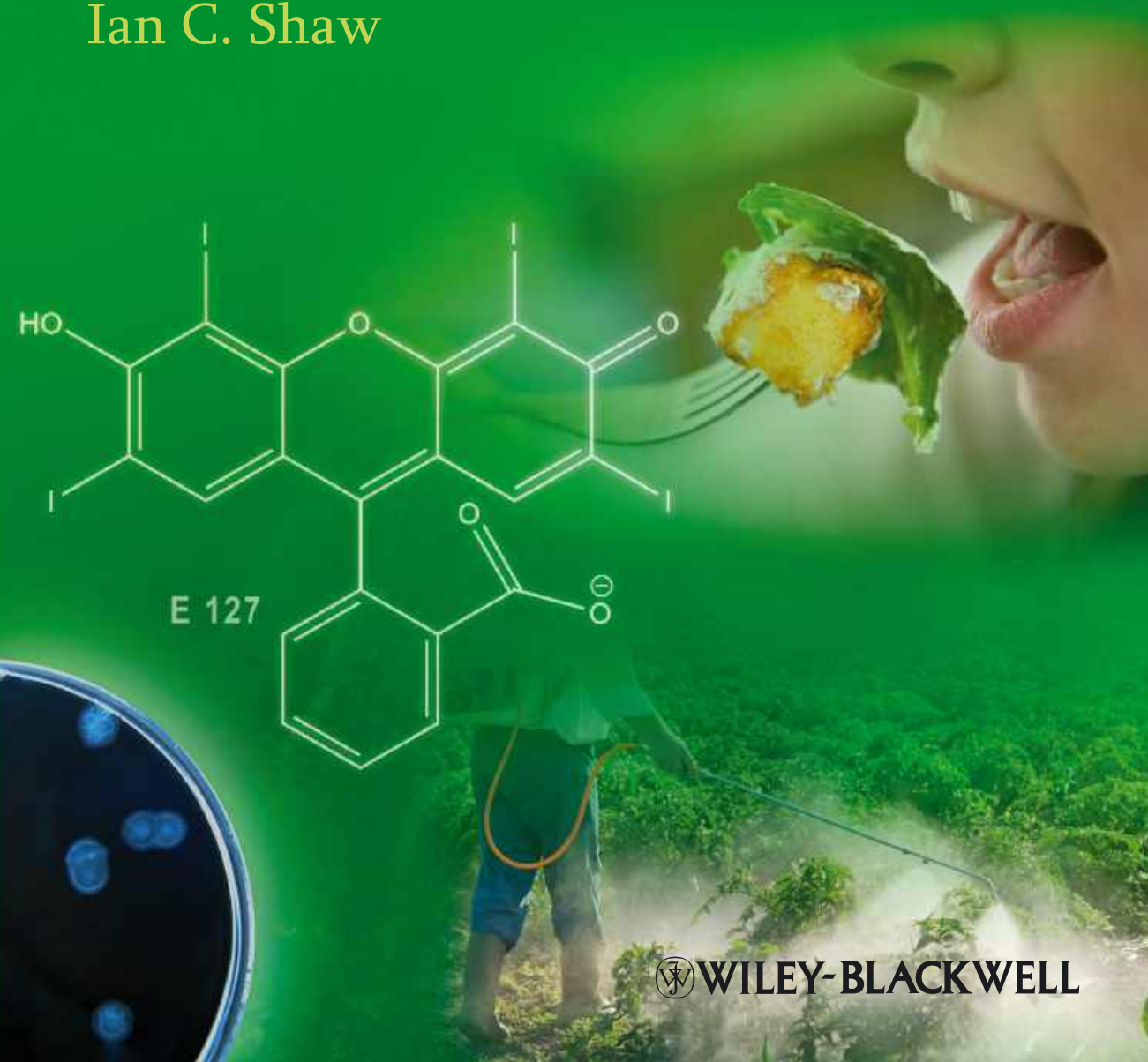
Food Safety

The Science of Keeping Food Safe

Ian C. Shaw



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Food Safety

This book is dedicated to the memory of two lovely ladies:
My mother
Audrey Shaw
28 November 1928 to 28 May 2009

My mother-in-law
Jeanne Zehms
6 May 1919 to 5 December 2010

They both inspired me in their own special ways.

Food Safety

The Science of Keeping Food Safe

Ian C. Shaw

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WILEY-BLACKWELL

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Contents

A colour plate section falls between pages 52 and 53

Preface

We expect our food to be safe; we certainly don't expect to be ill after eating a meal. However, it is important to remember that this is an affluent Western world expectation and that many undernourished people in the poorer parts of the world simply want to eat – the safety of their food is a secondary, or an even lesser, consideration.

Our desire for safe food, spurred on by food disasters like Mad Cow disease in the UK in the mid 1980s, has led to developed countries introducing legislation to ensure safe food – to make sure that food is fit for purpose.

In order to make food safe, we need to understand what makes it unsafe. Why do some microorganisms (pathogens) in food cause disease in their consumers, while others are harmless – or even beneficial? We need to minimise our exposure to food pathogens in order to minimise consumer risk. We need to understand why chemical food contaminants, like pesticides used in food production, can harm their consumers and we need to know the doses that are harmful so that we can set safe levels for chemical contaminants in food and so further minimise risk.

To store food we often use preservatives, otherwise harmful microorganisms might grow on the stored food; if we use chemical preservatives we must understand their potential toxicity to the consumer and make sure the chemical preservatives don't solve a microbiological problem, but introduce unacceptable chemical toxicity.

As consumers become more pernickety about their food they want it to look and taste exactly right – and by exactly right I mean how *they* think it should look and taste. To achieve this, colours and flavours are added to many pre-prepared foods. But are these additives safe? What are their effects on their consumers? Is using colours and flavours to enhance our food experience an acceptable risk?

Food is inextricably linked to health. If we eat too much fat or sugar we might become obese and our health will be significantly impacted – this might lead to heart disease or diabetes, both serious diseases. Some bacteria (e.g. Listeria) that might contaminate food cause serious diseases, even death. On the other hand, the contaminants and additives present in our food might affect our health in far more esoteric ways following very long-term exposure. For example, some food colours are known to cause cancer in rats at high doses, but what effects might they have on human consumers of infinitesimally tiny doses in food? Are these risks outweighed by the benefits of the chemicals? Is bright red cherryade worth the vanishingly low risk of its consumer contracting thyroid cancer? Do you *need* your cherryade to be bright red? Is any health risk associated with food colour acceptable – however small?

These are all fundamentally important questions – and there are many, many more – to which we should seek answers if we are to make our food safer. We need to understand the science that underpins food safety; we need to tease out the health effects of chemicals in our food and set these risks against their benefits. Is the risk of a bacterium growing in our food greater than the chemical used to kill it? Why is the chemical harmful to its consumer? Could we modify its molecule to make it less toxic, but maintain its bactericidal properties? These are some of the answers we might need to help us to produce and regulate our food and make it as fit for purpose as possible.

Over the last 50 or so years our understanding of food safety has grown to such an extent that we no longer accept food-borne illness as a consequence, albeit rare, of eating. Those responsible for food-borne illness outbreaks can fall foul of strict food legislation and find themselves subjected to heavy fines or, in rare cases, even imprisonment. Just 50 years ago this would not have been thought possible.

My book takes a trip through the world of food safety, from microbiological food pathogens, through chemical contaminants, natural toxins and the chemicals we use to colour, preserve and flavour our food. It grapples with the esoteric prion that causes Mad Cow disease which led to the collapse of the UK beef industry and prevents me as a Brit living in New Zealand from donating blood because of the perceived risk of transferring the prion to my fellow New Zealanders. It uncovers the controversy of 'organic' food and food irradiation. Finally, it looks at the laws that are used to make sure that when we eat our dinner or buy a snack on the street we don't contract a food-borne illness or expose ourselves to chemicals that might compromise our health in the future. This is a long journey flavoured with many examples from around the world; I hope you enjoy it!

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Christchurch, New Zealand
September 2012

Acknowledgements

I thank Wiley-Blackwell for asking me if I would write a textbook on Food Safety, and, in particular, David McDade for steering the idea to contract and Andrew Harrison for trying to keep me on schedule and for gently prompting me by e-mail when I missed deadlines by a country mile. He never once raised his e-mail voice even though this would have been justified on many occasions. In addition, I am indebted to Alison Nick for her supreme proof reading skills and frightening efficiency.

As with any wide-ranging textbook, there are subjects included that the author is less *au fait* with; in my case food legislation was the subject that I needed expert help with. I thank Keith Zehms and Sharon McIlquham for their advice on US law and John Reeves for his help with the New Zealand food legislation.

When I agreed to write the book and signed the contract on 21 January 2010 I could not, in my worst nightmare, have anticipated the devastation that the 4 September 2010, 22 February and 13 June 2011 Canterbury (New Zealand) earthquakes would bring to my life and environment. Much of this book was written during a period of regular and significant aftershocks, telecommunication failures, lack of internet, uncertainty about the stability of buildings, workmen everywhere, continual battles with the New Zealand Earthquake Commission and our insurers, and deep, deep sadness for the loss of our city and some of its people. Throughout this, my partner, David Zehms, gave me unwavering support and provided some semblance of emotional normality that allowed me to retire to my cracked and crumpled study to write this book – thank you David.

As you read, think of the people of Christchurch and Lyttelton, New Zealand, who have lost so much and have a long, hard road ahead.

I hope you enjoy my book.

Ian C. Shaw
Christchurch, New Zealand

Chapter 1
Introduction

Introduction

Food safety is a relatively recent 'invention'. It was introduced in the developed world to increase confidence in food. In our modern world it simply is not acceptable to have food that might make us ill. Sadly even now a good proportion of the world's people are very much more concerned about getting food and stemming their unrelenting hunger than they are about whether they might get a stomach upset as a result of eating the food. We must always remember these horrifying facts when we study food safety. Food safety and the legislation emanating from it are for the relatively rich countries that have the luxury of having sufficient food to allow them to make rules about what is safe to eat.

A brief history of food safety

Prehistoric times

The risk of eating in prehistoric times was very much more an issue of the dangers of catching the beast to eat than the ill effects suffered after eating it. To survive, cavemen had to eat and their animal instincts dominated their behaviour with respect to food. These instincts, no doubt, made them avoid food they had learned made them sick, but their overriding instinct was 'eat to live'. Some foods, however, might have been so toxic that they threatened the early man's survival. Behaviour that minimised consumption of toxic food would have been selected in because individuals that succumbed to toxins in their food simply did not survive. This is the raw material of Darwinian evolution and could be considered a very early manifestation of food safety issues! Whether this happened or not thousands of years ago is impossible to know, but we do know that modern-day animals avoid toxic plants in their diet. This might be because some of the toxins (e.g. alkaloids) have a bitter taste that warns the would-be consumer of the risk. Prehistoric man probably behaved in exactly this way which is why he was able to survive in such a harsh environment in which every day posed new and unknown food challenges.

Food Safety: The Science of Keeping Food Safe, First Edition. Ian C. Shaw.

This is hardly prehistoric food safety policy, but it illustrates our inborn survival instinct that extends to the food we eat. We have an innate desire not to eat something that will make us ill. This has not changed over the millennia.

Evolution of cellular protection mechanisms

It is important to remember too that our metabolic systems (and avoidance strategies) evolved during the tens of thousands of years of prehistoric times. Metabolism of toxins from food in order to reduce their toxicity and so make the food 'good' developed over millions of years. There are highly complex metabolic systems 'designed' to detoxify ingested toxins that evolved long before man, but the enzyme systems from the primitive cells

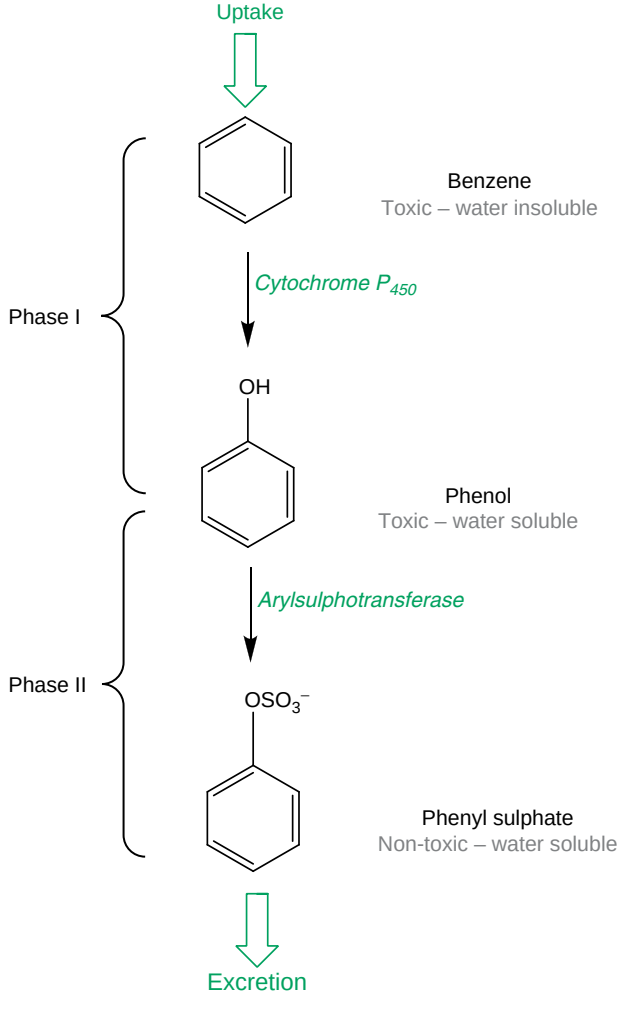


Figure 1.1 Phase I and II metabolism for a simple compound, benzene, showing how the molecule is detoxified, made water soluble and excreted (e.g. in urine).

in which they evolved were selected into the human genome through the evolutionary process and were inevitably expressed by the earliest hominids. These detoxification systems gave man an advantage because he could eat food that contained chemicals which if not detoxified would make the food too toxic to eat. These enzyme systems are now very well understood; they include the cytochromes P_{450} mixed function oxidases (termed Phase I metabolism) and the conjugating enzymes (termed Phase II metabolism) (Figure 1.1).

There are many food toxins that are detoxified by these systems, so making the food safe to eat (this will be discussed further in Chapters 7 and 8); for example, parsnips contain bergapten, a photosensitising toxin that also causes cancer (see Chapter 8, *Furocoumarins in parsnips, parsley and celery*); bergapten is detoxified by Phase I and II metabolism (Figure 1.2)

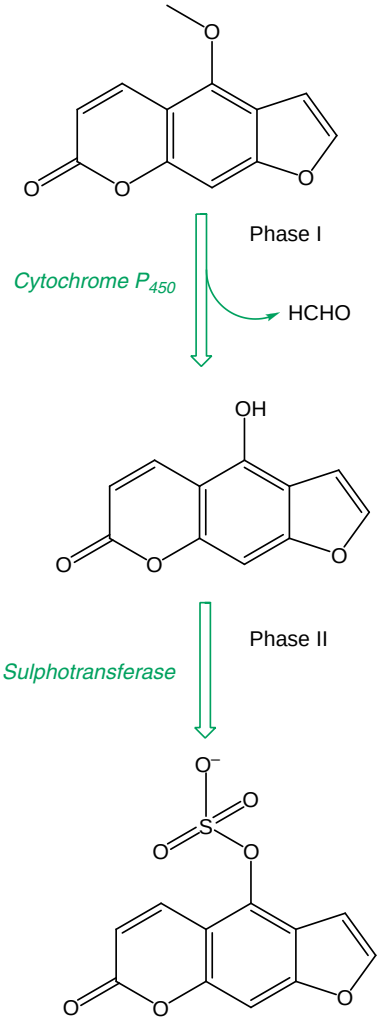


Figure 1.2 A proposed metabolic pathway for bergapten.

thus making parsnips safe to eat. These metabolic processes are the cell's internal food safety mechanisms and broaden the range of foods we can eat without suffering the ill effects that some of their components would cause.

There are significant differences in the susceptibility of different animal species to toxic chemicals; these are due to the evolutionary selective pressures under which the particular species developed. This means that safe foods for some species might be highly toxic to others. For example, the toxin in the swan plant (*Asclepias fruticosa*), labriformidin, is very toxic to birds but harmless to the monarch butterfly (*Danaus plexippus*) (see Chapter 8, *Why produce natural toxins?*).

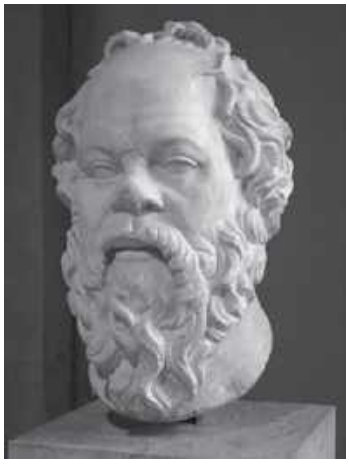
The monarch butterfly uses this differential toxicity as a means of protection. Its caterpillar eats swan plant leaves and incorporates labriformidin into its body; this makes it toxic and unpalatable to predatory birds. This interesting means of survival is by no means unique amongst animals. Indeed, some plants that are eaten by animals are very toxic to humans. For example, it would only take a few leaves of hemlock (*Conium maculatum*) to kill a person, but the skylark (*Alauda arvensis*) is unaffected by its toxin (Figure 1.3). Indeed, there have been cases of human poisoning in Italy following consumption of skylarks which (strange as it may seem) are a delicacy in that country. The toxin in hemlock is coniine (Figure 1.3) – it is very toxic; about 200 mg would be fatal to a human. Hemlock was the poison used to execute Socrates in 399 BC for speaking his mind in the restrictive environment of ancient Greece.

Tudor England (1485–1603)

In the 1500s I doubt whether many people thought about illness being linked to what they had eaten, but I imagine food-borne illness was prevalent in that rather unhygienic society. In fact spices were introduced into Tudor England to mask the putrid taste of some foods particularly meat – this is a 'head in the sand' approach where masking the bad taste was thought to take away the bad effects. Whether the Tudors thought that masking the taste of putrefying meat stopped them getting ill I cannot know, but they certainly thought that masking the terrible smells of putrid plague-ridden London prevented them catching fatal diseases like the Plague. The gentry used, amongst other things, oranges stuck with cloves, and ornate necklaces with receptacles for sweet-smelling spices and resins (pomanders – derived from the French *pomme d'ambre* meaning apple of amber; ambergris, a sweet-smelling substance produced by sperm whales was often used to scent pomanders) to waft in front of them to take away the evil smells as they walked the streets. This is hardly food safety legislation, but it might just be the beginning of people connecting off-food with illness – a key step in making food safe.

The times of King George III of England (1760–1820)

The Georgian era was a time of great social division. The rich ate well, if not exuberantly, and the poor just about found enough food to keep them alive. The idea that bad smells were associated with disease prevailed as



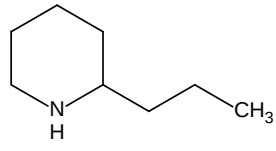


Figure 1.3 Socrates (469–399 BC), coniine, the poison from hemlock used to execute him, and the skylark (*Alauda arvensis*) which is unaffected by coniine. (Pictures from http://en.wikipedia.org/wiki/File:Socrates_Louvre.jpg, © Sting; http://en.wikipedia.org/wiki/File:Alauda_arvensis_2.jpg, © Daniel Pettersson; photograph of hemlock taken by the author.)

did the naïve thought that if the smell was masked, putrid food was good to eat. Susannah Carter, an American cookery author, described a 'method of destroying the putrid smell which meat acquires during Hot Weather' in her book *The Frugal Housewife, or, Complete Woman Cook*, published in New York in 1803. Some people must have been very ill after eating food prepared under this rather naïve food safety philosophy; i.e. bad smell means high risk and hiding the smell minimises the risk. I wonder if they connected their stomach upset with the food they had eaten? Probably not because such illness would be the norm in the 1700s and people probably simply took it for granted.

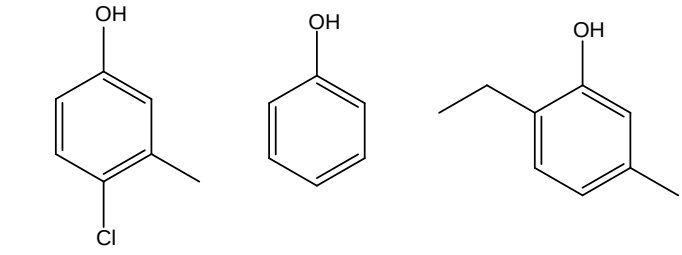
Figure 1.4 Louis Pasteur (1822–1895). (Picture from http://en.wikipedia.org/wiki/File:Louis_Pasteur.jpg.)

The 1800s – Pasteur's Germ Theory, Lister's antiseptics and the first refrigerators

In the mid 1800s in Europe there was a significant improvement in the understanding of disease and, in particular, public health. This was the time that the connection between microorganisms and disease was beginning to be understood. Louis Pasteur (1822–1895; Figure 1.4) proposed the Germ Theory of Disease while he was working at the University of Strasbourg in France in the 1860s. He later extended his understanding of 'germs' to propose that heating contaminated broths to a high temperature for a short time would kill the 'germs'. This is the basis of one of today's most important methods of assuring safe food – pasteurisation.

Disinfectants

Joseph Lister (1827–1912) followed Pasteur's work with his discovery of antiseptics. He showed that carbolic acid (phenol; Figure 1.5) killed germs and reduced post-operative infection. This revolutionalised surgery, which was often a sentence of death pre-Lister. The people of Victorian England embraced scientific development – they were fascinated by science and were keen to understand and use it. Lister's antiseptics were modified and developed and became the carbolic and creosote disinfectants that were used to keep Victorian (1837–1901) homes free of germs. There is no doubt that this 'clean' approach to living reduced food-borne illnesses in the kitchens of the Victorian upper classes. The lower classes were still scrambling to get enough food to feed their large families and probably knew nothing of the new-fangled theories of germs and antiseptics. A disinfectant fluid was



3-Methyl,4-chlorophenol (chloro m-cresol) | Phenol | 3-Methyl,5-ethylphenol (ethyl m-cresol)

Figure 1.5 Molecular structures of some of the components of Jeyes' Fluid, a very effective disinfectant introduced in Victorian times.

patented by John Jeyes in 1877 in London which was a product of the increased interest in 'germs' and antiseptics and was based on Lister's phenol. Jeyes' Fluid comprises 5% 3-methyl,4-chlorophenol (chloro m-cresol) and 5% alkylphenol fraction of tar acids (these were a by-product of the coal industry; Figure 1.5); it is still used today.

Refrigeration

It has been known for a long time that food keeps better when it is cooled. The Victorians equated this with suppression of the growth of spoilage germs and introduced complicated means of keeping their food cool. Refrigerators, as we know them now, were not introduced until the 1860s, but before then 'iceboxes' were used in which large chunks of ice kept the food cool. The production of ice was not an easy task either – this is a circular problem; without refrigeration it is difficult to produce ice. In the early days, ice was collected during the winter and packed into ice houses, then the ice houses were used for storage of perishable food. With good insulation the ice could be maintained for a good proportion of the year in temperate climates. Later ice was made using cooling chemicals and water. For example, when diethyl-ether evaporates it takes in heat, thus cooling its surroundings; the cooling property of ether was used to freeze water for iceboxes. There is no doubt that the increased availability of iceboxes increased the safety of mid-1800s' food. In the 1860s, the Industrial Revolution was under way; the developed world was enthralled by mechanical devices and commercial, large-scale manufacture. Long-haul transport became important as a means of moving products, including food, around and between nations; this led to a renewed interest in cooling devices both to keep food cold at home, and, perhaps more importantly, to allow food to be transported long distances without spoiling. Since the problem of food spoilage was more acute in hot countries, it is perhaps not surprising that it was a man from Scotland living in Australia who appreciated the need to cool food. This man was James Harrison (1816–1893) and he developed one of the first mechanical cooling devices based on the compression and expansion of a volatile liquid (when liquids evaporate – remember the ether example above – they take up heat). Harrison was granted a patent for the vapour-compression refrigerator in 1855. He used

Figure 1.6 A cow creamer. (Photographed with permission from the collection of Mrs S. Drew, Christchurch, New Zealand.)

this device to manufacture ice for the first attempt to transport meat from Australia to England in 1873. Unfortunately the ice melted before the ship arrived in England and the meat spoiled. It was not until 1882 that the first successful shipment of cooled meat was made from the antipodes to England and it went from New Zealand not Australia.

Refrigeration revolutionised food safety and continues to be used as one of the main ways we keep our food safe in the 21st century.

It is clear that the Victorians were aware of hygiene and its link to health. Mrs Beeton's *Book of Household Management* (published 1861) has many tips on hygiene; she advises suspending chloride of lime (calcium hypochlorite – $Ca(ClO)_2$)-soaked cloths across the room. Chloride of lime slowly liberates chlorine gas which is a powerful antiseptic. Such methods would have killed bacteria and therefore made food preparation more hygienic.

There are some good examples of the Victorians' concern about food hygiene. For example, they loved intricate, delicate china to accompany afternoon tea. Milk was served from creamers (small jugs) sometimes shaped like cows. Cow creamers (Figure 1.6) disappeared in the late 1800s because of concerns about hygiene – it was very difficult to clean them properly because of their intricate design.

Chemical preservatives

Food spoilage and food-borne illness can also be prevented by using naturally produced chemicals to kill bacteria or significantly reduce their growth rate. Some of these methods are very old. For example, fermentation; here 'good' microorganisms are used to produce natural preservatives in the fermented food. Salami manufacture relies upon fermentation. The acid products of the fermentation process (e.g. lactic acid) preserve the meat by inhibiting the growth of pathogens and spoilage bacteria which do not thrive in acid conditions (see Chapter 11, *Antimicrobial food preservatives*). On the

other hand, yoghurt is simply milk infected with good bacteria (traditionally *Lactobacillus bulgaricus* and *Streptococcus thermophilus* and more recently *L. acidophilus*); these bacteria colonise the milk so effectively that they prevent harmful bacteria growing. Yogurt production, as a means of preserving milk, has been known for at least 4,500 years and probably began in Bulgaria.

Some chemical preservatives are added to food to prevent food spoilage. Some of these preservatives have been used for thousands of years. Vinegar (acetic acid; ethanoic acid) produced by fermenting ethanol (originally from wine) is a good example; traces have been found in Egyptian urns from 3,000 BC and it is still used today to pickle vegetables (e.g. onions) and make chutneys, etc. The acidity of vinegar inhibits most bacterial and fungal growth, thus preventing food spoilage – the principle is the same as described above for food preserved by fermentation, but, in this case, the acid is added to the food rather than being produced by fermentation of the food (see Chapter 11, *Other organic acids*).

Sugar is also used as a preservative. If the concentration is high enough it too prevents bacterial and fungal growth by scavenging the water that microbes need to survive (sugars form hydrogen bonds with water, thus effectively removing the water from the system). Sugar, either in the form of refined sugar (sucrose) or honey (mainly fructose), has also been used for thousands of years to preserve food. Jam is simply fruit boiled with sugar and bottled aseptically. Sugar can also be used to bottle or can fruit which involves heating the fruit in a strong sugar solution in jars and sealing the jars aseptically. Both bottled fruits and jams will keep for years.

There are also many modern means of preserving food using gases (e.g. nitrogen) and chemicals (e.g. sodium benzoate) to inhibit microorganism growth, or using irradiation (see Chapter 12) to kill them. These techniques are associated with risks to the consumer and therefore are often controversial; we must not forget, however, that the risk of harm following exposure to a food pathogen is likely to be greater than the risk of the method of preserving the food (this will be covered in detail in Chapter 11). However, there is no doubt that pickling with vinegar and preserving in sugar represent a negligible risk to the consumer … unless, of course, you eat too much of the sugar-preserved food and your teeth decay and you become obese!

Sodium benzoate itself has a very low toxicity – no adverse effects have been seen in humans dosed up to 850 mg/kg body weight/day. However, in the presence of ascorbic acid (vitamin C) sodium benzoate can react to form benzene (Figure 1.7) which is a carcinogen. Since many foods that sodium benzoate might be used to preserve might also contain ascorbic acid, perhaps the risk is not worth the benefit. On the other hand, benzoic acid is present at low concentrations naturally in some fruits (e.g. cranberries) and they contain ascorbic acid too, so you cannot avoid the risk if you choose to eat these foods. Sometimes 'natural' is not good (See Chapter 8 for many more examples), but whichever way you look at it the risk is very low indeed (see Chapter 2).

For cats, the risk of cancer following benzene exposure via foods preserved with benzoate is significant because cats have very different routes of metabolism to humans and are unable to detoxify benzoate efficiently and so benzoate itself is toxic to cats. For this reason, the allowable level of sodium

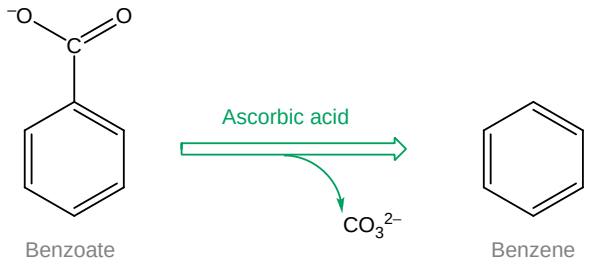


Figure 1.7 The oxidation of benzoate by ascorbic acid to form highly toxic benzene.

benzoate in proprietary cat foods is significantly lower than the corresponding level for foods intended for human consumption.

The influence of religion on food safety

Many religions are strict about what foods can be eaten and how they should be prepared. There is often little rationale for this except that it was decreed thousands, or more, years ago by the prophets or gods of the religion concerned. It is tempting to speculate that the reason that the food rules were originally introduced was because they constituted a simple means by which food was made safer to eat. There are good examples that illustrate this from Judaism and Islam.

The *Old Testament* prohibits the Jews from eating pork:

> And the swine, because it divideth the hoof, yet cheweth not the cud, it is unclean unto you: ye shall not eat of their flesh, nor touch their dead carcase. (*Deuteronomy* 14:8)

Similarly the Koran forbids pork consumption:

> He has only forbidden you dead meat, and blood, and the flesh of swine ….'

Banning pork was a very sensible food safety rule for a warm climate thousands of years ago. Pigs can be infected by the parasite *Trichinella* (see Chapter 5, *Trichinella sp.*) and it is likely that many more pigs were infected then than are infected now.

Trichinella is a roundworm (nematode) that infects pigs and spreads quickly via its eggs in infected animals' faeces. Consumption of undercooked *Trichinella*-infected pork can lead to human infection which leads to severe fever, myalgia, malaise and oedema as the *Trichinella* larvae infest the host's muscles. Modern meat production hygiene operated in most developed countries has reduced the incidence of human trichinellosis to very low levels – in the USA there were only 25 cases between 1991 and 1996, whereas in Asia and parts of eastern Europe there are still thousands of cases annually. Since the animal husbandry and meat production hygiene were primitive in the times of Christ and Allah it is very likely that most pigs were *Trichinella*-infected and therefore the risk of disease from eating pork was great. So what better food safety legislation than to ban pork consumption through the religious statutes?

The impact of space travel on food safety

The biggest impetus to make absolutely certain that food is safe was the introduction of space travel in 1960s USA. Astronauts must eat, but they simply cannot become ill while floating around in space, primarily because they usually do not have a doctor on board to treat them, and if they did the 'hospital' facilities would be rudimentary at best. There is a rather more pressing and pragmatic reason for not getting food-borne illness in the confines of a space craft orbiting the earth – most food-borne illnesses are associated with diarrhoea and vomiting and this is out of the question in a spaceship at zero gravity for obvious reasons. The developers of the US space programme realised the potential problems associated with unsafe food in space and therefore they formulated a series of extremely strict rules to ensure that the food consumed by astronauts would not make them ill. Producers of food for space travel had to ensure that it was sourced from reliable producers, that it was prepared under ultra-hygienic conditions, that it was cooked properly (to kill any pathogenic organisms that might be present) and packaged in a way that prevented later contamination (Figure 1.8). In addition, they developed a testing regime to check that astronauts' food was not contaminated with potential human pathogens. The system worked – as far as I am aware there has not been a serious incident of food-borne illness on any space mission so far.

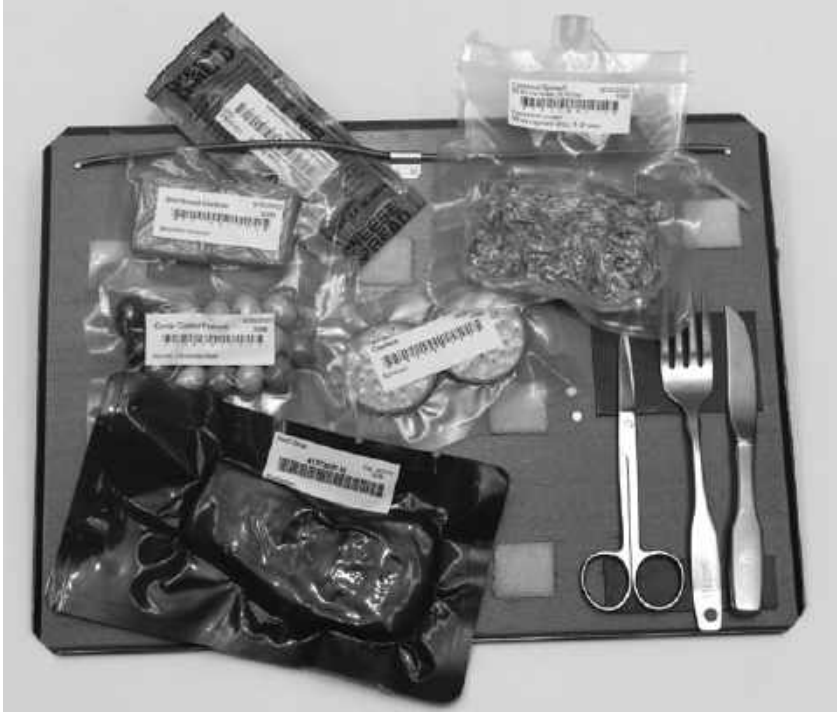


Figure 1.8 Space food used by US astronauts. It is sterilised and vacuum packed to prevent food-borne illness in space. (Picture from http://en.wikipedia.org/wiki/File:ISSSpaceFoodOnATray.jpg.)

The system that the US Space Agency formulated is the basis of modern food safety principles and has been adopted as the Hazard Analysis and Critical Control Point (HACCP) approach to minimising food-associated risk.

It is clear that making food safe by preventing the growth of spoilage and pathogenic organisms has been practised for a very long time. This is important because it allows food to be stored for times when it is less plentiful. We still use ancient food preservation techniques today to make some of our finest delicacies, including salami, yoghurt and cheeses. The idea that 'germs' in food might make the consumer ill is a much more recent (within the last 150 years) leap in understanding and the concept of chemical contamination causing illness is even more recent; these two facets of food safety form the basis of food legislation (see Chapter 16) in most countries.

In the following chapters we will explore what makes food unsafe, the processes that are used to make food safe and the laws that are in place to make it an offence to sell unsafe food. Food is safer now than it has ever been. Read on and you'll find out why.

Chapter 2
Food Risk

Introduction

Everything we do is associated with risk – nothing is risk free. There is a risk you will be killed crossing the road – in fact this is high relative to many of life's other risks, like dying from the ill effects of food. It is important that any risk is kept in perspective and compared to the general risks of everyday life if we are to assess it appropriately and determine how much money and time we spend minimising it. Governments through their regulatory authorities protect their countries' populations by introducing legislation to minimise risks. For example, most countries have laws that make the wearing of seat belts compulsory when travelling in a car; this significantly reduces the risk of dying following a car accident. In addition, most countries have laws that set rules for driving safely (e.g. stopping at a red light); these rules significantly reduce the number of accidents. So combining the road rules, which reduce accidents, and the seat belt laws, which reduce deaths following car accidents, results in a significant reduction in fatalities as a result of travelling in cars. Fines and even prison sentences are applied to 'persuade' people to follow the rules. This is an excellent example of successful risk management – identify the risk and reduce it.

Food risk has to be managed too because it is simply not acceptable for people to die as a result of eating. Acceptability (i.e. what level of risk we will accept) of becoming ill, or even dying, as a result of eating depends on an individual's perspective. A starving person would accept a far greater risk than someone who had too much food to eat; the benefit of the food to the former is survival and to the latter is pure pleasure. Clearly, benefit is an important consideration when determining what level of risk is acceptable.

This chapter will explore the following:

- What is risk?
- How is risk determined?
- Can risk be acceptable?

Food Safety: The Science of Keeping Food Safe, First Edition. Ian C. Shaw.

- Managing risk
- Food-related risk
- Food risk assessment
- Risk versus benefit
- How regulators minimise food risks

What is risk?

Risk is the probability of something going wrong. It is a word used in everyday conversation; a businessman might find a particular investment too risky (i.e. he fears losing his money), you might exclaim to a friend who asks you to dive off the highest board in the swimming pool, 'Wow, that's too risky for me!' In the latter example it is possible that if your diving skills are not very good you will injure yourself or even die if you dive off the high board. On the other hand, if you are an excellent swimmer who has dived off high boards many times before, your dive is likely to be an enjoyable experience. When you are standing on the high board (or perhaps before you climb the ladder), you make a risk assessment; 'Do I know what I am doing?' 'Will I hurt myself if I do this?' 'Do I want to do this?' The answers to these questions will determine whether you dive or not.

Similarly when you cross the road you assess the risk. You stand at the curb and check whether a vehicle is approaching; if it is, you decide whether you can get across the road without being hit. Most of us include a significant safety margin in this risk assessment to make absolutely certain we don't become one of our country's road traffic accident statistics.

This logical approach to assessing risk might be acceptable to a layman, but if we are to assess risk properly we need some numerical measurements of risk to base our decision on. This is termed quantitative risk. For example, 87% of lung cancer deaths in the UK (2002) were smoking related. This means that you are very much more likely to die prematurely if you smoke. This means that any sensible person deciding whether to smoke or not would regard the high risk of death from smoking as unacceptable and therefore would decide not to take that risk and would not smoke. This is a very simplistic approach to risk assessment; however, not only is there a need to express the risk in numerical terms to enable us to consider it properly, but also it is necessary to consider what factors determine risk and to measure them too.

The factors that contribute to risk

Humans have taken account of risk in their day-to-day decisions since they first walked the plains of the Serengeti about 2.5 million years ago. Arguably all animals are risk averse. My dog will not jump out of the back of my car, presumably because she thinks she might hurt herself if she does – she waits for me to help her down. Even though 'instinctive risk assessment' had been a part of human life since man evolved, it was not until the early 1500s that the Swiss philosopher and scientist Phillippus Aureolus Theophrastus Bombastus von Hohenheim (known as Paracelsus; 1493–1541) (Figure 2.1) defined risk in terms that we still use today.

Figure 2.1 Paracelsus (1493–1541) – the scientist who first defined risk. (From http://commons.wikimedia.org/wiki/File:Wenceslas_Hollar_-_Paracelsus_%28State_2%29.jpg.)

In his tome, *The Four Treatises of Theophrastus von Hohenheim, called Paracelsus*, he wrote:

Alle Dinge sind Gift	All things are poisons
und nichts ohn Gift	and nothing is not a poison
alien die Dosis macht,	it is the dose that makes
daβ ein Ding kein Gift ist	a thing safe

This was inspired thinking for its time because it recognised that the amount of poison ingested determines the effect – the dose makes the poison. Therefore even the most poisonous chemical will not cause harm if ingested at a low enough dose. For example, potassium cyanide (KCN) is very toxic – it would take only 100 mg to kill a person. Nevertheless drinking a glass of 0.0000001 M KCN (aq.) would cause no harm at all because the KCN dose is very, very low (i.e. in a 300 mL glass of 0.0000001 M KCN (aq.) there would be only 0.0065 mg of KCN). This principle applies to any risk situation. Therefore, returning to the example of crossing the road, the lower your 'dose' of car, the safer the crossing! In this situation, we, of course, aim for a dose of zero. The 'thing' we are exposed to is termed the hazard – the car and KCN are hazards. Risk is defined as follows:

$$\text{RISK} = \text{HAZARD} \times \text{EXPOSURE}$$

Exposure is used in place of *dose* because it applies to everything, whereas *dose* applies only to chemicals.

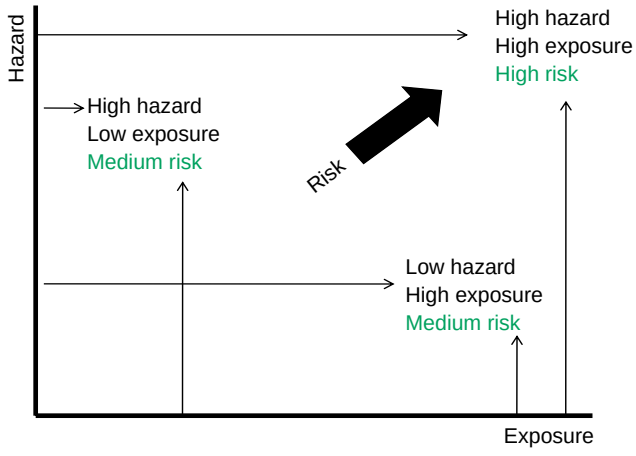


Figure 2.2 The relationship between hazard, exposure and risk – a low level of exposure to a high hazard or a high level of exposure to a low hazard both result in medium risk. The highest risk can only result from high-level exposure to a high hazard.

Therefore to determine the risk of a particular situation we need to know the hazard and measure the exposure to it; the risk associated with a particular hazard goes up with the exposure (Figure 2.2).

Measuring hazard

Hazard is an intrinsic property of something. If the 'something' is a chemical, hazard is a measure of its toxicity; if it is a pathogenic microorganism (e.g. a virus) the hazard would be less well defined, but would be a measure of how harmful the virus could be. For example, Ebola virus results in death and therefore has a very high hazard, whereas Norovirus (see Chapter 4) causes an unpleasant bout of gastroenteritis which rarely causes death and therefore it is a low–medium hazard. The risk associated with both chemical and microbiological hazards is determined by the exposure level. For example, if exposure to Norovirus is high (i.e. millions of viral particles) it is very likely that severe but short-duration gastroenteritis will result, but if exposure is very low (i.e. a few tens of viral particles) the body's immune system is likely to prevent infection and therefore gastroenteritis will not develop.

To measure chemical hazard, groups of animals are exposed to the chemical at different doses and the dose at which a toxic effect occurs is noted. If the toxic effect (end point) measured is death, the dose that kills 50% of one of the groups is the LD_{50} (lethal dose for 50% of a population). LD_{50} tests are rarely carried out now because they are considered inhumane and therefore LD_{50} has been replaced by the No Observable Adverse Effect Level (NOAEL). The NOAEL is determined using a non-lethal end point; for example, the effect of a test chemical on the liver which can be measured by a change in the serum activity of the liver enzyme glutamate pyruvate aminotransferase (SGPT). The NOAEL is the dose given to the group of animals immediately below the group showing the effect (e.g. raised SGPT) (Figure 2.3).