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What is a Healthy Food?*

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ABSTRACT

All foods can contribute to health, if used as part of a mixed diet; conversely, all foods can cause ill health if consumed unwisely; thus the question should have been 'What is a Healthy Diet?'

Health demands ingestion of numerous essential nutrients, yet none of these, or of the many thousands of the other components of food, may be consumed in excess without adversely affecting bodily functions.

Human behaviour patterns, whether derived by evolution, precept or tradition, permit mankind to avoid these hazards. This success is demonstrated by the very low incidence of nutritional deficiency disease in populations freely able to select their food.

A special present-day nutritional concern is the belief that over indulgence in certain food components, and consequent obesity, is a serious health problem. This is probably true for a large proportion of the population but the major, proven, cause of rapidly rising, diet-related, morbidity and mortality, is food-borne disease arising from contamination, not by additives or pesticides, but from pathogenic microorganisms. This phenomenon is the more distressing because it is avoidable, arising from neglect of simple hygienic precautions, especially in the kitchens of private homes and catering establishments.

In the early decades of this century newspapers and magazines, without the help of radio and television, did much to improve the health of the nation by increasing public awareness of the 'germ theory of disease' and the need for hygiene as well as cleanliness. How much more rapidly could the media of today help us to eliminate microbial dangers from our foods?

WHAT IS A HEALTHY FOOD?

What are we and why do we need to eat?

The human body is an association of thousands of chemicals which are

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continually being broken down and replaced. Some are present in minute quantities or removed slowly. Others are produced in large amounts, e.g. the metabolism of fat and carbohydrate involves the daily formation of between a pound and a kilogram of each of a number of organic acids including citric (E330), succinic (E297) and malic (E296) as well as some such as *cis* aconitic and alpha keto glutaric which are not approved as food additives.

The expenditure of 1500 kcal (about 6 MJ) on mechanical work, not an unusual need, may entail the synthesis and use of about 60 kg of adenosine triphosphate—a quantity equal to the body weight of many persons—during a working day!

LIFE ESSENTIALS

Body structure and function depend on numerous inter-related self-regulating chemical systems relying on external supplies of air, food and water. The basic requirements for oxygen, water and energy are shown in Table 1 which also notes that needs must be met at appropriate intervals. All

TABLE 1
Life Essentials

	Common intakes	*Dangerous or inadvisable intakes*		*Time to deficiency*
Oxygen	0·4 g/min	<0·2	> 1·6 g/min	Minutes
Water	1–4 litres/day	< 1	> 10 litres/day	Days
Energy	2 000 kCal/day	< 1 000	> 4000 kCal/day	Weeks
	8 MJ/day	< 4	> 16 MJ/day	

Water and energy intakes may vary several fold depending on type of environmental and physiological circumstances. Water toxicity depends on rate of intake compared with excretory ability.

chemicals, even if essential for life, can be toxic if intakes surpass ability to use or eliminate them and so permit harmful concentrations to arise. Hence foods, like other chemical mixes, can be injurious if taken to excess.

For present purposes food and diet are defined as follows.

Food

Any material containing substances other than gaseous oxygen, which, when introduced into the alimentary tract, may be used to provide energy and sustain vital functions. (This definition excludes medicines, water and condiments unless they supply energy as well as nutrients.)

The diet

The totality of food consumed over a period. It is often expressed as the mean daily consumption of food items and/or nutrients and energy.

A healthy diet must supply all of the many chemicals essential for life in amounts sufficient to meet needs over the periods in question yet none of these or of the other food components may be present in quantities which may in any way not permit full health and well being. (All essential dietary components need not be supplied every day. Most, depending on previous provision, body stores and rate of utilisation, are needed at less frequent intervals.)

Table 2 shows a commonly used method of classifying nutrients.

TABLE 2
Food Nutrient Classification

Energy
Protein
Electrolytes
Minerals
Vitamins—Fat soluble
—Water soluble
Trace elements
Others

ENERGY

Normally the greater part of bodily energy is derived from oxidation of fat and carbohydrate and, to a much lesser extent, from protein. Carbohydrate and most of the components of fat are not dietary essentials in strict terms, because they can be synthesised in the body, but are indirectly essential in that, in their absence, the extra protein needed to supply the energy normally derived from fat and carbohydrate could lead to harm. Tables 3–10 indicate the approximate amounts of essential nutrients which it is thought may safely be consumed by the average person as inferred from nutritional and epidemiological studies in man and animals, such as those considered by various authorities, e.g. FAO, DHSS and NRC. The levels noted are approximations because precise needs, and hazards, vary greatly with circumstances.

PROTEIN

In the native state some proteins are of little nutrient value while others (e.g. staphylococcal and clostridial toxins) are intensely poisonous. Denatured

TABLE 3
Protein and Amino Acids

	Common intake (g/day)	*Dangerous or inadvisable intake (g/day)*		*Time to deficiency*
Protein	40–100	<25	>?	Weeks–months
Essential amino acids				
Histidine	1–2	<?	>30	Weeks–months
iso Leucine	2–4	<0·6	>15	Weeks–months
Leucine	3–7	<0·8	>30	Weeks–months
Lysine	2–5	<0·6	>30	Weeks–months
Cysteine and Methionine	1–3	<0·5	>30	Weeks–months
Tyrosine and Phenylalanine	3–7	<0·8	>30	Weeks–months
Threonine	1–4	<0·4	>22·5	Weeks–months
Tryptophan	0·5–1	<0·15	>7·5	Weeks–months
Valine	2–5	<0·7	>30	Weeks–months

People have survived single i.v. doses of the size noted but chronic, safe consumption is probably much lower.
Sources: Milne (1968); DHSS (1969); FAO (1974); NRC (1980); Visek (1984).

and edible proteins, however, not only provide the nine essential amino acids, which cannot be synthesised in the tissues, but also the fixed nitrogen for the formation of the non-essential amino acids and many other nitrogenous compounds. The proportions of the different amino acids vary in the different food proteins. The quantitative needs for protein and amino acids are shown in Table 3 together with available information on toxicity. Protein deficiency can occur but protein toxicity has not been reported in normal persons—probably because it is difficult to obtain and hard to eat very large amounts of protein! There are, however, certain diseases in which problems can arise if protein consumption is not decreased. Little is known of the toxicity of amino acids because they are not readily available and are expensive while some have unpleasant tastes. People have survived the very large doses noted in Table 3 without permanent effects but with temporary disabilities (Milne, 1968; Visek, 1984).

THE ELECTROLYTES

Sodium (Na^+), potassium (K^+) and chloride ions (Cl^-) are vital to maintain the appropriate ionic environment in cells and tissue fluids. Safe and inadvisable intakes are shown in Table 4. Normally ionic concentrations are precisely regulated by controlled excretion but, under some circumstances

TABLE 4
Electrolytes

Element	*Common daily intake*	*Dangerous or inadvisable daily intake*		*Time to deficiency*
Na	2–7 g	< 70 mg	> 16 g	Hours–months
	100–300 mEq	< 3 mEq	> 700 mEq	
K	2–6 g	< 100 mg	> 18 g	Hours–months
	50–150 mEq	< 2·5 mEq	> 450 mEq	
Cl	3–12 g	< 100 mg	> 25 g	Hours–months
	100–500 mEq	< 3 mEq	> 700 mEq	

Sources: DHSS (1969); NRC (1980).

such as profuse sweating, diarrhea and vomiting, rapid losses of electrolytes may occur, when serious illness or even death may result.

MINERALS

Calcium (Ca), phosphorus (P) and magnesium (Mg) are important because their ions are concerned with the control of many aspects of metabolism while calcium and phosphate comprise the major mineral components of bone.

Advisable intakes are noted in Table 5. Harmful effects can follow consumption of large amounts of the soluble salts of calcium, magnesium and phosphates but are rarely observed. However, excess consumption of calcium for instance, in attempts to control osteoporosis might well lead to problems by decreasing zinc absorption from diets rich in fibre.

TABLE 5
Minerals

Element	*Common daily intake*	*Dangerous or inadvisable daily intake*		*Time for deficiency*
Ca	300–1 500 mg	< 400 mg	> ?	Months–years
P	1 000–2 000 mg	< 400 mg	> ?	Months–years
Mg	200–300 mg	< 250	> 5 000 mg	Months

Sources: DHSS (1969); FAO (1974); NRC (1980).

SULPHUR

Two sulphur compounds are essential, one is thiamine and the other is methionine from which all other required sulphur compounds can be derived.

TABLE 6
Fat-Soluble Vitamins

Vitamin		*Common daily intake*	*Dangerous or inadvisable daily intake*		*Time for deficiency*
A	(Retinol equivalents)	0·5–2·0 mg	<0·5 mg	>25 mg	Months–years
D		2–20 μg	<2·5	>25 μg	Months–years
E	dl tocopherol acetate	3–20 mg	<3 mg	>1 g	Years?
K		0·3–0·5 mg	<?	>?	Weeks—or never

Sources: DHSS (1969); FAO (1974); NRC (1980); Bender (1985); Owen (1971); Wooliscroft (1983).

FAT-SOLUBLE VITAMINS

The fat-soluble vitamins are listed in Table 6 together with common and inadvisable intakes applicable to normal individuals. There is, however, evidence to show that, in a minority of apparently normal persons, as little as ten times the normal intake of vitamin A or vitamin D (10 mg retinol or 25 μg cholecalciferol or ergocalciferol) may cause serious ill effects (Owen, 1971; Wooliscroft, 1983; Bender, 1985; Albert, 1987). For this reason the concentrations of vitamin A and D preparations on general sale are controlled to minimise the risk of overdosage.

It is commonly thought that vitamin E is harmless but toxic effects have been observed in persons taking large doses (Bender, 1985).

Vitamin K is normally synthesised by bacteria living in the gut and sufficient is absorbed to eliminate any need for dietary vitamin K except when, for any reason (e.g. treatment with certain antibiotics or alimentary dysfunction), microbial synthesis is interrupted or requirements are increased.

WATER-SOLUBLE VITAMINS

As a group the water-soluble vitamins display low toxicity (Table 7) probably because, being water soluble, they are readily excreted in the urine. However, enthusiastic believers in 'mega doses' of vitamins have clearly shown that very harmful effects, often long-lasting, can follow consumption of sufficiently large doses of ascorbic acid, folic acid, niacin, pyridoxine (vitamin B_6) and pantothenic acid (Owen, 1971; Wooliscroft, 1983; Bender, 1985; Dalton & Dalton, 1987). The fact that harmful reactions have not yet been reported after thiamine, riboflavin, vitamin B_{12}, or biotin may merely

TABLE 7
Water-Soluble Vitamins

	Common daily intake	*Dangerous or inadvisable daily intake*		*Time for deficiency*
Ascorbic acid	10–100 mg	< 10 mg	> 1 g	Weeks–months
Thiamine	0·4–2 mg	< 0·3 mg	>?	Weeks
Riboflavin	1–2 mg	< 0·5 mg	>?	Weeks
Niacin[a]	15–40 mg	< 10 mg	> 300 mg	Weeks
Vitamin B_6	1–2 mg	< 1 mg	> 50 mg	Weeks
Pantothenic acid	10–20 mg	< 4 mg	> 10 g	Months
Folic acid	0·1–2 mg	< 0·1 mg	> 10 mg	Months
Vitamin B_{12}	5–15 μg	< 1 μg	>?	Years
Biotin	50–300 μg	<?	>?	Years

[a] 60 mg tryptophan ≡ 1 mg niacin.
Sources: DHSS (1969); Owen (1971); FAO (1974); NRC (1980); Schaumberg *et al.* (1983); Wooliscroft (1983); Bender (1985); Albert (1987); Dalton & Dalton (1987).

be a reflection of the nasty taste (thiamine and riboflavin) or the high cost (vitamin B_{12} and biotin) of these substances.

TRACE ELEMENTS

All the trace elements shown in Tables 8 and 9 have been shown to be needed in the diet of higher vertebrates such as the rat and chick (Schwarz, 1974; Davies, 1981). Of these, nine have been demonstrated to be essential for man while it is almost certain that the others (manganese, arsenic, tin, vanadium, nickel and silicon) are also required because of their participation in vital reactions or their need for health in animals.

Because of their widespread occurrence in foods, dust and contaminants, and because of the small amounts needed, trace element deficiency, except for that of iodine and iron, is rarely encountered except under unusual circumstances in man. The necessary amounts of silicon and nickel, for instance, are so small that deficiency has only been induced in animals eating and drinking highly purified diets and breathing air filtered to remove all dust particles.

Table 9 summarises available information on body content, typical intakes, recommended requirements and levels of consumption thought to be inadvisable. This table clearly shows the lack of information on needs and safety levels for these essential, but potentially poisonous, elements (International Commission on Radiological Protection, 1974; NRC, 1980;

TABLE 8
Discovery of Trace Element Requirements

Iron	17th Century	Chromium	1959
Iodine	1850	Tin	1970
Copper	1928	Vanadium	1971
Manganese	1931	Fluorine	1972
Zinc	1934	Silicon	1972
Cobalt	1935	Nickel	1974
Molybdenum	1953	Arsenic	1976
Selenium	1957		

Sources: Schwarz (1974); Davies (1981).

Hazell, 1985; Nechay *et al.*, 1986; FAO/WHO, 1982, 1983). Application of the little information to hand is difficult because the presence of some of these elements, copper, zinc and molybdenum for instance, can modify the need for others. Furthermore, the availability, biological activity and toxicity depend greatly on chemical state. Trivalent arsenic and hexavalent chromium are highly toxic whereas pentavalent arsenic and trivalent chromium are much less so and are considered to be the forms best metabolised for essential functions. Though inorganic forms of tin are

TABLE 9
Trace Elements Essential for Man

Element	*Typical body content (mg)*	*Common daily intake (mg)*	*Recommended daily intake (mg)*	*Inadvisable daily intake (mg)*	
Iron	4200	11	10–18	<10 mg	>25–75
Fluorine	2600	1·8	1·5–4	<?	>20–80
Zinc	2300	9	15	<5	>30
Copper	100	1·5	2–3	<1	>10–35
Silicon	30	30	?	<?	>?
Arsenic	18	0·13	?	<?	>3·5
Selenium	13	0·06	0·05–0·2	<0·05	>0·2
Manganese	12	4·6	2·5–5	<2·5	>5
Tin	<17	4	?	<?	>140
Iodine	13	0·25	0·15	<0·15	>?
Nickel	10	0·18	?	<?	>?
Molybdenum	9	0·12	0·15–0·5	<?	>0·5
Chromium	1·5	0·03	0·05–0·2	<?	>?
Cobalt	1·5	0·007	nil	>6–8	
Vanadium	0·1	2·0	?	<?	>?

Sources: Int. Comm. on Radiological Protect. (1974); Walkiniew & Douglas (1975); Neathery *et al.* (1976); MAFF (1978); Spring *et al.* (1979); NRC (1980); Martindale (1982); WHO (1982); Buss (1983); Diplock (1985); Hazell (1985); Herbert (1987); Nechay *et al.*, (1986).

TABLE 10
Polyunsaturated Fatty Acids: The Essential Fatty Acids—Omega 6 Series[a]

Fatty acid		*Position of double bonds*		*Recommended consumption (% total energy)*	*Inadvisable intake*
		From COOH	*From CH_3*		
Linoleic	C18:2	9, 12	6, 9	8–10% total diet energy and	?
Gamma linolenic	C18:3	6, 9, 12	6, 9, 12		?
Dihomo gamma linolenic	C20:3	8, 11, 14	6, 9, 12		?
Arachidonic	C20:4	5, 8, 11, 14	6, 9, 12, 15	polyunsaturated/saturated fatty acid ratio >0·45	?

[a] Adverse effects not yet known but high levels of intake may increase incidence of gall bladder problems.
Sources: NACNE (1983); COMA (1984).

considered to be of low toxicity, some organo-tin compounds are exceedingly poisonous. For most of these elements little is known of the safety, or otherwise, of the many forms other than the simple ionic species in which they may exist. For these reasons consumption of trace elements is not advisable unless indicated by chemical analysis and expert clinical observations. Except under particularly abnormal circumstances such supplements are at best harmless though potentially hazardous.

ESSENTIAL FATTY ACIDS

Certain polyunsaturated fatty acids, the essential fatty acids (Table 10), linoleic, gamma linolenic and arachidonic acids, of the omega 6 series, have each been shown to fulfil the essential fatty acid needs of man (Hansen *et al.*, 1958). Little is known of the effects of prolonged high intakes of the essential fatty acids though under some circumstances adverse effects on vitamin E needs and liver function might be anticipated.

TABLE 11
Polyunsaturated Fatty Acids: Omega 3 Series

Fatty acid	*Position of double bonds*		
	From COOH	*From CH_3*	
Alpha linolenic C18:3	9, 12, 15	3, 6, 9	Beneficial effects claimed but toxicology not known
Octadecatetraenoic C18:4	6, 9, 12, 15	3, 6, 9, 12	
Eicosapentaenoic C20:4	8, 11, 14, 17	3, 6, 9, 12	
Eicosapentanoic C20:5	5, 8, 11, 14, 17	3, 6, 9, 12, 15	

Some other polyunsaturated fatty acids—those of the omega 3 series—have been identified in the tissues of higher vertebrates (Table 11). They have been shown to exert certain pharmacological effects (Salmon & Takahashi, 1985; Sanders, 1985) and are now recognised as nutrient essentials (Sanders, 1988). These, and other polyunsaturated fatty acids may modify blood coagulability, thrombus formation and blood lipid levels in ways thought to be favourable under some conditions. It is, however, not yet possible to predict with confidence the benefits to some—or risks to others—which might follow if consumption of omega 3 fatty acids (found in marine oils) were to be greatly increased.

TABLE 12
Major Fibre Components

Polysaccharides containing:
Arabinose
Mannose
Xylose
Galactose
Deoxy hexoses
Uronic acids
Glucose*
Lignin

* Retrograded or indigestible starch may be included under the heading of fibre.

FIBRE

Fibre is the term used to describe an ill-defined group of substances (Tables 12 and 13). They are not essential (breast milk contains none) but have been shown to benefit sufferers from certain common disabilities, notably constipation, diverticular disease and haemorrhoids (NACNE, 1983; COMA, 1984).

Fibre consists of an immense variety of polymeric substances ranging from lignin to carbohydrate polymers (other than those which are readily digestible).

Little is known of the way in which fibre may be of benefit; it may be partially broken down by microbial action in the lower gut to yield volatile

TABLE 13
Composition of Cell Wall of Wheat Bran

Component	*% dietary fibre (lignin + non-starch polysaccharides)*
Lignin	16·3
Deoxyhexose	0·3
Arabinose	19·7
Xylose	29·5
Mannose	0·5
Galactose	1·1
Glucose (non-cellulosic)	4·5
Glucose (cellulosic)	25·4
Uronic acid	2·6

Source: Selvendron & Dupont (1984).

fatty acids (McNeill *et al.*, 1978) and this may be an important feature of its action. Fibre can impede absorption of certain dietary components, e.g. carbohydrate, zinc, iron and calcium. This effect may be of assistance to diabetics and of little significance for normal persons but could have adverse effects on those with limited dietary supplies or special needs.

NON-NUTRIENT FOOD COMPONENTS

These may be considered under four major headings:

(1) Gross inedible components and contaminants
(2) Non-nutrient constituents and cryptic contaminants
(3) Substances formed during food preparation
(4) Materials intentionally added to food

(1) Gross contaminants

Gross contaminants and inedible material, husks, skins, pips, stones, earth, guts, feathers, maggots, fungal and bacterial growths are often associated with edible materials and are removed before consumption. Such contamination is rarely hazardous because it is obvious and usually renders food inedible.

(2a) Non-nutrient substances

These are often present, e.g. fibre and indigestible saccharides. These may modify gut functions; some, like fibre, beneficially by preventing constipation—others such as stachyose may act harmlessly but antisocially by inducing gas (notably methane) formation in the bowel. Still others, like lactose, for the majority of adult humans may cause diarrhea and even griping pains if consumed in more than modest amounts.

Poisons are present in many of our common food species, e.g. members of the Cruciferae, Leguminosae, Solanaceae and Umbelliferae contain potent toxins (Smart, 1976; Rodricks, 1978; Conning, 1983; Albert, 1987; Ames *et al.*, 1987; Fenwick, 1987). The amounts of these substances commonly comprise up to 5–10% of plant dry weight (Ames *et al.*, 1987) and are thought to act as part of the mechanisms which defend the immobile plant against attack, whether from bacteria, fungi or animals. Many types of biocidal activity, often of very broad, non-specific, types are displayed, *inter alia*, anti-proteases, haemagglutinnins, cyanogenetic glycosides, carcinogens and antithyroid agents amongst an amazing variety of chemicals. In spite of their presence our foods are generally safe to eat, and nutritious, because they are normally prepared in ways which past experience has proved safe,

e.g appropriate cooking of beans or processing of cassava. In other instances the poison may be rapidly eliminated and the food rarely eaten in amounts liable to cause harm. Potatoes, for instance, often contain about 15 mg total alkaloids per 200 g portion, about one sixth of the toxic dose for man (Ames *et al.*, 1987). The safety and acceptability of potato thus depends on the fact that few of us are able, or willing, to eat about 1·2 kg ($2\frac{1}{2}$ lbs) at one meal.

Numerous known carcinogens and mutagens have been identified, albeit in small amounts, in many foods, ranging from roast beef to lettuce (Ames, 1983).

Mechanical damage, storage under adverse conditions or microbial attack, may induce production by the plant, of phytoalexins, presumed to be defensive chemicals. These substances can, and do, produce adverse effects, including tumours in those handling these edible plants (Ames *et al.*, 1987; Anon, 1988*a*).

There is also clear evidence that food itself may be harmful—in the rat an increased food intake shortens life span and increases cancer incidence while, in man, obesity is associated with increases in certain types of malignant growth (Ames *et al.*, 1987).

(2b) Microbial contamination

Contamination of food by vegetative forms, spores and cysts of microorganisms and metazooan parasites can occur, often unnoticed but with potentially dangerous consequences.

In most instances microbial contamination leads to gross changes making the product inedible. However, the presence of fungi in hay and other foods for stock and of cereals and pulses for human consumption can lead to formation, within the crop, of a variety of toxins without any readily detectable change in the apparent quality of the food made from it. The best known of these dangerous substances are the aflatoxins, tricothecenes and ergot alkaloids (Conning, 1983), which have been responsible for disease and death in poultry, other farmstock and man. The aflatoxins are highly toxic and carcinogenic at concentrations of fractional parts per million (mg/kg). Even with all precautions aflatoxin is commonly present in maize and groundnut harvests at parts per billion (μg/kg) probably as a result of fungal infection before harvest but other cereals and pulses are far less liable to this problem (Conning, 1983).

(2c) Pesticides

Plants and animals must be protected from pests and diseases if safe and nutritious food is to be produced economically.

Animals may need vaccination and treatment with antibiotics while herbicides, insecticides and fungicides may be necessary to destroy weeds and control insect attack or microbial contamination of our food.

The harvested cereals, pulses and vegetables also need to be protected from these same agents, not only to prevent obvious loss but also to inhibit the formation of toxins by apparently minor fungal infections.

The artificial chemicals used must act specifically in being able to control the pests but without effect, at the doses used, on the plants or animals being protected, or on those who will consume them.

The use of pesticides is not permitted until thorough research has shown that their use is safe in the field and that residues, if any, left in the food will be without effect on the consumer.

(2d) Microbial pathogens

On animal products gross microbial growth normally leads to obvious corruption and consequent inhibition of consumption. For certain items—game birds, hare and venison—some microbial decay is deemed desirable. Such foods may well contain bacteria and toxins which would be dangerous if eaten raw but these contaminants are safely eliminated by the heat used to cook these delicacies.

Low acid foods, typically meat, poultry, fish and legumes, can support the growth of dangerous pathogens and toxin producers; thus care must be taken to ensure that all such foods are properly cooked to eliminate any microbes and their toxins. After cooking particular precautions must be taken to prevent contact with and contamination from the uncooked food and to ensure that the cooked products are not exposed to conditions under which harmful microorganisms may flourish.

Meat and fish may bear cysts of parasitic worms which can infect man-helminths, nematodes and cestodes. These are killed by cooking but differ from vegetative bacteria in that they are destroyed, rather than preserved, by freezing.

(3) Substances formed during food preparation

Heat treatment, whether in the kitchen or the factory, is the single most important process applied to food. The apparently simple methods, frying, roasting, steaming, boiling, grilling and the application of infra-red or microwaves in baking, can all, by raising the temperature, destroy vegetative organisms and thermolabile toxins. At the same time heat makes many foods easier to consume and their components more digestible. For

mankind this application of fire immensely increased the safety, the number and the nutrient value of available foods.

Heat not only destroys toxins but also, depending on conditions, may cause some changes in nutrient content. Apart from this, cooking also brings about numerous chemical reactions thus creating thousands of novel chemicals in the cooked food. Many of these substances are responsible for the tastes, aromas, flavours and colours of cooked dishes. Most of these chemicals occur at low concentrations but some are highly toxic, others are carcinogenic or mutagenic but very few have been subjected to toxicological investigation (Ames, 1983; Conning, 1983; MacLeod, 1984; Albert, 1987; Ames, 1987).

Fermentations have been carried out on foods since prehistoric times to change the texture and flavour and to preserve. The products are many and varied, bread and cheese, fermented meats and sausages, soya bean and fish sauces, to name but a few. In all instances the starting material is changed by microbial action which converts food components into new substances, e.g. alcohols, amino acids, organic acids and flavours *inter alia.* Many of the substances formed may be potentially toxic—few have been isolated and examined—but the traditional fermentation processes have been developed and continue because the products are useful and attractive while causing no more apparent harm than the unprocessed items.

(4) Substances added to foods

From the earliest times numerous ingredients—often with little or no nutrient value—have been added to foods to modify taste, colour, texture or other attributes. Could this be a manifestation of the omnivore's desire for variety? Hundreds of such additions have been and are being made today, some for flavour and aroma or colour, texture and appearance or to preserve foods or for miscellaneous purposes (Tables 14–16).

TABLE 14
Non-Nutrient Food Components which Convey Flavour and Colour, and their Sources

Flavour	*Colour*	*Source*
Bay	Spinach	Leaves
Angelica	Rhubarb	Stem
Ginger	Beet	Root
Vanilla	Paprika	Fruit
Mustard	Poppy	Seed

TABLE 15
Separated Food Components used for Texture and Appearance

Carbohydrates	Starch
	Sugar
	Gums
Proteins	Egg white
	Skim milk
	Gelatine
Glycerides	Oils
	Dripping
	Butter

TABLE 16
Preservatives and Miscellaneous Additives

Salt	Alcohol (brandy)
Honey	Phenols (smoke)
Nitre	Sodium bicarbonate
Yeasts	Baking powder
Vinegar	Pectin
Rennet	Lemon juice

Often it is inconvenient, or may be unattractive, to use the roots, bark, ground spice or leaves themselves; thus extracts, essences and the pure flavour chemicals or other active principles may be used instead and in some instances an artificial or synthetic substance may perform the required function. It is illegal to add to food anything which may injure health; thus, apart from traditional items or accepted foodstuffs, official approval is required before any substance can be added to foods even if that substance may be a normal component of many food items. Evidence as to need and safety must be presented before permission may be given. If approved for use in the countries of the EEC the substance will be given an E number to act as an identification code to denote its chemical nature and function (IFST, 1986; MAFF, 1987) and if added to food its presence must be noted on the label.

Many of the food additives given E numbers are chemicals which are to be found in numerous untreated foods and garnishes. It is perhaps fair to say that these constitute the major proportion of the few chemicals which have been critically tested for toxic effects, out of the many thousands which are present in our foods.

All ingredients incorporated into manufactured foods must be listed on the label; thus all food additives, whatever their nature, will be so declared but, if these same substances are by nature present, then listing is not a legal requirement. However, it has been pointed out (Anon, 1988*b*) that if all E number substances required declaration every pack of tomatoes would note the presence of the following:

Flavour enhancer	Monosodium glutamate	E 621
Colours	Carotene	E 160(a)
	Lycopene	E 160(d)
	Riboflavin	E 101
Antioxidant	Ascorbic acid	E 360

These could also be joined by:

Cellulose	E 460	Malic acid	E 296
Pectin	E 440	Fumaric acid	E 297
Alpha tocopherol	E 306	Succinic acid	E 363
Citric acid	E 330	Bicarbonate	E 500
		Carbon dioxide	E 290

INTOLERANCE TO FOODS AND FOOD COMPONENTS

Though food additives, like the other food ingredients, are generally safe in use, there is the occasional person who may be very sensitive to one or another of them in much the same way that some folk find that certain foods do not agree with them. It is difficult to state precisely how many people will respond badly to a particular food or food ingredient, but one in every one or two hundred will find that unpleasant reactions may be evoked by certain foods—milk, soya products, fish or eggs are not infrequently involved. Smaller numbers—about one in a thousand—may react to one or other of the food additives (EEC Scientific Committee for Food, 1982). Though the numbers may be small the inconvenience and distress caused to the sufferers is such that it is reasonable that *all* food ingredients, of whatever nature, should be disclosed so that persons finding themselves sensitive to any one will in future be able to avoid foods containing the offending items.

HOW DO ANIMALS SURVIVE?

Higher vertebrates, to survive, must acquire from the environment sufficient of each of the numerous life essentials while avoiding over consumption of any of them or even one of the thousands of other potentially toxic substances in their food.

Meeting these difficulties, even with the help of nutritionists and toxicologists, would appear a daunting task, yet the very existence of so many animal species shows that successful strategies have evolved.

(1) Selection of non-toxic foods

This is an apparently simple strategy but requires the ability to identify, capture and consume—a mode of existence adopted by carnivores.

This strategy is only hindered by the physical and chemical mechanisms evolved by potential prey species to defend themselves. The majority of plants and many animals only survive because they are poisonous or

offensive to potential consumers. The exceptions are those which are sufficiently fecund, well hidden, widely dispersed, agile or ferocious enough to escape or deter. Many slow animals—toads, puffer fish, slugs and insects—are intensely poisonous or repulsive while between 5 and 10% of the dry weight of most plants consists of chemical toxins.

(2) Specialisation

Numerous species, the Koala bear, the Giant Panda and countless insects have evolved the ability to eliminate, inactivate or metabolise and utilise the toxins and excess nutrients present in their single food source.

This strategy has advantages in that identification of food is simplified but has disadvantages in that the predator is wholly dependent on the prey and so if this is seasonal the predator must either migrate, like the whale and the swallow, or deposit food stores sufficient to overcome temporary starvation. Alternatively, survival capsules may be produced so that, though the individual may die, the species may continue in the form of eggs, cysts or pupae (as in many metazoan parasites and insects) until food is again abundant.

(3) The generalist or omnivore

Omnivores, such as man and the rat, habitually consume a wide variety of foods. This characteristic has advantages in that consumption of limited amounts of numerous items maximises the probability of obtaining a sufficiency of each of the numerous chemicals needed to support life while minimising the chance of ingesting harmful amounts of any one agent. There is also the advantage that survival is not dependent on the availability of a single prey species.

At the same time there are serious risks for the omnivore—many foods are poisonous and others nutritionally imbalanced; thus uncontrolled gluttony may at best be debilitating and at worst lethal.

To survive these dangers the omnivores appear to have evolved protective neurosensory mechanisms to prevent overindulgence on any one food without depressing the ability to eat other foods of different types, i.e. they are endowed with the ability to select a mixed diet.

THE CONTROL OF FOOD INTAKE

It is surprising that so little has been done to elucidate factors controlling food selection and consumption (Boakes *et al.*, 1987).

Most people, without conscious effort, maintain their weight within very narrow limits. Detailed investigations show that total food consumption may vary greatly from meal to meal and from day to day apparently showing little control of total food intake. There must, over the long term, be a precise match between food consumption and expenditure because if this balance were not better than 1% there would be big gains—or losses—in weight over a one year period whereas such changes are not normally apparent.

Consumption of nutrients other than energy also may change from day to day but may be excellent over long periods (Beaton, 1988). This phenomenon explains why short term dietary studies (over a few days only) may report major dietary deficiencies in populations for which there is no other evidence for lack of any essential food components (Emery *et al.*, 1988).

Studies of human behaviour have shown that the sensation of pleasantness and the appetite for a particular food declines rapidly as that food is consumed but this does not affect the appreciation of other foods in different organoleptic categories. At meals at which the second course was similar to the first, total food consumption was about 25–30% less than when the second helping was of a different character. Savoury foods were found to depress appetite for other savoury food but not to alter desire for fruits and sweets. Similarly consumption of fruit or sweet did not decrease relish for, or consumption of, savoury items though appetite for another sweet course was depressed (Rolls *et al.*, 1984).

These behavioural patterns widen the choice of food available for support of life and health by encouraging the consumption of numerous different foods, thereby maximising the chance of obtaining all nutrient needs without any excesses of potentially harmful substances.

It is tempting to speculate that better knowledge of the neurosensory mechanisms involved would offer keys to the control of appetite and its derangements, thus suggesting methods for the therapy of conditions such as *anorexia nervosa* and obesity.

WHAT ARE THE MAJOR DANGERS LURKING IN OUR FOODS?

Risk assessment

There are many hazards associated with food—these range from the penalties of overindulgence to malnutrition, from microbial disease to poisoning by food toxins. The perceived importance of these different

factors depends not only on circumstances but also on what is believed (in the face of firm beliefs facts are usually considered irrelevant!) (Cantley, 1987).

Roberts (1978) stated that the dangers should be assessed on the basis of mortality and morbidity depending on:

1. Severity, ranging from death to disability or minor discomfort.
2. The number of incidents.
3. The onset, whether an acute or chronic condition.

Ranking of hazards

Using these criteria mortality and morbidity data clearly show that, in the USA, the UK, and developing countries, food-borne disease of microbial origin (whether directly from pathogenic organisms or from microbial toxins) is by far the major hazard (Roberts, 1978; FAO/WHO, 1984; Gray, 1985; Strachan, 1986). In the UK many thousands of such incidents (mostly from meals in private homes) and numerous deaths occur every year (Roberts, 1988). Though many times less common there is still a problem of malnutrition mainly amongst the elderly unable to manage properly for themselves but also, increasingly, in families in which doting parents inflict unorthodox nutritional concepts on their children. Environmental contamination and natural food toxins can still present problems (Rodricks, 1978) but these are several orders of magnitude less than those of microbial origin. Lastly, pesticide residues and food additives can, on occasion, induce adverse reactions but the severity and incidence of these upsets is so low that actuarial assessment is impracticable (Upton, 1982). The major problem to be faced is indubitably that of food-borne disease.

Control of food-borne disease

Government health measures, based on medical and veterinary findings together with their appreciation by the general public, have done much to safeguard health and well being in our country. Examples are the control of infantile enteritis and elimination of bovine tuberculosis during the first half of the present century.

Control of infantile gastroenteritis

In the early 1900s total infantile mortality was about 130 per 1000 live births mainly attributable to summer diarrhea—infantile gastroenteritis—associated with the consumption of raw cow's milk on weaning from the

TABLE 17
Infantile Mortality of Children under 2 Years per 1000 Live Births in London

Period	*Deaths from diarrhea and enteritis*	*Total infantile mortality*
1911–15	72·0	128·5
1916–20	27·7	80·5
1921–25	21·2	61·7
1926–30	12·2	48·1
1931–35	11·4	47·7

breast. Table 17 (Wilson, 1942) shows the rapid decline in infantile mortality over subsequent decades, a decline attributable in large measure to the decrease in deaths from gastroenteritis from 72 down to 11 per thousand. This achievement has been ascribed to several factors (Wilson, 1942):

1. Replacement of raw by pasteurised milk.
2. Introduction of cold storage for major milk deliveries.
3. Decrease in flies through replacement of horse by mechanical transport.
4. Retailing of pasteurised milk in sealed bottles in place of the jug and churn.

To these should be added:

5. Replacement of liquid milk for infant feeding by roller dried (pathogen-free) milk powders (Sheill, 1912).

and

6. Public awareness of the dangers of 'germs' and the emphasis in newspapers and magazines of the importance of hygiene and cleanliness.

Of these measures the most important was undoubtedly the introduction of pasteurisation.

Pasteurisation is the term used to describe the destruction of vegetative organisms. The process does not sterilise, it eliminates vegetative microorganisms with minimum deleterious effect on food 'quality' as judged by taste, smell, colour, texture and nutrient value. The process is commonly effected by controlled heat-treatment but can also be brought about by irradiation with X or gamma rays.

The heat-treatment of liquids to destroy vegetative organisms was first reported by Pasteur in the mid 1860s to prevent souring of wine and beer.

Somewhat later the larger dairy companies applied the technique to improve the shelf life of milk—a highly perishable but costly commodity. The public health implications of the process were only recognised much later and many investigations were held to determine the precise times and temperatures needed to ensure efficient processing. In 1936 the first UK regulations were promulgated and, by 1944, 98% of the milk sold in London was pasteurised. Since 1983 sale of unpasteurised milk to the public has been completely prohibited in Scotland and only permissible under tight restrictions in England.

Control of bovine tuberculosis

Bovine tuberculosis accounted for a high mortality, mainly among children, amounting to between 1500 and 2000 deaths per annum in the early years of this century (Wilson, 1942). The problem was subjected to a two-fold attack: (1) the elimination of tubercular cattle and (2) destruction of mycobacteria by pasteurisation of the public milk supply. The success of these measures is shown by the disappearance of this disease from the UK shortly after 1950 (Galbraith & Pusey, 1984). The contribution of pasteurisation to this success is shown by the observation that the fall in incidence was most rapid in those parts of the country in which pasteurisation was more quickly and widely introduced.

Present problems

Outbreaks of disease are investigated by the Public Health Laboratory Service Communicable Disease Survey Centre. The work of this group (PHLS, 1988*a*,*b*) has clearly shown (Figs 1 and 2) a great increase in the

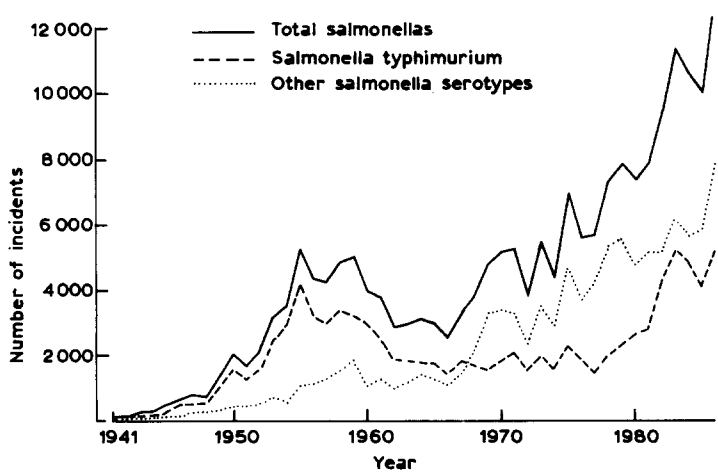

Fig. 1. Salmonellosis, England Wales, 1941–86. (Source: PHLS (1988*a*)).

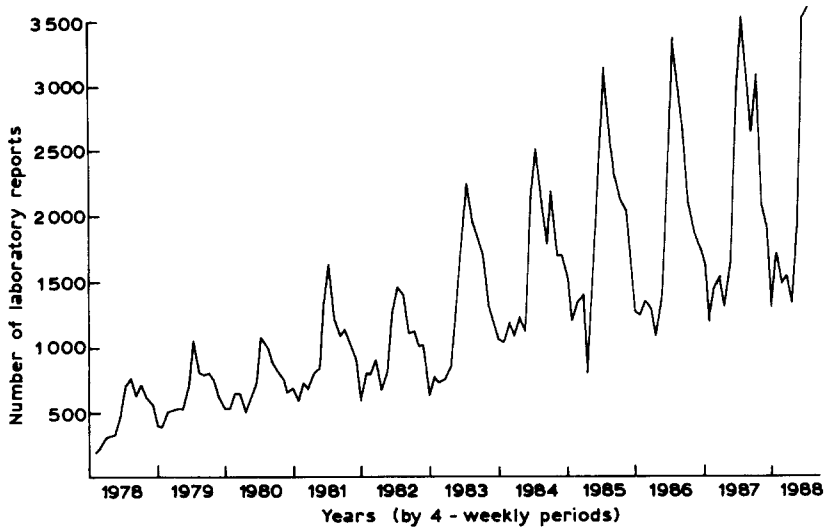

Fig. 2. Campylobacter enteritis: England, Wales and Ireland 1978–88. (Source: PHLS (1988*b*)).

incidence of food-borne infections. Those attributable to *Salmonella*, for instance, rose from less than 100 per year in 1941 to over 12 000 in 1987 while the number of *Campylobacter* infections has risen from less than 500 in 1977 to over 3000 in 1987. Food-borne *Listeria* infections are an even more recent cause for concern.

Several measures could be taken to eliminate these health hazards though some may be more difficult to implement than others and all require awareness and cooperation from the public:

1. Public awareness of the need for proper food handling in the home.
2. The elimination of sources of infection.
3. Prevention of contamination of food.
4. Destruction of pathogens in food.
5. Prevention of growth of infective organisms.

Source of infections

The vast majority of food-borne infections arise from contamination by animals (including man) of animal products—meat, poultry, fish, shellfish, cheese, frogs' legs, etc. The recent distressing increase in food-borne disease mainly arises from salmonellosis and other pathogenic conditions in animal populations. Such diseases are difficult to detect especially when the incidence is low and symptoms and signs not severe. The problem is aggravated because, under large scale production conditions, one infected animal can lead to contamination of many others. As a result it is common to find salmonellae and other pathogens in poultry and other carcasses.

The presence of some contamination by salmonellae and similar vegetative pathogens is of little consequence if the food is prepared properly and simple precautions taken (FDF, 1988; Anon, 1988*b*) i.e. cooked adequately and eaten promptly or, after cooking, protected from contamination and stored at refrigeration temperatures before consumption. The increasing incidence of disease clearly shows that these precepts are disregarded in many homes and catering establishments.

The successful elimination of bovine tuberculosis and infantile diarrhea as serious health hazards in this country shows how successful proper action can be in the prevention of food-borne disease and how effective is pasteurisation by removing the offending pathogens from the raw material.

Irradiation pasteurisation

Pasteurisation by thermal procedures applicable to liquids—wines, milk, beer and fruit juices—cannot be applied to solids, meat, fish, poultry, spices or cheese. Products of this type can, however, be pasteurised by irradiation to destroy vegetative pathogens in much the same way as thermal treatment. Over forty years of investigation have shown that gamma rays (from Cobalt 60) or X rays (from an electron beam) can destroy vegetative pathogens without detriment to nutritional value (FAO/IAEA/WHO, 1981; ACINF, 1987). In spite of the scientific evidence, many consumers have grave doubts about the safety, for them, of irradiation processes of which they have had no previous experience. This difficulty is exacerbated by the absence of any test to determine whether a food has, or has not, been properly irradiated. This should not be an insuperable problem because vegetative organisms will be present if the food has not been properly irradiated while over-treatment quickly leads to the appearance of off-odours (some foods acquire a 'wet dog' flavour) which would make such products unsaleable! Legal requirements for pasteurisation of milk could not be promulgated until methods were available to define the treatment; thus, though highly desirable from the public health point of view, irradiation pasteurisation may not be acceptable in spite of its obvious potential health benefits.

CONCLUSION

It is hard to answer the question as to what is a 'healthy food' because there may be no such thing and the phrase may merely describe an imaginary concept.

All foods can be 'healthy', i.e. support full health and well being if used as *part* of a diet.

Conversely every food could be unhealthy, no matter what its com-

position, if eaten in excess; furthermore no single food is able to meet all the different needs of all mankind at all stages and ages (breast milk alone, even if available, could not support health in adults).

There is truth in the old adage 'One man's meat is another man's poison'.

The question now should be 'What is a healthy diet?'. The answer would appear to be a *mixed* diet (Passmore *et al.*, 1979; Passmore, 1988) based on findings proved to be effective by tradition as well as supported by science, rather than to follow the latest fashionable and attractive but unproven hypotheses. Variety in food selection can eliminate many of the problems which could arise from nutrient deficiency, or presence of toxins, which may be associated with the use of a single staple food.

If overindulgence is the major problem then the advice of COMA (1984) and NACNE (1983) would appear to be admirable in that animals, and by analogy, men, consume less total energy, if the diet is high in fibre while decreasing salt, sugar and fat in food may be expected to decrease their appeal to many and so may assist them to combat gluttonous inclinations.

The consumption of unusual foods or unusual supposed nutrients is not to be encouraged. Substances which alleviate one condition may exacerbate others while the usually harmless, in untoward amounts, may prove by painful experience to have powerful toxic actions on apparently normal persons. It is chastening to recall that Vitamin D, the 'Sunshine Vitamin' was used for three decades, until the 1950s, before its dangers were recognised while sunshine itself—first adulated in the 1920s is only now commonly recognised to present dangers other than sunburn! Perhaps the best way to ensure healthy eating is to provide a good mixed diet while avoiding microbiological hazards. Most food-borne disease has been shown to result from thoughtless handling, or unsuspected risk, taken in the kitchen. It is well to remember the words of Foster (1978): 'We know how to prevent foodborne disease; all we need do is apply the knowledge. An outbreak of food poisoning simply means someone failed to follow good food handling practice.'

The media could play a major part now, as did the press in the early days of the present century, by calling attention to the dangers of microbial disease and how to overcome them. The large retail food stores provide excellent guides to safe food care while further, more detailed, information is available from most libraries (Hobbs & McLintoch, 1973; Hobbs & Gilbert, 1978; Hayes, 1981; Anon, 1988*b*; FDF, 1988).

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