Chapter 1

Understanding cancer

There are over 200 different types of cancer that can occur anywhere in the body, all having different causes and symptoms and all requiring different treatments (Cancer Research UK 2004). In the UK, the lifetime risk of developing cancer is one in three. Cancers affect people at different ages, with the risk rising significantly with age; 65% of cancers occur in people aged over 65 (Cancer Research UK 2004).

A cancer develops when a cell experiences a mutation that makes the cell divide more rapidly than usual, leading to an overgrowth of tissue (National Institutes of Health or NIH 2004). This overgrowth is often referred to as a tumour. Some tumours are not cancerous and are termed ‘benign’; those tumours that are cancerous are termed ‘malignant’. Uncontrolled growth of cells causes the tissue to look abnormal. If malignant cells are contained within the original tissue the cancer is called in situ, whereas if they invade neighbouring tissue the cancer is said to be invasive. One property of most malignant tumours is their ability to undergo metastasis – the spread of cancerous cells to other parts of the body (Tortora and Grabowski 2004).

Several factors may cause a cell to become cancerous, e.g. environmental agents in the food we eat, chemical agents, radiation and viruses. These agents are called carcinogens and cause mutations in the genetic make-up of the cell (Tortora and Grabowski 2004).

History of cancer and its treatment

The earliest description of cancer is from Egypt in the seventeenth century BC, when a physician wrote about a patient with cancer suggesting that there is no treatment. The Edwin Smith Papyrus describes tumours of the breast (American Cancer Society 2004).

In the fifth century BC, Hippocrates, the ‘father of medicine’, suggested in his writings that it is better not to apply any treatment in cases of occult (hidden) cancer because, if treated, the patients die quickly whereas if not treated the patient holds out for longer (Yarbro et al. 2000). It is
a widely held belief that the word *karkinos*, Latin for crab, was first applied to cancer by Hippocrates. *Karkinos* aptly describes the appearance of a malignant tumour, which is irregular in outline and uncontained, because crabs move in an erratic manner, sideways, often with sudden bursts of movement (Morgan 2001).

The Egyptians blamed cancer on various gods. Hippocrates explained all diseases as resulting from an imbalance of the four humours: blood, phlegm, yellow bile and black bile. A balance of these fluids resulted in health. In the case of cancer, an excess of black bile reportedly collected in various body sites (American Cancer Society 2004). In the Dark Ages little progress was made.

**Cancer epidemiology**

During the Renaissance, starting in the fifteenth century, scientists in Italy developed a greater understanding of the human body, mainly as a result of postmortem examinations and a clearer understanding of the organs of the body. In the seventeenth century, Bernardino Ramazzini, an Italian physician, noted the high incidence of breast cancer in nuns and suggested that it was in some way related to their celibate lifestyle. This launched cancer epidemiology, the study of the occurrence, distribution and control of the disease in the population. This was followed in the eighteenth century by the description of scrotal cancer in chimney sweeps by Sir Percival Pott, which led to many studies that identified a number of occupational carcinogens and then to public health measures to reduce cancer risk (Yarbro et al. 2000, American Cancer Society 2004).

**Cancer surgery**

Giovanni Morgagni laid the foundations of surgical oncology during his work with the pathology of cancer *post mortem* and John Hunter, a Scottish surgeon, applied this to practice (Yarbro et al. 2000). In the mid-nineteenth century, with the advent of anaesthesia, surgeons emerged who rapidly advanced surgical techniques for cancer: Theodor Bilroth in Germany, W. Sampson Handley in London and William Stewart Halsted in America. Their work led to cancer surgery designed to remove all of the tumour, as well as the regional lymph nodes, on the understanding that cancer was contained within anatomical compartments and metastasis spread via the lymph vessels. This thinking dominated cancer surgery...
for almost a century, until it was called into question by the work of twentieth century surgeons.

At the same time as Halsted and Handley, another surgeon, Stephen Paget, was researching metastatic spread and concluded that blood vessels, as well as the lymph, were involved and that the disproportionate spread of metastases to certain organs could not be the result of chance. Paget drew an analogy between metastases and seeds that were carried in all directions but could live and grow only on fertile soil; therefore cells from a tumour were able to grow only in certain organs – not just any organ where they came to rest. Paget’s work contributed to a new understanding of cancer that still applies today (Yarbro et al. 2000, American Cancer Society 2004). The understanding of metastasis became a key element in the recognition of the limitations of cancer surgery. It allowed doctors to develop systemic treatments that were used before and after surgery to destroy cells that spread through the body, enabling the use of less mutilating surgery (American Cancer Society 2004).

Cancer treatments

In 1878 Thomas Beatson, a graduate of Edinburgh University, discovered that the breasts of rabbits stopped producing milk after he removed the ovaries, drawing the conclusion that one organ (the ovaries) has control over the secretion of another separate organ. Beatson tested this by the removal of ovaries in women with advanced breast cancer and found that it resulted in some improvement. He discovered the stimulating effect of oestrogen on breast cancer even before the hormone itself was discovered. Beatson’s work provided the foundation for the modern use of hormone therapy in the treatment of breast cancer (Yarbro et al. 2000).

In 1896, Wilhelm Conrad Roentgen, a German physicist, discovered the X-ray, X being the algebraic symbol for an unknown quantity. Within 3 years, radiation was being used in the treatment of cancer. Unfortunately, radiation was recognized as a cancer-causing agent 7 years after its discovery, and a few years later a relationship with leukaemia was recognized (Yarbro et al. 2000). Today radiation is delivered with great precision to destroy tumours while preserving normal tissue (American Cancer Society 2004).

During World War II, naval personnel exposed to mustard gas as a result of military action were found to have severe bone marrow depression. In the studies that followed, a compound called nitrogen mustard was studied and found to have an effect on cancer of the lymph nodes. From this work, the development and use of chemotherapy drugs
resulted in the successful treatment of many people with cancer. Cancers that can be cured with chemotherapy include lymphoma, acute childhood leukaemia, testicular cancer and Hodgkin’s disease. Many other cancers can be controlled for long periods of time if not cured.

More recently, scientists’ understanding of the biology of cancer cells has led to the development of biological agents that mimic natural signals used by the body to regulate growth. This cancer treatment, commonly referred to as biotherapy or immunotherapy, has proved effective for the treatment of several cancers.

Cancer in the twentieth century

James Watson and Francis Crick received the Nobel Prize for their work in the discovery of the exact structure of DNA, which enabled scientists to understand how genes work and how they could mutate. There was already an understanding that cancer could be caused by chemicals, radiation and viruses, and that sometimes cancer seemed to run in families. With the study of DNA it was discovered that damage to DNA by chemicals and radiation or the introduction of new DNA sequences by viruses led to the development of cancer. It therefore became possible to pinpoint the exact site of damage to a specific gene; it was also discovered that these damaged genes can be inherited.

Cancer biology

The normal cell and cell cycle

The body is made up of about 200 different types of cell. Each cell is a functional unit and arises from existing cells by a process of cell division. The cell is made up of three key parts: the plasma membrane, cytoplasm and nucleus.

Plasma membrane

This is the cell’s outer surface which is made up of fats and proteins. The membrane allows substances to move in and out of the cell and restricts the movement of some substances. The function of the plasma membrane is determined by its protein make-up.
Cytoplasm

This is made up of all the cellular contents apart from the nucleus. There is a fluid portion, namely cytosol, within which are small organs termed ‘organelles’; each organelle has a characteristic structure and function (Tortora and Grabowski 2004).

Cytosol

This is the fluid inside the cell; it makes up 55% of the cell’s volume and is composed of water, solutes and particles. The cytosol is the site of many of the chemical reactions that allow cellular growth (Gabriel 2004, Tortora and Grabowski 2004).

Organelles

Each organelle is a functional unit where specific processes take place:

- Cytoskeleton: a network of protein filaments that maintains the shape and organization of the cell.
- Centrosome: made up of proteins important in cell division.
- Cilia and flagella: cilia are hair-like projections extending from the surface of the cell which propel fluids across the surface of cells. Cilia present in the cells of the respiratory tract sweep foreign particles trapped in mucus away from the lungs. Flagella are longer than cilia and can move entire cells; the only example in the human body is the tail of the sperm cell.
- Ribosomes: these are the sites of protein formation and they are made up of proteins and nucleic acids.
- Endoplasmic reticulum (ER): a network of folded membranes taking up half of the cytoplasm. It consists of rough ER and smooth ER. The rough ER is studded with ribosomes, and it processes and sorts proteins formed by the ribosomes. The smooth ER does not contain ribosomes and its function is the formation of fatty acids and steroids; it also plays a part in the inactivation of harmful substances including carcinogens.
- Golgi complex: made up of membranous sacs it stores packages and exports proteins from the rough ER to other parts of the cell.
- Lysosomes: made up of sacs containing 60 different digestive enzymes that break down the final products of digestion and allow them to be transported into the cytosol. Lysosomes also digest worn-out organelles (autophagy) and can destroy themselves (autolysis). Autolysis occurs in some medical conditions and also occurs after death when tissue deteriorates.
- Peroxisome and proteasome: peroxisomes detoxify harmful substances and proteasomes break down unneeded, faulty or damaged proteins.
- Mitochondria: these are the power house of the cell where energy is produced. Some cells contain thousands of mitochondria, but others just a
hundred, e.g. active cells such as the liver, kidneys and muscle have large numbers of mitochondria and use up lots of energy (Gabriel 2004, Tortora and Grabowski 2004).

Nucleus

This is a spherical or oval structure, usually the most prominent feature of the cell. Most cells have a nucleus with the exception of mature red blood cells; some cells, e.g. skeletal muscle cells, have several nuclei. The nucleus contains one or more spherical nucleoli that are clusters of protein, deoxyribonucleic acid (DNA) and ribonucleic acid (RNA), which engineers ribosomes.

Within the nucleus are most of the cell’s hereditary units or genes, which control cellular structure and direct cellular activities. Genes are arranged in a single row on chromosomes. Human body cells have 46 chromosomes; 23 are inherited from each parent cell. The total genetic information carried in a cell is called its genome (Tortora and Grabowski 2004).

Gene action or protein synthesis

The main job of the cell is protein production or synthesis. The proteins determine the physical and chemical characteristics of the cell and in turn of the organism. The DNA contained in genes provides instructions for making proteins. The information in the DNA is copied to produce a specific molecule of RNA, which attaches to a ribosome where the information from the RNA is translated into a sequence of amino acids that goes on to form a new protein molecule.

DNA consists of repeating building blocks called nucleotides. Each nucleotide is made up of three parts: a nitrogenous base, deoxyribose and phosphate.

The nitrogenous bases consist of one of the following nucleic acids: adenine, guanine, thymine or cytosine. The DNA is made up of two strands, similar to a ladder, which twist around each other, giving it the double helical structure. The strands consist of alternating deoxyribose and phosphate groups, and the ‘rungs’ of the ladder contain the nitrogenous bases made up of one of the nucleic acids, the ‘rungs’ being formed when the nucleic acids from each strand join together in sequence: adenine always paired with thymine and cytosine with guanine. About 1000 rungs of DNA make up a gene, and humans have between 35 000 and 45 000 genes.
Any change that occurs in the sequence of the nucleotides (known as adenosine, or A, guanosine or G, thymidine or T and cytidine or C) of a gene is known as a mutation. Mutations can result in the death of a cell, cause cancer or produce genetic defects (Gabriel 2004, Tortora and Grabowski 2004).

**Cell division**

Cell division replaces damaged, diseased and worn-out cells. There are two different types of cell division:

1. Meiosis or reproductive cell division produces sperm and eggs, the cells needed to form the next generation of sexually reproducing organisms.
2. Mitosis or somatic cell division produces two identical cells after division in all other body cells (Gabriel 2004, Tortora and Grabowski 2004).

**Mitosis**

A cell divides into two identical cells by replication of the DNA sequence so that the same genetic material can be passed on to the newly formed cells. The cell cycle is the term used for the sequence of events occurring from the time a cell forms until it duplicates and divides (Gabriel 2004, Tortora and Grabowski 2004).

**The cell cycle**

- Interphase: a state of high activity during which the cell replicates its DNA and manufactures additional organelles in anticipation of cell division. As DNA replication starts the helical structure of the DNA partly uncoils and the rungs of the ladder containing the nucleic acids separate. The nucleic acids then pair up with the newly synthesized DNA. This uncoiling and pairing continues until each of the two original DNA strands is joined to the newly formed DNA strand. The original has become two identical DNA molecules.
- Mitotic phase: mitosis or division of the nucleus occurs followed by division of the cytoplasm into two cells. The duplicated chromosomes segregate, one set moving into each of two separate nuclei (Tortora and Grabowski 2004).

Each human cell has the same DNA content but only a small number of genes are expressed, giving each cell a distinct structure and function – termed ‘differentiation’.
Carcinogenesis or cancer growth can be split into three stages:

1. Initiation: environmental, systemic or genetic changes to the gene.
2. Promotion: expression of mutant genetic information even after long periods of latency, sometimes by the initial promoter or carcinogen.
3. Progression: cells assume malignant behaviour, invade adjacent tissues and metastasize.

There are three main causes of cancer:

1. Environmental factors, diet, industrial pollution and viruses: studies of patterns of cancer around the world suggest that the key environmental factors related to cancer are smoking, diet, alcohol and sexual habits, and that some cancers may be avoidable if the environmental agent is removed.
2. Systemic factors, including the breakdown of the immune system.
3. Genetic factors that give rise to a degree of susceptibility for cancer.

Research suggests that cancer results from an interaction of factors at the cellular, genetic, immunological and environmental levels (Souhami and Tobias 1998, Yarbro et al. 2000, Gabriel 2004). Theories of tumour development suggest that cancer is an accumulation of mutations resulting from multiple events that act together.

Control of cell division is carefully maintained by opposing sets of genes, which promote and inhibit growth. Damage to genes is repaired by enzymes that are coded by DNA-repair genes. When damage cannot be repaired, the cell is destroyed. The number of times a cell is allowed to replicate is also controlled and limited. Growth-promoting genes are termed ‘oncogenes’ and growth inhibiting genes ‘tumour suppressor genes’. The terminology used to describe growth regulation reflects the fact that the genes were initially thought to be cancer genes; normal cellular genes are often termed ‘proto-oncogenes’, adding to the confusion!

Carcinogenesis or cancer growth occurs when these genes are damaged to such an extent that normal control mechanisms for growth spiral out of control. For cancer to develop, oncogenes (growth-promoting genes) must be activated, tumour suppressor genes (growth-inhibiting genes) inactivated, DNA-repair genes inactivated and cell death blocked, and the biological clock turned off so that cells can become immortal (Souhami and Tobias 1998, Yarbro et al. 2000, Gabriel 2004). This process occurs with damage to the genome as a result of environmental, systemic and genetic factors. Oncogenes become mutated and are activated, and tumour suppressor genes and DNA-repair genes are mutated and become inactivated. It is common for mutated tumour suppressor genes or DNA-repair
genes to be inherited, but inheritance needs to be from both parents for it to have an effect. Oncogenes are dominant genes and inheritance of a mutated gene is therefore necessary from only one parent for the effect to be shown.

Cancer may be thought of as a defect in the control of the cell cycle: tumour suppressor genes regulate checkpoints in the cell cycle, and if this does not occur growth is not controlled. It may also be thought of in terms of programmed cell death: mutated cells with defective DNA are induced to undergo programmed cell death or apoptosis. This process is defective in cancer cells (Souhami and Tobias 1998, Yarbro et al. 2000). Cancer can finally be thought of in terms of evolution and natural selection: the mutated gene eventually takes control until the host dies.

The effect on the cell

**Altered cell growth**
There is immortality, contact inhibition is lost (the property that stops normal cells growing when they come into contact with other cells), adhesion is lost (cells hold less firmly to each other), and there is loss of cell cycle control and programmed cell death. Cancer cells tend to be poorly differentiated, and in some cancer cells the tissue of origin cannot be confirmed.

**Cytological**
There is increased size and number of nucleoli, reflecting greater metabolic need and activity.

**Changes in cell membrane**
New surface antigens are exhibited.

**Metastasis**
Metastasis is the major cause of cancer death; in many cases metastatic spread occurs before the initial diagnosis of cancer.

Metastasis is the spread of tumour cells to a new site in the body via the bloodstream or lymph system (Gabriel 2004). To support tumour growth and movement, angiogenesis or formation of a blood supply occurs. Newly formed blood vessels provide nutrition and oxygen to the growing tumour, as well as being a potential travel route to other parts of the body. The degree of angiogenesis in the primary tumour is directly linked to metastatic spread and is the subject of research. Tumour cells also need to be motile (to move spontaneously without aid); again some
research centres on anti-motile agents to reduce metastasis. Tumour cells have enzymes that allow surrounding tissues to be invaded and immuno-suppressive factors that evade destruction by the immune system. Distribution of metastasis is not random and is probably related to tumour cell characteristics; tumour cells may lodge in the capillary beds of multiple organs, and certain microenvironments determine whether tumour growth is supported. Despite a lack of knowledge about the exact mechanisms of metastasis, research continues with a view to interrupting this process (Souhami and Tobias 1998, Yarbro et al. 2000, Morgan 2001, Gabriel 2004).

The classification and staging of cancer

Histology

This defines the tumour according to the tissue from which it arises:

- Carcinoma or epithelial tissue, e.g. the urinary tract, the colon, the respiratory tract
- Sarcoma or connective tissue, e.g. bone, muscle, blood
- Neural tissue, e.g. nerve cells.

Grading

This is a method of classification based on the histopathological characteristics of the tissue. One description of the microscopic appearance of cancer cells is the degree of differentiation or specialist function of the cell. All normal cells start as immature cells and when mature they carry out a specialist function, i.e. skin cells, bone cells or muscle cells. Normal cells that become cancerous lose their specialist function, and cancers that resemble the normal tissue of origin in appearance and function are well differentiated. Undifferentiated tumours show little or no resemblance to their tissue of origin and tend to be more aggressive. The grades are as follows:

- GX: grade cannot be assessed
- GI: well differentiated
- G2: moderately well differentiated
- G3: poorly differentiated
- G4: undifferentiated.
It is anticipated that a grade 1, well-differentiated tumour offers a good prognosis for the patient. Undifferentiated tumours show little or no resemblance to their tissue of origin and tend to be more aggressive. There are some problems with grading: several grades may exist within one tumour and the grade may vary over time (Souhami and Tobias 1998, Yarbro et al. 2000, Fawcett and Drew 2002).

**Staging**

Staging is used to establish the extent of disease at presentation. Staging is a clinical and histological tool and is one of the methods used to indicate prognosis and determine treatment options for solid tumours (Morgan 2001, Fawcett and Drew 2002). The TNM (tumour, node and metastases) system is one of several ways of staging cancers and is one of the most popular possibly as a result of its simplicity. T stands for primary tumour and the relevance of local invasion, N for lymph node spread and M for the presence of distant metastases (Souhami and Tobias 1998, Morgan 2001, Fawcett and Drew 2002) (Table 1.1).

<table>
<thead>
<tr>
<th>Stage</th>
<th>Size</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1</td>
<td>&gt;2cm in diameter</td>
</tr>
<tr>
<td>T2</td>
<td>2–5 cm in diameter</td>
</tr>
<tr>
<td>T3</td>
<td>&gt;5cm in diameter</td>
</tr>
<tr>
<td>T4</td>
<td>A tumour of any size with direct extension to chest wall or skin</td>
</tr>
<tr>
<td>N0</td>
<td>No palpable node involvement</td>
</tr>
<tr>
<td>N1</td>
<td>Mobile ipsilateral nodes</td>
</tr>
<tr>
<td>N2</td>
<td>Fixed ipsilateral nodes</td>
</tr>
<tr>
<td>N3</td>
<td>Supraclavicular or infraclavicular nodes or oedema of arms</td>
</tr>
<tr>
<td>M0</td>
<td>No distant metastases</td>
</tr>
<tr>
<td>M1</td>
<td>Distant metastases</td>
</tr>
</tbody>
</table>

**Incidence and risk factors**

Cancer is a major cause of illness in the UK, with more than 270,000 new cases diagnosed in the year 2000, 65% of these being diagnosed in people aged over 65. The main site-specific cancers are breast, lung, colorectal...
and prostate; together they make up over half of all new cases (Cancer Research UK 2004).

Breast cancer is the most common cancer in the UK, with over 40 000 cases per year; it is the most common female cancer and accounts for one in three of all female cancers. The second most common cancer in women is colorectal cancer with around 16 333 cases, followed by lung cancer with 15 200 cases (Cancer Research UK 2004).

In men the most common cancer is prostate cancer, making up 21% of all male cancers, closely followed by lung cancer at 18% and colorectal cancer at 14%.

The latest available mortality statistics indicate that, in 2002, 155 180 people were registered as dying from cancer. Cancer causes 26% of all deaths in the UK and 22% of those deaths are from lung cancer, with cigarette smoking identified as the single most important cause of preventable disease and premature death in the UK (Cancer Research UK 2004).

Cancer Research UK (2004) offers the following advice for reducing the risk for cancer.

**Smoking**

Smoking is the greatest avoidable risk factor for cancer, causing nine out of ten cases of lung cancer. Smoking is also a risk factor for cancer of the bladder, kidney, cervix, throat (pharynx and larynx), mouth, oesophagus, pancreas and stomach, and for some types of leukaemia. The three main components of cigarettes are nicotine, carbon monoxide and tar. Nicotine is not a carcinogen but is very addictive, carbon monoxide is a poisonous gas taken up by the bloodstream that impairs breathing, and tar is made up of various chemicals, many of which are known carcinogens. It is estimated that 70% of the tar in cigarettes is deposited in the lungs. Smoking is linked to socioeconomic status, manual workers being more likely to smoke than non-manual workers. The risk of getting lung cancer if you are a smoker is directly related to the number of cigarettes smoked: the higher the number, the higher the risk. This is also directly related to the length of time that the person has smoked. Risk of lung cancer is drastically reduced by stopping smoking; smokers who stop before the age of 35 have a life expectancy not significantly different from non-smokers, so the longer you do not smoke the more the risk is lowered.

**Alcohol**

Drinking too much alcohol increases the risk of mouth, throat and oesophageal cancer, and has been linked to breast cancer. Drinking and
smoking together also have an effect on the risk for these cancers. People
classed as alcoholics are at an increased risk from liver and bowel cancer.
Government guidelines for drinking recommend three to four units per
day for men and two to three units per day for women. One unit of alco-
hol equates to half a pint of beer or lager, a small glass of wine or a small
fortified wine (port or sherry).

Diet

One-third of all cancers are related to diet; research suggests that dietary
intake influences the risk of bowel, stomach, mouth, pharyngeal,
oesophageal and pancreatic cancers, and is linked to breast, prostate,
lung, cervical and bladder cancers. A healthy balanced diet should include
fruit and vegetables, at least five portions per day. Research also suggests
that eating fruit and vegetables protects against certain types of cancer.
Measurement of dietary components and their relationship to cancer are
the focus of long-term research. Dietary fibre found in cereals, vegetables
and fruit protects the bowel from cancer by reducing constipation, a
known risk factor for bowel cancer. Diets high in red and processed meat
are linked to an increased risk for bowel cancer and are also linked to
breast, lung, prostate and pancreatic cancers.

Obesity

Obesity is linked to some cancers, e.g. in women the risk of uterine can-
cer is higher. There is also a link to postmenopausal women and breast
cancer possibly as a result of higher levels of circulating oestrogen (a
known risk factor for breast cancer) linked to fat cells. Body mass index
(BMI) (height in metres squared, divided by weight in kilograms) should
be between 20 and 25, although this should not be looked at in isolation
– it is only a rough guide.

Exercise

A combination of regular exercise, a healthy BMI and a healthy diet
protects against a number of cancers. The recommended amount of
exercise for a healthy lifestyle is 30 minutes of moderate exercise every
day for 5 days a week. Moderate exercise includes brisk walking, swim-
mimg, cycling, vacuum cleaning, dancing, gardening and even pushing a
A combination of a healthy diet, healthy body weight and regular exercise has been shown to protect against a number of different cancers.

**Safe sex**

Some types of cancer have been linked to a virus transmitted through sexual intercourse. The human papillomavirus (HPV) has 70 different strains, some of which are more likely to cause cancer than others. The risk of cancer transmitted by HPV increases with many sexual partners, or by having unprotected sex. The use of condoms is advocated to reduce the risk.

**Safety at work**

The key risk factors for cancer in the workplace are asbestos, carcinogenic chemicals, and ultraviolet (UV) and ionizing radiation. Exposure to asbestos can also cause lung cancer (mesothelioma or cancer of the lining of the lungs), although there is a long delay between exposure to asbestos and disease development. Workers exposed to asbestos in the 1950s and 1960s, when regulations for the control of asbestos in the workplace were not in force, are only now showing signs of cancer development. Carcinogenic chemicals include: benzene, present in oil and gas and used in the petroleum industry; benzidine dyes, used in the textile industry; chromate pigments, used in the paint industry; and some herbicides and fertilizers. Carcinogenic chemicals are strictly controlled and all should carry hazard warnings by law. UV radiation, the harmful rays of the sun, increases the risk of skin cancer for people who work outdoors, unless sun protection cream and the covering up of exposed skin are encouraged. Ionizing radiation in high levels can cause cancers, in particular leukaemia. The nuclear industry, as well as the medical and dental professions, work with radiation sources that need adherence to strict health and safety guidelines. Ionizing radiation can be produced by X-ray machines or by gases such as radon, which are released naturally into the environment. It is estimated that 4% of all cancer deaths may be caused by exposure to a cancer-causing substance at work.

**Sun and UV light**

There are over 65,000 new cases of skin cancer reported every year in the UK, although the disease is almost totally preventable through methods.
of skin protection. Skin cancers are linked to prolonged exposure to the sun; UV radiation penetrates deep into the skin cells causing gene damage. People most at risk are those with a large number of moles or fair freckled skin, light coloured eyes and fair or red hair, particularly babies and children. People with dark skin that tans easily are less at risk. To protect skin from UV radiation: take care not to burn; avoid the midday sun; and cover up with loose clothing, a hat, sunglasses and suncream with a sun protection factor of at least 15, and avoid the use of sunbeds.

**Family history**

A small percentage of cancers (5–10%) are caused by faulty genes inherited from one parent. A familial history of cancer might be identified if there are several cases of the same type of cancer in one family, if cancers occur under the age of 50 or on the same side of the family, and if cancer types run together in families where there is a known link, i.e. breast and ovarian cancer.

In general it is important to know your own body and what is normal for you. Some body changes can be early warning signs of cancer. If action is swift there is a much better chance of successful treatment.

**Conclusion**

In the UK the lifetime risk of developing cancer is one in three with the risk rising significantly in people over the age of 65 (Cancer Research UK 2004). For health-care professionals working in this area, a clear understanding of the biology of cancer and its epidemiology and aetiology are necessary. This will help in caring for patients from prevention of cancer to its diagnosis and treatment.

**References**

An Introduction to Cancer Care