2 Food is Just One of Life's Risks

What is Risk?

Everything that we do is risky, even sitting in your chair at home reading this book has risk associated with it. The roof of your house might fall in and kill you, you might have an earthquake, or a meteor might hit your house. These risks are infinitesimally small, but they are nevertheless real risks. Despite this most of us would pay little attention to such minute risks. It is important that we keep risks in perspective so that we can decide what we need to worry about. As I discussed in Chapter 1, eating is a risky business – but how big is that risk? And how much should we worry about it?

Before we can decide whether we should be overly concerned about the risks associated with eating we need to understand what risk is. The Oxford English Dictionary includes in its definition of risk:

"exposure to mischance… exposed to danger… expose to chance of injury or loss."

It also uses the word *hazard* to describe risk. In scientific parlance *risk* and *hazard* are distinct and MUST not be confused. Hazard relates to the intrinsic deleterious properties of a chemical, living organism, or physical effect (e.g. radioactivity). So, for example, potassium cyanide has a very high hazard. Hazard can be measured in animal experiments by determining how much of a chemical is needed to kill a test species (usually ex-

pressed as the dose necessary to kill 50% of a test population of animals – LD50), or determining how much of the chemical is needed to cause a measurable effect (e.g. the change in the level of a hormone in an animal given a dose of the test chemical) – this is termed No Observable Effect Level (NOEL) and has now replaced LD50 tests because it is a far more humane way of checking toxicity. The LD50 for potassium cyanide by oral administration to rats is 10 mg/kg body weight (i.e. it takes a dose of 10 mg of potassium cyanide per Kg body weight of the rat – an average adult rat weighs about 500 g, so the lethal dose would be about 5 mg – this is the weight of a few grains of salt). Clearly potassium cyanide is extremely toxic. If we extrapolate the lethal dose in the rat to humans (average weight of a human is 70 kg), it would take about 70 mg to kill one of us.

Risk is related to exposure to the hazard and is a far more important way of expressing danger than talking about hazard. Unfortunately some politicians and action groups have not yet grasped this concept! If you have a bottle of potassium cyanide on the table in front of you it has an incredibly high hazard, but the risk to you is tiny because you are unlikely to eat it. If you don't open the bottle your exposure is zero and therefore the risk is zero.

Risk = Hazarad × Exposure

This simple equation is the basis of the science of risk.

In years gone by – before the authorities got worried about even the smallest risks – I used to demonstrate hazard and risk to my students by taking a bottle of potassium cyanide, weighing out 1 g and dissolving this into a litre of water. The concentration of that solution was 1000 mg/l. If I had drunk 70 ml I probably would have died. That was far too great a risk to take. So I took 1 ml of the solution and made it up to 1 l with water. This gave a solution with a potassium cyanide concentration of 1 mg/l. I happily drank a small glass full of the solution in the knowledge that I would have to drink 70 litres to kill me. This is an excellent illustration of hazard and risk. In the final example the cyanide is incredibly high hazard, but very low risk. And what's more I'm here to tell you the story!

An even simpler illustration of risk and hazard involves three vicious hungry animals in three cages, all have not been fed for a few days and so are very hungry (Fig. 2-1).

The animals represent equal hazards – they are all hungry and would eat you if given the chance. The risk from the animal in cage 1 is near to zero because barring some weird quirk of nature that resulted in the cage door opening, it would not be possible for the animal to get out to eat you, i.e. your exposure to the animal is zero. The chance of exposure to the animal in cage 2 is higher because its cage door is unlocked, therefore the

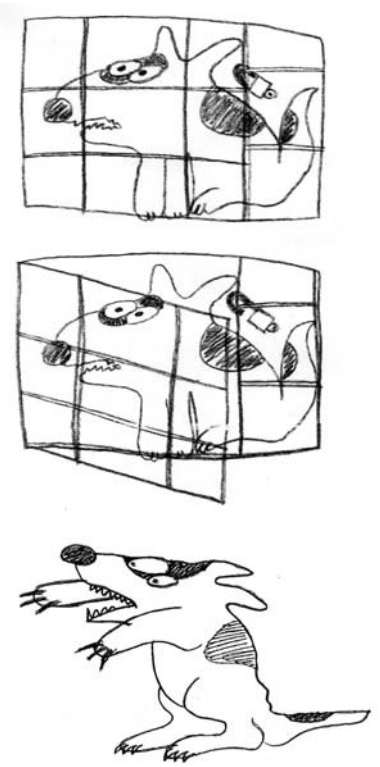


Fig. 2-1. Risk is determined by exposure to a hazard. In this example the hazards are identical – 3 vicious animals, but the risks are different. The first animal is locked in his cage therefore exposure is not possible – risk = 0; the second animal's cage door is unlocked therefore exposure is possible – risk = moderate; the third animal is not caged therefore exposure is inevitable – risk = high

risk is greater. The risk from the animal in cage 3 is enormous. His cage is open and he is free to leave and eat you.

Risk is a measure of the magnitude of the effect of our exposure to hazards. Crossing the road is a risk. We look both ways before crossing in order to minimise the risk, but still people get injured or killed crossing the road. It's the same with other risks, including the risks associated with eating. We attempt to minimise our exposure to hazards and so minimise the risk. We maintain good hygiene in our kitchens to minimise our exposure to disease causing bugs on our food. In industry they apply HACCP to minimise food risks. Our survival instinct makes us avoid risk, but it also often makes us exaggerate risk in our own minds – if it appears worse than it is you are more likely to avoid it! There is a difference between "real" risk (i.e. assessed using the risk equation) and assumed or perceived risk. Rare risks are usually perceived as worse than every day risks. So to most people crossing the road is safe – we think nothing about it, but flying in an aeroplane is more worrying. In reality you are much more likely to be killed crossing the road than you are in an air accident. The press helps us to perceive risks. They rarely publish articles about people being killed crossing the road, or in road traffic accidents because this is far too common to be interesting. On the other hand a hundred people killed in an air crash is front page news. So our perception of the risks is fuelled by the media.

BSE Risk

The BSE saga in the UK was a disaster for the beef industry, farmers were put out of work, and some even committed suicide. These were terrible times. The press reported every sordid event. They filled columns, and emblazoned headlines on numerous front pages of quality newspapers. You can understand why the British, and later the rest of the world, were terrified of BSE. They thought that a single mouthful of beef would give them nvCJD and that they would die a horrible death. The newspapers were reluctant to publish the real risk statistics. The risk of getting nvCJD was, and is, exceptionally small. Many mil-

lions of times less likely than dying in a car accident. But then this would not have sold newspapers. This might be a rather cynical view, but I suspect it is at least in part true. In the broad array of life's risks it seemed strange to me, as a risk scientist, that people were unhappy to eat beef, but did not give driving their car a second thought.

For us to respond appropriately to risks we must rank them. This helps to decide which ones to act upon; which ones are most important. Clearly BSE was important and had to be acted upon. This is unquestionable. It was an added life risk that we could minimise by legislation to prevent it re-occurring (I'll discuss this fully in Chapter 5).

Is Smoking an Acceptable Risk?

There are many other risks that people seem to accept, but why? Smoking is a good example. This is an addiction to a drug (nicotine) that is delivered in a rather bizarre way (i.e. in smoke) that simultaneously delivers highly hazardous chemicals (cancer-causing tars). The health risks associated with smoking are bronchitis, heart disease, asthma, and lung cancer. It seems strange that anyone would take this risk. In fact it seems utterly stupid! However, there is another side to the risk equation – benefit. Some people enjoy smoking. The benefit of smoking is enjoyment, pleasure. It is not possible to understand risk until we bring into the equation benefit. If the risk outweighs the ben-

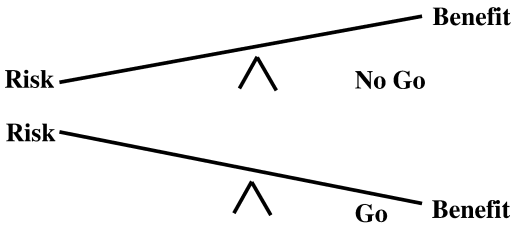
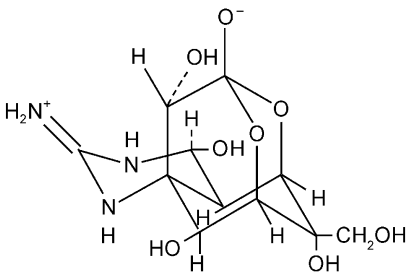


Fig. 2-2. Whether risk is acceptable depends upon benefit. If benefit outweighs risk the situation is acceptable. If risk outweighs benefit it is unacceptable

efit, the risk is unacceptable. If the benefit outweighs the risk the risk is acceptable. To smokers the benefit of a cigarette far outweighs its risk … and so they continue to smoke despite the potential effects upon their health (Fig. 2-2).

Food Risks

Food risks can be assessed in just the same way. To many Japanese people Fugu Fish is a great delicacy despite its potential toxicity. Fugu Fish contains tetrodotoxin (LD50 = 10 µg/Kg) which is intensely toxic – just 0.07 mg is likely to kill a human, this is equivalent to a small fragment of a grain of salt. The tetrodotoxin is present in the bile of the fish and when the chef prepares the fish meat for consumption he allows a tiny drop of the bile to contaminate the flesh. He does this because the toxin is a nerve poison and causes the consumer of the fish to get a tingly sensation on their lips. This is the benefit that the consumer gets to set against the risk of being killed by the toxin. The diners in Fugu Fish restaurants must really trust the chef. One mistake and they're dead. Clearly the Japanese government does not fully trust the chefs because they have recently introduced legislation to control the preparation of Fugu Fish so minimising the risk of harming its consumers – chefs now have to be trained



tetrodotoxin

Fig. 2-3. The Complex Molecular Structure of the Puffer Fish's Deadly Toxin, Tetrodotoxin

and pass an examination before they can prepare Fugu sashimi. They did this because every year about 8 people die of tetrodotoxin poisoning in Japan and this risk was not considered acceptable by the Japanese Ministry of Health & Welfare – quite right too! (Fig. 2-3).

Food associated risks are low compared to many of life's risks. Indeed the greatest risk associated with food is going to the shop to buy it. You are much more likely to get killed or injured on the way to the shop than you are to be harmed by the food that you buy.

HIGH RISK
Getting run over on the way to the shop
Choking on a Brussels sprout
Natural toxins
Pesticide residues
LOW RISK

Taken from "The risks of eating" Shaw IC (1999) Pesticides in Food. In: Brooks GT, Roberts TR (eds) Pesticide Chemistry and Bioscience. RSC, London

Continuing this comparison of food-associated risk with other life risks, it is possible to give risks numerical values by using the *risk* = *hazard* × *exposure* equation. These can be plotted on a graph to allow a comparison of risks to be made (Fig. 2-4).

The food-related illnesses are right at the bottom left hand corner of the graph, i.e. they are the lowest risks. So why are we worried about them?

I suppose the best answer to this question is that we worry because we feel that food should be safe. But this is relative, and in the context of life's daily risks that we accept without question, food is indeed very safe. Despite this, I'm pleased that we worry about food risks because it is possible to minimise them and so reduce life's risk burden upon us. For example, there is a world-wide problem with a relatively common food contaminant, the bacterium *Campylobacter jejuni*; it probably kills thousands of people around the world each year. Many of these will be in third world countries where food regulations are

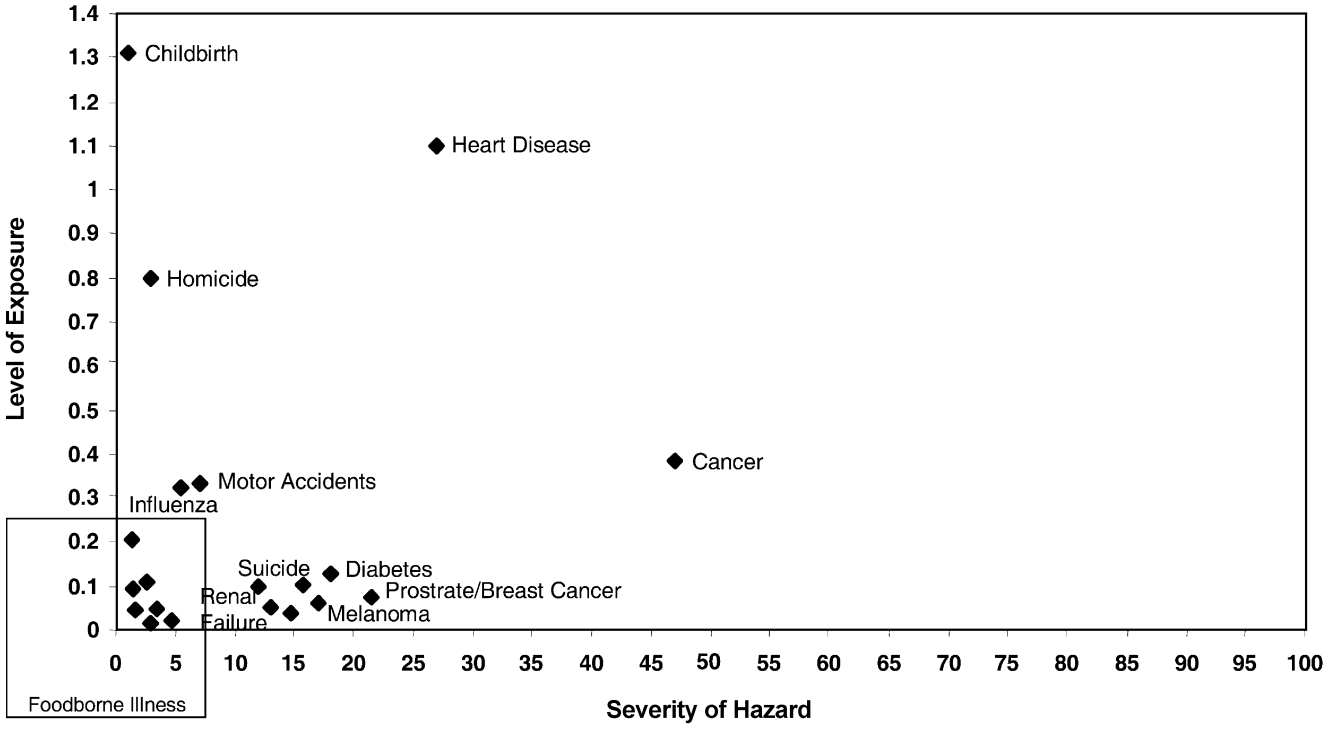


Fig. 2-4. The risks of being a New Zealander, showing that food poses one of the lowest risks (prepared in 2002 by Eva Harris, one of my summer vacation students)

scant or non-existent. However, in developed countries such as New Zealand, campylobacteriosis is still a significant food issue. Each year 1 or 2 people die in New Zealand of campylobacteriosis – in a population of only 4 million this is a significant number – and this is just the tip of an enormous iceberg, because for every death there are hundreds of cases of illness (I will deal with this more fully in Chapter 3). The question, of course, is "can we reduce the number of Campy infections and so reduce this risk?" The answer is almost certainly yes. Chicken has been suggested as the major source of Campy (the poultry industry disagree with this, and more work needs to be done to prove it one way or the other), so risk from Campy could be reduced by treating chickens to kill any Campy that they might be harbouring. In Iceland all chickens are frozen (this seems rather appropriate!) before selling for human consumption; freezing kills Campy – what a good idea. This (if it works, and we have every reason to believe that it will) is a very definitive intervention. However despite its scientific foundation, we might not like only being offered frozen chickens. Fresh chicken tastes better. Some of us might be prepared to accept the Campy risk for the better tasting fresh chicken – I certainly would. Choice is important, and for this reason most governments will not intervene in a draconian way unless the risk that they are reducing is enormous. Campy risk is not enormous. A better way of dealing with it is to educate the public about Campy. How can we reduce our chances of catching it? Campy is destroyed by heat, so proper cooking removes the problem. This is a far better solution than reducing the consumer's choice. Public education is the way forward. Tell people about hazards, and tell them how to reduce risks. They can then make up their own minds. That is exactly what this book is trying to do (Fig. 2-5).

If we identify hazards, assess risks based on exposure to the hazards, and then communicate exposure routes and means of avoiding them to the consumer, changed behaviour might result in reduced exposure and reduced risk. The hazard remains in place but its effect on the consumer is minimised. This is a cheaper option for regulators than trying to eliminate the hazard. This process is termed risk management. It relies on good consumer communication strategies.

Fig. 2-5. The risk spiral – communication about hazards leads to control of exposure and minimises risk (from Shaw, IC (2002) Making Food Safe to Eat, Food Technology New Zealand, November)

To illustrate this, I will return to the Campy example. Hazard elimination could involve selling only frozen chicken. On the other hand, risk management might involve alerting the consumer to the need to cook chicken well to kill Campy, or to the problems of cross contamination when cooking utensils are used to handle raw chicken and then are used to serve up the cooked meat. Both of these pieces of information will help the consumer to modify their behaviour so reducing exposure to this potentially lethal bacterium. It is simple, don't use the same tongs to handle raw and cooked chicken and so reduce the risk of getting campylobacteriosis. It might seem simple, but it can't be effective unless we communicate the hazards and how to minimise exposure to them to the consumer.

Risk Perception

The discussion above relates to the "real" risks associated with living. Crossing the road, meeting hungry lions, eating barbecued chicken, etc. However there is another very significant force at play when we consider risk – PERCEPTION. I can calculate risk and express it quantitatively, for example the risk of suffering from cancer in the UK is 1 in 220/year – if you live in

the UK you have a 1 in 220 chance of getting cancer (i.e. there are 267,000 newly diagnosed cases of cancer in the UK's population of 58,789,194 each year). As you get older the risk goes up, because cancer is a disease of advancing years. Did you expect the risk to be this low? What you thought the risk was is the perceived risk. Usually we perceive common risks as being low and rare risks as being high. So, it is likely that you thought the risk of getting cancer was higher than it is.

An everyday example illustrates this well. Try putting the following activities in risk order:

- Flying in an aeroplane
- Travelling in a car
- Dying from nvCJD from beef in the UK

There have been many studies on the perception of risk that show that many people would put flying in an aeroplane at the top of travel risk. So, let's look at the statistics. I'll use UK statistics to illustrate my point, but stats from all developed countries would show the same trend, Table 1.

The risk of death from car accidents is very much higher than the risk of death in an air accident. This only relates to one year in the UK, but shows clearly that no air-related deaths resulted from 73×1011 km travelled, but 1,687 people died on the roads over a total of 6.5×1011 km travelled (i.e. 91% less than air travel distance).

UK domestic travel/death statistics (1999). Data from National Statistics Online: http://www.statistics.gov.uk

Transport	Total distance travelled, km/year	Deaths
Car	6.5×10^{11}*	1,687
Air	73×10^{11}	0

* An average person in the UK travels 10,904 km/year, therefore assuming that everyone (population = 60 million) in the UK travels by car, the total distance travelled by car is $10{,}904 \times 60 \times 10^6 = 6.5 \times 10^{11}$ km.

Activity	Risk of death in the UK in 1999
Travelling in a car	1 in 35,714
Dying from nvCJD from Beef in the UK	1 in 4,000,000
Flying in an aeroplane	0

Assuming that everyone in the UK travels about the same distance each year by car, this gives a risk of dying in a car accident of 1 in 35,714/year. How does this compare with the risk of contracting nvCJD from beef in the UK? A lot of assumptions need to be made to do this calculation (e.g. that everyone eats beef – of course they don't), but the risk comes out to about 1 in 4,000,000 in 1999 (i.e. there were 15 newly diagnosed cases in 1999 out of a population of 60 million). This is not a good risk calculation because the "incubation" period for nvCJD is long – up to 12 years – and so occurrence of the disease in 1999 relates to exposure years ago. Nevertheless it gives us an idea of the magnitude of the risk, and it is very low; much lower than being killed in a car accident.

Using these risk values we can put the three activities that were discussed above in risk order (i.e. rank them), Fig. 2-2.

Most people wouldn't think twice about driving their car, but are likely to worry a little about air travel and eating beef. But they are wrong; they should worry very much more about driving their car than either of the other activities. Their per-

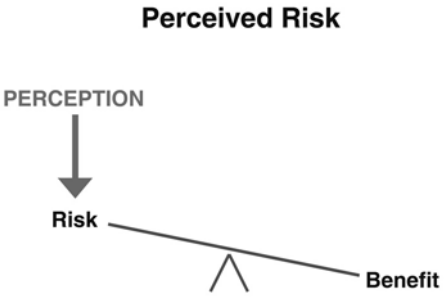


Fig. 2-6. The risk/benefit balance showing how perception adds weight to risk

ception of the BSE risk is far greater than reality – probably fuelled by an over zealous news media.

Perception often adds weight to the risk side of the risk/benefit balance, and therefore means that the risk seems to be greater and so is less likely to be outweighed by the benefit. For example, the threat of contracting nvCJD from beef in the UK meant that many people stopped eating beef even though they enjoyed it. The risk is nvCJD, the benefit is enjoyment, but their perception of the risk was greater than reality and therefore it outweighed the benefit in their minds (Fig. 2-6).

Paracelsus – the Grandfather of Risk

Risk science is not new, in fact a German scientist Phillipus Aureolus Theophrastus Bombastus von Hohenheim Paracelsus was its first, as far as we know, protagonist. Paracelsus was born in Switzerland in 1493, and died in 1541. He is famous for his risk philosophy discussed in his Four Treatises (Fig. 2-7):

Alle Dinge sind Gift	All things are poisons
Und nichts ohne Gift	There is nothing which is not a poison
Allein die Dosis macht,	It is the dose
Dass ein Ding	Which makes a thing safe
Kein Gift ist	

Roughly translated from Paracelsian philosophy this means, a little bit will do you no harm. Remember our discussion on cyanide where a tiny dose, even though its hazard is very high, will cause no ill effects – cyanide is poisonous, but a small dose won't hurt you.

Is There a Price on Risk?

The benefit side of the risk/benefit balance can have more than one component. For example it could include pleasure and price. If one of these changes it can tip the balance in favour of benefit.

Fig. 2-7. Theophrastus von Hohenheim Paracelsus (1493–1541) (reproduced from http://www.mhiz.unizh.ch/Paracelsus.html by kind permission of Dr. Urs Leo Gantenbein)

During the BSE epidemic in the UK there were significant fluctuations in the sale of beef. When it was suspected that BSE might cause disease in humans who consumed contaminated beef, sales of beef dropped off sharply. When it was confirmed that nvCJD resulted from consuming BSE beef, sales fell drastically and the UK beef industry collapsed. However the price drop that resulted from the decreased demand for beef meant that some people thought that beef was such good value that they were prepared to risk contracting nvCJD and eat beef anyway. Therefore sales of beef rose shortly after the price hit rock bottom. The power of price and enjoyment of eating beef outweighed the perceived risk of nvCJD. In this case the risk was acceptable at £1.95/lb! (Fig 2-8).

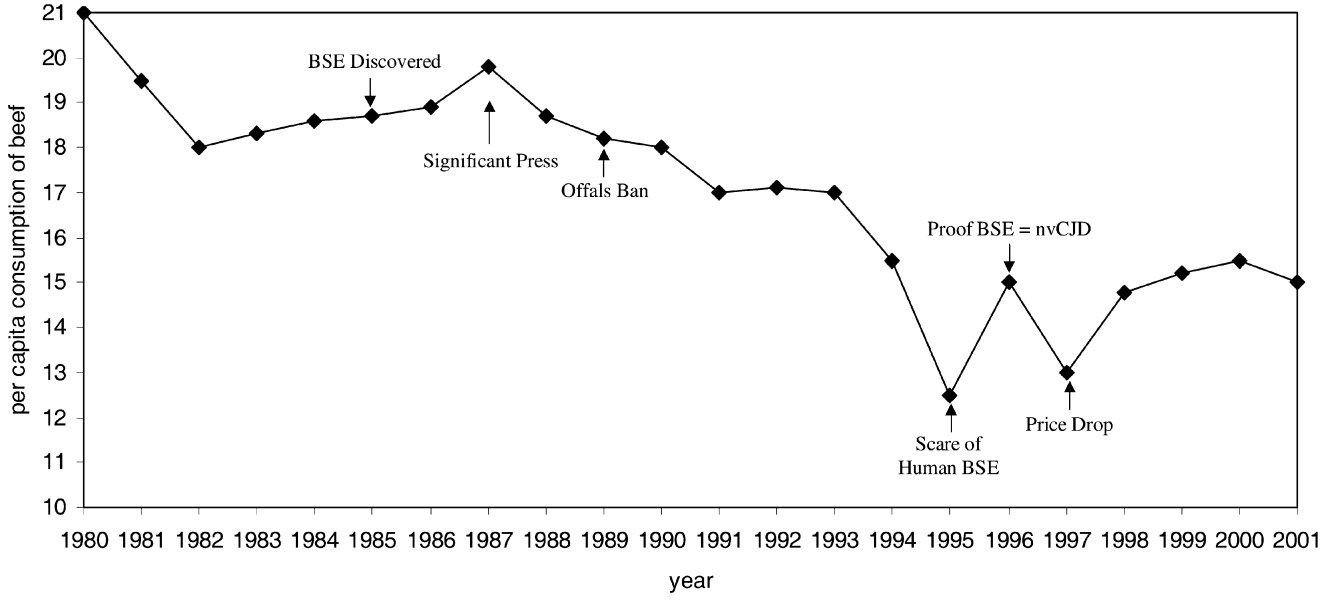


Fig. 2-8. Is there a price on risk? The changing price of UK beef driven by the Mad Cow saga and its effect on consumption. In 1997 the price got so low that people "forgot" their concerns about eating Mad Cow beef and stocked their freezers this increased demand drove the price up again (data from UK Agriculture Committee (2002))

Is Risk Increasing?

On the face of it, it looks as though life is getting more risky as time passes. More people die of cancer now than 100 years ago. This is partly because cancer diagnosis was rather hit and miss a century ago, and partly because cancer is a disease of old age and people are living longer now and therefore the susceptible population is greater. The fact that people are living longer means that risk must be decreasing. This is largely because medicine is better now than in years gone by. The risks associated with many diseases have reduced drastically. Tuberculosis (TB) is an excellent example. Just 75 years ago, before the introduction of sulphonamides, many people died of TB, now it is relatively easy to treat and therefore the death rate in the developed world from TB is much lower than pre-sulphonamides (Fig. 2-9).

Therefore, in general, life's risks are decreasing because of better medical care and medicines. This is why Queen Elizabeth II sends more congratulatory telegrams to her centenarian subjects than Queen Elizabeth I might have. Associated with this decreased risk of living there is a concomitant increase in some risks. The decreased risk is associated with intellectual and technological advances, e.g. medicine. With these come extra risks. The invention of the motor car is an obvious example. Despite all of this, we live longer now than we did 100 years ago and therefore the risks are being managed effectively to promote a longer life.

Amidst life's risks are food related risks. We have discussed some of these above – they are incredibly low in the context of many of our other daily risks. But the question is, are they increasing? A quick look back at Chapter 1 would persuade anyone that food related risks have diminished over the centuries. However we need to look more closely at the risks over the past few decades. We hear so much more about food related illness now than in decades gone by. We go to exotic holiday destinations and return with gastric upsets. Travel agents even warn us not to eat certain foods in some of these tropical havens. A few days in India or Indonesia without a very sensible approach to eating will prove my point. I speak here from bitter experience! There is absolutely no doubt that eating in developing countries

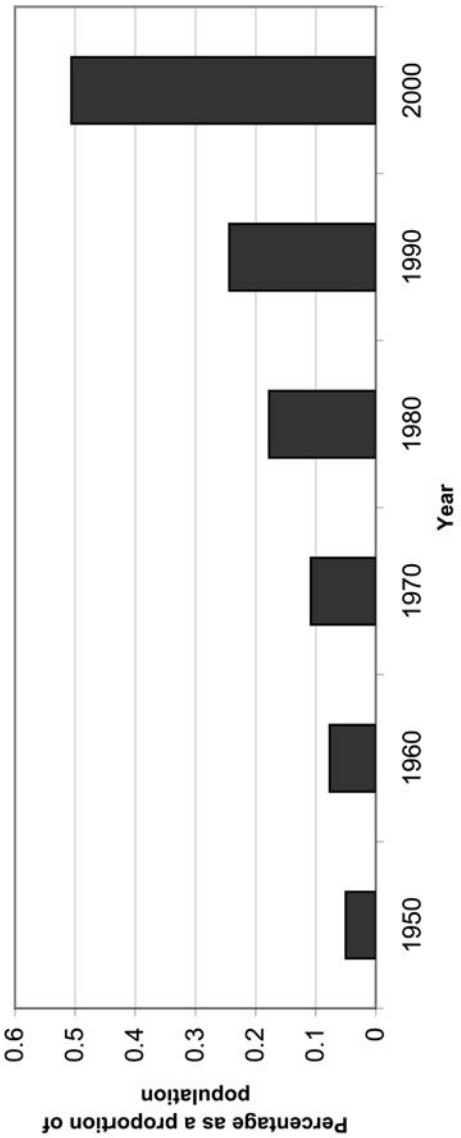


Fig. 2-9. The number of 100-year birthday celebratory telegrams sent by the Queen of England. This shows that a greater proportion of the population are living beyond 100 years so life must be getting less risky

is far more risky that eating in the developed world. Most developing countries do not have good food legislation. This is way down their list of priorities – feeding their people and combating terrible diseases is rightly above food safety. So if we travel to these countries we apply our own risk management procedures – don't eat uncooked, unpeeled vegetables, watch food that might be reheated, don't drink water unless it is bottled, don't have ice in drinks, etc. This minimises our exposure to microbiological hazards associated with food and water.

But what about the so-called developed world? How have food-related illnesses changed in the past few decades? The USA has an excellent organisation, the Centers for Disease Control and Prevention (CDC) that collects and collates (amongst a myriad other things) data on food poisoning. Since the USA is a good example of a developed country from the point of view of food safety issues, I will illustrate the changes in food-born illness over the past decades with information from CDC.

In the USA, about 76,000,000 food-related illnesses occur each year (this includes multiple incidents in individuals), of these 325,000 (0.43% of cases) result in hospitalisation, and there are 5,000 deaths (0.007% of cases). *Salmonella, Listeria* and *Toxoplasma* (a parasite, found in food, that causes neurological disease) account for 1,500 deaths per year. Looking at just one of these food-related infections, listeriosis, in the USA over a 7-year period shows a clear downward trend (Fig. 2-10).

Other diseases (campylobacteriosis and yersiniosis) show the same trend. This suggests that food-related illness risk is going down. But if we look further, other bacterial diseases associated with food are showing a gentle upward trend (Fig. 2-11).

This is a "swings and roundabouts" situation. Some food-related diseases are increasing, while others are on the way down.

Chemical Risks

All of our discussion so far has been about microbiological food risk. This is because far more is known about the diseases that

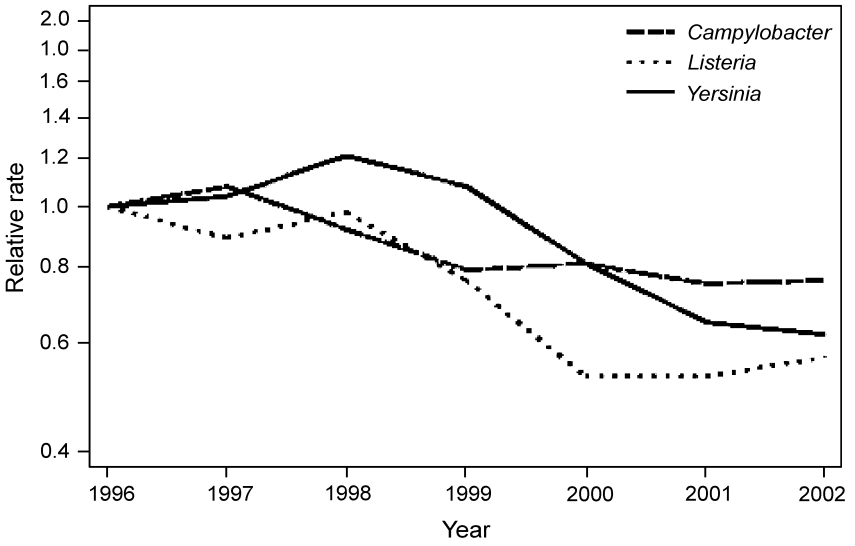


Fig. 2-10. Declining numbers of listeriosis, campylobacteriosis and yersiniosis cases in the USA (data from Mead et al. (2003), Food-related illness and death in the United States http:www.cdc.gov/ncidod/eid /vol5no5 /mead/htm)

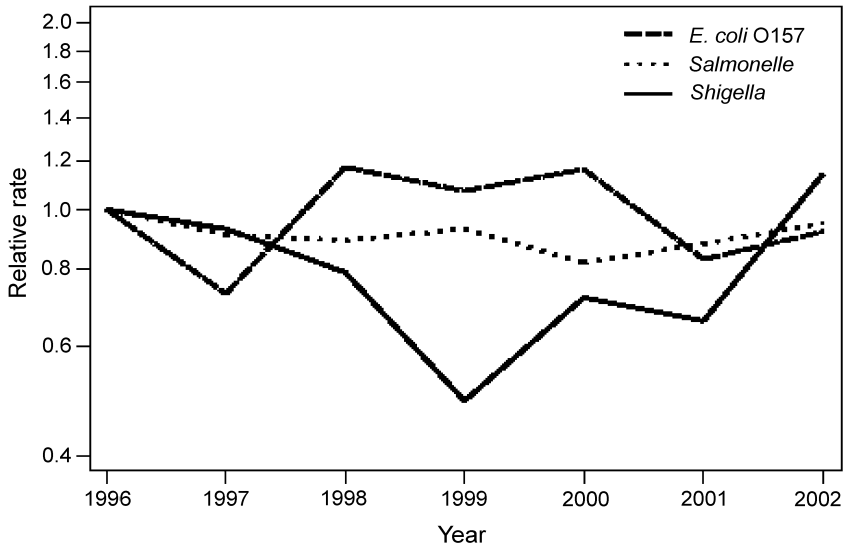


Fig. 2-11. Declining numbers of *E. coli, Salmonella* and *Shigella* cases in the USA (data from Mead et al. (2003), Food-related illness and death in the United States http:www.cdc.gov/ncidod/eid/vol5no5/mead/htm)

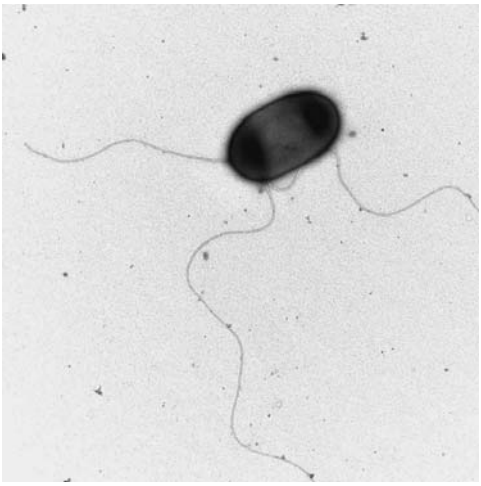


Fig. 2-12. Electron micrograph of *Listeria* [magnification approx 35,000X] (kindly provided by Phillipa Rhodes)

microbes cause. The symptoms of *Listeria* infection (i.e. listeriosis) are very well known; any doctor would recognise them and could prove their cause by taking a faeces sample and getting the lab to culture it to show the presence of *Listeria* (Fig. 2-12).

Chemical contaminants are very different. Most food-related bacterial and viral diseases are acute – i.e. the disease associated with the virus or bacterium occurs a day or two – or at most a few weeks after infection. It is not so simple for chemical contaminants. Indeed, even if a particular chemical contaminant could cause an acute effect, it is extremely unlikely that the levels in food would be sufficiently high to result in acute toxicity (i.e. exposure to the acute hazard is too low to result in an acute risk). For example, organophosphorus pesticide (OP) residues – from the use of OPs in crop protection or as vet medicines – in food would never result in the acute neurological effects (shaking, salivation, death) associated with very high doses of OPs. This is because residues in food are at exceptionally low levels (i.e. of the order of parts per million [ppm], or mg/kg) compared with the doses that would cause acute toxic effects.

Data from the UK's pesticide monitoring schemes show that some vegetables have very low residues of an OP called pirimiphos-methyl – used as an insecticide in crop production and grain storage. For example, a single pirimiphos-methyl level found in 1999 in bread was 0.1 mg/kg. If you assume that someone eats 250 g of bread in a day – this is a worst case scenario because it wrongly assumes that all bread contains pyrimiphos-methyl residues at 0.1 mg/kg (most would contain no residues at all), the daily dose of pyrimiphos-methyl would be 0.025 mg. Assuming that the person weighs 60 kg, their daily intake would be 0.0004 mg/kg body weight. The maximum dose that would not result in toxicity to a person if they ate it every day for their entire life (this is termed the Acceptable Daily Intake – ADI) is 0.01 mg/kg, and this includes a safety factor of 1,000. The bread that the UK found to contain residues of pyrimiphos-methyl would lead to a dose that is only 4% of the ADI and therefore would not result in toxicity; the risk is therefore very low indeed.

The same sort of calculation can be made for most pesticide residues in food. Indeed this argument has often been used to show that residues are of no health concern to the consumer. But what effect might the complex cocktail of pesticides that we eat in a lifetime have on our health? We don't know, and it is extremely difficult to predict or determine such effects because the cocktail changes with time as new pesticides are introduced and old ones phased out. So we certainly cannot say that pesticide residues will have no effect, but we can say that they are unlikely to have an effect based on the toxicity of individual pesticides in our food.

A simple approach to determining the possible additive effects, as a means of assessing risk, of pesticide residues is to look at a class of pesticides whose members all exert their effects by the same biological process (mechanism). If we determine risk based on the individual pesticides this gives us a low risk assessment, but since they are all acting by the same mechanism we should add up their effects to give an overall effect of exposure to the class of pesticides.

The OPs are an excellent example. They all kill insects by preventing nerve impulses being generated by inhibiting a spe-

cific enzyme (acetylcholinesterase – AChE) in the nervous system (see Chapter 7). Similarly their toxic effects on people are by the same mechanism. So if you get small doses of Propetamphos, pyrimiphos-methyl, Diazinon and a large number of other OPs, determining risk by looking at the individual OP intakes will not give a realistic assessment of the risk. The best way would be to add the concentrations together (or better, add together a measure of their relative biological activity) and determine an additive effect. It might be that each of the pesticide intakes is just below the ADI – this is unlikely, but will be used here as an example. Adding them together would exceed the ADI, and might result in harm to the consumer. I will discuss this again in Chapter 7, but for now it is important that we remember that chemical risks might appear lower than they really are because it is very difficult to assess them properly.

Newspapers Often Exaggerate Risk

Newspapers often exaggerate the risks associated with chemical residues in food and therefore increase the consumer's perception of risk. For this reason many people rate the risks of pesticides in their food higher than microbiological risk. This is simply wrong.

Your typical lettuce.... after 11 doses of pesticide

The front page newspaper headline from the UK's Guardian newspaper on 16 September 1999 reporting the "leaked" annual report of the Working Party on Pesticide Residues – beneath the headline was a picture of an apparently normal lettuce. The article was accompanied by a very funny cartoon showing a woman off to do her shopping wearing protective clothing! The article quoted statistics on pesticide residues frequency, but did not highlight their levels; it focused on hazard rather than risk – this is misleading.

A great deal of this over perception of the risk of pesticides is due to adverse press coverage. It is interesting that in a survey of experts (in this case toxicologists) and lay people (i.e. non-scientists). The experts rated pesticides as less toxic than did the lay group:

Statement: Residents of a small community observed that several malformed children had been born there during each of the past few years. The town is in a region where agricultural pesticides have been used during the past decade. It is very likely that these pesticides were the cause of the malformations.

Responses (%):	Strongly disagree	Disagree	Agree	Strongly agree	Don't know
Toxicologists	22.2	59.3	4.3	1.2	13.0
Lay people	3.9	23.4	39.5	9.0	24.2

Taken form Paul Slovic's The Perception of Risk, Earthscan Publications Ltd., London, 2000.

It is interesting that the experts do not regard pesticides as being the cause of the town's malformed children, whereas the lay people blame the pesticides. This is an important illustration that perception depends on knowledge. This is why we teach our children that cars are dangerous, in the hope that they will err on the side of safety when crossing the road. "Err on the side of safety" means over assess the risk. This is exactly what the lay group did in the above example.

In conclusion, risks are not always what you think they are. If the risk that you are contemplating is common place (e.g. smoking) you are likely to under estimate it. Whereas if the risk is rare (e.g. death from vaccination) you are more likely to over rate it. When students were asked to assess the risk of smoking-related deaths per year in the USA, they came up with 2,400 – the real value is 150,000. On the other hand they were asked to assess deaths from skiing, they thought 72 – the real figure is 18. I bet more of them smoke than ski!

Paracelsus should have the last word. *All things are poisons, there is nothing that is not a poison, it is the dose that makes a thing safe,* or in other words, everything is safe unless you take too much of it!

3
Bacteria in Food – Good or Bad?