxicants — poisonous substance is already present at the time the food is eaten.

Natural
Bacterial
Fungal
Algal, etc.

2.4 Foodborne infectious bacteria

Campylobacter — zoonosis (esp. poultry), multiplication $\geq 30^{\circ}\text{C}$, microaerophile

- Tends to cause sporadic illnesses, rather than outbreaks, affects young adults
- Usually diarrhea, occasional neurological complications
- Not a spore-former, labile in environment, killed by cooking

Clostridium botulinum — from soil, infects **infants** (rare)

- Rarely foodborne, but *C. botulinum* spores are found in honey
- *C. botulinum* also causes wound botulism (like tetanus) and foodborne intoxications
- Spores occur in the soil, are resistant to boiling

Clostridium perfringens — from soil or animals; spore-former; rapid growth at 45°C ; produces toxin (spore-associated protein) in intestine

- Pathogen of animals and humans (gas gangrene)
- Foodborne outbreaks most common in institutional feeding (e.g., dormitories)
- Gas produced by growing *C. perfringens* may be mistaken for boiling of food

Escherichia coli — various modes of pathogenesis (enteropathogenic, enteroinvasive, enterotoxigenic, enterohemorrhagic)

- All human-adapted except EHEC (bovine & ?) = topic of session 8
- May multiply in food (e.g., alfalfa sprouts)
- No spores: killed by thorough cooking or by irradiation
- *E. coli* O157:H7 is an “illegal adulterant” in raw ground beef (USDA-FSIS)

Listeria monocytogenes — “ubiquitous” (soil, sea, animals); “opportunistic” pathogen

- Illnesses usually mild, can cause abortions or stillbirths in humans
- Multiplies (slowly) in refrigerated foods
- “Illegal adulterant” in ready-to-eat foods in U.S. (all serotypes)

Salmonella spp. — principally zoonosis, can multiply well in many foods & environments

- Associated with many animal species, often as a commensal (no illness)
- Common non-food source is reptiles, especially pet turtles
- No spores: killed by thorough cooking or by irradiation

Shigella spp. — human-adapted, some growth possible in foods

- Human fecal contamination
- Bacillary dysentery — severe illness
- Special problems with parsley and cilantro

Streptococcus pyogenes — largely from humans

- Can cause severe illness (e.g., scarlet fever)
- Respiratory contamination more likely than fecal
- No spores: killed by thorough cooking or by irradiation

Vibrio spp. — from the sea, via shellfish

- Mostly three species: *cholerae* (causes cholera), *parahaemolyticus* (causes gastroenteritis), and *vulnificus* (deadly to some)
- Normal inhabitants (i.e., not contaminants) of warm marine waters
- Removed from shellfish by “depuration,” killed by cooking or irradiation

Yersinia enterocolitica — zoonosis, largely from swine (mimics appendicitis)

- Causes gastroenteritis
- Multiplies slowly in refrigerated foods
- No spores: easily killed by thorough cooking or irradiation

(*Brucella* spp.) — very serious zoonosis (sheep, goats, cattle, swine); controlled by “institutional measures” in U.S. (Session 6), so seldom foodborne here, but still a threat in developing countries; milk pasteurization

(*Mycobacterium* spp.) — very serious zoonosis (*M. bovis* transmissible to humans via raw milk = topic of session 12); controlled by “institutional measures” (Session 6), milk pasteurization in U.S.

2.5 Viruses — fecal-oral transmission; cannot multiply in foods; human host-adapted

Hepatitis A virus — one serological type, worldwide

- Illness mild or inapparent in children, may be severe in adults
- 4-week incubation complicates epidemiology, taking food histories
- Resists heat and drying, partially resists milk pasteurization

Noroviruses — caliciviruses, leading cause of reported foodborne illness in US, '98-'02

- Gastroenteritis — vomiting very common, may transmit virus
- Incubation 1–2 days, duration 1–2 days, fecal shedding may continue for a week after recovery
- Estimated to cause 2/3 of foodborne illnesses in U.S. (CDC)

Other

- Rotaviruses and astroviruses cause diarrhea most often in children; sometimes foodborne.
- Tick-borne encephalitis in Eastern Europe infects dairy animals (mostly goats), shed in milk, causes severe illness in those who drink raw milk or eat products made from raw milk.

2.6 For next time:

- Prions
- Parasites
- Intoxicants

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 Session 3. **Foodborne pathogens, continued**

The brief survey of foodborne disease agents will continue with prions and parasites as infectious agents, bacteria and molds that produce toxins in foods, and other causes of foodborne disease.

3.1 Foodborne pathogens

Infectious agents

Bacteria (last time)
 Viruses (last time)
 Prions
 Parasites

Intoxicants

Natural
 Bacterial
 Fungal
 Algal, etc.

3.2 Prions (see session 4)

- These are low molecular-weight proteins (called PrP^C) that occur in glycosylated form on the surfaces of cells, especially in the central nervous system.
- Normal folding is determined by their amino acid sequence, which is genetically controlled.
- When a prion becomes abnormally folded (PrP^{Sc}) for any of a number of reasons, it may become resistant to protease digestion and able to transmit its abnormal configuration to normal prions by contact.
- PrP^C PrP^{Sc} conversion occurs within a single animal, and accumulation of PrP^{Sc} in the brain causes incurable spongiform degeneration.
- Some spongiform encephalopathies are transmissible to other animals within a species and others to animals of another species.
- Bovine spongiform encephalopathy (BSE, or “mad cow disease”) is apparently transmissible among cattle, to some other ruminants, and to cats and humans. The human form of the disease is called variant Creutzfeldt-Jakob disease (vCJD) and is evidently acquired by eating parts of a bovine animal (not voluntary muscle or milk) that have high levels of PrP^{Sc} in them. Human susceptibility apparently has a genetic aspect.

3.3 Parasites

Protozoa — generally multiple host species

Cryptosporidium parvum and *hominis*

- Both broad-spectrum (*C. parvum*) and human-specific (*C. hominis*) species
- *C. parvum* is a common cause of diarrhea in calves
- Most often waterborne (huge outbreak in Milwaukee, 1993), sometimes foodborne; big problem in day-care centers for children
- No treatment — lifelong infection in AIDS patients

Giardia lamblia (= *duodenalis* = *intestinalis*)

- Broad host-spectrum, often present in surface water (lakes & rivers)
- Characteristic diarrhea due to blocked absorption — smelly
- More often waterborne than foodborne; also a problem in day-care centers

Toxoplasma gondii — important sequelae in newborns infected in utero

- Cat is definitive host; oocysts shed in feces for limited periods
- Tissue cysts occur in animals that ingest cat feces (usually accidentally)
- Humans infected from either source; waterborne outbreaks reported lately

Cyclospora cayetanensis — human-specific, oocysts sporulate slowly

- Gastroenteritis
- Imported to U.S. and Canada on produce (especially raspberries)

Roundworms— life cycles vary

Anisakids — from marine “finfish” & squid

- Definitive hosts are marine mammals (increasing)
- Rarely cause invasive infections
- Killed by cooking or (usually) freezing

Ascaris — human-adapted (also *A. suum*?)

- Eggs very stable in soil and in sewage sludge
- May be transmitted on produce

Trichinella — swine (rarely domestic in U.S.)

- Reservoir in rodents
- Carnivores (*bears*, *cougars*, etc.) more common food source in U.S.

Tapeworms — some life cycles complex

Diphyllobothrium latum — from freshwater fish; affects humans and other animals that eat fish raw

Taenia saginata — beef tapeworm

- Eggs shed in human feces infect cattle
- Cysticerci in beef killed by cooking or freezing

Taenia solium — pork tapeworm

- Eggs shed in human feces infect swine
- Cysticerci in pork killed by cooking or freezing
- Human cysticercosis from autoinfection or from eating fecally contaminated food

3.4 Natural intoxicants

- Mushrooms — some species deadly, some hallucinogenic
- Cyanogens produce cyanide in vivo from cassava (manioc), some varieties of lima beans, etc.
- Other: herbs used as “teas,” etc.

3.5 Bacterial intoxicants

Clostridium botulinum — from soil, marine sediment

- Spore-former, many types resist boiling
- Strictly anaerobe (can't grow in the presence of oxygen), needs pH > 4.6
- Nonproteolytic varieties most associated with seafoods, less heat-resistant, but grow slowly in the refrigerator
- Toxins destroyed by boiling, or even heating to 80–85°C

Staphylococcus aureus — saprophyte in foods (poor competitor)

- Salt-tolerant (ham a common vehicle)
- Bacteria are easily killed by cooking but produce heat-stable toxin (see table)

Bacillus cereus — from soil

- Spore-former, may withstand cooking
- Emetic or diarrheal toxin produced in food

3.6 Fungal intoxicants

Aspergillus and *Penicillium* toxins — most associated with grains (in field, more often in storage)

- Can cause severe illness in humans and animals
- Aflatoxins in cattle feed are shed in milk in slightly modified form
- Toxins are usually heat-stable

Fusarium toxins — grow mostly on grains in the field, can cause severe or fatal illness

Ergotism — *ergots* replace a grain of rye (or other grain species) growing in the field

- Large doses cause gangrene or convulsions and hallucinations in humans
- Apparatus is now available to remove the ergots from grain by sorting

3.7 Algal intoxicants

Shellfish toxins — certain dinoflagellate algae “bloom” in water occasionally

- Mollusks accumulate heat-stable toxin, can be deadly
- Prevention is based on coastal surveillance, public warnings

Waterborne toxins — some fresh-water algae (cyanobacteria) produce toxins that are hard to remove in drinking water treatment

3.8 Fish toxins

- Predatory reef fish show “food-chain” accumulation of toxins
- Tuna and some other species, held at too high temperatures, are substrate for bacterial production of histamine (illness resembles allergy)

3.9 “Chemicals” — chemical intoxications reported by CDC are almost all from seafoods (just described), rarely from acid foods or beverages attacking metal container surfaces.

3.10 Summary — looked at rest of common foodborne pathogens (not covered in previous lecture)

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Session 4 (in-depth): **Bovine Spongiform Encephalopathy**

(adapted from *Institute of Food Science and Technology (UK) Summaries and other sources*)

Spongiform encephalopathies have long been known to occur in sheep and goats (scrapie) and in humans. The most common spongiform encephalopathy in humans is called Creutzfeldt-Jakob disease (CJD). It occurs sporadically at the rate of one per million people worldwide; there are also familial and iatrogenic forms, plus some rarer varieties. A transmissible mink encephalopathy was reported in 1947; and a spongiform encephalopathy of North American cervids, chronic wasting disease (CWD) was recognized in 1967.

Bovine spongiform encephalopathy (BSE) was first recognized by the Central Veterinary Laboratory (CVL, UK) in late 1984 and identified as "spongiform encephalopathy" in a report dated 19 September 1985. From April 1985 to December 2006, 184,131 cases of BSE in cattle were confirmed in the UK, plus thousands in 25 other countries (Table 1 has data from 1987). Epidemiological study indicated that the vehicle of BSE was infected meat-and-bone meal (MBM) in cattle feed concentrates, the banning of which led to dramatic year-by-year reductions after 1993, despite failures in effective compliance and enforcement of the ban on Specified Bovine Offals in the period up to mid-1996. The number of EU-reported cases is surprisingly low, considering that 55,400 British breeding cattle were imported into other EU countries during 1985–90, plus thousands of tons of British MBM. In the spring of 2003, a first case of BSE was reported in Canada; and on December 23 of that year, the US reported its first BSE case, in a cow that had been imported from Canada; totals are now 3 and 9, respectively.

By 1994, TSEs were being observed in the UK in zoo ruminant and feline species and in household cats. Then, during 1994–95, 10 anomalous cases of CJD of a previously unrecognized pattern, reviewed by the UK CJD Surveillance Unit, led the Spongiform Encephalopathy Advisory Committee in March 1996, in the absence of other explanation at the time, to the conjecture that the UK cases were "most likely" to have resulted from ingestion of infected cattle brain or spinal cord before 1989, when they were banned from the food chain. By December 2006, there had been 158 deaths definitely or probably caused by vCJD in the UK, with 37 fatal cases in other countries (Table 2). Some countries suspect they have another form of BSE, and BSE infection of goats is suspected.

BSE-affected cattle appear nervous (there is no conventional *madness*), lose weight and have difficulty in walking; milk yields decline. The incubation period in cattle is generally 3–5 years, but the range may be wider.

The infective agent: A prion (PrP^{C}) is a small glycosylated protein molecule found mainly in the bovine brain cell membrane. It consists of 231 amino acid units. An infective prion (PrP^{Sc}) is not "live" — it has no associated nucleic acid. This is an agent that replicates and infects; it does not appear to follow the "rules" of microbiology nor of toxicology. "Prion" is a generic term. Different species have brain cell proteins of different conformations. For example, the human prion-protein amino-acid sequence differs at more than 30 positions from the cattle prion protein.

Protein molecules have three-dimensional, mainly alpha-helical corkscrew shapes. A PrP^{Sc} is a prion that has undergone a conformational change to a predominantly beta-sheet, flattened form and in the process become heat- and protease-resistant. When a PrP^{Sc} molecule reaches (by whatever route) the PrP^C in the brain or other cell membrane of a "host," it appears that the PrP^{Sc} molecule is able to act as a "template" to cause a PrP^C molecule to adopt a similar distorted shape — one might say, to "recruit" it. The newly converted PrP^{Sc} is able to act as a template to do the same to another PrP^C molecule; and so on. However, it should be noted that, once this process has started, subsequent PrP^{Sc} are of the host's amino acid sequence, and further recruitment is of PrP^C molecules of the same conformation.

In-vitro research suggests that "recruitment" can occur, though at a slower rate, even if there is a significant conformational difference between the infective prion and the host normal prion. The finding that vCJD host prion protein that is methionine/methionine homozygous at codon 129 is converted more rapidly than host prion protein that is valine/valine homozygous at codon 129, may have a bearing on the fact that all the victims who have died to date have been met/met homozygous at codon 129, suggesting that met/met has a shorter incubation period, rather than that val/val or met/val confer immunity [Since this was written, >6 years ago, there have still been no primary vCJD cases who were not methionine/methionine homozygous at codon 129 — the extended incubation period among other genotypes may be greater than one lifetime.].

The BSE infective agent can be detected in the brain, spinal cord, retina, trigeminal and dorsal root ganglia, tonsils, and distal ileum of symptomatic BSE-infected cattle; but extensive tests have failed to detect it in muscle meat or in milk. Potentially positive parts of the animal (Specified Bovine Materials) have been excluded from the food chain in the UK.

In the UK, a culling option was chosen, admittedly for political rather than scientific reasons, that entailed killing 79 healthy cows for each infected cow killed.

- BSE incidence: The continued dramatic year-by-year fall in the number of new cases of BSE in the UK (from the peak of 37,280 in 1992 to 338 in 2004) is “on track to bring the epidemic to an end.”
- BSE/vCJD: From research published from October 1996 onward, scientific evidence has been accumulating that BSE infectivity and new variant CJD (vCJD) infectivity carry the same "fingerprint." Although the scientific evidence does not prove a causal connection, the evidence is consistent with the transmissibility of BSE infectivity to humans.
- Beef and milk: Tests have not detected BSE infectivity in muscle meat (beef) or milk from confirmed BSE cows. Presently, animals slaughtered for meat in Europe must be under 30 months old (24 months in some countries), or their brains must be tested for BSE prions before the meat is released for human consumption. In the UK, cattle over 30 months old could not be eaten until recently.
- Blood: Recent research has indicated that human (but not bovine) lymphocytes circulating in the blood may carry infectivity. Blood products such as clotting factors are no longer being produced from the blood of UK donors but are being purchased abroad. In the US and many other countries, persons who have spent 3 months or more in the UK during 1980–1996 are disqualified as blood donors (Japanese, 1 day); a further ban has been applied to those who have spent 5 years in Europe since 1980. Since 2003, three

vCJD cases have been reported in the UK in which transfusion cannot be ruled out as the source of infection.

Measures imposed earlier by US agencies to prevent BSE in cattle or vCJD in humans from occurring in this country include:

- Prohibiting importation of live ruminant animals and most ruminant products from all of Europe (USDA-APHIS)
- Examining US cattle exhibiting abnormal neurological behavior to test for BSE (USDA)
- Prohibiting the use of most mammalian protein in the manufacture of animal feeds given to ruminant animals (FDA)
- Recommending that animal tissues used in drug products should not come from a country with BSE (FDA)
- Issuing guidelines asking blood centers to exclude potential donors who have spent 3 or more cumulative months in the U.K. between 1980 and 1996, etc., from donating blood (see above, FDA)
- Conducting intensive surveillance for any cases of vCJD among humans (CDC)
- Conducting research on BSE, CJD, vCJD, CWD, and related neurological diseases (NIH)

A recent concern in North America is the growing reported incidence of CWD in cervids. CWD was first reported in mule deer at an experimental facility in Colorado, but has since been found in elk and in whitetail deer in several western states, Canadian provinces, and in 2002, very extensively in Wisconsin. Both farmed and wild cervids are affected.

- Indications are that the agent of CWD contaminates soil, perhaps via feces of infected cervids, and is able to infect other hosts years later.
- Presently, the only known control method is depopulation. This is much more difficult with wild cervids (e.g., in Wisconsin) than with farmed animals.
- Although CWD is not known to be transmissible to humans (or other non-cervid species), surveillance has been established to detect human illnesses, and illnesses in predators of cervids, that might derive from CWD.
- Also, problems with disinfecting instruments used in surgery on humans with CJD have apparently led to transmission of prions to later surgical patients (= iatrogenic CJD). By analogy, there are fears that knives and other tools used in dressing hunter-killed cervids may later contaminate meat from domestic species that are slaughtered and dressed in the same facilities.

Events and measures since 12/23/03:

- As soon as the BSE diagnosis was announced, beef from the US was embargoed, just as we had embargoed beef from the 23 earlier BSE countries. Japan insists on 100% testing of slaughter animals (including veal calves), as they do, before opening their borders to our beef. The US is considering appealing this to the World Trade Organization. One company has volunteered to test all of its slaughter animals (to permit export to Japan), but has been refused by USDA.
- USDA ordered a several-state recall of all beef that might contain any part of “the cow.” New FSIS rules, enacted on an emergency basis, exclude from the human food supply:

downer cows, risk materials from cattle 30 months old at slaughter, and intestines from cattle of any age. New rules are estimated to cost the industry (and ultimately consumers) \$150 million. APHIS has stepped up testing of downers, animals dead-on-farm, and those with suspected neuropathies when inspected antemortem (carcasses of these animals cannot be passed until test results are received). Seven state laboratories (including ours in California) have been licensed to do rapid testing of bovine brainstems.

- FDA is banning mammalian blood, poultry litter, and plate waste from ruminant feeds. Feed production establishments must have separate facilities for production of ruminant feed if they use prohibited materials in the production of non-ruminant feed.

Table 1. Reported cases of bovine spongiform encephalopathy in the United Kingdom and other countries (as of 12/06)^a

Country	Total cases
United Kingdom	182,721
Republic of Ireland	1,590
Portugal	1,030
France	986
Spain	678
Switzerland	467
Germany	411
Belgium	133
Italy	140
Netherlands	82
Slovakia	20
Poland	50
Czech Republic	26
Japan	31
Denmark	15
Slovenia	7
Canada	9
Liechtenstein	2
Luxembourg	3
Austria	5
Finland	1
Israel	1
Greece	1
Sweden	1
United States	3

^aData from Food Standards Agency, UK.

Table 2. Chronology of variant Creutzfeldt-Jakob disease (vCJD) deaths in the United Kingdom and other European countries

Year of death in United Kingdom		Cases in other countries, by 4/07	
1995	3	France	22
1996	10	Canada	1
1997	10	Ireland	4
1998	18	Italy	1
1999	15	Japan	1
2000	28	Netherlands	2
2001	20	Spain	1
2002	17	US	3
2003	18	Portugal	1
2004	9	Saudi Arabia	1
2005	5		
2006	5		

(+ some still living)

Data from the UK Department of Health, World Health Organization, and ProMED.

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 Session 5. **Sources of pathogens in food**

Soil, water, air, plants, and animal and human wastes will be surveyed as sources of pathogens transmitted to humans via food. Production and transport of various classes of pathogens through the ecosystem to foods will be brought together into a matrix. We will consider which pathogens are of animal origin, and which contaminate foods in the pre-harvest phase.

5.1 Environmental matrix — interaction of people and animals with plants, water, and soil

5.2 Soil — enormous variations in ecosystems (more about microbial ecology in Lecture 6.)

Soil organisms — adapted to soil where they happen to be

- Heat, cold, freezing, high or low moisture, presence or absence of oxygen, nutrients
- Many dormant forms — spores, cysts, etc.
- Tend to out-compete introduced species
- Include some pathogens, such as *C. botulinum* and *Bacillus anthracis*

Organisms in soil — transients or visitors

- Pathogens from animal or (less likely in U.S.) human waste
- May persist, but seldom multiply in soil

5.3 Water

Water organisms — often adapted to a nutrient-poor environment

- Genera such as *Pseudomonas* produce many adaptive enzymes as needed, may sometimes be opportunistic pathogens.
- Water organisms of Genus *Vibrio* may be frank pathogens for humans, others such as dinoflagellate algae may produce toxins transmitted by seafoods.
- Some waterborne organisms can attack fecal pathogens that are introduced into water.

Organisms in water — transients or visitors

- Water-based waste handling (water-carriage toilets, flush manure systems) guarantees contamination of significant volumes of water.
- Nowadays, sewage and manure slurries are supposed to be treated before discharge to surface waters (lakes and rivers), but accidents sometimes happen. Treatment systems often use microorganisms to destroy pathogens and other pollutants.
- Most land-based pathogens can't multiply in water, and may die off slowly on their own, or by the action of indigenous water organisms.

5.4 Air — no “normal flora,” but almost never sterile

Spores — mold dissemination (and allergies)

- Found everywhere but the tops of the highest mountains
- Molds have an amazing array of enzymes to decompose things, and as pointed out in an earlier lecture, some are serious toxigenic pathogens.

Aerosols — finely divided droplets may contain bacteria and viruses.

- Droplets dry rapidly in air, but remaining solute may harbor bacteria that survive desiccation.
- Viruses transmissible by food and water are apparently not spread this way.

Dust — airborne solids may contain bacteria and other microbes.

- In California and some other areas, airborne dust may be finely powdered manure.
- Airborne soil dust is an alleged source of *C. botulinum* spores in infant botulism.

5.5 Plants — some produce toxic substances, but rarely (intrinsically) harbor agents infectious for humans or other animals; bacterial spores from soil are common.

Fertilization — use of animal manure or of human waste derivatives (e.g., septage, sewage sludge) as soil amendments **may** introduce infectious agents.

Irrigation — water re-use is necessary, but must be done **very** carefully.

- Drinking-quality water is usually too expensive for use in irrigation.
- Transport of pathogens from the roots of plants to edible, above-ground portions has been alleged repeatedly, but not really proven as yet.

Washing and icing produce at harvest add other opportunities for waterborne contamination.

5.6 Animal wastes

Zoonoses — some important agents of human disease are often of animal origin; examples: *Campylobacter*, *E. coli* O157:H7, *Salmonella*, *Yersinia*

Preharvest food safety — increasing pressure to eliminate zoonoses from the farm; easy to say!

- Wildlife animals (birds, deer, swine, etc.) are everywhere, defecating *ad libitum*
- Pathogens are by no means universal in food animals' manure, but animals shedding pathogens often give no hint that they are infected.

5.7 Human wastes

Urban wastewater & sludge — the water-carriage toilet and its ramifications

- We flush toilets to dispose of feces, and the water mingles with wastewater from other uses. Not all feces contain pathogens, but the entire volume of wastewater must be treated, at great expense, before discharge to surface water.
- Products of wastewater treatment are effluent (e.g., Sacramento's treated effluent goes into the Sacramento River) and *biosolids* (a.k.a. *sludge*), which are usually disposed to land or to landfills.

Wastewater treated on-site, septage — recycling: handle with care!

- Non-urban homes (farms and the “burbs”) tend to have on-site wastewater treatment systems, such as septic tanks. In some parts of California, these are poorly regulated.
- Improperly sited on-site systems can contaminate groundwater (i.e., someone's well) or surface water; septage (the material that is — or should be — pumped out of septic tanks) can be hard to dispose of safely. Used as a “soil amendment” in some places.

Direct contamination — human feces → human hands → food

- This is an important route for virus contamination of food.
- Any pathogen shed in human feces can contaminate food via unwashed hands, including eggs of *Taenia solium*.
- Fertilization of food crops with “nightsoil” (fresh human waste) is still common practice in some of the poorer parts of the world. It's all the fertilizer they have!

5.8 Primary vs secondary contamination

- Primary contamination is what happens pre-harvest, as in crops fertilized with pathogen-containing manure, or animals that are infected with zoonotic agents. This is sometimes unavoidable, so decontamination in processing can be important. We will look at pre-harvest contamination in session 6.
- Secondary contamination includes all the things that can happen to food between harvest and being eaten. Processing may decontaminate food, but other events may cause new pathogens to be added. These possibilities will be addressed in sessions 9 and 10.
- Pathogens that enter food may persist or die; only bacteria and fungi can multiply in food. The fate of pathogens in food will be discussed in session 7.

5.9 Summary — Foods are produced in complex ecosystems. Some pathogens that cause foodborne disease occur naturally in the environment, whereas others are produced in human and other animal bodies and contaminate food by way of soil or water. We try to minimize contamination before harvest, but it can't be entirely prevented.

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 Session 6. **Pre-harvest food safety**

There are many proposals to reduce or eliminate the presence of pathogens in agricultural products by the time of harvest. “Solutions” to some of the problems have already been announced, but may not prove valid. Available management options to promote pre-harvest food safety will be reviewed from the standpoints of scientific validity, practicality, and cost-benefit balance. We will consider how American agriculture, particularly animal agriculture, may be affected by these proposed innovations.

6.1 A goal: animals on (& leaving) the farm, consistently free of pathogens — at least of pathogens that threaten human health (i.e., zoonoses). This would **virtually** guarantee the safety of meat and animal products, but perhaps not “wholesomeness.”

Possibly included, by host species:

- Cattle — *Brucella abortus*, *Campylobacter coli*, *Coxiella burnetii*, *Escherichia coli* O157:H7 (or EHEC), *Mycobacterium bovis*, *M. avium* subspecies *paratuberculosis*, *Salmonella enterica*, *Taenia saginata*
- Small ruminants — *Brucella melitensis*, some others as for cattle
- Swine — *Ascaris suum*, *Brucella suis*, *Campylobacter coli*, EHEC(?), *Salmonella enterica*, *Taenia solium*, *Yersinia enterocolitica*
- Poultry — *Campylobacter jejuni*, *Salmonella enterica*

Not included: ubiquitous organisms such as *Bacillus cereus*, *Clostridium botulinum*, *Clostridium perfringens*, *Cryptosporidium parvum*, *Listeria monocytogenes*, *Staphylococcus aureus*, etc.

6.2 “Success stories”:

Brucella: skin test (B brand on left cheek of cattle for slaughter), ring test, vaccination

Mycobacterium bovis (reactors “suspect” from antemortem inspection, T brand on left cheek), skin testing, HTST pasteurization of milk. Outbreak in California — 2002.

Trichinella spiralis — cooking garbage for swine feed, serologic screening methods

6.3 Continuing challenges:

- *Salmonella* and *Campylobacter* in chickens (e.g., California Poultry Meat Quality Assurance Plan)

Sources of *Salmonella* and *Campylobacter* on the farm — *Salmonella* may be introduced to poultry ranches from many sources, but is seldom found in feed as purchased; carry-over from previous occupants of the poultry house is hard to prevent (dry cleaning is apparently more efficient than wet cleaning). The ecology of *Campylobacter* in poultry

ranches is poorly studied.

Carriage of *Salmonella* and *Campylobacter* by chickens — Intestinal carriage of *Salmonella*, and perhaps *Campylobacter*, is not prolonged. Some types of *Salmonella* that are likely to be found in chicken houses are almost never associated with human illness.

Schemes for prevention or eradication — “Competitive exclusion” tries to establish a *Salmonella*-free, adult microflora in chicks. The problem is that the microflora defies characterization (too many species, perhaps also dependent on relative concentrations), so production of the inoculum outside of chickens' intestines has not been successful.

- *Salmonella* in eggs (e.g., California Egg Quality Assurance Plan)

Advent of *Salmonella* Enteritidis Phage Type 4 (SE PT4) — Declared “epidemic” in Southern California in the mid-1990s; this may have been rhetorical overkill.

Sources & carriage of SE PT4 — supposed to be related to infection of the hen's reproductive tract (as opposed to the intestines), whereby the *Salmonella* occurs inside the eggshell. The mechanism has not been well described.

Control strategies — success? L.A. County, which reported a lot of SE PT4 in past years, says there was a real drop in salmonellosis in 2001 and that only 22% was SE. Most of the isolates are not phage typed; they say that eggs are a less frequent vehicle than in the past, suggesting transmission from eating chicken or from endemic presence in the community. No confirmed outbreaks of SE, regardless of phage type, were reported in all of California in 2004.

We are not alone!

- *E. coli* O157:H7 in cattle (see session 8)

Sources of *E. coli* O157:H7 on the farm — can infect wildlife species, as well as cattle. Birds can transport the agent, but evidently are not colonized.

Carriage of *E. coli* O157:H7 by cattle — Probably not carried for life, but there is at least a possibility that the rumen (and not just the intestines) is colonized. The major site of colonization seems to be the rectoanal junction. Shedding rates go up dramatically when cattle are stressed.

Opportunities for monitoring and eradication — environmental control is daunting; special attention to watering troughs is being advised and tested.

- *Cryptosporidium parvum* and the water supply — *C. parvum* is common in surface waters, as a result of shedding by many host species. Young calves produce large

quantities of oocysts, but cattle are certainly not the only source of oocysts in water. Deer, elk, feral swine, and California sea lions (among other species) have been shown to shed *C. parvum*. Existence of the human-specific species, *C. hominis* is now established.

- “Organic” farming — may lead to manure on vegetables, even though only composted manure is supposed to be applied to soil within 60 days of harvest. In a study of an egg ranch that had been contaminated with *Salmonella* Enteritidis PT4 from sewage by way of rodents, “free range” chickens were much more frequently colonized than caged layers.

6.4 Prognosis:

USDA — APHIS & FSIS seem to be working their way up to expecting pathogens to be absent from food-producing farms. The means to eliminate these pathogens are, as noted above, not readily at hand.

Public expectations of risk-free food: Some consumer advocates claim that, if farmers weren't so messy, food would contain no pathogens, and people wouldn't have to worry about how they handle and cook food at home.

Politics & science: At present, the move to eliminate pathogens from animal agriculture has more of a political than a scientific basis. Elimination of zoonotic agents that do not cause illness in food animals will be very expensive (if it can be done at all) and may be unnecessary.

“Willingness to pay”: There is a segment of consumers who say they are willing to pay much more for food if it is pathogen-free. However, there are substantial numbers of people in the U.S. (and many more in poorer countries) who cannot feed themselves adequately at present prices; for them, the risks of not eating outweigh the risks of eating. Interventions to reduce or eliminate on-farm pathogens should have a firm scientific basis before they are attempted.

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 Session 7. **Microbial ecology of foods**

Foods comprise a variety of ecosystems in which pathogens may occur. Pathogens may multiply (bacteria and fungi only), persist, or die. Important determinants of the fates of pathogens in foods, or any other context, include temperature, water activity, pH, E_h , nutrient availability, competing organisms, and the presence of antimicrobial substances. We will consider how these determinants act on pathogens, and how conditions in foods can be adjusted to enhance food safety. Remember that the microbes don't “know” they are in food — they are just responding to their immediate microenvironment.

Most raw (unprocessed) foods at room temperature are good substrates for microbial growth. Safe food preservation and storage depend on knowledge of the microbial ecology of the system.

7.1 Temperature — most infectious agents are adapted to body temperature ($\approx 37^\circ\text{C}$), but most grow well at room temperature, and some at considerably higher or lower temperatures. Organisms that produce toxins in foods may do so at temperatures quite different from that of the body.

Psychrotrophs grow *slowly* at refrigeration temperatures — *Listeria*, *Yersinia*, nonproteolytic *C. botulinum*.

- All of these grow faster at room temperature than in the refrigerator.
- Psychrotrophic infectious agents grow faster still in vivo at 37 C.

Optimum for *C. perfringens* is 45°C (doubling in 8–10 min), minimum for *Campylobacter* is 30°C .

“Danger zone” = 4°C (40°F) to 57°C (135°F) — new numbers

- As stated above, psychrotrophs will grow slowly at temperatures below 4 C.
- Most foodborne pathogens begin to die as temperatures approach 57 C.

7.2 Water activity — “ a_w ”

Defined as water available for microbial growth, based on water present and on binding by solutes such as salt or sugar; equilibrium relative humidity $\div 100$; range is 0 to 1.00

Minimum a_w for growth of some foodborne pathogens

<i>Salmonella</i>	0.93
<i>C. botulinum</i>	0.93
<i>Staphylococcus aureus</i>	0.86
(Most yeasts)	0.88
Most molds	0.75

Approximate a_w of some foods

Fresh fruit or vegetables	≥0.97
Fresh poultry or fish	≥0.98
Fresh meats	≥0.95
Juices, fruit & vegetable	0.97
Cheese, most types	≥0.91
Honey	0.54–0.75
Cereals	0.10–0.20
Crackers	0.10

Really dry foods may inactivate most viruses and protozoan cysts and oocysts in hours to days. The virus of hepatitis A and the noroviruses withstand drying.

7.3 pH — Most foods are acid or neutral, organic acids are more antimicrobial than inorganic acids.

Important minimum pH values for growth of microbes in foods

<i>Clostridium botulinum</i>	4.8–5.0
<i>Salmonella</i> (most types)	4.5–5.0
<i>Staphylococcus aureus</i>	4.0–4.7
Yeasts & molds	1.5–3.5

pH values of some foods

Egg white	7.6–9.5
Milk	6.3–6.8
Chicken	5.5–6.4
Beef	5.3–6.2
Cheeses, most	5.0–6.1
Tomatoes	3.7–4.9
Apples	2.9–3.5

Mold growth can *raise* pH of acid food.

7.4 Eh or oxidation-reduction potential — the basis of the aerobe-anaerobe-facultative classification of bacteria (molds are generally strict aerobes); positive mV values are aerobic.

Many foodborne pathogens are facultative: *Clostridium botulinum* is a strict anaerobe; bacterial pathogens are seldom strict aerobes.

Live foods (e.g., mushrooms) metabolize oxygen from air in packages; meat and fish bind oxygen. Either of these can create anaerobic conditions that permit growth of *C. botulinum*.

Packaging and atmosphere in the package are important.

- Controlled atmospheres in packages are often formulated to prolong product quality.
- A “hermetic seal” is supposed to keep everything that is put into a package inside, and everything else out. Seals on cans and some other packages are absolutely essential to the safety of the products. Fresh mushrooms, on the other hand, are usually prohibited from being sold in sealed containers.

7.5 Nutrient availability

Growth of bacteria and molds requires at least a carbon and a nitrogen source. Foods offer an abundance of these; microbial preferences differ, whereby some foods serve regularly as vehicles for certain pathogens. For example, no one has yet determined why the emetic form of *Bacillus cereus* food poisoning is so frequently associated with cooked rice.

Chemical complexity; physical form of the food

- We know that some bacteria can grow well on simple sugars and nitrogen sources, but this tends not to be true of pathogens, which often require complex nutrients (e.g., several different amino acids).
- Bacterial contaminants on the outsides of melons, oranges, etc., may not grow because they cannot get to the nutrients inside the fruit. Once the fruit is cut open, bacteria are likely to grow if the temperature is high enough to permit this. Molds, on the other hand, can often produce enzymes that can let them penetrate the fruit's outer covering, especially if any mechanical damage has occurred.

7.6 Competing organisms — each non-sterile food has a flora of its own; pathogens may compete, survive, or die.

Staphylococcus aureus is a poor competitor, so its vehicles are generally not raw meats or milk (which have many indigenous bacteria). An exception is milk from a cow with staphylococcal mastitis.

Clostridium botulinum doesn't seem to be subject to this constraint — indigenous bacteria are not known to prevent botulinum toxin formation, assuming that conditions are anaerobic.

In addition to competing for nutrients, some bacteria in food produce **bacteriocins** — heat-stable proteins that kill closely related bacteria

7.7 Antimicrobial substances — in foods, these may be:

- Intrinsic (e.g., lysozyme in egg white)
- Produced by other microbes (e.g., lactic acid in sauerkraut; bacteriocins)

- Added (e.g., nitrite in cured meats, which has been an important preventive for botulism; sulfites in wine)

7.8 Interactions

- Microbial **ecology** addresses the entire ecosystem, in this case, in a food.
- Conditions that barely permit **growth** of a toxigenic organism in food may not permit toxin production.
- Combinations of two or more limiting conditions may produce a safe result, even if neither of the conditions would suffice by itself (e.g., in cured meats, NaCl and nitrite, each at a level too low to be effective, combine to prevent growth of *Clostridium botulinum*).
- Modeling of the effects of the above factors on the growth of microbes in foods is increasingly being used to predict the safety of food formulations and processes — subject, however, to verification with real pathogens in prototype foods.

7.9 Summary — We have considered how the potential growth of bacteria and molds in foods is influenced by temperature, water activity, pH, Eh, nutrient availability, competing organisms, the presence of antimicrobial substances, and of course, time. All of these factors interact in complex ways, so that predictive modeling, no matter how sophisticated, must always be validated with real pathogens (or appropriate surrogates) in real foods. Knowledge of the life and death of foodborne pathogens is essential to the safety of the foods we eat.

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Session 8 (in-depth). **Enterohemorrhagic *E. coli* Food Poisoning and its Prevention**

(borrowed in part from an *Institute of Food Science and Technology (UK) summary*)

Foodborne illness caused by enterohemorrhagic *E. coli* (EHEC) was first recognized in early 1982. It does not appear to be a very common illness; but it is now regarded as important because serious infections, particularly in children, may result. The O157:H7 serotype is the predominant cause of human infections, but other serotypes have also been implicated. Infection may produce a mild diarrhea, or a severe or fatal illness. The infective dose may be very low, i.e. less than 10 cells.

Cattle appear to be the main reservoir of the agent, most cases being associated with ingestion of undercooked hamburgers and similar foods, or raw milk. However, other foods (**drinking & swimming water, apple juice, lettuce, alfalfa sprouts, baby spinach, etc.**) have also been implicated. The mechanism of transmission in the food chain is not fully understood but the contamination of meat from intestinal contents at slaughter is probably an important factor. More research is needed on the incidence of EHEC in cattle and meat products.

Prevention of EHEC illness in humans requires good slaughterhouse hygiene and heat treatment of raw meat and milk. EHEC is destroyed by heat; adequate cooking of meat (70°C [158°F] for 2 minutes internal temperature) and pasteurization of milk will protect consumers from infection from these sources. Hygienic food handling and good chilled storage conditions are essential and should ensure that other foods do not become contaminated. **For purposes of control by the Hazard Analysis-Critical Control Points (HACCP, session 10) system, irradiation is arguably the only authentic CCP.**

For most foods, testing for EHEC is not an effective control strategy and the setting of end product specifications is not appropriate. **[The preceding is the judgment of scientists in the U.K. The USDA declared *E. coli* O157:H7 an “illegal adulterant” in ground beef a few years ago and is now expanding testing, as well as the ban. A lot (≈ batch) of beef that yields O157:H7 is “unfit for human consumption” and must be recalled.]** The widely used standard methods for detection and confirmation of *E. coli* are not appropriate as many EHEC strains grow poorly or not at all at 44°C and usually do not produce β-glucuronidase. However, extensive research on isolation techniques has been carried out and various commercial kits have become available.

BACKGROUND

EHECs produce one or two Shiga toxins (ST), which are identical or similar to a toxin produced by *Shigella dysenteriae* type 1. The pathogenicity of these strains is not fully understood, but it involves adhesion to and colonization of the intestinal tract and the production of powerful toxins which act on the colon. There are at least three genetic determinants of virulence, all of which must apparently be present for the organism to function as EHEC. The infectious dose is not known, but it may be very low, i.e. less than 10 cells. Clinical signs range from mild diarrhea to severe bloody diarrhea (hemorrhagic colitis), and in some sufferers — most often young

children — include hemolytic uremic syndrome (HUS) and kidney failure, which can be fatal. In some outbreaks a high proportion of cases develop HUS, which is the most common cause of renal failure in children in the UK. In older adults, rarely, the infection may progress to cause thrombotic thrombocytopenic purpura, a condition like HUS but with neurological complications. Most EHEC infections in the UK and US arise from strains of serotype O157:H7, but other serotypes can produce ST and some have been shown to cause enterohemorrhagic illness. The predominance of O157:H7 is at least partly due to the fact that it is the only serotype routinely looked for. There is no specific treatment for EHEC infections at present; recovery from symptoms within 2 weeks is usual, but the organisms are often shed for longer periods.

OUTBREAKS

EHEC illness was first recognized early in the 1980s, and reports of outbreaks and cases are now increasing rapidly; there have been well-publicized incidents in many parts of the world. A large outbreak in the U.S. (1993) recorded 732 affected people: undercooked hamburgers were the vehicle. An outbreak of bloody diarrhea in a refugee camp in Malawi affecting 20,000 people is thought to have been caused mainly by EHEC; some cases were probably due to *Shigella*. Evidence from stored sera in the Netherlands has indicated that EHEC may have been the cause of HUS since at least 1974 in that country. A provisional UK figure of 656 cases was reported for 1994. In July/August 1996 there was a very large outbreak in Japan. Up to 26 August 1996, 9,578 people had been affected, many of them school children. Eleven people were reported to have died. Cases occurred in 40 prefectures, and several food sources were implicated — especially radish sprouts. A recent review from the U.S. Centers for Disease Control and Prevention estimates that over 100,000 EHEC illnesses occur annually in the U.S. (over 90,000 foodborne), of which about 1/3 are caused by STEC of serotypes other than O157:H7.

FURTHER

Many on-farm control measures have been tested, without great apparent success (session 6). The organism may be harbored in the rumen (including other ruminants than cattle), as well as the colon, and shed intermittently.

Many slaughterhouse measures are being imposed. None has yet been shown to be reliable. Testing of ground beef by USDA went on for 3 years, at 5,500 samples per year; samples positive were: 1995, 3; 1996, 4; 1997, 2. In 1998, FSIS increased the number and size of the samples; not surprisingly, the number of positive results and of recalls has increased. A modest-sized outbreak (15 confirmed human illnesses?) led to the recall of ~25 million pounds of ground beef and bankrupted the company. Federal ground beef recalls in 2002 totaled >5.3 million pounds.

Late in 1997, the U.S. Food and Drug Administration passed a regulation permitting irradiation of red meats, which would kill reasonable quantities of *E. coli*, as well as *Salmonella* and other pathogens that might be present. Irradiation of such products could not begin until USDA's FSIS passed a companion regulation, which finally occurred in February of 1999; the last of the needed regulations came out in February of 2000. The meat and poultry industries are showing

interest in irradiation, and retail establishments in some areas are now offering irradiated ground beef (mostly “pasteurized” with high-energy electrons), which is apparently selling well.

Associated Press, 2/29/00: Up to half the cattle in the nation's [U.S.] feedlots, far more than previously thought, are infected with deadly bacteria, the government said today. Researchers using new testing methods found *E. coli* O157:H7 present in rates varying from 1% in the winter months to as much as 50% in the summer. Previously, government scientists had thought the infection rate to be no more than about 5%. Cattle are exposed to the bacteria from manure in feedlots. [Cattle on range may have somewhat lower incidence of EHEC, but patterns of shedding and transmission are apparently comparable.]

The new figures were developed by the Agriculture Department's Agricultural Research Service and presented at a public session sponsored by USDA's Food Safety and Inspection Service. The study concluded that chances of *E. coli* getting into ground beef could be reduced by testing cattle hides and carcasses before the meat is processed. Testing now is done after the beef is ground. [Sampling and testing in this way would add greatly to the cost of beef and are not at all certain to prevent a significant portion of illnesses.]

Research done at a dozen packing plants during the fall of 1999 found the bacteria on 3.56% of the hides that were sampled and 0.44% of the carcasses. All the bacteria on the sample carcasses were eventually removed through steam pasteurization, hot water or organic acid rinses, the treatments typically used in packing plants. Samples in the industry study were taken at a rate of 1 per 300 carcasses.

A total of 38 confirmed outbreaks of *E. coli* O157 infection were reported to CDC in 1999 (the most recent year for which outbreaks have been described). These were reported from 30 states and affected 1897 persons. Two hundred one (11%) persons were hospitalized, 37 (2%) developed hemolytic uremic syndrome (HUS), and 4 (0.2%) died.

Over 70% of these illnesses occurred in two large outbreaks in New York and Illinois. Nine (24%) of the 38 outbreaks involved fewer than 5 persons, compared with 16 (36%) of 45 outbreaks in 1998. Three multistate outbreaks were associated with eating commercial frozen ground beef patties; one of these led to a nationwide recall of over 170,000 pounds and another to a nationwide recall of 250,000 pounds of ground beef. Contaminated drinking water was responsible for a large outbreak with two deaths at a county fair in New York. Romaine lettuce was the suspected vehicle in two community, multistate outbreaks. Five outbreaks occurred in restaurants, including one multistate outbreak involving a nationwide fast-food chain. Two outbreaks were associated with swimming at a community lake or beach. Seven (18%) outbreaks were attributed to person-to-person transmission in day care centers, and a vehicle was not identified in seven (18%) of the outbreaks. In 2000, a total of 4,528 *E. coli* O157:H7 illnesses were reported in people in the US.

During the summer of 2003, an *E. coli* O157:H7 outbreak involving five people was associated epidemiologically with tri-tip steaks that had been tenderized by needle injection of enzymes.

The supposition was that the needles had carried surface contamination into the centers of the steaks, which, having been tenderized, did not need to be thoroughly cooked for tenderness. Although the product had been processed in Chicago, a great deal of it had been distributed to restaurants in Western Canada, from which it was recalled by the Canadian Food Inspection Agency.

Reported incidence of EHEC in the U.S. in 2002 comprised 3,840 cases caused by serotype O157:H7, 194 cases caused by other serotypes, and 60 cases caused by strains that were not serotyped. USDA's Economic Research Service estimates that ECO157 illnesses in the US cost \$405 million, including \$370 million for premature deaths, \$30 million for medical care, and \$5 million in lost productivity.

A landmark event of 2006 was an interstate outbreak of ECO157 infections from baby spinach grown in the San Benito Valley of California. The outbreak comprised at least 205 cases in many states. All were infected with the same PFGE-pattern agent, which was also isolated from some packages of baby spinach. The source of contamination was not conclusively determined.

Another increasing source of EHEC is petting zoos; children are the most frequent victims (Associated Press, 4/2/05). One of the first large outbreaks occurred near Philadelphia in 2000; it included at least 16 children, and perhaps 45 more. In the summer of 2001, outbreaks at county fairs in Ohio and Wisconsin affected 84. In August 2002, 82 people (mostly children) were ill after attending a county fair in Oregon. The 2004 North Carolina State Fair yielded about 108 illnesses, and three fairs in Florida in the spring of 2005 have led to 22 illnesses, almost all in children.

What can you do to protect yourself, as a consumer?

- If you can't buy irradiated ground beef, cook your burgers thoroughly. There are those who say that a meat thermometer must be used (160 F at the center). Avoid cross-contamination, such as can happen when cooked burgers are put back onto the plate on which they were carried when raw.
- If you buy your vegetables at a farmers' market, where you are probably speaking directly with the producer, ask whether and how manure was used in their production. There is probably no hope of getting an accurate answer to such a question at a supermarket, but that is not necessarily a reason to avoid supermarkets. Washing produce at home probably accomplishes little, but it's worth a try.
- Wash your hands carefully after using the toilet or petting your critters and before preparing food or eating.
- Raw sprouts are OK most of the time, but they have spread *E. coli* O157:H7 and *Salmonella* in several recent outbreaks. They are probably not as much of a risk as driving on Interstate-80, but they are one of our more hazardous foods. Your call!
- Don't drink raw milk — outbreaks of *E. coli* O157:H7, *Salmonella*, and *Campylobacter* have been associated with this vehicle in recent years.

VMD 413. Veterinary Food Safety — Spring, 2007
Session 9. **Food processing methods**

Foods are processed (e.g., heated, chilled, ground, mixed) to preserve quality and enhance safety and convenience. In addition to direct treatments of the food, aspects of sanitation play an important role in “Good Manufacturing Practices.” We will consider how food processing and sanitation are used by the industry to meet its obligations in the domain of food quality and safety. **Note: Regulations are passed (enacted) by regulatory agencies; laws are passed by the U.S. Congress or state legislatures. Regulations cannot contradict laws. On the other hand, regulatory agencies are somewhat bound by scientific principles (via public action), but legislative bodies (e.g., U.S. Congress, state legislatures) are not.**

9.1 Inspection — doing what can be done to assure the quality and safety of raw material

- Grade A milk — the Pasteurized Milk Ordinance covers virtually everything that happens on the dairy, including the “harvest” process, which is two or three times a day milking; grade A dairy farms are subject to inspection.
- Despite increasing emphasis on pre-harvest food safety, the safety program in killing and processing four-legged food animals, poultry, and fish generally begins at the slaughter facility. Most of the program is governed by federal laws via the U.S. Department of Agriculture, especially the so-called “Megareg” (so named by USDA because it is perhaps the most massive regulation in the history of food law in the U.S.); state agencies may do some or all of the work, but the U.S. Congress requires that state inspection be at least as rigorous as federal inspection.
 - Methods of consumer protection:
 - 1) Eliminating diseased meat
 - 2) Esthetic factors
 - 3) Clean equipment and environment
 - 4) Labeling
 - Benefits of meat and poultry inspection
 - 1) Annual data show trends in animal health problems
 - 2) Prevention of human disease
 - 3) Increased marketability of products (across political boundaries)
 - 4) Consumer confidence
 - Antemortem inspection is intended to assess the health of the live animal and to detect conditions that might be less obvious after slaughter. Obviously, large animals (e.g., cattle) get more individual attention than small animals (e.g., broiler chickens). Animals are classified as passed, suspect, or condemned.
 - Postmortem inspection deals with slaughtered animals that were classified either “passed” or “suspect” on antemortem inspection. In some systems, inspection is done

by lots of carcasses rather than on each individual carcass. Inspection is done with eyes, nose, and hands, aided with a knife. Most of the observations are in the domain of gross pathology, with some tests for prohibited residues. In addition to conditions that might threaten the health of consumers, many decisions are based arbitrary standards called “wholesomeness.” End products of the inspection process are classified as “passed”; “passed, restricted”; or “condemned.” Additional portions of a carcass are classified as inedible because they are not ordinarily eaten by the intended consumer population. Condemned items must be carefully managed so that they are not marketed as edible. Although the “Megareg” is supposed to supplant organoleptic inspection eventually, carcass inspection at slaughter is still required by U.S. law.

- Eggs and many products not of animal origin are inspected for wholesomeness or classified for quality, or both. Some of these inspection and grading systems are operated by trade associations, rather than by government agencies. Time will not permit detailing these systems.

9.2 Processes convert raw food materials into products that are safer, more stable, or more useful to the consumer. For example, flour, yeast, and water are widely available; but most of us prefer to buy ready-baked (and perhaps ready-sliced) bread most of the time.

- Heat
 - *Blanching* processes are intended to inactivate enzymes or to drive gases from vegetables before freezing or canning; temperatures below the boiling point of water are usual.
 - *Pasteurization* processes are intended to kill the most important pathogens in a food without sterilizing the food; again, temperatures below the boiling point of water are usual.
 - *Boiling* is one of the oldest methods of cooking and is often used in commercial processing as well as in the restaurant or the home. Heat transfer from boiling water to food is reasonably optimal.
 - *Baking* is done at much higher nominal temperatures than the boiling point of water (e.g., $177^{\circ}\text{C} = 350^{\circ}\text{F}$). It is important to realize that, at atmospheric pressure, the temperature of food in the oven will not significantly exceed 100°C until almost all of the water in the food has evaporated away (at which point the food is likely to resemble a cinder).
 - *Retort processing* entails the use of pressure to raise the boiling point of water to, say 121°C (250°F). This allows reliable killing of spores of *C. botulinum*, assuming that the heat transfer properties of the product have been properly determined. Think of this every time you open a can!

- Chilling and freezing are common options in most American homes, but are less used in other countries and unavailable in the poorest parts of the world.

Keeping the temperature of a food low maintains or increases safety; most parasites are killed by freezing, whereas bacteria, molds, and viruses are likely to be preserved along with the food.

Lowering the temperature increases shelf life by days, and in frozen foods, even years. Nothing grows in ice, but some enzymes may remain active in frozen foods unless previously inactivated by blanching.

- Drying — lowers a_w
Air drying in the open is used for California raisins.
Hot air is used indoors to hasten drying.
Spraying into hot air is used to dry milk and other liquids.
Vacuum can draw water from foods in the frozen state (*sublimation*).
- Fermentation — microbial action
Acid production in making cheese, sauerkraut, etc.
Yeasts ferment sugars to produce CO₂ and alcohol in bread, wine, beer, etc.
Some molds are used in the production of specific foods (e.g., cheeses).
- Mechanical modification — peeling, slicing, grinding (comminution), mixing
- Chemical modification — acidification, preservatives
- Irradiation — disinfestation, control of sprouting or maturation, delay or prevention of spoilage, killing pathogens

9.3 Quality assurance — things done principally to make sure food is acceptable to consumers; safety measures are largely related but are increasingly treated as separate; “security” measures (anti-tampering, anti-terrorism) are being treated as yet another domain.

9.4 Packaging — intended to protect quality and safety

- Rigid, hermetically sealed containers
- Cans largely supplanted glass jars as containers for most foods, once problems with lead solder, chemically inert linings, and reliability of closures were surmounted.
- Most important is knowledge of heat transfer, so food can be safely processed in the final container.

- Flexible containers are being used increasingly, even for foods that require intense thermal processing, as polymers are developed that have the needed physical properties and do not leach material into the food.
- Carriers (boxes, crates, etc.) are used to store and transport several consumer-sized packages as one large unit. Occasionally, these have been a source of food safety problems.
- Atmosphere inside a package can be almost anything that is technologically desirable, given present-day methods. It is important that gas mixtures designed to maintain quality do not threaten safety (e.g., anaerobic atmosphere that might enhance botulism risk).
- Labeling is now regarded as a major aspect of packaging technology, whereby regulators are trying to find alternative vehicles for labeling information concerning foods sold in bulk. Classes of information include brand, product identity and quantity, preparation and cooking instructions, list of ingredients (exhaustive — for allergy prevention), and “nutrition facts.”

9.5 Food security — As with many other aspects of food and government, the processing sector has attracted special attention in the campaign for food security (i.e., prevention of malicious tampering with the product). The industry has taken this seriously, and much thought and training have been devoted to dealing with this. However, foods are usually much more accessible to tampering in storage and distribution and at retail than in processing.

9.6 Summary — Many things done to food between harvest and being eaten can affect safety. Inspection is intended to minimize opportunities for safety to be compromised, but most of the burden still lies with the food industry, in whose custody the food is until we bring it home. Food scientists apply knowledge of the microbial ecology of foods (among other things) to devise means of making and keeping food safe. Consumers need to understand enough of this so as not to undo the good stuff.

VMD 413. Veterinary Food Safety — Spring, 2007
 Session 10. **HACCP and risk analysis**

The quality, including safety, of products has traditionally been controlled by inspection of the final product. This is the case with food as well as with other products. Veterinary food (meat) inspection began almost 150 years ago, after veterinary pathologists and parasitologists had found ways to identify human pathogens in food animals. Meat inspection is still a large scale function of veterinary medicine.

10.1 Current meat inspection is based on 100% inspection of the live animals, their carcasses and organs, antemortem and postmortem. This system has not changed significantly over the last 100 years. The problem is that 100% inspection does not mean 100% detection of human pathogens, and this has led to a search for other ways to assure food safety. The inefficiency of the present inspection system is associated with changes in the ways meat (food) is produced, processed, marketed, and prepared as compared to 100 years ago.

10.2 HACCP was developed in the 1960s by the US food industry and NASA as a “zero-defect” approach to feeding astronauts. The bases of HACCP are that it is a process control rather than a product control and that it focuses control on steps in the processing system that are critical to consumer health.

- HACCP has won wide acceptance as a voluntary control program in the food industry, and is now a prescribed part of meat and poultry slaughter and processing in the U.S., as well as being required for seafood and juices. There is also a growing interest in using HACCP to control the safety of live animal production/pre-harvest food safety.
- There can hardly be HACCP without Good Manufacturing or Management Practices (GMP). Briefly, GMP is a description of all the steps (which should represent good practice) in a processing facility, while HACCP is a documentation that the steps important to consumer health are under control. Proposed GMPs for food *production* are often called GAPs (Good Agricultural Practices); at this point, GAPs are advisory, rather than mandatory. Sanitation standard operating procedures (SSOPs) are also a needed (and often required) prerequisite to HACCP.

10.3. **HAZARD ANALYSIS & CRITICAL CONTROL POINTS.**

Definitions:

- *Acceptable level* means the presence of a hazard which does not pose the likelihood of causing an unacceptable health risk.
- *Control point* means any point in a specific food system at which loss of control does not lead to an unacceptable health risk.

- *Critical control point*, as defined in USFDA Food Code, means a point at which loss of control may result in an unacceptable health risk.
- *Critical limit* means the maximum or minimum value to which a physical, biological or chemical parameter must be controlled at a critical point to minimize the risk that the identified food safety hazard may occur.
- *Deviation* means failure to meet a required critical limit for a critical control point.
- *HACCP plan* means a document that delineates the formal procedures for following the HACCP principles.
- *Hazard* means a biological, chemical, or physical property that may cause an unacceptable consumer health risk.
- *Monitoring* means a planned sequence of observations, or measurements of critical limits designed to produce an accurate record and intended to ensure that the critical limits are maintained.
- *Preventive measure* means an action to exclude, destroy, eliminate, or reduce a hazard.
- *Risk* means an estimate of the likelihood of a hazard occurrence; severity of outcome is often considered, too.
- *Sensitive ingredient* means any ingredient historically associated with a known biological hazard.
- *Verification* means methods, procedures and tests used to determine if the HACCP system in use is in compliance with the HACCP plan.

A HACCP plan begins by defining the product, describing the consumers for whom it is intended, forming a team for planning and implementation, and developing a detailed, step-by-step process diagram.

HACCP Principles

There are seven principles that are generally accepted as the core of HACCP. They are summarized below, with milk pasteurization as an example (even though the Pasteurized Milk Ordinance is just “evolving” into a HACCP system).

Principle #1: Hazard analysis

The hazard analysis accomplishes three purposes:

- (i) Hazards of significance are identified
- (ii) Likely hazards are selected (= risk assessment)
- (iii) Identified hazards can be used for developing preventive measures

Hazards are biological, chemical or physical in nature.

The potential risk of each hazard is assessed based on its likelihood of occurrence and its severity. The assessment is based on a combination of experience, epidemiological data and information in the technical literature.

Example: Raw milk has been known to be the vehicle for brucellosis, tuberculosis, Q-fever, campylobacteriosis, salmonellosis, and hemorrhagic colitis caused by *E. coli* O157:H7. These are *hazards*. In recent years, *Brucella abortus*, *Mycobacterium bovis*, and *Coxiella burnetii* (the agent of Q-fever) have largely been controlled at the herd level, whereas the other three illnesses still happen to people who drink raw milk (e.g., in California). Therefore, the significant *risks* associated with unpasteurized milk are campylobacteriosis, salmonellosis, and hemorrhagic colitis caused by *E. coli* O157:H7. Tuberculosis could be added to the list, as of 2002, in California.

Principle #2: Identify the critical control points (CCP) in food preparation

A CCP is a point, step, or procedure at which control can be applied and a food safety hazard can be prevented, eliminated, or reduced to acceptable levels. CCPs can be cooking, chilling, sanitation procedures, product formulation control (pH, salt, water activity), prevention of cross contamination, or employee and environmental hygiene.

Different facilities preparing the same food can differ in the risk of hazards; a *CCP Decision Tree* is of help in assigning CCPs — see diagram.

Example: Because milk cannot always be guaranteed to be free of pathogens, even when obtained from healthy cows, it is necessary to process the milk so that pathogens are killed before the product is packaged and distributed. Pasteurization is a long-established way to do this, and means exist to monitor the time and temperature of the process on a continuous, “real-time” basis, so it is a good choice as a *critical control point*.

Principle #3: Establish critical limits for preventive measures associated with each CCP

Critical limits are the boundaries for safety for each CCP and may be limits with respect to temperature, time, meat patty thickness, water activity, pH, available chlorine, etc. Critical limits may be derived from regulatory standards or guidelines, literature, experiments, and expert opinion.

Example: The most heat-resistant of milk-borne pathogens are *Mycobacterium bovis* and *Coxiella burnetii*, depending on the temperature of pasteurization. Legal standards for pasteurization of fluid milk in the U.S. specify 15 seconds at 72°C (the high-temperature, short-

time [HTST] process) or 30 minutes at 63°C (the low-temperature, long-time [LTLT] process) as minima. These are based on killing *M. bovis* and *C. burnetii*, respectively, and will kill all of the *Campylobacter*, *Salmonella*, or *E. coli* O157:H7 that could reasonably be expected to occur in raw milk. The ultra-high temperature process (Europe) is 2 seconds at 139°C. Therefore, depending on which process is chosen, these time and temperature values become the *critical limits*.

Principle #4: Establish procedures to monitor CCPs

Monitoring is a planned sequence of observations and measurements to assess whether a CCP is under control and to produce an accurate record. This record can be used in case of complaints about the product and is also used in the verification of HACCP. The measurements for monitoring are visual observations, temperature, time, pH, water activity, etc. The measurements must be done “on-line”; there is no time to wait for lengthy laboratory tests. There must be written documentation for who has the responsibility for monitoring.

Example: In HTST pasteurization, milk is sent to the heat exchanger with a positive-displacement pump. The number of revolutions per minute that the pump makes can easily be measured; this determines the time the milk spends in the heating unit. The temperature at which the milk comes out of the pasteurizer is continuously monitored and recorded (electronically and on a circular paper chart). These two records continuously determine that the critical limits are being met.

Principle #5: Establish corrective action to be taken when monitoring shows that a critical limit has been exceeded

There must be written instructions for actions to be taken (re-process, condemn, etc.) when critical limits have been exceeded and who has the authority for the action. Both disposition of the product and correction of the process must be specified. Sometimes regulatory agencies must be consulted.

Example: Milk that has not been legally pasteurized as it comes from the pasteurizer is not sent to packaging or further processing. Instead, a flow diverter sends it back to the feed tank to be pasteurized again. Meanwhile, a visible or audible signal alerts the operator to adjust the flow rate or temperature as necessary to meet the legal requirements. Both time and temperature are usually set slightly above the legal standard, as a margin of error. Warning devices go off before the CL is violated.

Principle #6: Establish an effective record-keeping system that documents the HACCP

This is necessary for internal audits and for verification of the HACCP system, sometimes by third parties. It is also important in case of consumer complaint.

Example: Processing charts are saved for every pasteurization run and must be shown to inspectors on request. Each is coded so that any batch of milk alleged to be under-processed can be matched to its corresponding pasteurization record. Records of corrective actions must also be made and stored.

Principle #7: Establish procedures to verify that the HACCP system is working

Verification is based on the HACCP documentation and may consist of internal audits or verification done by a third party. Verification may include laboratory testing of samples of food and/or the environment; the laboratory testing is sometimes called validation of HACCP.

Example: A supervisor should review the processing records regularly, to ensure that the process is being performed as specified and that corrective actions have been taken and recorded as needed; this is called *verification*. *Validation* can be done by sending samples to the laboratory — adequate pasteurization can be demonstrated by the absence of an enzyme called alkaline phosphatase, and proper handling of the pasteurized product until packaging (i.e., no recontamination) can be shown by properly low levels of coliform bacteria in the product. Validation essentially demonstrates that the process is accomplishing its intended purpose.

How can a food processing plant be sure that the raw materials used for processing do not in themselves present health hazards that cannot be eliminated during processing?

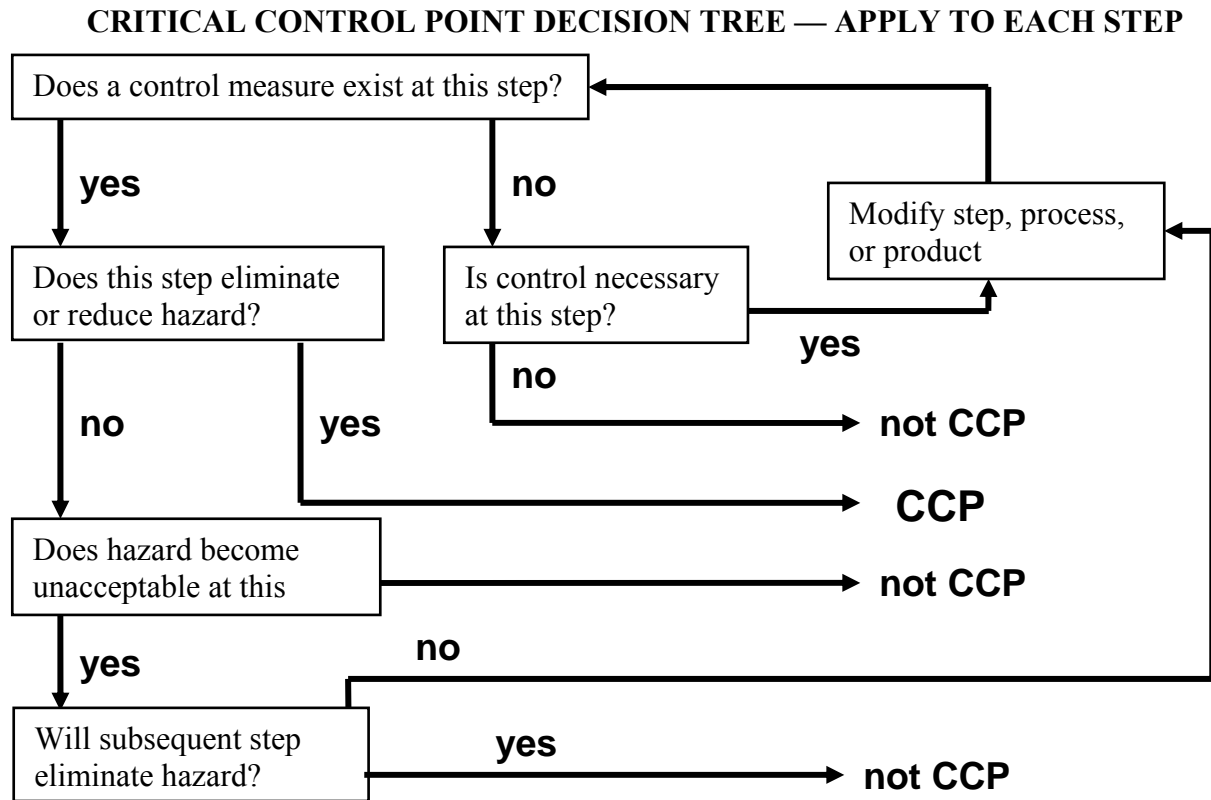
There are two ways: (1) specifications can be written for raw materials and checked by inspection and/or laboratory testing; (2) the processing plant can request that the supplier employs a HACCP system.

Attempts are being made to use the second approach in food animal production to assure raw product safety. According to the Food and Drug Administration, live food animals are defined as unprocessed food. Food safety in relation to animal production is known as *Animal Production Food Safety* or *Pre-harvest Food Safety*. This is really a unique branch of preventive veterinary medicine — unique in the sense that it attempts to prevent infection of food animals with human pathogens that rarely produce any clinical signs in animals.

10.4 Risk analysis involves three phases — risk assessment, risk communication, and risk management. In the HACCP context, it serves to choose, among the hazards listed for a food, those that are likely enough or severe enough to warrant preventive action. However, risk assessment is rapidly becoming a discipline unto itself, in which practitioners consider the levels of pathogens in foods, the quantities eaten by various groups of consumers, and vulnerability of the consumers to the potential disease. Once the risk of a given combination of food and pathogen has been assessed the information may be shared with the public or with decision-makers (risk communication), and a risk management strategy may be selected (often HACCP). Risk analysis will be revisited in session 12.

10.5 Applications — Adaptations of HACCP have been proposed in such diverse situations as production agriculture, surgical anesthesia, sewage treatment, and restaurant operation. There is seldom a problem identifying real hazards, but there can be serious difficulty identifying valid critical control points. Some years ago, the CCP criteria were mitigated by saying that, in addition to *eliminating* a hazard, a CCP could *reduce* or *avoid* a hazard. This has opened the door for many CCPs that require full time observation and (often) intense monitoring effort, but deliver much less than HACCP promises.

10.6 Summary — HACCP offers an organized way to monitor processes so as to eliminate health risks and deliver safe food to the consumer. Its authenticity and success depend on identification and use of valid CCPs. In its original application in the space program, it has an unblemished record of success for over 40 years.



VMD 413. Veterinary Food Safety — Spring, 2007
 Session 11. **Regulation of the food industry**

The U.S. government has regulated the food industry for about 100 years. State and local agencies, as well as international organizations, have also taken a hand. The depth and breadth of food safety regulation have increased enormously, whereby every aspect of the food system, farm to table, is now covered in some way. We will survey who does what in food regulation, and insofar as the question can be answered, “Why?” We will consider the facts that American agriculture constitutes an extremely successful partnership between agriculture and agribusiness, but that the term “free enterprise” does not mean what it once did in this context.

11.1 “Domains” of the federal government in food safety

USDA: meat and poultry, egg **products** (interstate and, to a certain extent, intrastate)

- Inspection: Food Safety and Inspection Service (FSIS), including labels; meat and poultry HACCP
- Protection, quarantine: Animal and Plant Health Inspection Service (APHIS)
- Preharvest food safety: FSIS (vs?) APHIS

FDA: foods other than meat and poultry (drop-in inspections, largely interstate commerce)

- Processing methods (Good Manufacturing Practices)
- Food additives (some USDA)
- Labels
- Foods on “common carriers” (planes, trains, ships)
- Animal feeds, chemical fertilizers
- Drugs for veterinary use (e.g., in food animals)
- Pasteurized Milk Ordinance (Conference of Interstate Milk Shippers) — published by FDA, enforced by states
- Food Code (adoption, enforcement by states)
- Seafood and juice HACCP
- Shell eggs

EPA: pesticide registry (includes sanitizers); drinking water standards; run-off into waterways

Bureau of Alcohol and Tobacco (Treasury Dept.): alcoholic beverages

FBI: Since ~September 11, 2001, food counter-terrorism.

11.2 State and local:

- Any of the above (enforcement of Pasteurized Milk Ordinance)
- Food service and retailing (enforcement of FDA Food Code?)
- In-state meat inspection

11.3 International:

- Codex Alimentarius (Food and Agricultural Organization of the U.N.) — internationally negotiated food specifications for international trade
- International Standards Organization (ISO 9000, 22000, etc.) — generally considered “advisory” until specifically adopted by national governments; often the basis for agreements between companies trading internationally.
- International Commission on Microbiological Specifications for Foods — non-government organization developing science-based recommendations for greater food safety at national and international levels
- Leagues of specific countries that have internal trade standards — European Union, NAFTA, Mercosur, etc.

11.4 Inspection by FSIS

- Continuous in-plant inspection by USDA employees, under veterinary supervision
- Has been largely clinical and gross pathology observation (“organoleptic,” sometimes called “poke and sniff” by consumer activists)
- Beyond ante- and postmortem inspection, inspectors are responsible for everything that happens during processing and packaging.
- Now requiring HACCP, testing for “generic” *E. coli*, sampling for *Salmonella* (testing by USDA); *E. coli* O157:H7 is an “illegal adulterant” in ground beef. The levels of *E. coli* and the frequency of detection of *Salmonella* are called “performance standards”; the principle of regulation on this basis has been questioned (Is there a scientific basis for the standards? Do they relate to public health [i.e., prevent foodborne diseases]?)’
- Plans to assign most of the HACCP-based inspection tasks to company (as opposed to USDA) workers have been fought by the inspectors' union. Present meat and poultry inspection laws require USDA inspection of every animal, ante-mortem and post-mortem, by USDA inspectors; this will continue until Congress changes the law.

11.5 Inspection by FDA

- “Drop-in” inspections, often at 3–5-year intervals
- During normal operating hours
- Inspectors can go anywhere in the plant and take samples, but not photos (still true?). Can examine thermal processing records and HACCP files, but not other operating records.

11.5 Future directions

- HACCP for fish and juices, then meat and poultry and all FDA-jurisdiction foods — old inspection systems **out**, eventually
- Implies the existence of one or more critical control points; critical limits at CCPs require “**real-time**” or “**on-line**” measurements
- Federal version includes an unprecedented focus on end-product testing for microbial pathogens; inspection of records
- “Farm-to-table” presents real problems — stress on the agriculture-agribusiness relationship
- Rhetoric: Pathogen-Reduction Program, “Megareg,” “illegal adulterant,” “Food Safety Initiative,” “science-based” regulation, activist demands for increased testing
- “Performance-based standards” — put arbitrary limits on levels of “generic” *E. coli* (not O157:H7), frequency of occurrence of *Salmonella* on carcasses; not-quite-full-cycle in 30 years.
- Mandatory recalls — USDA claims that voluntary recalls do not provide enough consumer protection.
- Consumer education — “Fight Bac” is intended to alert consumers to what they can do to keep food safe. Although the federal government has funded the development of programs, the state and local governments are supposed to pay for printing and delivery of materials and instruction.

11.6 Government is inevitably involved in enforcement of food safety regulations. Many of the rules have derived from political decisions that were intended to keep the voters happy. Although the food industry still bears the brunt of the food safety task, many believe that it is government that keeps food safe and that greatest safety is best achieved by an adversarial relationship between the food industry and government. Since September 11, 2001, there is increasing talk of “partnership” between the food industry and government, at least to protect against terrorism.

VMD 413. Veterinary Food Safety — Spring, 2007
 Session 12 (in-depth). **Milk and Milk Products — Risk Analysis**

This discussion section has two purposes:

1. Consider consumer risks from milk and its products
2. Learn about *risk analysis* as an approach to dealing with public health risks

We'll start with a brief overview of Risk Analysis as a formal system. It comprises:

1. Risk *assessment* — determining the probability and severity of a hazard
2. Risk *management* — devising a means of dealing with the hazard
3. Risk *communication* — sharing information (in both directions) with the public

Risk assessment: milk and milk products

Hazards from milk may be chemical, microbiological, and possibly physical

- Chemical hazards may be intrinsic to milk (casein [the principal milk protein] as an allergen or lactose [milk sugar] as a substance that some people cannot digest) or extrinsic (e.g., antibiotics used in treating cattle infections or toxic substances from cattle feed).
- Microbiological hazards may come from the udder, the cow's (or other dairy animal's) intestines, the human milker, or the environment on the farm or at the processing facility.

Milk is good for most people most of the time, so its benefits outweigh the risks in most circumstances. Risk assessment tries to determine how likely a hazard is to occur and how severe the consequences will be if it does occur.

- The likelihood that milk will contain casein and lactose is 100%. Fortunately, not very many people are at risk: casein allergies occur principally in children, who often outgrow them, and lactose intolerance usually depends on age and ethnicity, so at-risk persons have an opportunity to learn how to deal with it.
- The likelihood that milk will contain antibiotics or toxins is much less; the US FDA has jurisdiction both as to veterinary antibiotics and what is fed to cattle. Dairymen can ignore the rules, but most of them choose not to, either out of public spirit or fear of the consequences if caught. Humans exposed to antibiotics in milk may have acute allergic reactions, whereas toxins (e.g., aflatoxin) in milk are likely to be at low enough levels that any adverse effect would be from chronic exposure.
- The likelihood that pathogenic microorganisms will occur in the milk of infected animals varies widely and will be discussed in more detail below. Contamination of human and environmental origin is a result of lapses in sanitation; some of these are straightforward, and others are subtle. Human infections from milk bacteria are usually transient, but most have potential life-threatening syndromes or long-term sequelae.

Risk management: milk and milk products

Management is possible for each of the risks described above. Some strategies are based on excluding the risk, others on processing for safety, and still others on truth in labeling.

- Exclusion strategies are based on withdrawal times for antibiotics, feed formulations that avoid toxins, control of *Brucella* and *Mycobacterium* infections, and sanitation to keep other pathogens out during and after milking.
- Processing strategies are based principally on pasteurization to kill other bacterial pathogens that might occur in raw milk. When compulsory pasteurization was introduced in New York City between 1899 and 1910, the infant mortality rate went down from 12 to 3.8 per 1,000. Lactose intolerances can be mitigated by lactase treatment, or by fermentation of milk to make yogurt or cheese.
- Truth in labeling includes indicating that lactose or casein (now to be called “milk sugar” and “milk protein,” apparently) are in a product or (where legal) that milk (fluid, or made into cheese) has not been pasteurized. In the latter case, a great deal turns on the legal definition of pasteurization.

The decision to require these kinds of risk management generally lies with government. How much protection is enough is a perception shared by government and the consuming public.

Risk communication: milk and milk products

One hopes that government, industry, the consuming public, and scientists are all communicating in these matters. Some groups claim to speak for consumers, whereas some trade groups have different perceptions. The U.S. government has chosen to require pasteurization of milk shipped interstate, and most states require pasteurization for milk sold at retail. Exemptions for milk from the “family cow” are common and are often abused. Government has not done as good a job as they might of communicating risks of raw milk, whereby people who regard pasteurization as unnatural are still determinedly putting themselves and their children at risk. Scientists, physicians, and veterinarians should do what they can to communicate such risks, though few in the U.S. today have seen someone with bovine tuberculosis or brucellosis, the worst of the milk-borne diseases.

Specific microbial hazards from milk:

- *Mycobacterium bovis* in cows’ milk was once a commonplace. Those who drank unpasteurized milk from tuberculous cattle were at risk of systemic tuberculosis, which often caused bone deformities such as hunchback. The original time-temperature combinations for legal pasteurization were predicated on killing *M. bovis*. Control of bovine tuberculosis is generally institutional at this time, but outbreaks still occur, as in California in 2002.
- *Brucella abortus* in cows’ milk, and *B. melitensis* in the milk of goats and sheep, were once also common in the U.S. These are capable of causing severe febrile disease in humans, with lasting sequelae. Institutional control has been largely successful, but outbreaks still occur somewhere in the U.S. with some frequency. There is also a continuing concern over *Brucella* spp. in wild ruminants that may have access to dairy species.
- *Coxiella burnetii* occurs in cows’ milk, although most human infections result from aerosol exposures to parturient sheep and goats. The key findings on the incidence and heat resistance of *C. burnetii* that were produced here at UCD led to a slight increase in the temperature for low-temperature, long-time pasteurization of milk. Disease in humans from ingestion with milk is probably milder than that from inhalation.

- *Campylobacter* spp. is not often reported to be associated with cattle, yet outbreaks of human campylobacteriosis have repeatedly resulted from drinking raw cows' milk. Source cattle are generally healthy. Humans experience gastroenteritis, typically lasting a few days. More severe illnesses, as well as arthritic and neurological sequelae, sometimes occur.
- Enterohemorrhagic *E. coli*, usually type O157:H7, has many alternate vehicles, but its transmission via raw milk is well known, as established in session 8. The source cattle are generally healthy, but bloody diarrhea in humans is sometimes followed by kidney failure. *Salmonella* spp. have caused outbreaks when transmitted via raw milk. Although the gastroenteritis only lasts a few days in humans, arthritic sequelae are known.
- *Mycobacterium avium*, supsp. *paratuberculosis* (MAP) is not known surely to be transmissible to humans, in which it is alleged to cause Crohn's disease. It causes a late-onset enteropathy in cattle and may occur in milk. It is alleged to be more heat-stable than *M. bovis*, whereby a small fraction of the bacteria might survive pasteurization.
- Hepatitis A virus does not infect cattle, so it cannot be shed in milk. Infected people may contaminate milk if they milk cattle manually, with fecally contaminated, unwashed hands. Inactivation in pasteurization is likely to be 90 to 99% (i.e., incomplete).
- Prions of bovine spongiform encephalopathy have been intensively sought in the milk of cattle late in the incubation of BSE. None have ever been demonstrated. Despite the extreme unlikelihood of prions' presence, concern for the severity of variant Creutzfeldt-Jakob disease in humans keeps this agent in the limelight. Temperatures of pasteurization surely would not inactivate prions. Further, as pointed out in session 4, some people are apparently not at risk.

Microbial hazards from milk in processing:

- *Staphylococcus aureus* has been known to take over cheese vats in which the starter culture failed. The resulting cheese contained staphylococcal enterotoxin, which can cause severe gastroenteritis. Making "processed" cheese from such product would not apply enough heat to deal with the very stable toxin.
- *Listeria monocytogenes* can occur in raw milk but is not known to have been transmitted by that vehicle. It has caused outbreaks via soft cheeses that seem most likely to have been contaminated because *Listeria* colonized the environment in the cheese plant.
- *Salmonella* spp. has been known to colonize a factory in which non-fat dry milk was "instantized." Dry milk that arrived at the plant without *Salmonella* was contaminated when it left. An outbreak of over 16,000 illnesses apparently resulted from cross contamination of fluid milk in a pasteurization facility.

Milk and milk products are highly nutritious for people, but also for microorganisms. Our Pasteurized Milk Ordinance in the U.S. is intended to guarantee that milk is produced in a clean farm environment, properly pasteurized, and packaged and distributed in sanitary way. Because this is a human activity, mistakes sometimes happen. All the same, the PMO provides a system for managing the risks that are known to be significant. The tendency of some members of the public to circumvent the PMO represents a failure of risk communication.

VMD 413. Veterinary Food Safety — Spring, 2007
Session 13. **Summary and review**

During twelve lectures in ~3 weeks, we have surveyed the U.S. food industry and considered what kinds of foodborne illnesses occur and what can be done to prevent them. In this lecture, we will review the material that has been covered and add a few “afterthoughts.” Questions are appropriate at any time; plan to ask questions about the entire course.

U.S. food industry: production, processing, storage and distribution, food service, retailing.

Foodborne diseases: infections, intoxications, allergies, intolerances, idiopathic illnesses

Incidence, costs

Causes: bacteria, viruses, molds, protozoa, parasites, algae, “chemicals”

Food contamination: from animals, humans, soil, water, air

Strategies for preventing foodborne disease

Preharvest food safety: control at the farm level

HACCP: process control

Food Safety Initiative, 1997: many proposed changes

Regulatory approaches: mandated HACCP, performance standards

“Food security” = counter-terrorism

Microecology of food: life or death of bacteria and molds, based on environmental factors in the food

Food processing: many ways to improve convenience, usefulness, safety

Epilogue: Eating and Enjoying It (**not on final exam**)

“Das Leben ist zu kurz um schlechten Wein zu trinken.”

Bucking “prevailing wisdom”

GLOSSARY

ACUTE—having a relatively sudden onset of symptoms or a rapid clinical course; does not refer to the length of the incubation period or the onset time

AGE-ADJUSTED INCIDENCE—the number of persons contracting a given illness, adjusted for changes in the age distribution of the population over the period in which comparisons are made

ALLERGY—a disease state, caused by exposure to a particular chemical to which certain individuals have a heightened sensitivity (hypersensitivity), that has an immunologic basis

CARCINOGEN—a substance which, when introduced into the body, causes cancer

CARRIER—a person who harbors, and at least occasionally sheds, a pathogen or parasite over prolonged periods of time, usually without displaying evidence of the disease the agent ordinarily causes

CHRONIC—having a relatively gradual onset of symptoms or a prolonged clinical course; again, does not refer to the length of the incubation period or the onset time

CONSUMER—the person who ingests a food product

CONTAMINATION—introduction of foreign microorganisms or disease agents into a foodstuff

CROSS-CONTAMINATION—contamination of one food from another (example: thawing chicken drips *Salmonella* on salad)

DANGER ZONE—the temperature range 40° to 135°F, in which bacterial disease agents can grow in foods

DECONTAMINATION—thermal (or other) food treatment that destroys a contaminant (example: pasteurization of liquid eggs to kill *Salmonella*)

DEPURATION—a process by which molluscs are supposed to cleanse themselves of microbial contaminants; shellfish are held in decontaminated seawater, which they are expected to siphon until all disease agents have been eliminated

DIARRHEA—abnormal frequency and liquidity of fecal discharges

DINOFLAGELLATE—a microscopic, marine algal form which is a major constituent of plankton; some of these produce toxins that are accumulated by molluscs or other seafood species.

DISEASE—any abnormal condition in the body, whether of intrinsic or extrinsic cause, that is perceptible to the host or to a clinician

DYSENTERY—inflammation of the intestines, with frequent stools containing blood and mucus

ENTEROTOXIN—a toxin with specific action on the intestinal lining

FOMES (pl. FOMITES)—an inanimate object that transmits an infectious agent

GOOD MANUFACTURING PRACTICE (in the processing of food)—following well established and prescribed procedures intended to assure a safe and wholesome product

HOST—a living organism (often human) in which a microbe multiplies or a toxin exerts its effect

INCIDENT—(said of foodborne disease) the occurrence of one or more cases of illness in consumers

INCUBATION PERIOD—see "onset time"

INFECTION—a condition in which a microbe or parasite establishes itself and grows or multiplies in the host's body

INTOLERANCE—a disease state caused by a chemical that is toxic to certain individuals only because they exhibit some genetic deficiency

INTOXICATION—an adverse reaction by the body to a foreign (toxic) substance, whether the substance was produced within or outside the body

IRRADIATION—food processes using ionizing radiation (e.g., gamma rays or accelerated electrons) for any of several purposes, including destruction of microbial disease agents

JAUNDICE—a symptom of liver disease characterized by yellowish appearance of the patient

MISHANDLING—treatment of food in such a way as to lead to the introduction into or production of a disease-causing agent; degradation of food quality through improper treatment

NORMAL FLORA—those microorganisms that are usually or unavoidably present in the body of a host or in a food

ONSET TIME (and INCUBATION PERIOD)—the time that elapses between the first introduction of a disease-causing agent into a host's body and the beginning of overt symptoms of illness

OUTBREAK—the occurrence of two or more cases of a disease, associated in time and place so as to suggest a common source

PATHOGEN—agent or factor that causes disease and is therefore said to be pathogenic

PREPARATION (of food)—the final stage(s) of readying a food to be eaten, whether commercially or in the home; usually done in a kitchen

PROCESSING (of food)—treatment of food, usually on a commercial scale, to increase its usefulness, stability, or acceptability

PRODUCTION (of food)—the growing, usually under human supervision, of the basic animal or vegetable material of a food

RECONTAMINATION—contamination after decontamination (example: virus on roast pork)

RESERVOIR—recurrent source of a pathogen

RETAILING (of food)—the display and sale of food in a store for later consumption off the premises

SERVICE (of food)—final preparation and sale or giving of food for consumption on the premises (e.g., in a restaurant or cafeteria) or elsewhere (e.g., a take-out); can also include outdoor group feeding at picnics, etc.

SOURCE (of a contaminant)—where a foodborne disease agent originates (example: human intestines, *Entamoeba histolytica*)

SPORES—inactive or resting form of a bacterium

STORAGE AND DISTRIBUTION (of food)—the holding and transportation of food so that it will be available when and where it is needed, ideally without any alteration in the quality or safety of the product

TEMPERATURE ABUSE—holding of food at a temperature (and for a sufficient time) to allow the multiplication of the pathogens or spoilage organisms that it contains (example: beef gravy with *C. perfringens* spores in it)

VECTOR—a living animal that transports an infectious agent

VEGETATIVE CELLS—the reproductive form of a bacterium

VEHICLE—food or water in which a cause of disease is transmitted to a consumer

VIRULENCE—the capacity of a pathogenic organism to cause disease

ZOONOSIS—an infectious disease that is transmissible from animals to man; the human host is often a "dead end," in that the disease is not transmitted from person to person.