State of Art

Food Safety and Environmental Pollution

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Introduction

Food safety and environmental pollution is an important public health issue, as illness due to contaminated food is perhaps the most widespread health problem worldwide. The recent publicity related to Bovine Spongiform Encephalopathy (BSE) or mad-cow disease in Europe, the discovery of dioxin and other chemical contaminants in chicken and some fish, the use of antibiotics in animal husbandry, the presence of bacteria and the advent of genetically modified organisms have heightened consumer awareness and concerns about food safety.

Globalisation of trade in food, urbanisation, changes in lifestyles, international travel, environmental pollution and natural and manmade disasters are adding to the complexity of food safety **(World Health Organization , 2000),.** For instance, the World Tourism Organization estimated that in 2000 a record 698 million people travelled internationally. Tourist arrivals will top one billion by 2010 and 1.56 billion by 2020 **(World Tourist Organization , 2001)** with huge implications for the spread of food-borne diseases. With increasing international travel many outbreaks of food-borne diseases can rapidly spread across national boundaries.

In order to ensure standards in food safety and quality, the Food and Agricultural Organization and the World Health Organization have developed the Codex Alimentarius (Food and Agricultural Organization and World Health Organization , 1999), as an international reference for health authorities, food control officials, manufacturers, scientists and consumer groups. The Codex Alimentarius addresses standards related to food labelling, food additives, contaminants, methods of analysis and sampling, food hygiene, residues of veterinary drugs in foods, and pesticide residue in foods.

Magnitude of the Problem

Contaminated food causes illness and suffering particularly among children, the elderly and other vulnerable populations. Food-borne diseases also lead to financial losses related to absenteeism from work, expenditures on medical care, costs of investigation of outbreaks and containment, and legal and other expenses **(World Health Organization , 1999)** .

It is difficult to estimate the true extent of food-borne illnesses worldwide. These continue to be a widespread and growing public health problem in developed and developing countries **(World health Organization , 2000).**

-Annually up to 30% of people in developed countries suffer from food-borne diseases, resulting in many hospitalisations and deaths.

-The World Health Organization estimated that in 1998 alone 2.2 million people, including 1.8 million children, died from diarrhoeal diseases, largely attributed to contaminated food and water.

-Developing countries face a growing problem of food-borne diseases including those caused by parasites.

-Food safety problems are the underlying cause of the high prevalence of diarrhoeal diseases and deaths in many developing countries.

Threats to food safety

Threats to food safety involve a complex chain of factors and sources such as environmental and industrial pollution, agricultural practices, food production practices, and even cultural practices such as consumption of raw or under-cooked foods. Threats include:

Microbial hazards

Poor food storage and unhygienic cooking and handling practices are important factors in the spread of microorganisms and food-borne illness. The major food-borne diseases are caused by: *Salmonella, Campylobacter, E-coli, Listeria and Vibrio cholera*. Food-borne diseases caused by microorganisms generally manifest in abdominal pain, fever, vomiting and diarrhoea. The outcome can be fatal unless fluid and electrolytes are replaced.

Chemical hazards

The use of pesticides and chemicals can threaten food safety. Chemical contaminants of food can include toxins such as aflatoxins, Persistent Organic Pollutants (POPs) such as dioxins and polychlorinated bephenyls (PCBs), or metals such as lead and mercury that can have adverse health effects. Chemical contamination of food with tin or fungal toxins can cause acute vomiting. Aflatoxin can increase the incidence of liver cancer. Some pesticides have carcinogenic and mutagenic actions. High levels of mercury contamination of fish

can cause foetal abnormalities and have adverse impact on the nervous system **(World Health Organization , 2001).**

Changes in animal feeding practices, such as the use of infected meat and bone meals in animal feeds, can result in threats to food safety such as the spread of BSE through food.

Antibiotics in animal feeds

Low levels of antibiotics are sometimes added to animal feed in order to prevent and cure illness. By weight, half of all antibiotics are given to livestock and fish in a prophylactic attempt to prevent disease **(World Health Organization , 2000),.** This practice has raised concern about transfer of antibiotic resistance to human pathogens. The problem needs more attention in the face of growing antimicrobial resistance.

Genetically Modified (GM) foods

The safety of GM foods often referred to as "Frankenstein Foods" has become of great concern. Assessment of GM foods should consider health benefits as well as potential negative health consequences. Crops modified to resist pests, foods with allergens removed or food with an increase of essential nutrients are possible examples of health benefits. The safety of GM foods needs to be assessed and findings communicated to the consumers.

Irradiation and Food Safety

Irradiation is a physical treatment of food with high-energy, ionising radiation. It can be used to prolong the shelf life of food products and/or to reduce health hazards associated with certain products due to the presence of pathogenic micro-organisms.

In the United States, foodborne infections cause an estimated 76 million cases of illness and 323,000 hospitalizations annually--more than one in 1 in 1000. The economic burden is substantial, estimated at up to $6.7 billion annually in patient-related costs for treatment of bacterial infections alone (Buzby et al.,). Five pathogens account for much of the most severe illness: *Salmonella*, *E. coli* O157 and other Shiga toxin-producing *E. coli*, *Campylobacter*, *Listeria*, and *Toxoplasma* cause an estimated 3.5 million infections, 33,000 hospitalizations, and 1600 deaths each year **(Mead et al.,).**

These goals include reducing the national incidence of infections with *Salmonella*, *E. coli* O157, *Campylobacter*, and *Listeria* to 50% of their 1997 incidence. Reaching those goals means preventing 50% of foodborne diseases now occurring. This will require new approaches for prevention.

Hazards With Food Irradition

Irradiation damages food by breaking up molecules and creating free radicals. The free radicals kill some bacteria, but they also bounce around in the food, damage vitamins and enzymes, and combine with existing chemicals (like pesticides) in the food to form new chemicals, called unique radiolytic products (URPs) **(US Government Accounting Office 1990).**

Some of these URPs are known toxins (benzene, formaldehyde, lipid peroxides) and some are unique to irradiated foods. Scientists have not studied the long-term effect of these new chemicals in our diet. Therefore, we cannot assume they are safe Irradiated foods can lose 5%-80% of many vitamins (A, C, E, K and B complex). The amount of loss depends on the dose of irradiation and the length of storage time.

Most of the food in the American diet is already approved by the U.S. Food and Drug Administration (FDA) for irradiation: beef, pork, lamb, poultry, wheat, wheat flour, vegetables, fruits, shell eggs, seeds for sprouting, spices, herb teas. (Dairy is already pasteurized). A food industry petition currently before the FDA asks for approval for luncheon meats, salad bar items, sprouts, fresh juices and

frozen foods. Another petition before the USDA asks for approval for imported fruits and vegetables.

Irradiation damages the natural digestive enzymes found in raw foods. This means the body has to work harder to digest them. **(Public Citizen's Critical Mass Energy and Environment Program, 2000).**

Patel F. (2000) investigated the effect of gamma irradiation on lipase activity of the groundnut (peanut) during germination. The dose levels were 10, 30, 50, 70, 90 and 120 Kiloroentgens, which convert to .1, .3, .5, .7, .9 and 1.2 kGrays. These doses range from one-eightieth to one-eighth the maximum dose requested in the Australia/New Zealand application to allow irradiation of oilseeds. The suppression of lipase activity is for almost all doses dose-dependent.The difference in the level and rate of lipase activity between control and irradiated seeds begins at day 6 and continues through the germination period. The authors state "irradiation of 50 kr [.5 kGray] and above induced damage to the active centers [of lipase production]." "In general the growth of irradiated seeds was poor (only epicotyls are developed) and seeds irradiated to higher dosage levels, i.e., 70 kr [.7 kGray] and above did not grow at all," although lipase increased without germination in these seeds. The authors attribute the abnormal timing of lipase production and failure to germinate to irradiation damage to the nucleus and mitochondria. An other study compared irradiated fungus spores and wheat at doses of 0.0, 1.2, 2.6, 4.6, 6.7 and 10.2 kGray for spore viability, wheat usability in baking, and germination. Germination was tested by storing the seeds on germination paper in the dark for 7 days at 20ºC, then planting them in soil in early spring. Table 3 ("germination test," the 'easiest' test) shows that *no* normal germination occurred at any irradiation dose. The percentage of seeds that did not germinate increased from 0.5% (control group)to 6.0% (10.2 kGray).The remaining seeds germinated abnormally **(Sitton ,1995).**

There are many similar studies on deliberately retarding the sprouting of potatoes, the spoilage of oysters, etc.

If unlabeled, raw foods that have been irradiated look like fresh foods, but nutritionally they are like cooked foods, with decreased vitamins and enzymes. The FDA allows these foods to be labeled "fresh."

-Irradiated fats tend to become rancid.

-When high-energy electron beams are used, trace amounts of radioactivity may be created in the food.

-Science has not proved that a long-term diet of irradiated foods is safe for human health.

-The longest human feeding study was 15 weeks. No one knows the long-term effects of a life-long diet that includes foods which will be frequently irradiated, such as meat, chicken, vegetables, fruits, salads, sprouts and juices (Donald Louria,1990).

-There are no studies on the effects of feeding babies or children diets containing irradiated foods, except a very small and controversial study from India that showed health effects.

-Studies on animals fed irradiated foods have shown increased tumors, reproductive failures and kidney damage. Some possible causes are: irradiation-induced vitamin deficiencies, the inactivity of enzymes in the food, DNA damage, and toxic radiolytic products in the food.

-The FDA based its approval of irradiation for poultry on only 5 of 441 animal-feeding studies. Marcia van Gemert, Ph.D., the toxicologist who chaired the FDA committee that approved irradiation, later said, "These studies reviewed in the 1982 literature from the FDA were not adequate by 1982 standards, and are even less accurate by 1993 standards to evaluate the safety of any product, especially a food product such as irradiated food." The 5 studies are not a good basis for approval of irradiation for humans, because they showed health effects on the animals or were conducted using irradiation at lower energies than those the FDA eventually approved **(FDA , 1992).**

-The FDA based its approval of irradiation for fruits and vegetables on a theoretical calculation of the amount of URPs in the diet from one 7.5 oz. serving/day of irradiated food. Considering the different kinds of foods approved for irradiation, this quantity is too small and the calculation is irrelevant.

-Even with current labeling requirements, people cannot avoid eating irradiated food. That means there is no control group, and epidemiologists will never be able to determine if irradiated food has any health effects.

-Science is always changing. The science of today is not the science of tomorrow. The science we have today is not adequate to prove the long-term safety of food irradiation.

-Irradiation covers up the increased fecal contamination that results from speeded up slaughter and decreased federal inspection, both of which allow meat and poultry to be produced more cheaply. Prodded by the industry, the USDA has allowed a transfer of inspection to company inspectors. Where government inspectors remain, they are not allowed to condemn meat and poultry now that they condemned 20 years ago.

-In 2000, seven meat industry associations submitted a petition to USDA to redefine key regulations relating to contamination. If accepted by USDA, this petition would permit unlimited fecal contamination during production, as long as irradiation was used afterwards **(Federal Register ,2000).**

-Labeling a public policy decision unrelated to science--is necessary to inform people so they can choose to avoid irradiated foods.

-Because irradiated foods have not been proven safe for human health in the long term, prominent, conspicuous and truthful labels are necessary for all irradiated foods. Consumers should be able to easily determine if their food has been irradiated. Labels should also be required for irradiated ingredients of compound foods, and for restaurant and institutional foods.

-Because irradiation can deplete vitamins, labels should state the amount of vitamin loss after irradiation, especially for fresh foods that are usually eaten fresh. Consumers have the right to know if they are buying nutritionally impaired foods.

-Current US labels are not sufficient to enable consumers to avoid irradiated food. Foods are labeled only to the first purchaser. Irradiated spices, herb teas and supplement ingredients, foods that are served in restaurants, schools, etc., or receive further processing, do not bear consumer labels. Labels are required only for foods sold whole (like a piece of fruit) or irradiated in the package (like chicken breasts). A radura is required. The text with the declaration of irradiation can be as small as the type face on the ingredient label. The US Department of Agriculture requirements have one difference: irradiated meat or poultry that is part of another food (like a tv dinner) must be disclosed on the label **(FDA and USDA regulations,1999).**

Electron- beam irradiation:

Electron- beam irradiation today means nuclear irradiation tomorrow.

-Many foods cannot be irradiated using electron beams. E-beams only penetrate 1-1.5 inches on each side, and are suitable only for flat, evenly sized foods like patties. Large fruits, foods in boxes, and irregularly shaped foods must be irradiated using x-rays or gamma rays from nuclear materials.

-Countries that lack a cheap and reliable source of electricity for e-beams use nuclear materials. Opening U.S. markets to irradiated food encourages the spread of nuclear irradiation worldwide**(David ,1990)**

Irradiation using radioactive materials is an environmental hazard. The more nuclear irradiators, the more likelihood of a serious accident in transport, operation or disposal of the nuclear materials.

Food irradiation facilities have already contaminated the environment. For example, in the state of Georgia in 1988, radioactive water escaped from an irradiation facility. The taxpayers were stuck with $47 million in cleanup costs. Radioactivity was tracked into cars and homes. In Hawaii in 1967 and New Jersey in 1982, radioactive water was flushed into the public sewer system **(Pacific National Laboratories ,1999)**

People may become more careless about sanitation if irradiation is widely used. Irradiation doesn't kill all the bacteria in a food. In a few hours at room temperature, the bacteria remaining in meat or poultry after irradiation can multiply to the level existing before irradiation.

Some bacteria, like the one that causes botulism, as well as viruses and prions (which are believed to cause Mad Cow Disease) are not killed by current doses of irradiation.

Irradiation encourages food producers to cut corners on sanitation, because they can 'clean up' the food just before it is shipped **(Jensen ,2000).**

Irradiation does nothing to change the way food is grown and produced.

Irradiated foods can have longer shelf lives than nonirradiated foods, which means they can be shipped further while appearing 'fresh.' Food grown by giant farms far away may last longer than nonirradiated, locally grown food, even if it is inferior in nutrition and taste. Thus, irradiation encourages centralization and hurts small farmers.

The application of ionizing irradiation (e.g. gamma rays from 60 Co or 137 Cs) to raw fruits and vegetables is a means of extending shelf-life **(Thayer *et al.*, 1996).** Decay caused by indigenous microflora and post-handling contaminants

can be eliminated or delayed by dose levels that do not adversely affect the sensory qualities of many fruits and vegetables. Dose levels of 1 to 3 kGy, depending upon the type of fruit or vegetable, are sufficient to kill large numbers of most moulds, yeasts and bacteria naturally present on produce as it is taken from the field **(Farkas, 1997).**

conditions of fruits and vegetables, and environmental conditions (e.g. relative humidity and temperature) surrounding fruits and vegetables at the time of treatment. Extensive studies to determine the efficacy of irradiation treatment in disinfestation and abating post-harvest fungal diseases have been reviewed . *Botrytis*, *Rhizopus* and *Mucor* species are among the major spoilage moulds of raw fruits and vegetables, and are inactivated by dose levels of 1.2 to 3.0 kGy. A dose of 3 kGy, for example, will extend the shelf-life of strawberries stored at 5°C by about 1 week . Guidelines for disinfestation and quarantine treatment of raw fruits and vegetables have been issued The effectiveness of ionizing irradiation in killing microorganisms capable of causing human illness has been studied but most investigations have been done using *in vitro* conditions or foods of animal origin **(Monk *et al.*, 1995).** Vegetative cells of pathogenic bacteria are sensitive to irradiation, D 10 values being in the range of 0.2-0.8 kGy, depending on environmental conditions. Foodborne parasites are also generally sensitive to irradiation. With the exception of some nematodes such as *Aniostrongylus cantonensis*, *Gnatostoma spinigerum* and *Anisakis* sp., a dose below 1 kGy is sufficient to control infection caused by protozoa, trematodes and cestodes **(Loaharanu and Murrell 1994).** Higher doses are required, however, for inactivation of viruses. D 10 values for inactivating viruses may be more than 10-fold higher. Therefore, irradiation cannot be used for inactivation of viruses. Nevertheless, ionizing irradiation could be an extremely effective tool in reducing populations of pathogenic microorganisms and parasites from the surface of raw fruits and vegetables. Its application on a large scale for the purpose of reducing the risk of disease associated with the consumption of potentially contaminated raw fruits and vegetables would appear to have exceptional merit. There is a need, however, to evaluate the tolerance of most fruits and vegetables to the radiation doses required for controlling various Factors influencing lethality include the level of sensitivity of the target insect or microorganism(s) to irradiation, post-harvest and pretreatment pathogenic microorganisms.

Prohl et al., (2002) evacuated about 50,000 people from the settlements in the 30-km zone around the reactor in the period 3-11 d after the accident. As no countermeasures were implemented in the early phase, people continued to consume milk and some leafy vegetables. In this paper, average effective ingestion doses are modeled for evacuees. Input data for the assessment are the 137Cs activity per unit area, the ratios of the radionuclides relative to 137Cs, the mean day of evacuation, and intake rates for milk and green vegetables. The transfer of radionuclides from deposition to humans is estimated by modeling radionuclide interception by vegetation, weathering, and the time-dependent transfer of radionuclides to milk taking into account site-specific agricultural practices. Depending on the evacuation day and site, the estimated ingestion doses for the settlements are in the range of 20 to 1,300 mSv and 3 to 180 mSv for infants and adults, respectively. 131I is by far the most important isotope, the ingestion dose due to 133I is more than one order of magnitude lower. The most exposed organ is the thyroid, inducing more than 80% and 50% of the ingestion dose for infants and adults. The ingestion doses are compared to the doses due to inhalation and external exposure. The internal dose exceeds the external by a factor of about 2-10 for adults and 2-40 for 1-y-old infants depending on site and evacuation day. The thyroid doses assessed for the evacuees are consistent with results achieved in studies performed in areas outside the 30-km zone.

Nuclear Worker hazards:

In the Spheres of Influence article in the July issue of *EHP*, Parascandola (2002) presented our concerns about the validity of extrapolating cancer risks from studies of A-bomb survivors to nuclear workers as a matter of differences in dose rate between the two populations. Our primary critiques of using A-bomb data, however, concern biases that arise from selective survival and dose misclassification **(Wing et al. 1999)**, issues that are not mentioned. **Stewart (1985, 1997, 2000)** presented evidence of dose- and age-related selective survival in the Japanese cohort assembled for cancer studies 5 years after the nuclear bombing of Hiroshima and Nagasaki. This evidence is in concordance with basic biological principles of heterogeneity in susceptibility and may help explain the inability of A-bomb survivor studies to detect the impacts of *in utero* radiation exposures on childhood cancers, effects that have been demonstrated

repeatedly in low dose studies **(Doll and Wakeford 1997)**. Age-related selective survival also helps to explain the reported decrease in radiation-cancer dose response among A-bomb survivors exposed at older ages, an observation that deviates from expectations based on the increased sensitivity of older adults to other physical, chemical, and biological agents, evidence of age-related decline in DNA repair capacity, and evidence from some studies of nuclear workers **(Richardson et al. 2001; Wing 2000)**.

Epidemiologic studies depend on accurate exposure classification for valid dose-response estimation. In addition to selective survival in a population subjected to nuclear attack and subsequent devastation of public health infrastructure, radiation-cancer dose-response estimates from A-bomb studies are further affected by a lack of individual dose measurements and the use of dose reconstruction based on interviews conducted in an occupied nation by a scientific team funded and directed by the U.S. government **(Wing et al. 1999)**. The ability to elicit accurate information on location, position, and shielding was affected not only by traumatization of the survivors and their domestic stigmatization but by their distrust of medical teams working under occupation forces **(Lindee 1994)**.

As Parascandola (2002) noted, we believe that findings from carefully conducted epidemiologic studies of badge-monitored nuclear workers exposed to chronic, low-level ionizing radiation should be considered in implementation of the Energy Employees Occupational Illness Compensation Program Act. Medical practices regarding exposures of pregnant women to diagnostic X rays were changed decades ago on the basis of low-dose studies, even though their findings were not predicted from studies of A-bomb survivors. The question today is, will A-bomb studies continue to dictate estimates of cancer risks in adulthood, despite evidence of bias and the availability of alternative epidemiologic data? The large number of highly exposed survivors in the study, cited as a major strength, may actually be a weakness if it encourages scientists and policy makers to confuse statistical precision with valid dose-response estimates that depend on an absence of selective survival and correct exposure classification.

Pathogenic Microorganisms

A wide variety of microorganisms can cause foodborne illnesses. These illnesses range from relatively mild, self-limiting gastroenteric infections to severe, sometimes lethal, infections and intoxications. For example, enteric bacteria, such as pathogenic *E coli* and *Salmonella*, can cause a variety of infections that are sometimes lethal **(Reed , 1993 and Reed , 1994).** Certain bacteria (eg, *Staphylococcus aureus* and *Bacillus cereus*) produce toxins in food that cause self-limiting illness relatively soon after the food is ingested . In contrast, the neurotoxins produced by *Clostridium botulinum* act more slowly but cause an illness with a high mortality rate if not promptly treated .Toxigenic molds can produce toxins in certain foods, and several types of human viruses, most notably hepatitis A, can be transmitted in food.

Sourses of microorganisms :

They are widely distributed in our environment. Some common living places of microbes are air, water, soil, plants, animals, human beings, sewage, fertilizer, food ingredients, processing equipment, and packaging materials. The microbes found in food include those associated with the raw material, those acquired during harvesting, handling, and processing, and those surviving any preservation treatment and storage**(Marriot, 1994)** . Food handlers can transmit microorganisms. In fact, humans are the major source of food contamination **(Banwart , 1989).** Their hands, hair, breath as well as their unguarded coughs and sneezes can contaminate food.

Microorganisms Hazards

Microorganisms may have different functions in food. They may remain inert, serve a useful function, cause spoilage, or become a health hazard **(Barckett ,1994).** Useful microbes are those which produce desirable changes in food, such as converting milk to cheese, grape juice to wine, and cabbage to sauerkraut. Spoilage microbes are those that cause deterioration which is manifested by alterations in the appearance, texture, odor, or flavor of the food. Pathogenic microbes are those toxigenic and invasive microorganisms which can multiply in food and/or the human body and cause foodborne illness.

Pathogens of most concern:

Salonella :

The antigenic scheme for classifying salmonellae recognizes more than 2300 serovars and, while all can be considered human pathogens, only about 200 are associated with human illness.

Animal husbandry practices used in the poultry, meat and fish industries, and the recycling of offal and inedible raw materials into animal feeds, has favoured the continued prominence of *Salmonella* in the global food chain **(D'Aoust, 1997).** Hygienic conditions during the production, harvesting, transport and distribution of raw fruits and vegetables from some countries may not always meet minimum hygienic requirements, thus facilitating contamination on arrival in another country. Application of night soil, untreated sewage sludge or effluents, or irrigation water containing untreated sewage to fields and gardens can result in contamination of fruits and vegetables with *Salmonella* and other pathogens. Washing fruits and vegetables with contaminated water and handling of produce by infected workers, vendors and consumers in the marketplace helps the spread of pathogenic microorganisms, including *Salmonella* **Potenski et al., (2003).**

Salmonellae have been isolated from many types of raw fruits and vegetables (Wells and Butterfield, 1997). Outbreaks of salmonellosis have been linked to a diversity of fruits and vegetables, including tomatoes. The pathogen can grow on the surface of alfalfa sprouts (Jaquette *et al.*, 1996), tomatoes and perhaps on other mature raw fruits and vegetables, making it imperative to use hygienic practices when handling them.

Escherichia coli:

Escherichia coli is common in the normal microflora of the intestinal tracts of humans and other warm-blooded animals. Strains that cause diarrhoeal illness are categorized into groups on the basis of virulence properties, mechanisms of pathogenicity, clinical syndromes and antigenic characteristics. The major groups are designated as enterotoxigenic, enterohaemorrhagic, enteropathogenic, enteroinvasive, diffuse-adhering and enteroaggregative **(Doyle *et al.*, 1997).**

All foods can become contaminated with one or more of these groups while in the field or during post-harvest handling. Sources and mechanisms of contamination are similar to those described for *Salmonella* and *Shigella*.

Enterotoxigenic *E. coli* is a cause of traveller's diarrhoea, an illness sometimes experienced when individuals visit countries with food and water hygiene standards different from their own **(Hussein and Omaye, 2003).**

Campylobacter:

Campylobacter jejuni is a leading cause of bacterial enteritis in many countries. Reservoirs of this pathogen include several wild animals as well as poultry, cows, pigs and domestic pets (Nachamkin, 1997). While consumption of food of animal origin, particularly poultry, is largely responsible for infection, *Campylobacter* enteritis has also been associated with the consumption of raw fruits and vegetables (Harris 1986). Although *Campylobacter* does not grow at temperatures below 30 °C and is sensitive to acid pH, it can survive on cut fruits for sufficient time to be a risk to the consumer (Castillo and Escartin, 1994).

Listeria monocytogenes :

Listeria monocytogenes is present in the intestinal tract of many animals, including humans, so it is not surprising that the organism can also be found in the faeces of these animals, on the land they occupy, in sewage, in soils to which raw sewage is applied and on plants which grow in these soils. The organism also exists in nature as a saprophyte, growing on decaying plant materials, so its presence on raw fruits and vegetables is not rare . Surveys of fresh produce have revealed its presence on cabbage, cucumbers, potatoes and radishes in the United States and Canada **(Odumeru *et al.*, 1997),** tomatoes and cucumbers in Pakistan, and bean sprouts, sliced cucumbers and leafy vegetables in Malaysia **(Schuenzel and Harrison 2002)** .

Staphylococcus aureus:

Staphylococcus aureus is known to be carried in the nasal passages of healthy food handlers and has been detected on raw produce **(Abdelnoor *et al.*, 1983)** and ready-to-eat vegetable salads **(Houang *et al.*, 1991).** However, enterotoxigenic *S. aureus* does not compete well with other microorganisms normally present on raw fruits and vegetables, so spoilage caused by nonpathogenic microflora would probably precede the development of the high

populations of this pathogen that would be needed for production of staphylococcal enterotoxin.

***Clostridium* species:**

Spores of *Clostridium botulinum* and *Clostridium perfringens* can be found both in soil and on raw fruits and vegetables. The high rate of respiration of salad vegetables can create an anaerobic environment in film- wrapped packages, thus favouring the growth of *C. botulinum* and botulinal toxin production. Botulism has been linked to coleslaw prepared from packaged, shredded cabbage and chopped garlic in oil **(Ader and Beuchat , 2002).** Studies have revealed that *C. botulinum* can produce toxin in polyvinyl film-packaged and vacuum-packaged mushrooms **(Malizio and Johnson, 1991)**. It is important that the permeability characteristics of packaging films minimize the possibility of development of anaerobic conditions suitable for outgrowth of clostridial spores. Recognizing that anaerobic pockets may develop in tightly packed produce, even when films have high rates of oxygen and carbon dioxide permeability, an additional measure to prevent growth of *C. botulinum* is to store produce at less than 3°C.

Bacillus cereus:

Spores of enterotoxigenic strains of *Bacillus cereus* are common in most types of soil. Some strains can grow at refrigeration temperatures. Foods other than raw fruits and vegetables are generally linked to illness implicating *B. cereus*. Illness associated with eating contaminated soy, mustard and cress sprouts has, however, been documented **(Portnoy *et al.*, 1976)**. Human illness tends to be restricted to self-limiting diarrhoea (enterotoxin) or vomiting (emetic toxin). However, emetic toxin-producing strains have produced liver failure and death by the foodborne route.

***Vibrio* species:**

Vibrio species are generally the predominant bacterial species in estuarine waters and are therefore associated with a great variety of fish and seafoods. There are 12 human pathogenic Vibrio species, of which Vibrio cholerae, V. parahaemolyticus and V. vulnificus are of greatest concern **(Oliver and Kaper, 1997).** *Vibrio cholerae* is the causative agent of cholera, one of the few foodborne diseases with epidemic and pandemic potential. Carriage of the

organism by infected humans is important in transmission of disease. Water can become contaminated by raw sewage.

Ingestion of water containing *V. cholerae* or of foods that are washed with contaminated water but not disinfected can lead to widespread transmission of cholera (Mintz *et al.*, 1994). An outbreak of cholera linked to the consumption of unpasteurized coconut milk has been documented **(Taylor *et al., 1993).***

Viruses:

Viruses can be excreted in large numbers by infected individuals. Although viruses will not grow in or on foods, raw fruits and vegetables may serve as vehicles for infection. Many food-associated outbreaks of hepatitis A have been recorded. In most instances, these outbreaks have not appeared to depend on the stability of the virus in the food.

Mattick et al., (2003) determined the microbiological quality of washing-up water and the environment in domestic and commercial kitchens. Chicken meals were prepared by people without food safety training in their own kitchen (n = 52) or by trained staff in a commercial kitchen (n = 10). Study participants then washed-up, cleaned the kitchen and completed a food hygiene questionnaire. The temperature and microbiological quality of the washing-up water, and the presence of pathogens in dishcloths, tea towels and other kitchen samples was determined. Of the raw chickens used in meal preparation, 96 and 13% were naturally contaminated with Campylobacter or Salmonella spp., respectively. In domestic kitchens, two of 45 sponges, dishcloths or scourers and one of 32 hand- or tea towels were contaminated with Campylobacter after washing-up and cleaning but none of the tap or sink swabs yielded pathogens. The mean washing-up water temperature in the domestic kitchens was 40.7 degrees C, whereas in the commercial kitchen it was 44.7 degrees C (P = 0.04). Study participants who used hotter water (>/=40 degrees C) had lower levels of bacteria in their washing-up water. The aerobic plate counts of the washing-up water samples in domestic homes were usually between 105 and 106 CFU ml-1 but those associated with the commercial kitchen were consistently lower (P = 0.01). Despite this, Campylobacter was detected in one of 10 washing-up water samples from the commercial kitchen but in none of the samples from domestic kitchens. Pathogenic microorganisms can be recovered relatively frequently from the kitchen environment.

Hussein and Omaye (2003)showed that verotoxin-producing Escherichia coli (VTEC) have emerged in the past two decades as food-borne pathogens that can cause major outbreaks of human illnesses worldwide. The number of outbreaks has increased in recent years due to changes in food production and processing systems, eating habits, microbial adaptation, and methods of VTEC transmission. The human illnesses range from mild diarrhea to hemolytic uremic syndrome (HUS) that can lead to death. The VTEC outbreaks have been attributed to O157:H7 and non-O157:H7serotypes of E. coli.

Potenski et al,.(2003) studied Mutants of Salmonella Enteritidis selected following exposure to the sanitizer chlorine or to the preservatives sodium nitrite, sodium benzoate or acetic acid show resistance to multiple antibiotics (tetracycline, chloramphenicol, nalidixic acid, and ciprofloxacin). Complementation experiments with a functional marR restored antibiotic susceptibility of selected mutants to levels similar to wild-type strains, suggesting that mar mutation was responsible for resistance.

Wilson (2002) Between 1995 and 2000, investigated the levels of contamination of raw retail chickens (n = 1,127) with salmonella and campylobacter. The levels of contamination over the 6-year period were 11 % (95 % CI +/- 6.5%) for salmonella, and 57% (95% CI +/- 95%) for campylobacter. S. Bredeney (20%) and S. Enteritidis (18%) were the dominant serovars. Although salmonella contamination was higher than in an earlier survey we conducted (7%), since 1998 it has declined to 6%. Many S. Enteritidis isolates (43%) were associated with one large integrated poultry organization that appears to have successfully managed the contamination, and the serovar has not been isolated since 1998. Contamination ranged from 0 to 44% between different producers. There was no significant difference between producers contributing large and small numbers of samples, although some small producers had much poorer contamination rates than others. S. Bareilly, S. Bredeney, S. Enteritidis and S. Virchow showed associations with particular producers. Campylobacter contamination remains high. Contamination ranged from 47 to 81% between different producers..

Garlic is known to have antimicrobial activity against several spoilage and pathogenic bacteria. However, the fate of Salmonella, Escherichia coli O157:H7, and Listeria monocytogenes in garlic butter has not been reported. This study

was undertaken to determine the viability of these organisms in garlic butter as affected by the type of raw minced garlic added to the butter, storage temperature, and storage time. Unsalted butter at 40 degrees C was combined with raw minced jumbo, elephant, or small-cloved garlic at a 4:1 butter/garlic ratio (wt/wt), inoculated with mixed-strain suspensions of Salmonella, E. coli O157:H7, or L monocytogenes, and stored at 4.4, 21, or 37 degrees C for up to 48 h. All pathogens retained their viability at 4.4 degrees C, regardless of the presence of garlic. The addition of garlic to butter enhanced the rates of inactivation of all three pathogens at 21 and 37 degrees C. The most rapid decline in pathogen populations was observed at 37 degrees C. The inactivation of L. monocytogenes occurred more slowly than did that of Salmonella or E. coli O157:H7. The inactivation of Salmonella and L. monocytogenes was more rapid in jumbo garlic butter than in elephant or small-cloved garlic butter. It is concluded that Salmonella, E. coli O157:H7, and L. monocytogenes did not grow in unsalted butter, with or without garlic added (20%, wt/wt), when inoculated products were stored at 4.4, 21, and 37 degrees C for up to 48 h. **(Adler and Beuchat, 2002)**

Girones (2002) defined and studied,shellfish-growing areas from diverse countries in the north and south of Europe and the microbiological quality of the shellfish was analyzed. Human adenovirus, Norwalk-like virus, and enterovirus were identified as contaminants of shellfish in all the participating countries. Hepatitis A virus was also isolated in all areas except Sweden. The seasonal distribution of viral contamination was also described. Norwalk-like virus appeared to be the only group of viruses that demonstrated seasonal variation, with lower concentrations occurring during warm months. The depuration treatments currently applied were shown to be adequate for reducing Escherichia coli levels but ineffective for the elimination of viral particles. The human adenoviruses detected by PCR correlate with the presence of other human viruses and could be useful as a molecular index of viral contamination in shellfish.

How to control microorganisms?

Four basic systems are often used to control microbes in food. They are (1) prevent contamination; (2) remove contaminants; (3) inhibit growth; and (4)

destroy contaminates[1]. In most cases, two or more of these systems are used in combination to control the microbial level in food. For example, optimum quality and shelf-life of fresh-squeezed citrus juice can be achieved by (1) good hygiene and sanitation practices to prevent contamination; (2) effective sorting, washing, sanitizing, and extraction to remove fruit surface microbes; and (3) proper refrigeration to inhibit the growth of microbes during storage, distribution .By identifying factors that affect the number of microorganisms in washing-up water and the kitchen environment, evidence-based recommendations on implementing domestic food hygiene can be made.

The first decade of the 21st century will be faced with continuing dilemmas posed by emerging diseases and will need to provide solutions through the balancing of science, education, and politics **(Bryan , 2002).**

Antibiotic Resistance

Antibiotic resistance is the ability of a bacterial cell to resist the harmful effect of an antibiotic. This resistance may be represented by several different systems, and a given bacterial cell may have one or more of these systems available.

*The bacterium may have a system that prevents entry of the antibiotic into the cell.

*The bacterium may have a system that destroys the antibiotic if the antibiotic gains entry into the cell.

*The bacterium may have a system that associates with the antibiotic inside the cell and therefore blocks the action of the antibiotic.

*-The bacterium may have a system that pumps the antibiotic back out of the cell before the antibiotic can act within the cell. Low levels of antibiotics are sometimes added to animal feed in order to prevent and cure illness. By weight, half of all antibiotics are given to livestock and fish in a prophylactic attempt to prevent disease **(World Health Organization , 2000),.** This practice has raised concern about transfer of antibiotic resistance to human pathogens. The problem needs more attention in the face of growing antimicrobial resistance.

There is growing concern over the consequences of over-use of antibiotics in livestock operations. Persistent use of antibiotics leads to the development of resistance in bacterial populations. Once a particular type of bacteria has developed resistance to an antibiotic, that antibiotic can no longer be used to combat the infectious organism.

In livestock industries, farm operators not only treat their animals with antibiotics for disease, but they also add antibiotics to the feed to promote growth. This long-term overuse of antibiotics in livestock production is now contributing to the development of resistant pathogens. This poses a problem for managing animal health, and it also may impact human health-antibiotic resistance can be passed between different types of bacteria and may therefore create resistance to antibiotics that humans depend on.

The US produces approximately 50 million pounds of antibiotics each year and 40% of that is given to animals, usually as a feed additive to promote growth. **(Grady and Denise , 1999)** More and more evidence shows, however,

that infectious bacteria are quickly developing resistance to even the newest, most powerful antibiotics.

Antibiotic Hazards:

Researchers have published disturbing reports that antibiotic resistance in Salmonella and Campylobacter, two human pathogens, is on the rise **(Grady , 1999)** and evidence is mounting that these resistant bacteria can be passed from chickens and pigs to humans through the food chain **(Seyfarth , 1997).** This poses a great health risk to the human population because it makes it easier for humans to become infected with resistant pathogens for which there are few effective treatment options.

It takes years to develop, test and gain approval for new antibiotic drugs. So while pharmaceutical companies are slowly developing potent new classes of antibiotics, resistance is developing at a rate faster than the drug companies can develop replacements. For example, within the last few years there has been an emergence of bacteria resistant to vancomycin-a last defense drug for some illnesses, including deadly blood infections and pneumonia caused by Staphylococcus bacteria **(Smith , 1999)**-and there is evidence that resistant bacteria may have been passed to humans in the meat products from livestock who were fed a similar antibiotic for growth purposes. Likewise, a rise in antibiotic-resistant Campylobacter infections in humans has occurred in conjunction with the increased use of new classes of antibiotics such as the fluoroquinolones in animal production.

As early as 1969, policy makers in other countries were calling for an end to the use of certain antibiotics as growth promoters in livestock (Witte , 1998) . In 1997, the World Health Organization issued a report re-emphasizing those recommendations, yet livestock regulatory agencies failed to respond. In January 1999 the US Food and Drug Administration (FDA) proposed a policy for addressing the growing concern over antibiotic use in food animals. Unfortunately, the proposed framework was weak on two key points:

*It focused mainly on evaluating new drug approvals while ignoring the millions of pounds of approved antibiotics that are already used for livestock production on a regular basis.

*It did not sufficiently address the risk of antibiotic resistance.

The FDA proposed a category and ranking system for antibiotics based in part on each drug's relative importance in human medicine. The most important drugs are those which treat serious diseases in humans and for which there is no alternative cure-these are listed as "Category 1" antibiotics. However, the FDA's proposal would allow even some Category 1 antibiotics to be used in livestock as long as the level of resistance that develops does not exceed a given "threshold" level **(National Research Council ,1999)** Many people fear that even a limited use of Class 1 antibiotics will increase the chance that bacteria will develop resistance. When that happens, it may be too late to preserve the effectiveness of these important life-saving drugs in human medicine.

As an alternative, the National Research Council has reported that adopting simple production changes such as lowering stocking densities (less overcrowding), controlling stress, and improving hygiene could reduce the need for antibiotics without affecting output. And now a broad coalition of environmental, farm, and public health groups are endorsing a new bill introduced to the US Congress (H.R. 3266) which would limit the sub-therapeutic use of certain antibiotics in livestock **(World Health Organization ,1997)**

Antibiotic resistance and the intestinal flora :

Academic medicine breeds resistant microbes - Microbial resistance to antibiotics is a growing threat to patients. Academic medicine is breeding a destructive device, the hospital strain. A dangerous microbe, resistant to all antibiotics, that evolves in a Darwinian fashion **(Zajicek , 1993)** . Microbe breeding does not differ from cattle breeding, where the best parents are selected for mating. Here, antibiotic treatment selects parents that endow their progeny with antibiotic resistance. Microbe breeding is unintended and unwanted and can be averted only by a total ban on antibiotics, which is unthinkable since medicine and our society are addicted to antibiotics. The hospital strain highlights an inadequacy of academic medicine, total subjection to technology. By breeding hospital strains, academic medicine is dangerous to patients. The threat may, however, be alleviated in several ways: by boosting our resistance to microbes, e.g., vaccination, by relying more on the wisdom of our body in handling microbes **(Zajicek ,1994)** and by mobilizing our own flora.

Pasteur and other microbe hunters taught us that microbes are enemies that should be destroyed. Yet these tiny creatures are responsible for our existence. They appeared on earth one billion years after its formation. For 3.5 billion years they prepared our planet for the evolution of all life-forms **(Atlas and Bartha , 1993)**. Their most important achievement was the maintenance of oxygen and carbon dioxide in the atmosphere, without which life would not have evolved. Today they are the first and most important link in the food chain of our planet. Solar energy trapped by plants is converted mainly into cellulose, that is indigestible by us, since we lack cellulose-degrading enzymes. Cellulose is degraded solely by microorganisms. **The intestinal eco-system** - Microbes are everywhere, and inhabit our skin and intestine. The newborn baby receives its microbial flora from its mother, that already infects it at birth. Bifido-bacterium and Lactobacilli are the first to colonize its gastrointestinal tract. Then come the facultative anaerobes, such as Escherichia coli and Streptococcus fecalis, that are followed by strictly anaerobic bacteria, e.g., Bacteroides. At the time of weaning, populations of obligate anaerobes become dominant. The development of our personal microbial community, known as primary succession, involves an ordered sequential change in the microbial populations of the community. Succession ends when the community consists of up to 400 different species, and attains homeostasis. It is stable, resists any change in its content, and repels unwelcome microbes like the hospital strain.

Eco-system evolution is a complex process that repeats itself in every growing child. By infecting the baby, its mother transmits to it the best microbial populations that she has gathered. It is a vertical inheritance of a protecting eco-system that is adapted later to the needs of the adult. In the intestine, anaerobic bacteria outnumber aerobic bacteria by 100:1 or 1,000:1 (Wells et al., 1988). Since anaerobes preferentially colonize the intestinal mucous layer, they might hinder aerobes from accessing the mucous layer and crossing it.

Antibiotics induce an ecological catastrophe in the intestine - Antibiotics disrupt our stable ecosystem, that either adapts to the trauma or is invaded by resistant microbes. It is an ecological catastrophe like water pollution. Changes observed in the "ailing" ecosystems are called secondary succession. Clinically, this catastrophe is manifested by "antibiotic associated-diarrhea", a pseudo-membranous colitis caused by Clostridium difficile **(Roffe , 1996)** . Its

overgrowth is also induced by combination chemotherapy, and radiotherapy of the lower abdomen. In 20% of treated patients diarrhea may relapse several times, particularly following vancomycin, and metronidazole treatment. Treatment, called bacteriotherapy, or biotherapy, attempts to restore the original eco-system. This idea was raised last century, by Metchnikov, who noted that lactobacilli inhibit the growth of putrefactive organisms. He concluded that fermented milk, e.g., yogurt, kefir, or buttermilk, might be good for diarrhea. Today, live bacteria, e.g., L. acidophilus and L. bulgaricus, in dried form, are given as granules or packed in capsules. Their effect was examined in several clinical trials (6). Other treatments include enema of mixed microbe populations, and biotherapy with yeast.

Apparently some microorganisms may activate heterocyclic amines, and make them carcinogenic . If so, by preventing their access to the intestine, our eco-system might also protect us against cancer. This could be also the rationale for diets rich in fibers. These examples illustrate the importance of our microflora, a yet neglected organ, that waits to be explored **(Bocci , 1999)** .

Another concers of Pathogenic resistance:

Mary et al., (2003) studied the use of antimicrobial growth promoters in Danish food animal production which was discontinued in 1998. Contrary to concerns that pathogen load would increase; they found a significant decrease in *Salmonella* in broilers, swine, pork, and chicken meat and no change in the prevalence of *Campylobacter* in broilers. Antimicrobial growth promoters are antimicrobial drugs added to animal feed to enhance growth and improve feed efficiency of food animals. In the United States, the use of antimicrobial growth promoters also includes elements of prophylaxis, which are not allowed in Europe. Antimicrobial growth promoters have been widely used in Danish food animal production since the 1970s. On February 15, 1998, the Danish cattle and broiler industries, reacting to consumer concerns over food safety, voluntarily stopped the use of all antimicrobial growth promoters. The pig industry stopped using the growth promoters in pigs over 35 kg; all use was phased out in 1999.

Despite concerns that no longer using the growth promoters would have a wide range of negative effects (e.g., an increase in disease and death, poor growth rates, increased feed consumption, increased fecal shedding, and

enhanced shedding or carriage of foodborne pathogens such as *Salmonella* or *Campylobacter*), producers in the broiler and pig industries discontinued the use. Studies have shown that antimicrobial drugs reduce part of the intestinal flora while potentially decreasing pathogen shedding **(Girard et al., 1976 and Jarolman , 1976)**). For these reasons, producers believed that removing antimicrobial growth promoters could cause human pathogenic intestinal bacteria in food animals to increase. Another concern was that increased fecal shedding could lead to contamination of carcasses at slaughter and increased risk for foodborne infection in humans.

Contrary to concerns that withdrawal of antimicrobial growth promoters would cause an increase in pathogen load, we found a decrease in *Salmonella* prevalence in broilers, chicken, swine, and pork and no change in the prevalence of *Campylobacter* in broilers. Previous studies on this topic have shown mixed results. Two observational studies found that penicillin given to swine increased total bacterial and *Enterobacteriaceae* counts. Other experiments found that avoparcin increased *Salmonella* shedding in broilers and excretion rates had a dose-response effect with increasing concentrations of avoparcin **(Barrow et al., 1989)** . A series of experiments in broilers showed that avoparcin, nitrovin, tylosin, flavomycin, and lincomycin caused increased shedding of *Salmonella* in most experiments, while virginiamycin and bacitracin had little or no effect and sodium arsenilate decreased shedding . Holmberg et al. found that both avoparcin and monensin reduced shedding of *S.* Infantis in broilers but a combination of the two increased shedding . Bolder et al. showed that flavophospholipol and salinomycin decreased *Salmonella* shedding in broilers but had no significant effect on the shedding of *C. jejuni* **(Bolder et al., 1999).**

This study is unique because it included a large sample of animals under natural conditions, included both animals and products, and examined the combined effect of many antimicrobial growth promoters. However, several factors should be kept in mind when interpreting the results. First, our analysis cannot elucidate the impact that withdrawal of an individual antimicrobial growth promoter had on a particular pathogen or in a particular species. In addition, since avoparcin was withdrawn in 1995, any immediate effects seen from its discontinued use will be demonstrated in P1 of our study instead of P2. Despite a change in sampling methods for broilers in June 2000 and swine herds in July

2001, these data were included in analysis. Both changes increased the sensitivity of sampling, in theory leading to a higher prevalence. Since this change occurred during P2, it would tend to bias our results toward the null; thus, including these samples gives our study a more conservative result. Finally, our study only describes the prevalence of *Salmonella* and *Campylobacter* after the withdrawal of antimicrobial growth promoters. Effects such as productivity, changes in therapeutic antimicrobial drug use and economic impact are described in another study **(Emborg et al., 2001)**

Our findings only show a temporal relationship between withdrawal and reduction, and one should be cautious not to infer causality. The fact that the decrease was seen before and during the use of antimicrobial growth promoters suggests that other factors play a role. The most obvious of these factors is the effect of the ongoing surveillance and control programs in food-producing animals. Programs in broilers and swine, described each year in the Annual Report on Zoonosis , have been in effect since the late 1980s and mid-1990s and have made a substantial impact on reducing the prevalence of *Salmonella* in primary food production. What is clearly shown from this analysis is that *Salmonella* and *Campylobacter* rates have not increased in food animal carriers since antimicrobial growth promoters were withdrawn in 1998. This finding, combined with evidence that the withdrawal has taken place without remarkably noticeable effects on the productivity in broilers **(Emborg ,2001)**. and swine, is of particular importance in light of the emerging problem of antimicrobial drug-resistant human pathogenic organisms, which are associated with the use of antimicrobial growth promoters.

Mad Cow Disease

Mad Cow disease, or its scientific name Bovine Spongiform Encephalopathy (BSE), is a fatal brain-wasting disease in cattle which was first identified in the United Kingdom (UK) in 1986. The disease has an incubation period lasting 4-7 years, but ultimately is fatal for cattle within weeks of its onset **(Joan and Mary , 2000)**

BSE is one of a number of Transmissible Spongiform Encephalopathies (TSEs) - a family of diseases in humans and animals which are characterized by sponge-like lesions in the brain. Other examples of TSEs are found in sheep, deer, elk, mink and even the feline species. In deer and elk, TSE is commonly referred to as "Chronic Wasting Disease" and in sheep the disease is known as "scrapie." It is widely believed that cattle in Britain developed BSE as a result of being fed the rendered carcasses of dead sheep that were infected with scrapie **(Canadian Press, Sunday, 2001).**

prion theory :

There is a great deal of speculation as to the original cause of Mad Cow disease. According to the widely-held "prion theory", the BSE agent is composed largely, if not entirely, of a self-replicating protein referred to as a "prion." Another theory suggests that the agent is virus-like and possesses nucleic acids which carry information. Strong evidence collected over the past decade supports the prion theory, but the ability of the BSE agent to form multiple strains is more easily explained by a virus-like agent.

Epidemiological studies conducted in the UK suggest it is spread through cattle feed prepared from carcasses of other ruminants - any of a group of even-toed, hoofed, cud-chewing mammals, including cattle, deer, and elk. No one knows for sure how the first cow (or cows) got BSE, but we know it spread throughout Britain and eventually the world through the cannibalistic practice of making cattle feed out of the bits of cattle (offal) that are not fed to humans. Like a "chain-letter", offal from a Mad Cow infected many more cattle and offal of those cattle infected many more.

According to British customs figures, more than 200,000 tons of potentially contaminated feed were exported around the world.(**United Press**

International, 2001) The UK Sunday Times reported that Prosper de Mulder, Britain's largest rendering company, exported potentially contaminated material to Canada. In a worldwide alert the UN Food and Agriculture Organisation reported that meat and bone meal from Europe was imported by more than 100 countries since 1986, including Canada. All those countries are at risk, said the report, and added "All countries which have imported cattle or meat and bone meal that originated from Western Europe, during and since the 1980s, can therefore be considered at risk from the disease." As of December 2000 approximately 180,000 cases of Mad Cow disease were confirmed in the UK(World Health Organisation , 2001) Cases of BSE have been confirmed in cattle in Belgium, Denmark, France, Ireland, Luxembourg, Netherlands, Portugal, Switzerland, Germany, Spain, Liechtenstein, Italy, Belgium, Greece, Czech Republic, Poland, Slovakia, Slovenia, Finland, Austria, Israel and Japan **(World Organisation for Animal Health , 2001)** In December 2000, the World Health Organisation issued a warning of "Global Exposure to BSE" and urged that "all countries must prohibit the use of ruminant tissues in ruminant feed"; In other words - stop cannibalistic feeding practices. Cattle are herbivores - not cannibals.

Jakob disease (vCJD):

New variant Creutzfeldt-Jakob disease (vCJD) -- or "Human Mad Cow disease" -- is also a fatal brain-wasting disease. Characterized by dementia and loss of motor control, this hideous disease was first identified in the United Kingdom in 1996. It is widely accepted in the scientific community that the most likely cause of vCJD is from exposure to the BSE agent via "dietary contamination by affected bovine central nervous system tissue", or in more simple terms, from eating infected meat. Originally it was believed that Mad Cow disease could not jump from cattle to humans. The hypothesis of a link between vCJD and Mad Cow disease was first raised because of the association of these two in time and place. Experts quickly observed that the agent responsible for vCJD is consistent with the agent that causes Mad Cow disease in cattle; In other worlds - it was the same disease.(World Health Organisation, 1996) Scientific studies have since confirmed that vCJD and BSE are indeed the same disease.

vCJD is classified as a Transmissible Spongiform Encephalopathy (TSE) because of characteristic spongy degeneration of the brain. The first person known to develop symptoms of what turned out to be vCJD became ill in 1994. Early in the illness, patients usually experience psychiatric symptoms, which most commonly take the form of depression or, less often, a schizophrenia-like psychosis. Early in the illness, unusual sensory symptoms, including "stickiness" of the skin, are experienced in approximately half of all cases. Neurological degeneration, including unsteadiness, difficulty walking and involuntary movements, occurs as the illness progresses. By the time of death patients are completely immobile and mute. vCJD is often confused with CJD, or "Creutzfeldt-Jakob disease", another brain-wasting human TSE disease which causes sponge-like degeneration of the brain. However, CJD, which afflicts approximately one in a million people worldwide, is caused by a hereditary predisposition.

According to Mad Cow disease expert Dr. Steven Dealer, like cattle, thousands - perhaps millions - of people may have been infected before the disease was first identified in 1996. As of June 1st, 2001, there were 104 confirmed cases of vCJD. What worries Dr. Dealer is the alarming increase in new cases being diagnosed. There were 15 people diagnosed with vCJD in 1999 and 42 more diagnosed in 2000. Because no one knows the length of the incubation period, which is currently speculated to be as long as 40 years, it is uncertain how many people will ultimately be infected. Dr. Dealer estimates that as many as 5 million people will contract the fatal disease in the United Kingdom alone **(U.K. government, 2001)**

Insecticide Causes Mad Cow Disease:

Pharmaceutical interests in the UK are ignoring new scientific research that shows the insecticide used in the UK government's own warble-fly campaigns triggered the UK surge of 'Mad Cow' disease. Latest experiments by Cambridge University prion specialist, David R. Brown, have shown that manganese bonds with prions. Other researchers work shows that prions in the bovine spine -- along which insecticides are applied -- can be damaged by ICI's Phosmet organophosphate(OP) insecticide -causing the disease. British scientists have led the current theory that an infectious prion in bonemeal fed to cattle causes

bovine spongiform disease (BSE). Infectious prions are also claimed to cause new variant Creutzfeld-Jakob Disease (CJD) in humans -from ingesting beef. But the infectious prion theory serves to obscure a tragic chemical poisoning scandal behind the majority of BSE cases.The new work proves that the prions can bond with manganese in animal feeds or mineral licks. These manganese prions cause the neurological degeneration seen in BSE. By a similar process, prions in human brains are damaged by lice lotions containing organophosphate. This can result in neurological diseases like CJD and Alzheimers -later in life **(U.K. government, 2001)**

Brown (1999) sugested how prion protein bonds benignly with copper, but lethally with manganese. Even natural variations in relative environmental availability of manganese versus copper can trigger prion degradation.

The CJD and BSE symptoms mirror 'manganese madness', an irreversible fatal neuro-psychiatric degenerative syndrome that plagued manganese miners in the first half of the last century

Another Ugly Side of Beef :

When Health Secretary Stephen Dorrell addressed the British House of Commons on March 20, 1996, he was ashen-faced for good reason. Like numerous other government representatives, for years Dorrell had been reassuring the nervous British public that the beef in their hamburgers, kidney pies and Sunday roasts was as safe and sound as the Pound sterling. Dorrell had also steadfastly insisted that no connection existed between any human illness and Mad Cow Disease, the incurable dementia that has killed 160,000 British cows since 1985. On March 20, however, a somber Secretary Dorrell faced the legislative body, and the nation, and proceeded to eat his very words. A government advisory committee, he explained, had concluded that Mad Cow Disease was indeed the "most likely" cause of a recent outbreak in young British adults of a similar fatal disease**(Darnton , 1996)**

McCarthy(1996) wrote, "If Mad Cow Disease can cause an international panic and political heartburn in the British Parliament because 10 Brits may possibly have contracted a rare nerve disorder from tainted beef, it should follow that the heavens themselves would be shaken by humanity's outcry over the proven deadliness of meat that's available everywhere."

Oulton (1995) showed that in a 1990 press stunt, the British Minister of Agriculture John Gummer appeared on television munching hamburgers with his four-year-old daughter. Next, despite evidence that everything from household cats to zoo animals had contracted fatal dementia by eating infected cattle products, the government faithfully insisted that cattle represented a "dead-end host," meaning the disease would stop at cows and not infect other species . Finally, and probably worst of all, though it was suspected early on that feed laced with infected sheep and cattle was behind Mad Cow Disease, the British government waited until 1989 to prohibit the practice.

Mad Cow Disease has shown an uncanny ability to jump from species to species. One immediate step we can take to reduce the odds of this deadly dementia threatening cows and humans in North America is to end the practice of feeding cows to cows. England banned the practice in 1989, and the World Health Organization is now endorsing a ban for all countries **(Lawrence , 1996)**

Can BSE be passed on to humans?

In the early 1990s, British scientists became aware of a new illness that had many symptoms similar to those of classical CJD, but it was different in several ways. Most notably, it affected much younger people around the ages 28 or 29. In 1996, researchers confirmed that the illness was a new form or variant of Creutzfeldt-Jacob Disease (vCJD). Instead of occurring sporadically in the population, however, this form of CJD appeared to be caused by consuming beef and beef products from cattle infected with Mad Cow Disease. In the following years, strict measures were put in place in the U.K. and elsewhere to control the spread of BSE among cattle and minimize the risk to human and animal health. This greatly slowed the spread of the disease among cattle in Britain. To date, 94 cases of vCJD have been reported in the U.K., one in the Republic of Ireland, and three in France. All these cases are believed to be related to the consumption of British beef.

Bellagamba et al., (2003) studied recent European bovine spongiform encephalopathy crisis has focused attention on the importance of adopting stringent control measures to avoid the risk of the diffusion of mad cow disease through meat meal-based animal feedstuffs. Potential adulteration of such feedstuffs with bone particles from terrestrial animals is determined by

microscopic examination by law before the release of these feedstuffs for free circulation in the European Community. This study describes a DNA monitoring method to examine fish meal for contamination with mammalian and poultry products. A polymerase chain reaction (PCR) method based on the nucleotide sequence variation in the 12S ribosomal RNA gene of mitochondrial DNA was developed and evaluated. Three species-specific primer pairs were designed for the identification of ruminant, pig, and poultry DNA. The specificity of the primers used in the PCR was tested by comparison with DNA samples for several vertebrate species and confirmed. The PCR specifically detected mammalian and poultry adulteration in fish meals containing 0.125% beef, 0.125% sheep, 0.125% pig, 0.125% chicken, and 0.5% goat. A multiplex PCR assay for ruminant and pig adulteration was optimized and had a detection limit of 0.25%.

Hooper (2003) suggests that such inhibition might promote the neurotoxic properties of the prion protein (the causative agent of mad cow disease) and its conformational conversion to the infectious form, thus raising the question as to whether proteasome inhibitors might facilitate the development of prion diseases.

Genetically Modified Foods

It is important to assess the risks for health when novel foods are ingested by people and animals. The important principle here is that long term health risks will not be adequately assessed by the testing procedures and regulatory frameworks proposed by ANZFA. Recent research suggests that health risks are greater than previously thought. These risks arise in a number of ways. Given the huge complexity of genetic coding, even in very simple organisms such as bacteria, no one can possibly predict the effects of introducing new genes into any food. Therefore there is no way of knowing the overall, long-term effect on health. **This is because:**

* the transposed gene may act differently when working within its new host
* the original genetic intelligence of the host will be disrupted
* the new combination of the host genes and the transposed gene will have unpredictable effects **(Schubbert et al. , 1994).**

Cross-species transfers being made between unrelated species, such as the transfer of modified agrobacterium genes, viral genes, and antibiotic resistant genes, would not happen in nature and may create new toxins, diseases, and weaknesses. In this risky experiment, the general public will be the ultimate guinea-pig. Biotechnology industry advocates claim their methods are precise. In fact, there is a random element in all current gene insertion methods. Genetic research shows that many weaknesses in plants, animals and humans have their origin in tiny imperfections in the genetic code. Therefore, side-effects and accidents are inevitable, and some scientists have assessed the risks to be unlimited **(Jager and Tappeser , 1995).**

Genetically Modified Hazards

Unpredictable health damaging effects and new diseaseWhen genetic engineers insert a new gene into any organism there is a "position effect" which entails an unpredictable pattern of genetic function. The protein product of the transposed gene may carry out unexpected reactions and produce toxic products. There is also serious concern about the dangers of using genetically engineered viruses as delivery vehicles (vectors) in the generation of transgenic plants and animals. This could destabilise the

genome and lead to horizontal gene transfer to other species, including mammals. This risk arises because recent research suggests that disabled viral material used in recombinant DNA techniques can recombine with other viral material in the human or animal gut to produce new active forms of viral material. This may cause dangerous new diseases, resistance to antibiotics, and severe immune reactions. Genetic engineering also interferes with RNA editing and molecular folding which may cause the formation of prion-based diseases similar to BSE_mad cow disease (Greene and allison, 1994).

Dr Joseph Cummins, Professor Emeritus of Genetics at the University of Western Ontario warns: "Probably the greatest threat from genetically altered crops is the insertion of modified virus and insect virus genes into crops. It has been shown in the laboratory that genetic recombination will create highly virulent new viruses from such constructions. Certainly the widely used cauliflower mosaic virus is a potentially dangerous gene. It is a pararetrovirus meaning that it multiplies by making DNA from RNA messages. It is very similar to the Hepatitis B virus and related to HIV. Modified viruses could cause famine by destroying crops or cause human and animal diseases of tremendous power **(Inose and Kousaku , 1995) .**

The process of genetic engineering can introduce dangerous new allergens and fatal toxins into foods that were previously naturally safe. Already, one genetically engineered soybean was found to cause severe allergic reactions **(Nondlee et al., 1996).**

Genetically Modified and food processing:

Special consideration of enzymatic food processing agents and byproducts of biotechnology and novel additives in foods :

In addition to genetically engineered organisms themselves, hundreds of biotechnology byproducts are beginning to be used as processing agents and additives in foods. These include rennets, baking aids, sweeteners, micronutrients and vitamins, and fat splitting and juicing enzymes. Most of these products have not been assessed adequately for their long term effect on health. Many are substituted for natural derivatives without precautionary labelling to inform the public of a change of process or ingredients. It is scientifically

incorrect to assume, as ANZFA appears to do, that, because a product is present in small quantities, its effect on health will negligeable. It important that these novel products are also labelled clearly as derived from gene technology. For example, a six-fold increase in reported cases of asthma among German bakery workers following the introduction of enzymatic baking aids needs careful investigation and assessment **(Mark and Fortin, 2001)**

Informed comment :

Dr Michael Antoniou, Senior Lecturer in Molecular Pathology at a London teaching hospital says, 'the generation of genetically engineered plants and animals involves the random integration of artificial combinations of genetic material from unrelated species into the DNA of the host organism. This procedure results in disruption of the genetic blueprint of the organism with totally unpredictable consequences.The unexpected production of toxic substances has now been observed in genetically engineered bacteria, yeast, plants, and animals with the problem remaining undetected until a major health hazard has arisen. Moreover, genetically engineered foods or enzymatic food processing agents may produce an immediate effect or it could take years for full toxicity to come to light." Because genetically engineered foods reproduce themselves and can never be recalled from the environment, Dr Antoniou warns of an unprecedented health risk for humanity.

Commercial implications:

Consumer safeguards in the composition of regulatory bodies.The present composition of ANZFA does not take proper account of consumer preferences. Mechanisms should be put in place to take proper account of consumer wishes. The present system, where small committees of scientists and civil servants decide what foods can be sold to the public, has not proved adequate to protect the public . Previous mistakes with thalidomide, tryptophan, DDT, etc are examples of novel products approved by scientists who lacked natural caution and then rushed to market for commercial reasons with tragic results. In many cases GMO approval committee members have close links to the biotechnology industry.They may derive their funds for research from biotechnology companies or act as paid consultants to food industries. The Natural Food Commission is recommending that ANZFA is made responsive to consumer wishes by

mechanisms such as referenda on the acceptability of biotechnology and increased consumer representation on committees (Ye et al., 2000).

Marc and Fortin (2001) studied commercial reasons to steer clear of biotechnology in New Zealand and Australia. This year modified soybeans and maize in USA were the first genetically modified crops to reach the market in large quantities. Up to 2% of this year's crop was involved. Plans called for these crops to be mixed with their conventional counterparts in the grain elevators and sold without labelling or distinguishing the product. However, widespread consumer opposition in Europe and North America has caused a change of heart. In the last month, manufacturers such as Nestle and Unilever have responded to public pressure by announcing that they will not use US soybeans in their products. The EU itself has banned the genetically modified corn because of concerns in 13 member states over health hazards. Public opposition has featured persistently on the front pages of newspapers in Switzerland, Denmark, Norway, Sweden, Austria, and Germany. Massive new markets have opened up for products that can be guaranteed free of genetic engineering. Companies specialising in the testing and sale of such products have posted record order books as retailers and manufacturers start to take account of consumer preferences. Australia and New Zealand rely heavily on exports of primary produce. Any perception that we are introducing genetically engineered foods will affect overseas assessment of our products. Conversely, a moratorium on the introduction of genetically engineered foods will improve export opportunities to European markets .

Plant Biotchnology (GM)

IS at a crossroads in Canada. Agriculture and the agri-food industry account for about 14 percent of Canada's employment, and the introduction of genes in plants offers important new opportunities for plant improvement. In Canada, canola, corn, potato, soybean, wheat and flax varieties have been approved for release. Over the last five years, the increases in acreage for a few genetically modified (GM) crops have been spectacular (a fifteen-fold increase in acreage between 1996 and 1998) **(James , 1998)**. Agricultural biotechnology is second only to biomedical biotechnology in terms of biotech investments (28 % and 40 %, respectively). Predictions for the growth of the agbiotech sector range from a few percent to as much as 40 percent per year.

However, many are concerned with the risks to human health and the environment associated with the release and cultivation of such plants in the environment. Industry and governments have invested heavily in biotechnology research, but the public is concerned, scientists do not agree on key issues, trade partners threaten to refuse shipments of GM crops, and regulatory agencies are increasingly under pressure for accountability given recent food safety incidents (abroad). Farmers are caught in the middle of this imbroglio and hesitate on whether they should plant GM crops. Indeed, it appears that 2000 will be the first year in which there is a decrease in acreage **(U.S. Department of Agriculture , 2000)**

Improved plants

There are virtually no food products on supermarket shelves that have not been improved by plant breeders (fiddleheads and wild blueberries are examples of a few remaining unimproved plants). Most wild forms of the plants we consume daily are significantly different from what is currently commercialized: both yield and food quality have been improved since the transition from hunter-gatherer societies to farming societies where farmers selected and collected seed from the best plants in their fields. Wild lettuce is bitter and unpalatable, wild tomatoes are not nearly as sweet as current varieties and most consumers would reject them. The yield of corn has almost doubled in the last 40 years and almost tripled in the last 100 years. World food production has doubled since 1960 while productivity (amount of food per hectare) has tripled **(National Science and Technology Council ,1995)** This somewhat parallels the growth in population over the same time period.

Plant biotechnology

Approximately 25 years ago, several research groups discovered that soil bacteria could transfer genes from bacteria to plant cells. The bacteria (several species of *Agrobacterium*) have all the "machinery" for naturally transferring segments of DNA to a plant. Genetic engineers take advantage of this capacity of bacteria to transfer genes to introduce new genes into plant cells. In this way, genetic engineering makes it possible to introduce foreign genes into plants, eliminating the fertility barrier that separated most plants from each other and from animals and microbes. In theory, any segment of DNA from any living cell can be inserted and the trait for which it codes can be expressed in plant cells.

The first plant product derived from biotechnology to be put on supermarket shelves was the FlavrSavr tomato (in 1995, developed by Calgene). This tomato variety was created to satisfy consumer demand for a flavourful product year round. By increasing the tomato's firmness, it could be left to ripen on the vine and still be transported to market without the losses associated with a soft ripe tomato. The tomato's firmness was increased by reducing the activity of an enzyme (polygalacturonase) involved in the fruit's softening. This reduction was achieved by making a copy of the gene, inverting it and reintroducing it in tomato plants. The expression of a second, inverted, copy of a gene generally leads to reduced gene expression of the original (resident) copy. In genetic terms, the manipulation involved was an inversion; it is ironic that this product was the subject of controversy since inversions occur naturally in all organisms **(Gianessi et al., 2000).**

One question which remains largely unanswered in the opinion of many is whether the new food products pose any threat to human health. The ingestion of foreign DNA is an unlikely source of risk, as we ingest DNA daily as part of our regular diet of plant, animal and microbial products. In addition to inserting genes, most genetic manipulations also introduce an antibiotic resistance gene as a "marker," to discriminate transformed from untransformed plants in the course of experimentation. The ability of transformed plants to resist antibiotics is useful as untransformed plants die in the presence of the antibiotic, thereby simplifying the task of identifying transformed plants **(Losey et al., 1999).**

Proponents of GM food suggest that a canola plant that is resistant to herbicides should produce the same oil as a non-GM canola plant. Others state that this can not be assumed to be the case since it has not been established scientifically. This is perhaps the crux of the battle behind whether the "precautionary principle" (where a GM product poses a threat, precautionary measures should be taken even if cause and effect relationships have not yet been established) should be used by regulatory agencies instead of the principle of "substantial equivalence" (if there is no apparent difference between the GM food and its traditional counterpart, it is assumed to be safe). Most regulatory agencies have so far operated using the principle of substantial equivalence. The Canadian Food Inspection Agency correctly states that "Establishing substantial equivalence is not a safety assessment in itself **(Canadian Food**

Inspection Agency , 2001) .The Clinton administration has recently emphasized the need for "science-based" assessment of new food products. Regulatory agencies use data submitted to them by the developers of these new products and from the scholarly literature to judge whether a new GM plant can be released for field trials; the agencies generally do not have the resources to conduct their own in-house tests.

The possibility that GM foods could induce allergic reactions has been widely mentioned; this has not been shown so far for any GM plant on the market. Protocols exist for testing for some allergic reactions, but more research is needed to standardize and implement them. One of the most widely criticized GM plants has been soybean modified to contain a brazilnut protein involved in allergenicity (these plants have never been put on supermarket shelves). In many cases, more experimental analyses are needed.

GM plants and the environment.

The large majority 92 % **(Ferber, 1999)** of GM crops planted in 1999 were modified for only two characteristics: either herbicide resistance or insect resistance. Despite research efforts, relatively few genes are available for genetic engineering. We still do not know which DNA sequences code for the vast majority of traits of economic importance (such as yield and quality) and modification of those traits has so far largely been out of the reach of genetic engineers.

Herbicide resistance:

Herbicide resistance, which is conferred by a gene that detoxifies the herbicide, has been amenable to genetic manipulations for several years. Because the gene will only degrade one type of herbicide, and both the herbicide and the herbicide degradation genes are proprietary, farmers must purchase both the GM plant with the herbicide-resistance trait and the matching herbicide, likely from the same company. There was initially concern that this might create a monopoly situation, but other companies now offer competing combinations of herbicide-degrading GM plants and herbicide.

Proponents of GM crops show field data which suggest that use of herbicide-resistant GM plants can lead to reduced herbicide use in the field. These reductions, if indeed there are any, vary significantly between crops,

regions and weed population pressures. For herbicide-resistant (Roundup Ready) soybean, different investigators report marginal (i.e., 4 %) increases **(Gianessi and Carpenter , 2000)** or decreases **(U.S Economic Research Service , 2001)** in herbicide use per hectare. On the other hand, GM soybean allows farmers to substitute Roundup for more hazardous and long-lasting herbicides such as acetochlor. It also reduces the need for farmers to till the soil, thus reducing erosion. It is now possible to sow herbicide-tolerant soybean directly into undisturbed soil and apply a post-emergence herbicide, thus conserving soil moisture, improving crop performance and reducing water and wind erosion. Competition in the herbicide market has also resulted in price reductions for an estimated U.S. $278 million savings for U.S soybean growers, or 28 percent of total herbicide expenditures. Products with more direct consumer benefits will likely be developed. An example is the introduction of genes that produce beta-carotene (the precursor of vitamin A) in rice: GM rice now has a light golden-yellow color and contains sufficient beta-carotene to meet human vitamin A requirements from rice alone **(Ye et al., 2000)**

Heavy Metals

Heavy metals' toxicity is the result of their interactions with the enzymatic systems from the animal cells or some constituents of cells' membranes. The interactions of heavy metals with usual elements from diet (Cu, Zn, Fe, Ca, Se) have an important role in acute and chronic toxicity. Population can be contaminated with heavy metals by ingestion of contaminated or polluted food or water. The concentration of heavy metals in food products is varied, depending on their origin, storage conditions and processing technologies.

In this work, lead, cupper, cadmium and zinc were determined over 1 year in different food products: milk and dairy products, meat products, bread and bakery products and beverages. Samples were first submitted to a wet digestion and after that were measured by atomic absorption spectrometry. The reliability of the approach was confirmed by analysing data from literature**(Coultate,1990)**

Essential Metals:

Minerals and trace elements occur in the body in a number of chemical forms, such as inorganic ions and salts or as constituents of organic molecules, for example proteins, fats, carbohydrates and nucleic acids.

The minerals that are considered essential in the human diet are sodium, potassium,chloride, calcium, magnesium, phosphorus, iron, copper, zinc, manganese, selenium,iodine, chromium, cobalt, molybdenum, fluoride, arsenic, nickel, silicon and boron.

Toxic Metals:

A number of other chemical elements occur in food, e.g. aluminium, strontium, lead, tin, mercury, cadmium and many of them are toxic.Toxic metals may reach our food from a number of sources. The more important of these are: l.the soil in which human or animal feed stuffs are grown; 2.sewage sludge,fertilisers, and other chemicals applied to agricultural land; 3.the water used in food processing or cooking; 4.contaminating dirt (e.g. soil on unwashed vegetables) and 5.the equipment, containers and utensils used for food processing storage or cooking. According to their nutritional role the metals from food products can be divided in two categories: essential metals (their absence

or even their insufficiency in human diet induce after a period of time some modifications of metabolic process and will appear some diseases, e.g. Na, K, Ca, Cu, Zn, Mn) and unessential metals (like Pb, Hg, Al, Sn,Ag). When the metals' intake is smaller than the possibility to eliminate through urine,gastric juice or other physiological, their behaviour is like some chemical impurities that are present for a short time in the human body without biochemical perturbations.

Heavy metals Hazards:

For both categories, increasing of metal concentration in food over the limits can cause toxic effects for consumers of these products. The gravity of toxic effect depends on nature, quantity and chemicals form of metals from the food product and it depends on metal concentration in food, on body resistance and on synergetic orantagonistic effects of other chemical contaminants.

Severity of toxic effects of all elements increases with dose. A normal diet supplies about 0.01 0.04 µg Cd/day/g; the intake of cadmium will be dependent on the consumed fond amount. Among the most important food possible contaminated with cadmium are pork meat, fish, milk and beer. The sources of contamination with lead are: lead piping and lead-lined tanks in domestic water supplies, canning and using the pottery glaze for storage the beverages. The total lead intake from food and beverages has been estimated for adults in various industrialised countries to be 250 - 300 µg/day **(Coyer et al., 1994).** Copper is not as toxic as one might imagine; large doses of copper sulphate are emetic, and a dose of 100 g is reported to have resulted in liver and kidney damage. The daily intake from normal adult diets is between 1 and 3 mg, which roughly corresponds to the intake level recommended by most authorities. The conditions for this metal to become toxic are high acidity of food and long contact time. The distribution of zinc in our foodstuffs has much in common with copper. Typical diets have been calculated to supply between 7 and 17 mg/day, of which about 20% are absorbed. The potential for zinc absorption to be reduced by the presence of large quantities of fibber in the diet does not seem to be realised in the case of ordinary European diets .

In current research we determined lead, copper, cadmium and zinc content from 36 bread and bakery samples (14 bread samples and 22 bakery products: rolls, muffins) and from 17 samples of pastes. the presence of heavy metals has been praised in fruit and vegetables because of the absorption from soil. We analysed 20 samples of vegetable products. From animals we analysed 20 samples of milk products and 47 samples of meat products. the heavy metals content in beverages is very low .

Heavy metals in Some Foods:

On the basis of the monitoring measurement results of Krakow's environment a long-time evaluation has been presented, with regard to lead presence. The pollution with lead in a last few years was displayed in atmospheric air, soil and drinking water samples, as well as in some vegetables and food products. It was concluded that a characteristic trend regarding lead pollution of the environment could be observed;a decrease of lead level in air and in vegetables was presented, in soil and milk as well as in dairy products--remained low. Only in lettuce and parsley leaves the exceeding of the permissible lead content was confirmed. At present, in spite of the decrease of the heavy metals level in the environment, lead is still present and the health hazard to men exists, but to a lesser degree **(Lutynski , 2003)** .

Dragun et al.,(2003) determined the concentrations of cadmium, lead, mercury, and metalloid arsenic in the samples of some dietetic products marketed in Croatia, and to compare the values obtained with maximum allowed amounts (MAA) according to the law. Methods. Metal and metalloid concentrations were measured in 30 dietetic products from the group of industrial food supplements and food additives. The measurements were performed by the method of atomic absorption spectrometry. The concentrations of cadmium, lead, mercury, and arsenic were compared with the maximum allowed amounts for these substances in corn and corn products, and their estimated daily intake with the recommendations of the World Health Organization. Results. Two out of 30 samples contained cadmium, 5 samples contained lead, and as many as 16 samples contained mercury in concentrations exceeding maximum allowed amounts. The concentration of

arsenic was below maximum allowed amount in all samples. In total, the concentrations of metals exceeding maximum allowed amount were found in 17 out of 30 samples. Extremely high contamination with heavy metals was detected in a sample based on zinc oxide, in which the concentration of cadmium (0.418 mg/kg) was four times higher than the maximum allowed amount, and of lead (6.074 mg/kg) 15 times higher than the maximum allowed amount. The highest concentration of mercury (1.117 mg/kg), 35-fold maximum allowed amount, was found in a ginseng-based sample. Conclusions. Cadmium, lead, mercury, and arsenic were present in some dietetic products in concentrations exceeding maximum allowed amounts. Dietetic products control should match respective legal provisions of the European Union requirements, and requires continual monitoring.

Filazi et al., (2003) evaluated levels of five heavy metals [copper (Cu), lead (Pb), cadmium (Cd), chromium (Cr) and nickel (Ni)] in liver and muscle tissues of fish (Mugil auratus) collected from the Black Sea at Sinop-Icliman, Turkey. Sampling and analysis methods are described. Variations of heavy metal concentrations with seasons are discussed. Cr and Ni concentrations were below the limits of detection (< 0.05 and 0.1 microg/g dry weight) in all tissues and seasons. Cu, Pb and Cd were detected within these limits, as microg/kg dry weight, in liver tissue: 0.49-1.30, 0.60-1.21 and 0.15-0.50, and in muscle tissue: 0.30-1.00, 0.57-1.12 and 0.10-0.40, respectively. Cu, Pb and Cd concentrations in these tissues were elevated and the highest heavy metal concentrations were found in the liver. While Cu, Pb and Cd concentrations were highest in fish tissues collected in August 2000, the lowest concentrations of these metals were observed in fish tissues collected in May 2000. Pb had the highest level observed in fish tissues. According to the Turkish Food Codex Regulation's residue limits, the cadmium level determined in fish tissues was high (0.1 microg/g) and the lead level, especially in liver tissue, was high in August (1 microg/g), while other metals (Cu, Cr and Ni) were within the maximum residue limits.

Veeken and Hamelers (2002) found that separately collected organic fraction of municipal solid waste, can be reused for soil conditioning after composting. In this way, environmentally harmful waste management strategies, such as landfilling or incineration, can be reduced. However, frequent application

of composts to soil systems may lead to the accumulation of heavy metals in soils, and therefore legal criteria were laid down in a decree to guarantee the safe use of composts. The heavy metal content of biowaste-composts frequently exceeds the legal standards, and thus raises a conflict between two governmental policies: the recycling of solid waste on the one hand, and the protection of natural ecosystems and public health on the other hand.

Schober et al., (2003) studied humans exposed to methylmercury, a well-established neurotoxin, through fish consumption. The fetus is most sensitive to the adverse effects of exposure. The extent of exposure to methylmercury in US women of reproductive age is not known. To describe the distribution of blood mercury levels in US children and women of childbearing age and the association with sociodemographic characteristics and fish consumption. The 1999-2000 data from the National Health and Nutrition Examination Survey, a cross-sectional survey of the noninstitutionalized US population. In 1999-2000, 1250 children aged 1 to 5 years and 2314 women aged 16 to 49 years were selected to participate in the survey. Household interviews, physical examinations, and blood mercury levels assessments were performed on 705 children (56% response rate) and 1709 women (74% response rate). Blood concentration of total mercury: Blood mercury levels were approximately 3-fold higher in women compared with children. The geometric mean concentration of total blood mercury was 0.34 micro g/L (95% confidence interval [CI], 0.30-0.39 microg/L) in children and 1.02 microg/L (95% CI, 0.85-1.20 microg/L) in women. Geometric mean mercury levels were almost 4-fold higher among women who ate 3 or more servings of fish in the past 30 days compared with women who ate no fish in that period (1.94 microg/L vs 0.51 microg/L; P<.001). They concluded that , measures of mercury exposure in women of childbearing age and young children generally fall below levels of concern. However, approximately 8% of women had concentrations higher than the US Environmental Protection Agency's recommended reference dose (5.8 microg/L), below which exposures are considered to be without adverse effects. Women who are pregnant or who intend to become pregnant should follow federal and state advisories on consumption of fish

VanderJagt et al., (2001) determined levels of lead in the milk of 34 Fulani women in Plateau State in northern Nigeria. The Fulani are nomadic, semi-pastoralists who inhabit the western Sahel, including the countries of Mali, Burkino Faso, the Republic of Niger, Nigeria and Chad. The mean age of the women in this study was 26.0 +/- 7.5 years and their mean body mass index (BMI, kg/m2) was 19.4 (range, 14.5-24.0). Their average parity was 4 (range, 1-10). Milk was collected 2-24 weeks postgestation (mean, 11 weeks). Fifteen of the 34 milk specimens contained measurable lead (limit of detection, 4.6 microg/dL); the milk of the other 19 women (56%) did not contain a detectable level of lead. The data were not normally distributed. The median lead concentration of all 34 milk specimens, determined by end on plasma-axial view spectrometry, was 6.7 microg/dL (range, < 4.6-130 microg/dL). Given the mean weight of the 34 infants in the study (4.6 kg) and assuming that each infant consumed 0.7 L/day of milk, the average lead intake of these exclusively breastfed infants was 9.9 microg/kg/day, a value which is twice the daily permissible intake (DPI) of 5.0 microg/g/day set by the World Health Organization in 1972. These data indicate that some exclusively breastfed Fulani infants in the Jos Plateau are at risk of injury from lead derived from their mothers' milk, and raise questions about the actual blood levels in these nursing infants and the source(s) of the maternal lead.

Pesticides residues

One of the early actions of the regulatory authority included the bans of organochlorine insecticides such as DDT, aldrin, dieldrin, endrin and HCH (, and). All of them (as well as lindane and chlordane) have been included as "banned for use in agriculture List of Products Whose Consumption and/or Sale Have Been Banned, Withdrawn, Severely Restricted or not Approved by Governments (1994) but they are banned for all uses. The economic reforms in 90s have resulted in novel problems in pesticide regulation. Previously, a centralised trade organisation functioned as the main national focus for pesticide import and distribution . After decentralization, responsibilities for import and distribution of pesticides have been dispersed among a number of small units, many of which lack the required experience. A large part of the farming population has insufficient education and training in plant protection. To cope with the increased potential hazards associated with pesticide use in Bulgaria, the system for registration and classification has been revised **(Tasheva, 1995).**

Twenty years later (1990-1996) DDT and HCH (and) were not found in the River Iskar in Bulgaria. Only lindane has been identified in 3 points ranging 0.006-0.008 g/l. The fact that DDT (and its metabolites) and HCH (and) are not detected in the recent studies is a logical sequence of their 30-year period of ban. The presence of lindane more likely depends on its migration capacity due to its weakest adsorption ability and highest volatility if compared with the other persistent organochlorine pesticides **(Vasilev ,1997).** Daily intake of DDT and lindane by drinking water and food has been surveyed **(Georgiev, 1974).** The daily intakes of DDT and lindane were estimated to be 55.6 and 22.4 ml g/person/day, respectively. These levels are much below than Acceptable daily intake and comparable with the levels found in West European countries such as Italy, The Netherlands etc.

Pesticides Exposure:

Foods :

• Although many pesticides act at the same site no calculations are made to determine multiple residual exposure in diets. Many food products will have a number of pesticide residues. Agriculture Canada reports that the average peach

in Canada has 31 pesticide residues **(Neidert et al., 1994)** . The majority of these act at the same sites; the parasympathetic and central nervous systems. Although the residue of one pesticide may not exceed the maximum allowable level, a number of pesticides of the same class, acting at the same physiological sites, will have a cumulative and possibly toxic effect.

WATER

Pesticides are commonly found in water consumed by both rural and urban populations. Groundwater was found to have residues of 39 pesticides and their degradation products in a study of U.S. states and Canadian provinces.

Allowable pesticide levels for water are calculated on the basis of adult exposure and toxicity but again the pediatric population is exposed to a considerably greater total amount of residues that are potentially toxic because they are consuming on average 4 times the amount of water per kg of body weight.

Residues of pesticides that are "severely restricted" because of their serious effects on human health were also found in significant quantities in the water sources.

Residues enter the water supply as they are leached from soil into ground water after home, lawn, roadway and agricultural spraying **(National Research Council , 1993)**

Pesticides Hazardes:

The most widely used pesticides function by disrupting neurological cellular function. The systemic toxic effects after acute exposure are well documented and the Committee on Pesticides in the Diet of Infants and Children CPDIC conclude that emerging data suggest that neurotoxic and behavioural effects may result from low level chronic exposure to organophosphates and carbamate pesticides. These are commonly used pesticides in Canada; found in food, lawn and garden pesticides and household products.

The recent study on Home Pesticide Use and Childhood Cancer **(Leiss et al., 1995)** demonstrated a significant correlation between yard treatment with pesticides and pediatric soft tissue sarcoma and between pesticide strips and pediatric leukemias. The pesticide used in pest strips has been shown to be a carcinogen in animals and this strong association with leukemia in children is

disturbing given their common use and accessibility to infants and children. This occurs directly or indirectly following application on lawns, lawn furniture and play structures and through storage in the home.

There are many studies linking exposure to these herbicides with cancer. Studies from Sweden have suggested that workers exposed are at an increased risk of developing soft-tissue sarcoma, Hodgkin's disease and non-Hodgkin's lymphoma. **Hoar et al.,(1986)** found that exposure to herbicides on greater than 20 days per year resulted in a 6 fold increase in non-Hodgkin's lymphoma. These findings coincide with the Findings of increased incidence of NHL in caretakers of golf courses and previous studies on farmers.

These pesticides are chlorine containing compounds including DDT, aldrin, dieldrin and lindane. The organochlorines act through disruption of neurotransmission. PCB's, which are not used as pesticides, are also organochlorines with similar human action and thus have the potential for an additive effect.

• The greatest concern with the organochlorines are the long term effects. The U.S. EPA has concluded that DDT, DDE and DDD are probable human carcinogens. On this basis both Canada and the U.S. banned the organochlorines however, they continue to be very prevalent posing long term health risks.

• The organochlorines are still widely used in developing countries including Central and South America, India, China and many other countries. Products imported from these countries are obvious sources of DDT and other organochlorines. They are also transported in air, oceans and bioaccumulate in organisms **(Davies, 1988)** • Food is the primary route of exposure. Foods which may contain DDT include: meat, fish and poultry, dairy products and root and leafy vegetables. Fish from the Great Lakes Basin and inland waters are a large food source of organochlorine exposure.

• A study of the concentrations and dietary intake of selected organochlorines in fresh food composites grown in Ontario demonstrated that organochlorine residues were detected in all of the food composites. This included all types of fresh food grown in Ontario including beef, poultry, fruits and vegetables (it did not include fish). The Findings suggest that consumption of

eggs and meat is also a significant source of exposure to the majority of organochlorine chemicals studied.

• The provincial and federal health departments report that there are instances where maximum allowable levels of DDT intake may be exceeded in breast-fed infants.

• Similarly, they report that people living near hazardous waste sites have been found to have an increased exposure to organochlorines in large part because of leaching into the soil (Health and the Environment: A Handbook

Recently in the country was introduced phenylpyrazole insecticide fipronil acting on the same receptor (GABA receptor regulating chloride channel) as cyclodienes including endosulfan. It appeared to be less toxic than cyclodienes with a rapid metabolism and elimination in mammals, less toxic for fish than pyrethroids with moderate accumulation.

Use of biopesticides still represent a small percentage (about 3.8%) of total pesticide use in Bulgaria. The majority of biopesticides are based on Bacillus thuringensis. Due to their relatively high prices in comparison with agrochemicals their use is only supplementary in wheat, maize, cotton, sugar beet, rice, horticulture, fruit production and forestry **(Tasheva, 1995).**

- The amounts and variety of pesticides now used are far greater than in any other time in history.

- Both quantitative and qualitative differences in toxicity of pesticides exist between children and adults. Infants and children may develop toxic outcomes from smaller quantities due to different metabolic rates, greater absorptive areas, diets more concentrated with certain foods high in pesticides but they may also have outcomes such as neurological, behavioural, endocrinological that are not seen in adults due to critical windows of exposure both in utero and during certain growth phases.

- Current regulatory systems look only at the average exposure of the entire population. As a consequence, variations in dietary exposure to pesticides and health risks related to age and to other factors such as geographic region and ethnicity are not addressed.

- Diet is an important source of exposure to pesticides **(National Research Council , 1993)**

The Committee on Pesticides in the Diet of Infants and Children (CPDIC) concluded that the population is at great risk from the existing allowable levels of pesticide residues and that the data strongly suggest that exposure to these neurotoxic compounds at levels believed to be safe for adults could result in permanent loss of brain function when it occurs during prenatal and early childhood periods of brain development .1 Toxicologists agree that by extrapolation from hazard assessment studies conducted primarily in rodents, pesticides have the potential to produce toxicity in humans, a potential that includes many different toxic end points. Recent studies have investigated nonoccupational human exposure, such as those presented by **(Leiss et al., 1995)** who demonstrated an association between yard treatments and soft tissue sarcomas (odds ratio 4.0) and the use of pest strips and leukemias (OR 1.7–3.0) in children. Similar findings have been reported by **Gold et al.(1979)** who report an association between exposure to insecticide extermination and brain tumors (OR 2.3) 3; **Lowengart et al., (1987)** who report an association between household pesticides and leukemia (OR 4.0) and garden pesticides and leukemia (OR 5.6) and most recently **(Davis et al., 1993)** who found odds ratios up to 6.2 for several pesticide specific exposures among children with brain cancer.

The CPDIC has demonstrated that infants and children are particularly at risk of consuming toxic amounts of pesticides . This data is transferable to our Canadian population and we need to ensure that our public health system includes methods of determining maximal allowable levels of pesticides in foods based on human health outcomes , that there is education and restrictions on home and institutional pesticide spraying where children and adults can be exposed to acute and chronically toxic levels of pesticides and support the use and development of nontoxic alternatives. for Health Professionals , 1995

Pesticides and Infants:

A survey of the pesticide content of 25 commercially available infant formulae and 30 weaning foods available in New Zealand was undertaken in 1996. It included a representative mixture of imported and New Zealand manufactured infant foods. Three different pesticide screening techniques were used: a high-sensitivity organochlorine screen was carried out on all infant

formulae, while a multiresidue screen (organochlorine and organophosphorus pesticides, synthetic pyrethroids, carbamate pesticides, fungicides and herbicides), and a specific screen for dithiocarbamate fungicides were both carried out on all weaning foods and on soy-based infant formulae. All results are expressed on a ready-to-feed basis. Extremely low levels of residues of three organochlorine compounds (p,p'-DDE, p,p'-DDT and dieldrin) were detected in infant formulae samples. Residues of p,p'-DDE were found in seven of 20 milk-based infant formulae at concentrations ranging from 0.03 to 0.5 microgram kg(-1). Residues of p,p'-DDT were found in one imported milk-based infant formula at 0.7 microgram kg(-1), and dieldrin residues were detected in four of five soy-based infant formulae at concentrations ranging from 0.05 to 0.08 microgram kg(-1). The multiresidue pesticide screen detected low levels of residues of two organophosphorus pesticides; azinphos-methyl in one soy-based infant formula at a level of 22 microgram kg(-1) and pirimiphos-methyl in two cereal-based weaning foods at concentrations of 5 and 14 microgram kg(-1). None of the other approximately 140 pesticides (including fungicides and herbicides) included in the multiresidue screen were detected in any weaning foods or soy-based infant formulae, at a detection limit of 10 microgram kg(-1). No residues of dithiocarbamate fungicides were detected in any product analysed, at a detection limit of 100 microgram kg(-1) **(Food Safety Programme, Institute of Environmental Science and Research , 1998) .**

An environmental pesticide fate study was conducted in soil profiles collected from 23 rice field sites in an important Mediterranean wetland (Albufera Natural Park, Valencia, Spain) from April 1996 to November 1997. Temporal and spatial distribution of 44 pesticide residues in an alluvial Mediterranean soil (gleyic-calcaric Fluvisol, Fluvaquent) were monitored. During this period, the levels of pesticide residues in different soil horizons (Ap1 0-12 cm, Ap2 12-30 cm, ApCg 30-50 cm, C1gr 50-76 cm, and C2r 76-100 cm) were investigated. In addition, information was agricultural pesticide application practices and soil characteristics. Distribution throughout the soil profile showed that pesticide concentrations were always higher in the topsoil (Ap1 horizon), in the autumn season, and in the border with citrus-vegetable orchard soils (calcaric Fluvisol, Xerofluvent). Chlorpyrifos (organophosphorus), endosulfan (organochlorine), and pyridaphenthion (organophosphorus) insecticides were, respectively, the most

detected of all the pesticides investigated. These results were associated with processes, such as nonleaching, transport by movement into surface waters, retention, volatilization, and chemical and biological degradation in the topsoil, as well as with direct and indirect exogenous contamination sources **(Gamon et al., 2003).**

An unknown peak was detected in a GC chromatogram of many kiwi fruit extracts during analysis for pesticide residues. It was identified by GC/MS as diphenyl 2-ethylhexyl phosphate (DPEHP), used as a plasticizer and flame retardant. The concentration of DPEHP was investigated in 15 samples of kiwi fruit, and it was detected at between 0.02 and 0.14 microgram/g in 10 of the samples. It might be due to migration of DPEHP into the fruit from the printed portion of the polyethylene terephthalate (PET) package **(Tamura et al. , 2002)**.

Parasites

The major modes of transmission of protozoa include consumption of surface water, exposure to contaminated recreational water, animal-to-person contact and person-to-person contact .However, the epidemiology of protozoa most commonly associated with human infections, namely *Giardia, Entamoeba, Toxoplasma, Sarcocystis, Isopora, Cryptosporidium, Eimeria* and *Cyclospora*, is not fully understood **(Goodgame, 1996).** While the life cycles of each of these parasites differ, all require passage through an animal or human host. Shedding of cysts or spores into faeces which may then, directly or indirectly (e.g. via sewage or irrigation water), contaminate raw fruits and vegetables occurs on a global scale; it may be common in countries where hygienic conditions (especially water quality) are compromised. Expanding poverty, urban migration, food exportation, air travel and immigrant populations in conjunction with other societal and environmental changes make the distinction between so-called tropical and western parasitic diseases less clear-cut than in the past.

Parasites Infections

Outbreaks of protozoan infections in humans have been linked to raw fruits and vegetables. Epidemiological evidence has implicated an asymptomatic food handler as the probable source of *Giardia lamblia* and raw sliced vegetables as the vehicle of transmission in an outbreak of giardiasis **(Mintz et al, 1993)**. Raspberries **(Centers for Disease Control and Prevention, 1996 ; 1997a,b; Jackson *et al.*, 1997),** lettuce **(Centers for Disease Control and Prevention, 1997b),** and basil have been the implicated vehicles of transmission in outbreaks of *Cyclospora cayetanensis* infection, and unpasteurized apple juice has been linked to outbreaks of cryptosporidiosis caused by *Cryptosporidium parvum* . A survey of vegetables has revealed the presence of *Cryptosporidium* oocysts on cilantro, lettuce, radish, tomato, cucumber and carrot. The current status of knowledge about *Cryptosporidium* and its significance in food and beverage production has been reviewed by **Donnelly and Stentiford (1997).** The presence of these protozoa on raw fruits and vegetables is likely to be due to contact with animal or human faeces, sewage, water containing untreated

sewage and sludge from primary or secondary municipal water treatment facilities.

While *Cryptosporidium, Giardia* and other parasites in water are quite resistant to chlorine and other disinfectants, little is known about the efficacy of these disinfectants in killing or removing parasites from the surface or tissues of fruits and vegetables. Surveys have shown that there is a high incidence of the parasitic roundworm, *Ascraris*, in the sewage sludge of many cities **(Jackson *et al.*, 1977).**

Parasites Hazards:

Foodborne trematode infections are major health problems, with an estimated 40 million persons, mainly in eastern and southern Asia, being affected **(Abdussalam *et al.*, 1995).** Infection takes place through the consumption of raw plants, or raw or undercooked freshwater fish or shellfish, containing the infective cyst (metacercaria) stage of these parasites. Watercress is a major source of *Fasciola hepatica* infection **(World Health Organization, 1995).** Over 300,000 clinical cases of fascioliasis may have occurred in more than 55 countries in Africa, the Americas, Asia, Europe and the western Pacific from 1970 to 1990. Large endemic areas have been reported more recently in Bolivia, Egypt, Iran and Peru **(Abdussalam *et al.*, 1995).**

A wide range of other aquatic plants may support metacercariae. Conditions for transmission of *Fasciolopsis buski* are present in areas of cultivation of water caltrop, water chestnut, water hyacinth, water bamboo, water mimosa, lotus and duckweed. If animal manure or effluent from livestock pens or abattoirs is used as fertilizer for these plants, it introduces *Fasciolopsis* to the aquatic environment. Plantborne trematodes encyst as metacerceriae on the surface of plants or on debris floating on the water surface. Plants that grow in water are also believed to serve as hosts for encystment of metacercariae of certain intestinal flukes (World Health Organization, 1995). Non-aquatic plants such as lettuce, alfalfa, mint and sugarcane which may be eaten raw have also been implicated in human trematode infections.

Strategies for controlling foodborne trematode infections associated with eating raw plant materials will be shaped by ecological and environmental factors. The use of non-composted animal manures or effluent from livestock

pens for the purpose of fertilizing aquatic plants should cease. Freezing, heating and irradiation can be effective in killing trematodes. The effectiveness of chemical treatments in killing trematodes attached to plant surfaces is not well understood.

The legal use of various treatments differs from country to country. Described here are disinfectants and disinfection treatments that are in current use or that have potential for killing microorganisms on whole and cut raw fruit and vegetables, as well as surfaces that are in contact

Thomas (2002) showed that the salmonid hosts during the summer has arisen as a result of evolutionary pressures. At this time, the gut environment is particularly inhospitable because of the temperature-related enhancement of the host's immune mechanism and the increased gut turnover rate. In contrast, the larval stages in the immunologically and metabolically more benign intermediate host would be under less intensive selective pressures. It is postulated therefore that evolutionary pressures have caused the parasites to leave the definitive host and concentrate their reproductive efforts in the intermediate hosts during the warmer months. Evidence is given in support of the hypothesis that the parasite populations are regulated in a density-dependent manner and that the regulatory mechanisms may involve the host's immune mechanisms and intraspecies competition and interspecies competition of an exploitative or interference nature . This indicates that the introduction of novel parasites or new genetic strains of host fish could result in harmful epidemics. Despite causing tissue damage, there was no evidence of parasite-induced mortality among the salmonids in the Teifi. This finding is in accord with the generally accepted view that most freshwaters are not troubled by parasite problems. although parasites are present in abundance. In fact, parasite abundance in the salmonid fish in the Teifi was positively correlated with the condition factor and the adipose index. Two testable hypotheses were advanced to explain these observations. First, the more dominant well-conditioned fish in the hierarchy are more likely to acquire parasites because they ingest more food items and spend more time in sheltered habitats with depositing sediments where transmission mainly occurs. Second, the parasites may release factors that stimulate the host's immune and endocrinological systems to produce factors that enhance somatic growth and inhibit reproduction of the host. This benign relationship is considered to be

indicative of long-term coevolution. The sex of the fish had a significant influence on the abundance of the parasites

Parasites in Fish:

Marcogliese (2002) demonstrated that Helminth parasites of fish in marine systems are often considered to be generalists, lacking host specificity for both intermediate and definitive hosts. In addition, many parasites in marine waters possess life cycles consisting of long-lived larval stages residing in intermediate and paratenic hosts. These properties are believed to be adaptations to the long food chains and the low densities of organisms distributed over broad spatial scales that are characteristic of open marine systems. Moreover, such properties are predicted to lead to the homogenization of parasite communities among fish species. Yet, these communities can be relatively distinct among marine fishes. For benthos, the heterogeneous horizontal distribution of invertebrates and fish with respect to sediment quality and water depth contributes to the formation of distinct parasite communities. Similarly, for the pelagic realm, vertical partitioning of animals with depth will lead to the segregation of parasites among fish hosts. Within each habitat, resource partitioning in terms of dietary preferences of fish further contributes to the establishment of distinct parasite assemblages. Parasite distributions are predicted to be superimposed on distributional patterns of free-living animals that participate as hosts in parasite life cycles. The purpose of this review is first, to summarize distribution patterns of invertebrates and fish in the marine environment and relate these patterns to helminth transmission. Second, patterns of transmission in marine systems are interpreted in the context of food web structure. Consideration of the structure and dynamics of food webs permits predictions about the distribution and abundance of parasites. Lastly, parasites that influence food web structure by regulating the abundance of dominant host species are briefly considered in addition to the effects of pollution and exploitation on food webs and parasite transmission.

Gomez-Bautista et al., (2000) detected Cryptosporidium parvum oocysts in mussels (Mytilus galloprovincialis) and cockles (Cerastoderma edule) from a shellfish-producing region (Gallaecia, northwest Spain, bounded by the Atlantic Ocean) that accounts for the majority of European shellfish production. Shellfish

were collected from bay sites with different degrees of organic pollution. Shellfish harboring C. parvum oocysts were recovered only from areas located near the mouths of rivers with a high density of grazing ruminants on their banks. An approximation of the parasite load of shellfish collected in positive sites indicated that each shellfish transported more than 10(3) oocysts.

Water in Egypt:

Although industrialization is considered the cornerstone of the development strategies due to its significant contribution to the economic growth and hence human welfare, however, in most developing countries it led to serious environmental degradation. The earnest intentions are now not only targeting the qualitative and quantitative treatment of the industrial wastes but also attempting to avert their hazards to human health and restoring the quality of the environment.

Industrial and Agricultural Activities :

By the beginning of fifties, heavy industries were born in Egypt along the Nile Delta and in Cairo and Alexandria metropolitan areas. Chemicals, food, metal products, and textiles are the most prominent branches in Egypt (Hamza and Gallup, 1982). Undoubtedly, the impact of industrial pollution in Egypt appears in all environmental media: air, water, and land. Industrial releases to surface or groundwater are considered the major chemical threat to the agricultural land. The worst industrial waste liquids are those heavily laden with organic or heavy metals or with corrosive, toxic or microbially loaded substances. Such waters endanger public health through the direct use as well as through feeding with fish that live in the polluted streams **(Abdel-Dayem, 1994).**

In Egypt, food industry uses the largest volumes of water. Several studies revealed that untreated industrial wastes of more than 350 factories were discharged directly into the Nile and the Mediterranean, most of them released explicitly known toxic and hazardous chemicals such as detergents, heavy metals and pesticides **(RNPD, 1989).** Waterways used to receive about 85% of the industrial water withdrawals back. In addition, the Nile and its waterways currently suffer from the discharge of contaminated agricultural wastewater. The discharge of oil and grease originates from navigation and untreated domestic wastewater **(Abdel-Shafy et al., 1987).** Some groups of chemicals, such as carcinogenics, mutagenics and neurotoxins, are even unaffected by the usual methods of water treatment. The threats imposed by chemical discharges comprise contamination of drinking water supplies, phyto- and aquatic toxicity,

destruction of agriculture as well as fisheries, bioaccumulation, and biotransformation **(El-Kholy, 1993).**

Some spectacular threats to water resources and land are now quite obvious, e.g., in Helwan (south of Cairo): air pollution with cement dust, nitrogen and sulfur oxides, carbon monoxide, and other airborne pollutants resulted in the death of almost all the trees **(Hindy et al., 1990).** The industrial wastewater discharged from Helwan area amounts to some 45 million m^3/yr (RNPD, 1989).

In Shoubra El Khaima (north of Cairo) huge volumes of untreated industrial wastewater are daily discharged into agricultural drains. The textile industries representing 48.3% of the total number of industrial plants are the main contributors (almost 52%) to organic load. *Table 4* shows the distribution of organic load among the various industrial sectors **(El-Gohary, 1994)**.

The metropolitan area of Alexandria accommodates a multitude of industries in the vicinity of surface waters, e.g., in Amiria at the Lake Marriott, near the Mahmoudia Canal, etc. Out of 1243 industrial plants 57 were identified as major sources of marine pollution either directly or indirectly via Lake Marriott. Paper, textile and food industries contribute 79% of the total organic load **(Hamza and Gallup , 1982).**

As it might be expected, the mid-stream conditions of the Nile are still, on an average, at a fairly clean level owing to dilution and degradation of the pollutants discharged. The riverbanks, however, are much more polluted **(El-Gohary, 1994).**

Inefficient production in some industries (e.g., oil and soap) generates waste that contains raw material as well as products, a costly burden to the national economy and the consumer **(Abou-Elela and Zaher, 1998).** Evidently, application of an integrated preventive environmental strategy to processes and products to reduce risks to humans and the environment" **(Weston and Lambert, 1990).** Obviously, cleaner production is the unique answer for the industrial pollution in Egypt.

Type of load	Miscellaneous	Oil and soap	Starch yeast glucose	Pulp and paper	Metal Ind.	Plastic and rubber	Textile and dyeing	Total load
COD (kg /day)	1366.9	7006	3239.4	2322.3	11676.3	236.7	26372.3	52219.9
BOD (kg /day)	244.9	4568	1148	661.7	1257.7	77.9	8533.9	16492.1

Organic load contributed by the various industrial sectors in Shoubra El-Khaima **(Based on El-Gohary , 1994)**

COD : Chemical Oxygen Demands as organic load

BOD : Biological Oxygen Demands (5-day test) as organic load

Sewage, domestic and rural wastewater:

The alarming increase in the discharge rates of municipal and domestic wastes rendered the occasional primary treatment of urban sewage even insufficient to prevent further deterioration of vital water streams **(Parker, 1987).** Furthermore, secondary treatment cannot be satisfactory in emphasizing the quality of wastewater for reuse or in preventing further pollution with pathogenic bacteria and other microorganisms **(Cairncross, 1989)**. In the Nile delta, Bahr-El-Baqar is a paradigm of highly polluted waterways. As such, mixing drainage water with the freshwater for irrigation purposes makes the use of this water risky for public health.

Admittedly, the unused drainage water led into the lakes and the sea transfers its pollution burden to the coastal and marine ecosystems. Typhoid, paratyphoid, infectious hepatitis, and infant diarrhea are some endemic diseases indicating deterioration of water quality in Egypt. Despite the assiduous endeavours for public awareness through the media, the prevalence of Bilharzia substantiates the lack of rural sanitation against the traditional contamination of surface waters with human wastes, i.e., urine and faeces.

Water Quality:

Although agricultural, industrial and human needs essentially depend on the availability of freshwater, yet the present networks allow eventual mixing up of almost all agricultural and agrochemical drains as well as industrial and domestic wastes. Actually, the burden is too heavy to be tackled by the present water treatment plants. Accordingly, the agricultural network, irrigation and river systems do heavily suffer from excessive water wastes along their passage up to the northern lakes and seacoast. This problem imposes hazardous effects on the health of the rural people beside its vigorous impacts on the national economy. The current top concerns of wastewater management are **(Gaber, 1994):** (1)

effluent quality as its standards has become stricter and stricter over the years; (2) noise and odour nuisance control, as urban expansion has pushed built-up areas out to the doorstep of many once-remote treatment plants (e.g., gigantic treatment plant Al-Gabal Al-Asfar in Cairo); (3) disposal of sludge as a fertilizer in agriculture or as an ingredient of compost due to its content of phosphate, nitrogen, and organic matter, which is, however, made almost impossible by the presence of non-degradable heavy metals; and (4) the high cost.

Impact of pollution:

The above-mentioned aspects manifest that water pollution affects not only public health but also the economic factors relevant to water quality and natural resources of reusable waters. Based on this concept, in Egypt an appropriate law was issued for the protection of irrigating and drained waters from pollution. However, several obstacles largely hinder, so far, the optimal and effective acting of the law. In this context two problems can be noted:

The existing change in water quality as a result of pollution. The river Nile and the co-ordinated canals receive enormous amounts of biological and chemical pollutants. Before constructing the developed controlling system, assimilation of the river was possible within the period of the yearly flood. Hence, the Egyptian new regulation ought to restore the healthy state of the river in terms of physical, chemical, and biological characteristics. Ironically, all respectably taken measures failed in accomplishing the objectives and the studies undertaken confirmed the continuous deterioration of water quality all over the network of irrigation.

Impact of water pollution on processes of water treatment. Due to the alteration that occurred in the quality of river Nile and its canals, the conventional processes to treat drinking water by means of the currently implemented designs desperately fail in removing several biological and chemical pollutants.

WaterTreatment:

The current national plan for wastewater treatment, though highly expensive and ambitious, yet, fails in encountering the pressing environmental burdens. This is particularly true in the cities of complex industries, e.g., 10^{th} of Ramadan, northern Cairo. Primary treatment is always insufficient to accomplish

the objective for clean environment **(Cairncross, 1989).** Thus, it is important to adapt inexpensive and simple technology systems regardless to their large area requirement, particularly for sewage treatment (i.e., artificial wetlands and gravel bed hydroponics). The water treatment plants have got to face the following problems that largely affect the quality of water produced:

-The relatively high levels of alum dose and the relevant problems in terms of aluminum residues in water and the duly high expenses of water produced.

-The relatively high levels of added chlorine to raw water (prechlorination) to reduce total counts of bacteria and fungi and similarly the added chlorine to the filtered water (postchlorination). The high level of bacteria is ascribed to the drained sewage, which leads also to growth of fungi and to increased amounts of nitrogenous and phosphorous salts. As the dose of chlorine increases, it leads to increased concentration of organochlorinated compounds that are known as carcinogenic and mutagenic. Therefore, the well-known trend to replace chlorination by ozonation for disinfection is actively suggested.

-The currently implemented processes of water treatment are inefficient in removing residues of pesticides and organochlorinated pollutants. Furthermore, they are also insufficient in removing parasites, viruses, and other non-parasitic microorganisms. As a result, these residues of chemical and biological pollutants may persistently remain in drinking water.

*The growing levels of biological and chemical pollutants in raw water impose heavy burden on the efficiency of sand filters leading to blockages and development of microbial colonies, such as Nematode larvae which may eventually be present in drinking water.

Recommendations

• Be aware of the possibility of acute or chronic toxicity secondary to both local lawn spraying, home application and food intake.

• Educate patients regarding the known health concerns associated with pesticides.

• Encourage alternatives to pesticides including buying organic products

• using alternatives to pesticides for lawn and garden care as well as indoor pest management . The Toronto Environmental Alliance (TEA) has a Green Thumb Project where volunteer homeowners trained in chemical-free lawn maintenance will educate any interested homeowner.

• ensuring that local governments and business are not exposing patients or your community pesticides by local spraying or pesticide application. Cootie St. Luc, a Montreal area town has passed a bylaw prohibiting general applications of pesticides. Patients should be encouraged to organize similar endeavours in their communities.

*The water quality monitoring programs of the Nile water, drainage water and groundwater need to be integrated into one national efficient monitoring network, directed to stimulation, coordination and integration of already ongoing monitoring activities. The central functions should include detection of gaps and weak points and providing guidelines on how to supplement new activities in the ongoing monitoring activities.

*A central agency responsible for the management of municipal wastes, collection and treatment should be established.

*A well coordinated information system is needed to help the planners and decision makers to make proper water quality assessment in order to manage the water resources on an environmentally sound basis.

*The scientific and technical capabilities of the integrated planning for sustained and environmentally sound use and development of water resources should be enhanced.

Analytical and mathematical tools for an integrated management of water resources, with emphasis on water quality models should be implemented and applied.

*More effort should be directed to redesigning chemical processes to reduce waste of raw materials, to produce less by-product and to assure systems that recycle, purify or, otherwise, find use of by-products.

*The energy consumption should be controlled. Sometimes over half of the energy is wasted. This leads to the thermal pollution of waterways implicating the reduction of dissolved oxygen that renders the streams hardly capable of sustaining aquatic life.

*More emphasis should be placed on the cost/benefit aspects of low-waste technologies. Shortage of capital, lack of qualified labor or incentives of short-term returns should not therefore limit the level of technology acquired by industry. The bad condition of the equipment in some factories often results in the production of excessive waste as well as loss of raw materials.

*Industrial development projects that control pollution of the natural resources, particularly water supplies, will have a very large net positive economic impact and therefore should be given priority.

*The protection of water against pollution can be achieved better through control of pollution at the source. The challenge to industry is practically and economically accomplishing this task, without an excessive burden to itself.

*Legislation sensible to environmental control should depend on a thorough knowledge of the existing situation and careful assessment of its likely impact on the development.

*Detailed studies to minimize the quantity and improve the quality of wastewater discharged should be carried out for each industry. Research must take the objective of sustainability into consideration. Low input use and low levels of chemicals must have priority in research.

*Toxicological research should be promoted towards the early prediction of any chronic side effect of new compounds, particularly of carcinogens. Guidelines for management of their use and disposal are required.

*Integration of old and new lands in the distribution of resources and marketing is a positive sign of conservation of water supplies. Sustainability of water resources should be kept in terms of quality and quantity.

*The reuse of efficiently treated sewage water after/or without mixing with freshwater for land reclamation and irrigation of sandy soils should be optimised.

To avoid any pollutants to the food chain cycle, suitable plants should carefully be selected.

*Water should be introduced in the economic accounting of the various agricultural uses. Hence, a system of cost recovery to maintain the irrigation system can be established.

*A better distribution of the population among the regions is required to limit the deterioration of the environment and urban encroachment on natural resources, particularly water supplies.

*The use of proper and low technologies in agriculture limits the pollution of water canals and soil.

*The use of water hyacinth plants as animal food after mixing with grains **(Abdel-Sabour et al., 1997)** or to convert them to compost and biogas by means of anaerobic treatment is desirable (Abdel-Sabour et al., 2001).

*The foreign aids on which the Egyptian scientific research system is highly dependent are in jeopardy. Therefore, the creation of powerful self-support research system is of sensible top priority.

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سلامة الغذاء وتلوث البيئة

تقرير مقدم من

د/ طلعت محمد سحلول

الأستاذ المساعد فى التغذية وعلوم الأطعمة

بقسم الاقتصاد المنزلى - كلية التربية النوعية بدمياط - جامعة المنصورة

و ذلك ضمن متطلبات الحصول على

درجة أستاذ فى علوم الأطعمة

إلى

اللجنة الدائمة لترقية الأساتذة و الأساتذة المساعدين فى الاقتصاد المنزلى

مايو 2003

المقـــدمـــة

مما لاشك فيه أن الارتباط بين التلوث البيئى الذى زاد فى الآونة الأخيرة ، والغذاء وسلامته من أهم متطلبات الصحة العامة للإنسان فى كل بقاع المعمورة . فقد واكب هذا التلوث انتشار المرض الناجم عن تلوث الغذاء ، بل واستحداث أمراض لم يعرفها الإنسان من قبل ومن أمثلة ذلك مرض جنون البقر والأمراض الناتجة عن المواد الكيماوية الملوثة للبيئة بالإضافة للدايكسونات كما تلوثت الدواجن واللحوم والأسماك والخضر والفاكهة بالميكروبات ، هذا بالإضافة للاستخدام الواسع للمضادات الحيوية فى معالجة الماشية ، وهذا يضاعف من حاجة المستهلك لمزيد من الوعي فيما يتعلق بغذائه ، كما أن زيادة حركة وتنقل الأفراد بين العالم استوجب أيضا مزيد من الحرص . فقد رصدت المنظمة العالمية للسياحة ٦٩٨ مليون سائح عام ٢٠٠٠ سيصلوا لحوالى ١,٥٦ بليون عام ٢٠١٠ . مما يجعل من تلوث البيئة وبالتالي عدم أمان الغذاء مشكلة مؤرقة لهم .

لذا ومن أجل مواجهة ذلك فإن كلا من منظمة الأغذية والزراعة (FAO) ومنظمة الصحة العالمية (WHO) عام ١٩٩٩ أصدروا مواصفات عالمية شملت : - مضافات الأغذية ، كتابة البيانات وجودة الغذاء ، وطرق أخذ العينات ، والطرق المختلفة لتحليل الأغذية ومواصفات الغذاء الصحى والمتبقى من المعالجات البيطرية ، والمبيدات .

وترجع أهمية هذه المشكلة لكون الغذاء غيرالصحى مسبباً للكثير من الأمراض وخاصة بين الأطفال والمسنين وناقصى الحصانة – كما أن هذا يؤدى لخسارة اقتصادية بسبب نفقات العلاج والحجز فى المستشفيات والتغيب عن العمل . وقد رصدت منظمة الصحة العالمية عام ١٩٩٨ حوالى ٢,٢ مليون شخص ماتوا بسبب الإسهال منهم ١,٨ مليون طفل ، كما أن معظم الدول النامية تعانى من الأمراض الطفيلية ، كما تمثل المشاكل المرتبطة بتلوث الغذاء معظم وفيات الأطفال .

و يوجد سلسلة من العوامل والتلوثات البيئية سواء الصناعية منها أوالزراعية تهدد سلامة الغذاء منها الميكروبية التي تسبب آلام المعدة والحمى والقيء والإسهال وأمراض قاتلة ، كما أن الملوثات الكيماوية الناجمة عن الأفلاتوكسينات والملوثات العضوية الدائمة فى التربة POPs والديوكسينات والزئبق وخلافه كلها تؤثر فى الكبد والكلى وتؤدى للسراطانات المختلفة ، هذا بالإضافة للمضادات الحيوية التى تضاف فى تغذية الماشية ولو بكميات صغيرة للمعالجة ينجم عنها سلالات مقاومة لتلك المضادات ، أضف لذلك مخاطر الإشعاع والمواد المشعة على الغذاء وبالتالي صحة الإنسان ، والتعديلات الوراثية المختلفة فى الغذاء . كل ذلك يستدعى مزيد من الدراسة مع تعريف المستهلك بتلك المخاطر .

وكانت نسبة الحبوب الغير نامية ٠,٥ % فى عينة المقارنة فى مقابل ٦ % فى أقل تركيزاً من الإشعاع ولذا فإن الأغذية المشعة تصبح شبيهه تماماً بالأغذية المطبوخة رغم أن بيانتها تقول أنها طازجة . كما ثبت أن تشعيع الدهون يسرع من تزنخها .

ولو أن الإشعاع الإلكترونى المستخدم كان شديداً فإنه يؤدى لتخليق نواتج جديدة فى الغذاء . علماً بأن أكبر مدة للدراسة على الإنسان كانت لمدة ١٥ أسبوع فماذا يحدث لو أن المدة امتدت ... ؟؟

ولا يوجد دراسات على أطعمة الأطفال الداخل فيها مواد مشعة ، أما الدراسات على الحيوانات التى تناولت أغذية مشعة نتج عنها ظهور أورام خبيثة وفشل كلوى وتليف كبدى ، بالإضافة لذلك فإن الإشعاع عطل نشاط الإنزيمات فى جسم الحيوانات وإتلاف DNA .

ويذكر أحد علماء الـ FDA أن الموافقة على استخدام الإشعاع كان عام ١٩٨٢ من خلال ٥ دراسات على الحيوانات فى ذلك الوقت وهى فقط من ضمن ٤٤١ دراسة التى أعطت نتائج إيجابية فى تأثير الإشعاع على الغذاء .

علماً بأن المستهلك لا يعرف التفرقة بين الأغذية المشعة وغير المشعة دون ذكر بيانات تفصيلية عنها تذكر نوع الإشعاع ومدة التعرف ونسبة التالف من الفيتامينات المختلفة والإنزيمات وخلافه .

كما أن استخدام الإشعاع يغطى على المعاملات والتداول غير النظيف للإستخدام سواء أثناء ذبح اللحوم والدواجن أو نقلها وتداولها .

واستخدام الشعاع الإليكترونى حالياً يعنى إشعاع ذرى غير آمن فهو يخترق حوالى ١ – ١,٥ بوصة من كل جانب ولا يناسب فقط غير الأسطح ، فالأغذية الكبيرة الحجم كالفطائر المحشوة والفاكهة الكبيرة والأغذية المعبأة فى صناديق يحب أن يستخدم معها أشعة إكس أو جاما . والدول الفقيرة فى استخدام الشعاع الإلكترونى قد تستخدم المواد النووية مما يشجع انتشار الإشعاع الذرى فى العالم ، ويؤدى بالتالى لأخطاربيئة كبيرة .

علماً بان الإشعاع لا يقتل كل البكتريا فى الغذاء بل يتبقى بعض البكتريا تستطيع أن تتكاثر وتقتل فى فترة قصيرة للإعداد الأصلية ،كما أن بكتريا التسمم الغذائى البيوليتى لا تتأثر بالإشعاع وأيضاً فيروس البريون المسبب لجنون البقر . علماً بأن استخدام الإشعاع يجعل المنتجين فى غفلة عن الجوانب الصحية .

المخاطر الميكروبية

أنواع مختلفة من الميكروبات لها القدرة على إمراض الإنسان وهذا وبما يتراوح بين الإصابة المتوسطة والتى تؤدى للإخلالات الهضمية والشديدة الوطأة وهى أحياناً قاتلة وهى إما بالعدوى أو التسمم ، وعلى سبيل المثال البكتريا التى تستوطن فى الجهاز الهضمى مثل E.coli والسالمونيلا التى تسبب أنواع من العدوى واحياناً تصبح قاتلة . أنواع لأخرى من البكتريا مثل ستافيلوكوكس أيريس ، وباسلس سريس تنتج التوكسين فى الغذاء نفسه والذى يسبب التسمم فوراً بعد تناوله وهضمه وعلى العكس من ذلك التوكسين المخى (neurotoxin) الذى نتجه . كلوستريديم بوتيلينم يكون تاثيره بطىء فى إحداث المرض مع معدل وفيات أعلى إذا لم يتم التغلب عليه ،كما أن الفطريات يمكنها أن تنتج أفلاتوكسينات ، و بعض الفيروسات يمكن أن تقتل الإنسان عن طريق الغذاء مثل الفيروس الكبدى A.

مصادر الميكروبات :

مصادرها متعددة حيث تصل الغذاء من الهواء والتربة والماء والنباتات والحيوانات والإنسان ، مياة الصرف والمخصبات ، الملوثات الغذائية ، أجهزة المعاملة ،ومواد التعبئة . كما أن الميكروبات تصل للغذاء أثناء حصاده وتداوله ومعاملاته المختلفة . كما أن الإنسان أكبر مصدر لتلوث الغذاء من خلال الأيدى والشعر والتنفس والكحة الغير محماه وكذا العطاس Sneeze .

والميكروبات عامة تؤدى إلى تغيير الطعام بل وافساده بتغيير الصفات الطبيعية والكيماوية بحيث يصبح تالفاً أو تفرز سمومها فيصبح ممرضاً .

الميكروبات المرضية المختلفة : -

السالمونيلا Salmonella :

وهى عبارة عن ٢٣٠٠ سلالة منها ٢٠٠ سلالة ممرضة للإنسان وهى تلوث اللحوم والدواجن والأسماك المصنعة . كما أن التلوث فى الاغذية النباتية يكون مصدره الأساسى الماء المختلط به الصرف الصحى الغير معالج حيث يتم غسيل الخضر والفاكهة وتداولها فى الأسواق يزيد أيضا من محتواها من الميكروبات . علما بأن هذه الميكروبات يمكنها أن تنمو على أسطح الخضروات المختلفة وهى تسبب عموما الحمى التيفودية.

بكتريا القولون E-Coli :

وهى مستعمرة للأمعاء فى الإنسان والحيوانات ذات الدم الدافىء منها أنواع تسبب الإسهال وانتاج السموم المختلفة حسب أنواعها وكل الأطعمة تصاب بنوع أو أكثر منها وطريقة تأثيرها شبيهة بالسالمونيلا والشيجلا ، وقد لوحظ أن المرض بها يزداد إذا تناول شخص طعام أو ماء لدولة أخرى تختلف فى المواصفات الصحية عن بلده .

Compylobacter Jejuni

من الميكروبات التى تسبب إلتهاب الأمعاء فى كل الدول . ويوجد هذا الميكروب بأعداد كبيرة فى الحيوانات البرية والدواجن والابقار والخنازير بينما العدوى فى الغذاء تأتى من الأغذية ذات الأصل الحيوانى كما أنه مرافق أيضا للفواكهة والخضروات ، وهو لا ينمو على درجة حرارة أقل من ٣٠ م . كما أن حساس للحموضة .

Staphylococcus aureus:

ويوجد فى الأنف والدمامل كما يوجد فى اللحوم ومنتجات الألبان والخضروات ويفرز توكسين معوى يؤدى للإسهال والقئ وألام المعدة والتسمم به يظهر بعدحوالى ٣ ساعات من تناول الغذاء بل يعتبر الأكثر انتشارا وتستمر الحالة من ١ : ٣ أيام ونادراً ما يسبب الوفاة .

Clostridium spcies :

ومنها ميكروبات التسمم الغذائى Cl.perfringens , Cl.botulinum ومصدره التربة ويوجد فى الخضروات والفواكهة . كما أن معدل التنفس فى سلطة الخضروات يمكن أن يكون وسط مناسب لنمو اكللورستريديا وبالتالى يفرز توكسين ، كما أن الدراسات أظهرت أن استخدام البولى فينايل فى تغليف الأغذية تحت تفريغ يساعد Cl.botulinum فى النمو وإفراز التوكسين ولذلك من المهم لأن تلوث مواد التعبئة ذات صفات تمنع وجود ظروف لا هوائية حيث تساعد هذه العبوات التى بها بعض النفاذية لإخرج CO_2 وجعل الوسط هوائى علماً بأنه يفضل الحفظ على ٣ م على الأكثر .

Bacillus carus :

يلوث الغذاء من التربة ، ويستطيع أن ينمو على درجة حرارة الثلاجة وهو مرتبط غالباً بالفاكهة والخضر والتى ينجم عن تلوثها المرض وأحياناً يسبب الإسهال أو القىء ، كما أن التوكسين المقىء سلالاته تسبب فشل فى الكبد والوفاة بهذا المرض .

Vibrio spcies

وهو من البكتريا المنتشرة فى المياة وهى مرتبطة بالأسماك البحرية ، كما أن كلا V.cholera , V.paraheamolytica يسببا الأوبئة ويلوثا الماء من الصرف الصحى وتناول الماء الملوث بهذه البكتريا أو الأغذية المغسولة بهذا الماء يمكن أن يؤدى إلى انتشار واسع للكوليرا .

Virses

لا تنمو الفيروسات فى أو على الأغذية ولكن يمكن أن تفرز من المصابة بالعدوى وتعتبر الخضر والفاكهة ناقلة للعدوى كما أن أغذية عديدة مسئولة عن الإلتهاب الكبدى A .

وفى أحد الأبحاث عن مدى مسئولية الأفراد وتدريبهم على إنتاج الطعام الآمن حيث تم تكليف عدد ٥٢ فرد من غير المحترفين على أعمال المطاعم مقابل ١٠ أفراد مدربين لاكتشاف الفرق فى نسبة

التلوث بين أداء المجموعتين وقد كان وجود الميكروبات المرضية بنسبة ٩٦ % . مع الطهاة المنزليين بينما المحترفين (المدربين) كان التلوث ١٣ % .

فى أحد الأبحاث الحديثة وجد أن أحد سلالات E-Coli والتى تنتج توكسين انتشرت فى السنوات العشرين الأخيرة Vetrotoxin – Producing Escherichia Coli (VTEC).

والتى تسبب حالياً المرض للإنسان فى كل بقاع العالم مما ينبغى إحداث تغيرات فى المعاملات الغذائية . وهذا الميكروب يسبب من الإسهال المتوسط لتحلل الدم والذى يؤدى للوفاة .

وفى بحث آخر وجد أن الثوم له تأثير فعال لوقف نشاط كل من السالمونيلا واليستريا والإيشيريشيا كولاى وذلك على درجة ٢١ ، ٣٧ م ولم يختلف عن عينة المقارنة على ٤,٤ م وكان معظم الميكروبات التى ماتت تلك التى عوملت على درجة ٣٧ م وكان تأثير الثوم على وقف النشاط بطىء فى حالة اليستريا عنه فى حالة الميكروبين الآخرين .

البكتريا المقاومة للمضادات الحيوية

هى قدرة البكتريا على مقاومة التأثير الضار للمضادات الحيوية وهذه المقاومة لها صور مختلفة مثل :-

- امتلاك البكتريا لنظام يمنع دخول المضاد الحيوى للخلية .
- امتلاك البكتريا لنظام يحطم هذه المضادات الحيوية إذا أفلحت فى دخول الخلية .
- إمتلاك البكتريا لنظام داخلى يتوافق مع المضاد الحيوى داخل الخلية وبما يحجب فعالية المضاد الحيوى .
- كما أن البكتريا قد تحوى نظام يقذف بالمضاد الحيوى خارج الخلية قبل أن يؤثر فى الخلية .
- وإن كان بعض المضادات الحيوية تضاف لأغذية الحيوانات حتى تجنبها بعض الأمراض . وقد أدت هذه العملية إلى أن اكتسبت الميكروبات الممرضة للإنسان مناعة . والخطورة هى فى زيادة أعداد الميكروبات ذات القدرة على مقاومة المضادات الحيوية مما حيد كثير من المضادات المستعملة والتى لم يصبح لها قيمة فعلية كما كانت فى السابق – فمن ضمن ٤٠ مليون دولار تصرف على المضادات الحيوية فى الولايات المتحدة الأمريكية سنويا نجد أن ٤٠ % منها يذهب لحيوانات المزرعة كمضافات للأغذية لتحسين نموها .

وقد رصدت أحد الأبحاث الحديثة أن كلا من السالمونيلا والكامبيلوباكتر وهما ميكروبين مرضيين للإنسان قد زادت بصورة ملحوظة مقاومتهما للمضادات الحيوية . وهذ المقاومة ممكن أن تنتقل من الدواجن والخنزير للإنسان خلال السلسلة الغذائية . وبما يفرضه من مخاطر صحية جسيمة للبشر . علماً بأن الامر يأخذ سنوات لاستحداث مضادات حيوية جديدة . كما رصدت فى السنوات

الأخيرة إنبثاق سلالات من ستافيلوكوكساى مضادة للفانكوميسين وهو ذو التأثير الحاسم للقضاء على الميكروبات

و هذه السلالات أدت إلى تلوث الدم وإلتهاب رئوى ، كما ثبت أيضاً أن هذه الأصناف المقاومة يمكن أن تصل للإنسان عن طريق اللحوم ومنتجاتها ، علماً بأن العدوى بالكامبيلوباكتر بدأت فى الظهور كميكروب ممرض بعد زيادة استخدام المضادات الحيوية .

كما أنه منذ عام ١٩٦٩ ودول كثيرة تعهدت أن توقف بعض المضادات الحيوية التى تساعد فى تنشيط نمو الحيوانات إلا أنها لم تلتزم مما دعا WHO عام ١٩٩٨ ليشددوا من جديد مطالباً كل الحكومات لمراعاة ذلك . وبما دعا FDA الأمريكية عام ١٩٩٩ لوضع أطر معينة لإدارة هذا النوع إلا أن مقترحاته كانت ضعيفة فى نقطتين : -

- أنها ركزت على المضادات الحيوية الجديدة متجاهلة ما تم الموافقة عليه من مضادات حيوية سابقة .

- أنها لم تنصب بصورة كافية على مخاطر المضادات الحيوية .

حيث أن FDA ركزت على المضادات الحيوية التى لها أهمية طيبة للإنسان مع العلم بأن يصعب التفريق بين المضادات لحيوانية وأخرى بشرية ، مما يعطى الفرصة لزيادة تلك السلالات المقاومة .

ومما هو جدير بالذكر أن الميكروبات المقاومة للمضادات الحيوية تؤدى إلى تدمير الميكروبات المفيدة فى الأمعاء والتى تعمل توزان مع الميكروبات الضارة حيث يستلزم معالجة تلك الميكروبات بالمضادات الحيوية التى لا تؤثر فيها ولكن تدمر الميكروبات المفيدة فقط وبما يخل من التوازن الذى يجنب الإنسان من هموم السرطان وأمراض الضغط العصبى والقلب والكبد والكلى .

ولقد شبه العلماء زيادة الميكروبات غير المرغوبة فى الأمعاء وتغلبها على الميكروبات المرغوبة مثل تلوث الماء ومن ضمن هذه الميكروبات اللاهوائية وغير المرغوبة كميكروب كلوستريديم ديفيسل كما أن هذا الميكروب يزيد نشاطه وتغلبه على الميكروبات بعد العلاج بالمضادات الحيوية فانكوميسين وميتاروندزول وبما يخل بالنظام البيئة الأصلى للأمعاء . Intestinnel eco-system ولإعادة التوازن للأمعاء لابد من الرجوع إلى متشنكو ف رائد البروبيوتك بكتريا وهى الثورة الجديدة فى إعدة التوازن لبيئة الأمعاء . وذلك بإضافة بعض السلالات النافعة من اللاكتوبسلالى والبفيد وبكتريا والتى تقطن الأمعاء وتحافظ على التوازن البينى بها .

كما وضح أن بعض هذه الميكروبات غير المرغوبة فى الامعاء تنشط بعض الامينات الحلقية وجعلها مسرطنة مما ينبغى معه أن تكون الأغذية غنية بالألياف .

فى أحد الأبحاث تلاحظ أنه منذ عام ١٩٩٨ قامت بعض شركات تربية الماشية والدواجن (كرد فعل لقلق المستهلكين وبصورة تطوعية) بإيقاف استخدام منشطات النمو من المضادات الحيوية .

ولقد كان معروفا أن المضادات الحيوية المفروض أن تقضى أو تقلل الميكروبات المرضية ولكن فى الأبحاث الحديثة ثبت أن البنسلين الذى أعطى للخنزير زاد عدد البكتريا الكلية والأنتروبكتريسيه كما أن دراسة أخرى أظهرت أن أقوبرسين زاد من السلمونيلا فى الدواجن .

مرض جنون البقر

Mad Cow Disease

واسمه العلمى Bovine Spongiform Encephalopathy (BSE) أى الماشية ذات المخ الإسفنجى ،وهو مرض خطير يحدث فى مخ الأبقار ويؤدى إلى حالة من الجنون تستمر لفترة بعدها ينفق الحيوان وقد تم التعرف على المرض عام ١٩٨٦ فى اللملكة المتحدة ويعتقد أن فترة الحضانة لهذا المرض من ٤ – ٧ سنوات ، يصبح بعدها قاتل للماشية خلال أسابيع . كما يعتبر هذا المرض واحد من مجموعة Transmissible Spongiform Encephalopathies(TSEs) وهى مجموعة الأمراض التى تصيب الإنسان والحيوان ، وتتميز بأنها تسبب نوع من التدمير فى المخ وتجعله شبيه بالإسفنج . ويوجد أمثلة منه فى الأغنام والجمال والقطط البرية . كما أن TSEs يسمى فى الماشية Scrapie أى المرض المدمر الذى يحولها إلى فتات . والبعض يفسر إنتشار BSE فى بريطانيا إلى تناول الأبقار فى غذائها بعض منشطات النمو من جثث الأغنام النافقة والمصابة بهذا المرض . كما أن هناك إتفاق تام فى تصور الإصابة بحدوث تغير فى بروتين المخ المسمى بريون Prion theory وبالتالي يصبح البريون غير طبيعي ومؤذى وإن كانت النظرية الأخرى تفترض وجود فيروس شبيه بالبريون وبه أحماض نووية ، تحمل المعلومات ، وله القدرة على مضاعفة سلالاته .

ويرى علماء الأوبئة أنه انتشرمن خلال الحيوانات المجترة ومنها الماشية والإبل والغزلان ، ولا أحد يعرف ما هى أول بقرة أصيبت بالجنون ولكن المعروف أن المرض بدأ فى بريطانيا وذلك بسبب أكل الأبقار للحوم بإضافة جزء من مخلفات المواشي أثناء تجهيز غذاءها ، لذا فإن فضلات البقر المصاب بالجنون أصابت أخرى وانتشر المرض من خلال تلك السلسلة . وتبعاً لمعلومات بريطانية فإن ٢٠٠,٠٠٠ طن من تلك اللحوم والمحتمل تلوثها بمرض جنون البقر تم تصديرها لدول مختلفة. كما أن حوالى ١٠٠ دولة من دول العالم قد استوردت من بريطانيا أغذية حيوانية تحوى لحوم وعظام أبقار منذ عام ١٩٨٦ ، وبالتالي فإن كل هذه الدول تعتبر واقعة تحت طائلة الخطر ،كما أن دول غرب أوربا

حدث بها أيضاً حوالى ١٨٠ ألف حالة جنون بقر فى بلغاريا والدانمارك وفرنسا وأيرلندا ولكسمبورج وهولندا والبرتغال وسويسرا وباقي أوربا بالإضافة لإسرائيل . ولذا فإنه فى ديسمبر ٢٠٠٠ فإن منظمة الصحة العالمية أصدرت تحذير من تفشى مرض جنون البقر ، وألزمت الدول بتحريم تغذية الماشية على الأنسجة الحيوانية ومسحوق العظام .

مرض جاكوب (vCJD) Jakob disease, Creutzfeldt-Jakob disease

(جنون البقر البشرى)

فقد وجد أن هناك شكل آخر لجنون البقر يصيب الإنسان وأطلق عليه جنون البقر البشرى Human and cow disease ، وهو أيضا مرض شنيع ومهلك يصيب المخ ويؤدى لإختلال عقلى وفقدان فى التحكم فى الأمور الحياتية . وقد تم التعرف على هذا المرض فى بريطانيا أيضاً ولكن عام ١٩٩٦ . ومن هنا تم الربط بين مرض جاكوب ومرض جنون البقر من حيث التشابه التام والفترة التى ظهر فيها.

ولذا اعتبر أن التغذية بملوثات البقر المصاب سواء من أنسجة مخه أو حتى أكل لحمه وسيلة لنقل المرض من الحيوان للإنسان حيث لا يوجد أى احتمال لأن يقفز المرض من الحيوان للإنسان بوسيلة أخرى غير التناول فى الغذاء . ولحدوث المرض فى نفس الفترة ونفس المكان ، اعتبر البعض أن المرضين وجهين لعملة واحدة أى أن جاكوب هو نفسه جنون البقر . ولذا أصبح جاكوب مصنف كأحد (TSE) وكلاهما يتميز بوجود تدهور إسفنجى فى خلايا المخ . ويعتقد البعض أن أول مريض ظهرت عليه علامات هذه المرض كان عام ١٩٩٤ وقد ظهرت عليه فى البداية اعتلالات نفسية مختلفة وأعراض إنفصام فى الشخصية متبوعة بإضطراب عقلى ، وأعراض حساسية ،ولزوجة بالجلد ثم تدهور فى الأعصاب المخية ثم مع الوقت وقبل الوفاة مباشرة يفقد الحركة وحاسة السمع .

ويتوقع أحد خبراء فى مرض جنون البقر أنه مثل ما حدث فى جنون البقر أن هناك آلاف أوملايين قد أصيبوا من تناول لحومه المصابة خلال الإنتشار الوبائى فى عام ١٩٩٦ وهم معرضون جميعهم للإصابة ،حيث تم اكتشاف ١٥ حالة عام ١٩٩٩ ، ٤٢ حاله عام ٢٠٠٠ ولا يعرف بالضبط ما هى مدة الحضانة لهذا المرض فقد تصل حسب رأى بعض الخبراء إلى ٤٠ سنة وقد يصل بالتالى هؤلاء فى بريطانيا لحوالى ٥٠ مليون شخص .

وقد اظهرت دراسة حديثة فى المملكة المتحدة وجود علاقة وثيقة بين جنون البقر والمبيدات الحشرية وخاصة تلك التى استخدمت فى القضاء على الذباب ، وقد جاء بالدراسة أن البريون يرتبط مع المنجنيز فى العمود الفقرى للماشية عند استعمال المبيدات المشتملة على الفوسفور العضوى (OP) مما يؤدى لحدوث المرض . بحث آخر وضح أن إرتباط البيرون بالمنجنيز يؤدى لتلف المخ ومثله بريون مخ الإنسان الذى قد يتأثر باستخدام ليسيونات القضاء على القمل والتى تحوى الفوسفور

العضوى مما قد يؤدى لحدوث مرض جاكوب وبحث آخر يظهر أن البريون يمكن أن يرتبط بالنحاس ولكنه لا يكون مؤذى للمخ كما يحدث فى حالة ارتباطه بالمنجنيز .

وقد أقرت الحكومة البريطانية أن جنون البقر فى أغلب الإحتمالات قد أصاب الصغار والشباب . مع العلم أن هذا المرض لم ينتشر بالولايات المتحدة الأمريكية حيث تتغذى الماشية فيها على الأعشاب الطبيعية، ولذا قررت كل دول العالم تحريم أن يأكل البقر نفسه حتى يتوقف هذا الرعب .

الأطعمة المعدلة وراثيا

مما لاشك فيه أن هناك خطورة فى الأطعمة المهندسة وراثياً المتناولة هذه الأيام ، حيث أن الخطورة الناتجة عن طول مدة التناول لم تختبر رغم الإجراءات والتشريعات التى قامت بها كل المنظمات المرتبطة بالغذاء وحديثاً ظهرت أساليب وراثية جديدة لتعديل الغذاء منها ما يستخدم البكتريا والفيروسات لإدخال صفات وراثية جديدة للغذاء ، لذلك لا يعرف ما هو أثر ذلك على المدى الطويل ، وهذا يرجع لأن :

- الجين الوراثى المرسل للغذاء قد يعمل بطريقة مختلفة فى العائل الجديدة .
- كما أن الصفات الوراثية الأصلية للعائل قد يحدث بها تلف .
- كما أن إتحاد الجين المرسل مع جين العائل لا تعطى نفس التأثيرات الأصلية .
- علماً بأن الجينات المرسلة لإحداث التعديلات الوراثية تكون بين أنواع غير متشابهة

(غير مرتبطة بعلاقة مع بعضها) وذلك مثل الجينات المرسلة للنباتات وجينات الفيروسات وكذا جينات الميكروبات المقاومة للمضادات الحيوية لا تحدث فى الطبيعة ، ولذا فإنه يمكنها تخليق توكسينات جديدة وأمراض جديدة بل وإضعاف مع امتداد الوقت، وكل علماء التكنولوجيا الحيوية يؤكدوا على أن هناك جانب عشوائى ولو صغيرجدا فى كل وسائل الإدماج. – كما أن الباحثين فى مجال الوراثة بينوا أن هناك ضعف سواء فى النبات أو الحيوان أو الإنسان إذا تم تعديله وراثياً ناتج عن نقص فى الكود الوراثى مما جعل البعض يؤسس على ذلك إحتمالات الخطورة المستقبلية على الإنسان.

كما لا يمكن التنبؤ بالتاثيرات الصحية والأمراض الجديدة ، وذلك عندما يدخل الجين المرسل لكائن ما هنا لابد من وجود تأثير للوضع الجديد مما يستلزم التنبؤ بنموذج جينى ذو وظيفة وراثية أخرى ، فالبروتينات المنتجة بالهندسة الوراثية لها تفاعلات مختلفة وتنتج توكسينات من تلك التفاعلات . كما أن الفيروسات التى تعمل على نقل الجينل للنبات من الحيوان ، فإنها قد تفقد هذا الجين وتوصل جين آخر لأنواع أخرى . هذه المخاطر زادات لأن الباحثين حديثاً ووجدوا مادة الفيروس التى تستخدم فى اتحاد DNA يمكن أن تتحد مع فيروسات أخرى فى النبات أو الحيوان مما يؤدى إلى انتاج نسخة جديدة نشطة من الفيروس، وهذا قد يؤدى إلى فيروسات جديدة ، أمراض جديدة ، ميكروبات مقاومة

للمضادات الحيوية ، وتغير في الأجسام المناعية . كما قد تؤدى الهندسة الوراثية إلى حدوث تداخلات لـ RNA ينجم عنه تشوهات في الجزئ مثل ما يحدث في البريون الموجود بالمخ والذى يؤدى تغيره إلى أمراض مثل جنون البقر أو جاكوب في الإنسان ، كما أنه من المخاطر المحتملة دخول جينات من الفيروس نفسه داخل النبات أو الحشرة المراد تغييرها وراثياً قد يحدث مزج للجينات . بحيث ينجم عن ذلك فيروسات جديدة وهذا يذكرنا بفيروسات استجدت مثل فيروس الإلتهاب الكبدى B وفيروس HIV .

كما أن الهندسة الوراثية قد ينجم عنها تدمير للمحاصيل الأصلية أو قد تسبب أمراض للإنسان أو الحيوان لقدراتها المروعة .

والهندسة الوراثية للأغذية تحمل أخطار أكثر من الأغذية التقليدية لإمكانية إحداثها أنواع جديدة من الحساسية والسموم في الغذاء الذى كان آمناً من قبل ، كما رصد بالنسبة لنوع من فول الصويا منهدس وراثياً حيث ثبت أنه يسبب الحساسية .

وأكثر من ذلك أن هناك مئات من المنتجات المهندسة الوراثية وآلتي تستخدم في المعاملات الغذائية كمضافات للأغذية مثل المنفحة المستخدمة في تجبن اللبن والمواد المساعدة في الخبيز والمحليات والفيتامينات ومنشطات النمو وعصائر الإنزيمات ، ولا يعرف حتى الآن تأثير أى منها على المدى الطويل والكثير منها يتم خلطه مع الأنواع الأصلية دون ذكر ذلك في البيانات الخاصة به وبما يختلط على المستهلك . وإن كان قد ظهر زيادة في حالات الربو asthma بين عمال المخابز في ألمانيا مما جعلهم يعيدوا النظر في إنزيمات الخبيز المهندسة وراثياً .

كما أن المنظمات الغذائية المختصة لم تقف عند رغبات المستهلكين بأخذ آراءهم في هذه التكنولوجيا الحديثة ، ففى السابق كانت مأساة الثاليدوميد ، والتربتوفان ، DDT والتى سوقت لأسباب تجارية وثبت بعد ذلك أضرارها الصحية ، ولذا ينبغى استفتاء المستهلكين في مدى تقبل ذلك بعد توضيح كل ما يتصل بهذه التكنولوجيا .

وقد شهد نهاية القرن والعشرين ،زدياد في الذرة المهندسة وراثياً في أمريكا وأوربا ، وبعد شكوى المستهلكين من مشاكل في القلب بدأت شركات كبيرة مثل شركة نستلة وينيفلر تعلن أن منتجاتها خالية من الأغذية المهندسة وراثياً ، كما أنهم لن يستخدموا فول الصويا المهندس وراثياً في منتجاتهم . كما حرمت السوق الأروبية الذرة المهندسة وراثياً لظهور ١٣ حالة مرضية خطيرة مرتبطة بذلك .

كما ظهرت أصوات ومنظمات في كل دول أوربا تطالب بإعطاء ضمانات من الجهات المنتجة تؤكد لهم سلامة هذه التكنولوجيا .

الهندسة الوراثية للنباتات

من سنة ١٩٩٦ إلى ١٩٩٨ إزدادت المنتجات المهندسة وراثياً فى كندا ١٥ مرة ، كما زادت الإستثمارات الخاصة بهذه التكنولوجيا بحوالى ٤٠ % سنويا .

وتظهر فى الأسواق حالياً أنواع من الأغذية النباتية المتوحشة ذات الاحجام الضخمة كما أن معظمها غير مستساغ مثل الخس الكبير الذى يزداد فيه المرارة والطعم الغيرمقبول كما أن الطماطم الضخمة والمتنوعة فى الشكل والطعم جعلت المستهلك يرفض التعامل معها .

كما ظهر أثناء تطبيق تقنيات الهندسة الوراثية أنه أثناء نقل بعض الصفات الوراثية عن طريق البكتريا Agvobacterium فإنه قد حدث بطريقة ميكانيكية نقل أجزاء من DNA الخاص بها للنبات مما قد كون جينات جديدة داخل النبات تعمل كعائق لإثمارها .

المعادن الثقيلة

Heavy Metals

ترجع سمية المعادن الثقيلة لتفاعلها مع النظام الإنزيمي للخلايا الحيوانية أو بعض مكونات غشاء الخلية . كما أن المعادن الثقيلة يمكن أن تحل محل معادن الغذاء العادية وتتبادل معها المواقع .

كما أن التلوث بالمعادن الثقيلة يمكن حدوثه من تناول بعض الأغذية أو الماء الملوثين بها . كما أن تركيز هذه المعادن فى الأغذية حسب نوعها ، ظروف حفظها ومعاملاتها التكنولوجية . وعلى رأس هذه المعادن الثقيلة الرصاص والنحاس والكادميوم والزنك .

والعناصر المعدنية الهامة فى تغذية الإنسان هى الكالسيوم والصوديوم والبوتاسيوم والفوسفور والحديد والكلور والنحاس والزنك والمنجنيز والسيلينوم والأيودين والكروميوم والكوبلت والملوبدنيوم والفلوريد والأرسنيك والنيكل والسيلكون والبورون .

والجانب الآخر من العناصر المعدنية عبارة عن الألومنيوم والإسترنشيوم والرصاص والقصدير والزئبق والكادميوم وكثير منها سام للإنسان .

وهذه العناصر السامة يمكن أن تصل للغذاء من مصادر مختلفة منها : -

١- التربة التى ينمو فيها غذاء الإنسان والحيوان .

٢- مرسبات الصرف الصحى ، والمخصبات وكثير من المواد المستخدمة فى الأراضى الزراعية .

٣- الماء المستخدم فى معاملات الأطعمة وطهيها .

٤- الأتربة الملوثة للخضروات الغير مغسولة جيداً .

٥- الاجهزة والأوعية والأوانى فى المعاملات والطهى والحفظ .

وغياب المعادن الضرورية للجسم أو نقصه لفترة ينجم عنه بعض الأمراض .

أما المعادن غير الضرورية (المعادن الثقيلة) إذا كانت بكميات ضئيلة فيمكن التخلص منها خلال الجهاز البولى حيث أنها تعتبر مثل بعض الكيماويات الملوثة الموجودة فى الجسم لفترة قصيرة دون مشاكل أو تأثيرات كيموحيوية . وكلا النوعين زيادة تركيز أى منها فى الغذاء عن الحدود المطلوبة يؤدى إلى تأثيرات سامة لمن يتناولها ، كما أن تأثيرها يتوقف على طبيعتها وكميتها وشكلها الكيماوي وتركيزها فى الطعام .

ويوجد تأثير شديد لكل معدن بزيادة الجرعة ، فالكادميوم يعتمد تأثيره على المتناول . فالأغذية العادية يكون المتناول يومياً من الكادميوم بها ٠,٠١ - ٠,٠٤ مليجرام / كادميوم /يوم . والأغذية المحتمل تلوثها بالكادميوم لحم الخنزير والسمك واللبن والبيرة . أما مصدر التلوث بالرصاص فهى مواسير الرصاص ووصلات ومحابس التنكات من الرصاص ومن مصادر ماء الصرف والعلب المعدنية أو استخدام الخزف الزجاجي المستخدم فى حفظ المشروبات . وقد كان الرصاص المتناول من الطعام من المشروبات للبالغين فى مختلف الدول الصناعية ٢٥٠ – ٣٠٠ مليجرام / يومياً . أما بالنسبة للنحاس فليس بدرجة السمية كما يتصورها البعض . فالجرعة التى تبلغ ١٠٠ مليجرام / يوم يسبب تلف الكبد والكلى أما المتناول اليومى الطبيعى للشخص البالغ بين ١ – ٣ مليجرام ويصبح النحاس شديد السمية إذا كانت حموضة الطعام مرتفعة وعلى طول مدة تواجده ، أما الزنك فوجوده بالطعام مرتبط بوجود النحاس والأطعمة العادية بها من ٧ – ١٧ مليجرام يومياً حيث يمتص منها حوالي ٢٠ % ويقل امتصاصها أكثر فى حالة وجود كميات أكبر من الألياف .

ويلاحظ أن نسبة وجود الاملاح الثقيلة مرتفع فى الفاكهة والخضروات لإمتصاصها من التربة .

ولقد انتشر التلوث بالرصاص فى الآونة الاخيرة بصورة كبيرة وذلك بدءاً من ماء الشرب والهواء والتربة والخضروات المختلفة والمنتجات الغذائية الاخرى .

وفى دراسة بكرواتيا على تركيز المعادن الثقيلة بحوالى ٣٠ عينة من الأغذية المختلفة ومقارنتها بما سمحت بها منظمة الصحة العالمية وجد من ضمن الثلاثين عينة عينين تحوى كادميوم وخمسة تحوى رصاص وستة عشر زئبق بتركيزات أعلى من المسموح به ، أما الأرسنيك فكان أقل وكانت العينات التى بها زيادة عن المسموح به ١٧ عينة من الثلاثين . وكان التركيز التالى من كل من المعادن الثقيلة كالآتى :

الكادميوم ٠,٤١٨ مليجرام / كجم وهو أكثر أربع مرات من المسموح به ، والرصاص ٠,٠٧٤ مليجرام / كجم أكثر ١٥ مرة من المسموح به ، أما الزئبق فكان أكثر تركيز له ١٠,١١٧ مليجرام / كجم وهو اكثر ٣٥ مرة من المسموح به .

وفى دراسة أخرى تم تقدير نسبة تواجد خمس معادن ثقيلة وهى Pb ، Cd ، CR ، Ni ، Cu فى كبد وعضلات الأسماك فى البحر الأسود بتركيا وقد أظهرت النتائج أن كلا من الكروم والنيكل كان أقل من معدلاتها الطبيعية ، أما المعادن الثلاثة الأخرى وهى الرصاص والكادميوم والنحاس فكانت تركيزاتها مرتفعة ، بينما نفس هذه المعادن كانت أكثر إرتفاعا فى الأنسجة فى أغسطس ٢٠٠٠ وكان أقل تركيز لها فى مايو ٢٠٠٠ .

وفى دراسة حديثة عام ٢٠٠٣ وجد أن الجنين أكثر تأثراً بالزئبق مثيل والذى يعتبر من التوكسينات المخية من خلال استهلاك السمك الملوث به ، وقد قدرت نسبته فى دم السيدات بثلاث مرات من دم الاولاد . حيث بلغ ٠,٣٤ ميكرجرام لكل لتر لدى الاولاد بينما وصل ١,٠٢ ميكروجرام / لتر من السيدات . وبما يكون مردوده مضاعف لدى الجنين ، كما وضح من البحث أن معدل زئبق المثيل يزداد أربع أضعاف فى السيدات اللاتى يأكلن ٣ مرات من الأسماك فى آخر ٣٠ يوم مقارنة بالسيدات اللاتى لا يأكلن السمك نهائى فى نفس الفترة ، علماً بأن ٨ % من السيدات كان تركيز الزئبق فى دمهم أكثر مما هو مسموح فى المواصفات الأمريكية .

وفى دراسة أخرى تم تقدير الرصاص فى لبن الأم لبعض السيدات فى مناطق مختلفة بنيجريا ، حيث وضح من الدراسة أن نسبة الرصاص فى لبن الرضاعة يتجاوز الحدود المسموح بها فى ٥٦ % من الفتيات . وأن معدل الرصاص فى اللبن الماخوذ عامة يصل فى المتوسط ٩,٩ ميكروجرام / كجم / يوم علما بأن منظمة الصحة العالمية تحدد المسموح به ٥,٠ ميكروجرام / كجم / يوم . وهذا يبين أن لبن الام فى هؤلاء السيدات مصدرتلوث للأطفال وماذا لو قيس مقدارالرصاص فى دم هؤلاء السيدات .

بقايا المبيدات

Pesticides residues

فى العقود الثلاثة الأخيرة من القرن العشرين تم تحريم كثير من المبيدات سواء كانت مبيدات حشرية مثل DDT والألدرين ، HCH ومبيدات أخرى ، كل هذا بالإضافة للندين والكلوردان اللذان تم تحريمهما فى الزراعة مع تشديدات صارمة بهذا . وهذا التقييد تم الاتفاق عليه من قبل الحكومات عام ١٩٩٤ . ولكنه على مستوى التجارة ظل تداول هذه المبيدات بين التجار فى كل الدول وظلت منتشرة وخاصة بالوحدات الصغيرة ، حيث أن الكثير من العاملين فى مجال الزراعة ليس لديهم تعليم وتدريب كافي يؤهلهم لحماية النبات ، وبعد ٢٠ سنة من تحريم هذه المبيدات فى بلغاريا بدأت تختفي آثار معظمها من مياة الأنهار كما أن المتناول كم كل من DDT واللندان أصبح أقل مما هو مصرح .

انتشار المبيدات : -

تصل المبيدات للإنسان عن طريق طعامه فكثير من الاطعمة تحتوى على المبيدات المتبقية بالتربة . فقد ذكر تقرير الزراعة الكندى أن أحد أنواع الفاكهة احتوى على ٣١ مبيد الغالبية من هذه المبيدات تعمل فى نفس الموقع بالجهاز العصبى المركزى وتعتبر كلها ذات تأثير سام .

المـــاء :

لقد وجد أن المبيدات موجودة فى الماء بصفة عامة سواء فى الحضر أو المدينة . أما الماء الجوفى فقد وجد به ٣٩ مبيد حشرى وهذا من خلال دراسة أمريكية كندية . علماً بأن المسموح به من المبيدات تم قياس سميته بالنسبة للبالغين و مراجعة ذلك من خلال طب الأطفال الذين يتناولوا أكثر أربع مرات لكل كيلو جرام من أجسامهم بالمقارنة بالبالغين . ورغم التقييد الذى تم بالنسبة للمبيدات فإن المتبقى منه فى الماء يعتبر خطير على الصحة حيث أنه يرشح من التربة المخزن بها للماء ومن الطرق والمشروعات ورزاز الزراعة .

مخاطر المبيدات الحشرية

أهم مخاطره هو التأثير فى وظائف المخ ، وقد تم حصر التأثيرات الضارة للتعرض له . كما حددت لجنة تغذية الاطفال والمهتمين بالمبيدات أن وجود الفوسفور العضوى والكربمات ولو بكميات قليلة فإن لهما تاثير سام على مخ الاطفال ، كما أن أحدث الأبحاث فى سرطانات الطفولة أثبتت وجود علاقة بين الورم الخبيث فى الأنسجة ورش المبيدات الحشرية فى الأماكن المفتوحة وهذا أدى أيضا لزيادة الليوكيميا بين الأطفال .كما ثبت أن المبيدات المرشوشة على مبيدات الأعشاب وعلاقتها بالسرطان .

كما ثبت فى أحد الأبحاث أن التعرض للمبيدات لمدة ٢٠ يوم فى السنة يزيد من الليوكيميا ٦ مرات . ويعتبر الغذاء المصدر الأول لنقل المبيدات الحشرية ولذا فإن الغذاء يحتوى DDT فى اللحم والسمك والدواجن ومنتجات الألبان والخضروات الورقية والجذرية ، كما أن المبيدات من الكلورينات العضوية والتى ثبت أيضاً أنها مسرطنة موجودة فى كل الأغذية الطازجة شاملة اللحوم والدواجن والخضروات والفواكه والبيض .

حديثاً استحدثت مبيدات حشرية مثل الفنايل بيروزول يعمل على نفس المراكز وهو أقل سمية من السيكلودينات حيث أنه يحدث له تمثيل سريع وسرعة إزالة فى الثدييات كما أنه أقل سمية للأسماك عن البيروثرويدز مع معدل تراكم متوسط .

والأفضل من ذلك إستخدام المبيدات الكيموحيوية إلا أنها لا زالت تمثل نسبة ضئيلة لم تتدعى ٣,٨ % من المبيدات والتى تعتمد على استخدام الميكروبات مثل Bacillues thuringensis إلا أن

سعرها مرتفع مع مقارنتها بالكيماويات وهى تستخدم حالياً فى القمح والذرة والقطن وبنجر السكر والأرز والبساتين وإنتاج الفاكهة

وترى جمعية وقاية الاطفال من أخطار المبيدات أن هذه الملوثات تبدأ فيما قبل الولادة من خلال تراكمها وتمتد خلال مرحلة الطفولة حتى تصل إلى وجود أورام فى المخ . كما أثبت علماء آخرون وجود علاقة بين المبيدات الحشرية المستخدمة فى المنازل وزيادة الليوكيميا فى الأطفال بزيادة مقدارا أربع مرات عن المعدل الطبيعى . وآخرون أوجدوا نفس العلاقة بين المبيدات المستخدمة فى الحديقة وزيداة الليوكيميا للأطفال بزيادة قدرها ٥,٦ مرة عن الطبيعى . وآخرون وجدوا هذه النسبة تصل إلى ٦,٢ فى سرطان المخ .

لذ يجب مراعاة الآتى : -

- الوعى الكامل بالملوثات التى يمكن أن تنقل هذه المبيدات للطعام.
- زيادة الوعى بما تسببه هذه المبيدات للصحة العامة .
- التشجيع على إيجاد ومبيدات بديله لتلك الكيمياوية .
- يجب مراعاة المرضى الذين يتأذون من الرش العشوائى للمبيدات وكذا الاطفال .

ومن المدهش أن كل أغذية الأطفال والألبان المجففة خصيصاً للأطفال والأغذية المجهزة للأطفال المفطومين كلها ظهر بها مبيدات حشرية .

الطفيليات

يعتبر استهلاك المياة السطحية أو التعرض للتلوث أو الاستحمام فى الماء أهم الطرق لانتقالها كما أن انتقالها يحدث أيضا من الحيوان للإنسان أوحتى ملامسة شخص لآخر .

وعلم الأوبئة الخاص بالطفيليات يهتم بالعدوى التى تحدث للإنسان ومنها أنواع كثيرة مثل الأنتوميبا ، التكسوبلازما ، والساركوسيتس والأيزوبورا والكونتوسيرديوم والجياردیا .

ودورة حياة هذه الطفيليات مختلفة عن بعضها وكلها تحتاج المرور بالحيوان أو الإنسان كعائل وتنقل هذه اليرقة عن طريق البراز والذى قد يكون مباشرة أو غيرمباشر عن طريق الصرف الصحى أو ماء الرى إلى الفاكهة والخضروات حتى تتم الدورة . وهذا شائع فى مل الدول التى بها الإهتمام بالنواحى الصحية والنظام (خاصة جودة المياة) لا تلقى لإهتمام الكافى.

وامتداد الفقر والإنتفال للمدن وتصدير الغذاء والهجرة مع أساليب وتغيرات فى السلوك البينى مما أصبح معه الفروق بسيطه بين الشعوب .

وازدياد العدوى بالطفيليات مرتبطة بتناول الفاكهة الطازجة والخضروات وخاصة منها الورقية أو تلك التى تستخدم فى السلطات مثل الخس والطماطم والخيار والفجل والجرجير والجزر .

والطفيل Cryptospordium وجوده فى الغذاء يؤخذ كدليل على وصول براز الإنسان أو الحيوان أو الصرف الصحى الغيرمعالج للخضروات والفاكهة .

كما أن هذا الطفيل والطفيليات الأخرى مقاومة تماماً للكلورين أو المطهرات الأخرى فلا تقتل أوتزال من الأنسجة السطحية للخضروات أو الفاكهة . وبعمل مسح للصرفالصحى وجد أن الطفيل المسمى بالدودة المدورة Ascracis موجودة بأعداد كبيرة فى كل العينات .

أما الطفيل الترماتودا المعدى فهو المسبب لمعظم المشاكل الصحية فهو موجود عند حوالى ٤٠ مليون شخص فى جنوب شرق أسيا ، حيث تحدث العدوى منه بتناول النبات طازجاً أو من تناول الأسماك الغير مطهية جيد من أسماك الماء الغذب أو الأسماك الصدفية والذى تكون محتوية الطور المعدى لهذا الطفيل .

كما أن الطفيل المسمى Fasciola hepatica وهو أحد التريماتودات حيث يوجد ٣٠٠,٠٠٠ مصابين به فى ٥٥ دولة فى أفريقيا وأمريكا وآسيا وأوربا والدول الاكثر تلوثا به بوليفيا ومصر وإيران وبيرو وتحتاح سركاريا الفوشيولا لنياتات مائية معينة تتواحد فى الدول التى بها أنهار ولا تقاوم هذه النباتات . وللتحكم فى هذه الطفيليات يجب اتباع الأساليب البيئية المناسبة ومنها عدم استخدام السماد الحيوانى قبل تحلله واستخدامه فى تسميد النباتات كما أن التجميد والتسخين والتشعيع يمكن به القضاء على التريماتودا والمعاملة المناسبة تختلف من دولة لأخرى .

المياة فى مصر

على الرغم من أهمية التقدم التكنولوجى فى كل مجالات الحياة . والإسهامات المختلفة للزراعة والصناعة فى زيادة رفاهية الشعوب إلا أن هذه الأنشطة أدت إلى التدهورات البيئية المختلفة ، ولذلك فإن موضوع الساعة هو استعاضة البيئة المائية فى مصر لمقوماتها التى تجنب الإنسان المخاطر الصحية المختلفة .

ففى السنوات الخمسين الأخيرة نشطت الصناعة فى مصر وخاصة بدلتا النيل والقاهرة والإسكندرية سواء فى الصناعات الكيماوية أو السجية أو الصناعات الغذائية . وقد زادت الملوثات الناتجة عنها سواء فى الهواء أو الماء أو الأرض وانطلقت مخلفات المصانع للماء السطحي والجوفي وبما يهدد الأرض الزراعية ، وقد حملت مخلفات المصانع الكثير من الملوثات سواء كانت معادن ثقيلة أو توكسينات ومواد آكلة ومجاميع ميكروبية مختلفة . كما أصبح الماء يعرض صحة الإنسان للخطر من خلال شربه أو أكل السمك الناتج منه والذى يحمل كل هذه الملوثات فى أنسجته .

وفى مصر يستخدم كميات هائلة من المياة فى الصناعة ، ويلقى حوالى ٣٥٠ مصنع مخلفاتهم فى النيل أو البحر المتوسط وبما تحتويه من مواد سامة وخطرة كالمبيدات والزيوت والشحوم العضوية والبلاستيك وخلافه .

كما أن حوالى ٤٥ مليون متر مكعب بما يحتويه من مركبات نيتروجينية وكبريتية وأول أكسيد الكربون تلوث النهر تأتى من مدينة حلوان جنوب القاهرة – كما أن ملوثات شبرا تمثل ٥٢ % من الملوثات العضوية التى تصب فى النيل ، وكذلك ملوثات الأسكندرية والتى تلقى فى بحيرة مريوط وترعة المحمودية . والتى تحوى مخلفات صناعة الورق والغزل والنسيج والصناعات الغذائية .

كما تحوى مخلفات المصانع لبعض النواتج الخام التى تسبب تبخرها وتفاعلاتها فى الماء ونواتج أخرى تسبب درجة أكبر من التلوث .

المجـــارى والصـــرف الصحـــى

كميات هائلة من الصرف سواء فى الريف أو الحضر مع عدم القدرة على معالجة مخلفات الصرف الصحى مما ادى إلى تلوث نهرالنيل بكل الميكروبات المرضية . وفى الدلتا يعتر بحر البقر مثل حى للتلوث الشديد حيث تصب فيه مجارى القاهرة الكبرى .

لذا فإن البحر والبحيرات المختلفة فى مصر أصبحت مصدر للعدوى بالأمراض مثل التيفود والباراتيفود بالإضافة للبلهارسيا .

جـــودة الميـــاة

حيث أن المياة الملوثة هذه تستخدم فى الزراعة مما أدى للتدهور المستمر فى الإنتاج الزراعى ، كما أن الوسائل المختلفة المستخدمة حالياً فى تنقية المياة غيرمناسبة وغيركافية حيث لا يتم إزاله الملوثات تماماً .

تأثير التلوث

لا يؤثر التلوث فقط فى الصحة العامة ولكن فى الاقتصاد القومي بانخفاض غلة الفدان وعدم قدرة منتجاتنا الزراعية على المنافسة والتصدير وبالتالى عدم قدرة الفلاح على تغطية نفقات الزراعة، مما جعل البعض يهجر الأرض الزراعية ويتركها للبوار . كما أن نظام الرى الدائم الموجود حاليا، وخاصة بعد انشاءالسد العالى لا يتيح الفرصة لإصلاح التدهورالبيئى للمياة التى تعانى منه الأنهار والترع فى كل أنحاء مصر .

معالجة المياة

الخطة القومية لمعالجة المياة مكلفة جداً وخاصة فى الأماكن المكدسة بمخلفات الصناعة إلا أن هناك فى المدن الجديدة عامة محاولة لمعالجة الآثار البيئية ، ففى العاشرمن رمضان يتم عمل معالجات بيئية لمخلفات الصناعة وإن كانت أيضا غير كافية حاليا .

فمثلاً المعالجات التى تتم على المياة تواجه بعقبات منها : -

١- زيادة معدل المعادن الثقيلة بالمياة .

٢- وجود كميات كبيرة من الكلورين وهو مشابه للكلورين المضاف فى تنقية المياة الخاصة بالشرب ورغم أنها تقتل عدد كبير من الميكروبات الموجودة بالمياة إلا أن الأعداد الكبيرة المتبقية تعزو لزيادة معدلات الصرف الصحى بالمياة . كما أن مركبات الكلورين العضوية تؤدى للأمراض السرطانية والوراثية .

٣- كما أن معاملات المياة لإزالة المبيدات والكلورينات العضوية غير فعالة ،كما أنها أيضا غير فعالة فى إزاله الطفيليات والفيروسات والميكروبات المختلفة ، ولذا فإن هذه المبيدات لابد أن تنتقل لماء الشرب .

٤- زيادة معدل الملوثات الكيماوية والبيولوجية يزيد أعباء التكلفة حيث يسبب انسدادالرمل المستخدم فى ترشيح المياة ويزيد مستعمرات البكتريا ويرقات النيماتودا فى مياة الشرب .

توصيات لابد أن تؤخذ فى الإعتبار

١- مراقبة جودة مياة النيل من حيث مياة الصرف والمياة الجوفية بتكاثف كل الأجهزة الحكومية لمعرفة نقاط الضعف فى هذا الصدد .

٢- لابد من وجود وكالة منوطة بإدارة عملية الصرف ووضع أسلوب مناسب لمعاملات المواد المنصرفة .

٣- وضع نظام معلوماتى يساعد المخططين فى وضع نظام مناسب لتنفقية المياة .

٤- عمل التحليلات المختلفة وحساب التكلفة والأدوات المطلوبة لإدارة مصادر المياة .

٥- إعادة تصميم الوسائل الكيماوية والتى تقلل المواد الخام فى الصرف وبما يقلل النواتج الثانوية مع عمل نظام تدوير المخلفات .

٦- التأكيد على الفائدة المرجوة من تكلفة المعالجة وتدريب العمال وتوعيتهم .

٧- لابد من التأكد من مصدر المياة ومتابعتة من خلال الدول التى يمر بها نهر النيل .

٨- لابد من وضع القوانين والتشريعات المناسبة لحماية الماء من التلوث والمتابعة الصارمة من الجهات المعنية بمتابعة التنفيذ .

٩- عمل الدراسات المختلفة قبل القيام بالمشروع وفى كل الصناعات لتقليل أو الحد من التلوث بالمخلفات السائلة .

١٠- دراسة السموم وتأثيراتها الجانبية والمواد المسرطنة ووضع الإرشادات المختلفة للتحكم فيها .

١١- إعادة استخدام المياة العاملة دون خلطها مع المياة العذبة للاراضى الجارى إصلاحها أو الأراضى الرملية أو لزراعة الاشجار أو أى زراعات لا تدخل فى طعام الإنسان أو الحيوان .

١٢- يجب الإستخدام الإقتصادى للمياة فى الرى حتى لا يساء إستخدامه وينتج عنه كميات كبيرة فى الصرف .

١٣- التقليل من استخدام التكنولوجيا فى الزراعة يقلل التلوث .