Part 1

Introduction to oncology

Chapter 1

What is cancer?

Cancer is not a single illness but a collection of many diseases that share common features. Cancer is widely viewed as a disease of genetic origin caused by mutations of DNA that make a cell multiply uncontrollably. The description and definitions of cancer, however, vary depending on the perspective as described below.

Epidemiological perspective

Cancer is a major cause of morbidity in the UK, with around 267 000 new cases diagnosed in 1999. There are more than 200 different types of cancer, but four of them—breast, lung, colorectal and prostate—account for over half of all new cases. Overall, it is estimated that one in three people will develop some form of cancer during their lifetime. In the 10-year period 1989–1998, the overall age standardized incidence rates for cancer increased by 1.6% in men and 6.3% in women. The fastest-growing cancers in men were malignant melanoma and prostate cancer, while in women, they were kidney cancer, non-Hodgkin's lymphoma and breast cancer.

Cancer incidence refers to the number of new cancer cases arising in a specified period of time. Prevalence refers to the number of people who have received a diagnosis of cancer who are alive at any given time, some of whom will be cured and others will not. Therefore, prevalence reflects both the incidence of cancer and its associated survival pattern. Overall, it is estimated that approximately 2% of the population of the UK (around 1.2 million people) are alive, having received a diagnosis of cancer. The single cancer that contributes most to this is breast cancer, with an estimated 172 000 women alive who have had a diagnosis of breast cancer.

Sociological perspective

Patients with cancer adopt a medically sanctioned form of deviant behaviour described in the 1950s by Talcott Parsons as 'the sick role'. In order to be excused their usual duties and to be considered not to be responsible for their condition, patients are expected to seek professional advice and to adhere to treatments in order to get well. Medical practitioners are empowered to sanction their temporary absence from the workforce and family duties, as well as to absolve them of blame. This behavioural model minimizes the impact of illness on society and reduces secondary gain that the patient benefits from as a consequence of their illness. As Ivan Illich pointed out, however, this sets up physicians as agents of social control by medicalizing health and contributing to iatrogenic illness—'a medical nemesis'. Of all the common medical diagnoses, cancer probably carries the greatest stigma and is associated with the most fear. The many different ways in which cancer affects people have been explored in the literature (see Table 1.1).

Table 1.1 Top 10 cancer books (in the authors' opinion).

	Title	Author
1	Cancer Ward	Alexander Solzhenitsyn
2	A Very Easy Death	Simone de Beauvoir
3	The Doctor	Anton Chekov
4	Age of Iron	JM Coetzee
5	The Cancer Journals	Audre Lorde
6	Patrimony, a True Story	Phillip Roth
7	Before I Say Goodbye	Ruth Picardie
8	Aids As Metaphor	Susan Sontag
9	The Black Swan	Thomas Mann
10	Dangerous Parking	Stuart Browne

Experimental perspective

In the laboratory, four characteristics define a cancer cell in culture:

1. clonal (all derived from a single parent cell)
2. grows on soft agar, in the absence of growth-factors
3. crosses artificial membranes in culture systems
4. forms tumours if injected into nude mice.[1]

Histopathological perspective

Cancer is usually defined by various histopathological features, most notably invasion and metastasis, that are observed by gross pathological and microscopic examinations. Staining for laminin may assist the histopathologist in identifying local invasion by tumours that breach the basement membrane. In addition, a number of microscopic features point to the diagnosis:

1. morphology different to normal cells
2. tumour architecture is less organized than that of the parent tissue
3. increased nuclear DNA and nuclear : cytoplasmic ratio
4. hyperchromatic nuclei with coarsening of chromatain and wrinkled nuclear edges
5. multinucleated or with macronucleoli
6. numerous and bizarre mitotic figures.

1 Nude mice are a mutant strain that lacks a thymus gland and T lymphocytes, and thus fail to reject a xenograft (transplant from another species). Nude mice are hairless owing to a mutation at a linked locus.

Cancers may be heterogenous with cells of varying sizes and orientation with respect to one another, despite their clonal origin.

Molecular perspective

The molecular features that identify cancer are described in 'Six steps to becoming a cancer' in Chapter 2:

1. grow without signal (self-sufficiency in growth stimuli)
2. do not stop growing (insensitivity to inhibitory stimuli)
3. do not die (evasion of apoptosis)
4. do not age (immortalization)
5. feed themselves (neoangiogenesis)
6. spread (invasion and metastasis).

How to read a histology report

The diagnosis of cancer is most commonly established following a histopathological report of a biopsy or tumour resection. A histopathological report should include both gross pathological features (tumour size, number and size of lymph nodes examined) and microscopic findings (tumour grade, architecture, mitotic rate, margin involvement and lymphovascular invasion). The grade and stage of a cancer are important prognostic factors that may influence therapy options (Box 1.1).

A histopathological definition of cancer

Malignancy is usually characterized by various behavioural features, most notably invasion and metastasis. The histopathologist, however, may have to identify a cancer without this information. Cancers are composed of clonal cells (all are the progeny of a single cell) and have lost control of their tissue organization and architecture. In addition to the natural history, a number of physical properties help to differentiate between benign and malignant tumours (Table 1.2). There is, however, no single histological feature that defines a cancer; nor indeed is there any single histological feature that separates benign from malignant tumours. In general, benign tumours are rarely

Box 1.1: Histopathology definitions

Quantitative changes

Too small

Atrophy
An acquired **diminution** of growth due to a decrease in the size or number of constituent parts of a tissue, such as decrease in size of the ovaries after the menopause.

Too big

Hypertrophy
Increase in the size of an organ or tissue due to an increase in the **size** of individual cells; for example, pregnant uterus.

Hyperplasia
Increase in the *size* of an organ due to an increase in the **number** of cells; for example, lactating breast.

Qualitative changes

Metaplasia
Replacement of one differentiated cell type by another. This implies changes in the differentiation programme and is usually a response to persistent injury. It is reversible so that removal of the source of injury results in reversion to the original cell type, such as metaplasia of laryngeal respiratory epithelium in a smoker. The chronic irritation leads to an exchange of one type of epithelium (normal respiratory columnar epithelium) for another (the more resilient squamous epithelium).

Dysplasia
Dysplastic changes do not necessarily revert to normal once the injury is removed. Dysplasia is usually considered to be part of the spectrum of changes leading to neoplasia, like cervical dysplasia initiated by human papillomavirus infection persists after eradication of the virus.

Invasion
The capacity to infiltrate the surrounding tissues and organs is a characteristic of cancer.

Metastasis
The ability to proliferate in distant parts of the body, after tumour cells have been transported by lymph or blood or along body spaces.

Table 1.2 Histological features of benign and malignant tumours.

Features of malignancy	Features of benign tumours
Macroscopic features	
Invade and metastasize	Do not invade or metastasize
Rapid growth	Slow growing
Not clearly demarcated	Clearly demarcated from surrounding tissue
Surface often ulcerated and necrotic	Surface smooth
Cut surface heterogenous	Cut surface homogenous
Microscopic features	
Often high mitotic rate	Low mitotic rate
Nuclei pleomorphic and hyperchromatic	Nuclear morphology often normal
Abnormal mitoses	Mitotic figures normal

life-threatening but may cause health problems on account of their location (by pressure or obstruction of adjacent organs) or by overproduction of hormones. In contrast, malignant tumours usually follow a progressive course and, unless successfully treated, are frequently fatal.

In situ or invasive?

Invasive cancers extend into the surrounding stroma. Tumours, on the other hand, which exhibit all the microscopic features of cancers but do not breach the original basement membrane, are termed *in situ* (non-invasive) cancers. Examples include *in situ* breast cancer confined to the ducts (ductal carcinoma *in situ* (DCIS)) or lobules (lobular carcinoma *in situ* (LCIS)). Similar preinvasive cancers have been found in many organs such as cervix, anus, prostate, and bronchus and are believed to represent a stage in the progression from dysplasia to cancer (Figs 1.1 and 1.2).

Histopathologist's nomenclature

The suffix *oma* usually denotes a benign tumour (although it simply means 'swelling' and some *omas* are not tumours, e.g. xanthoma). If a tumour

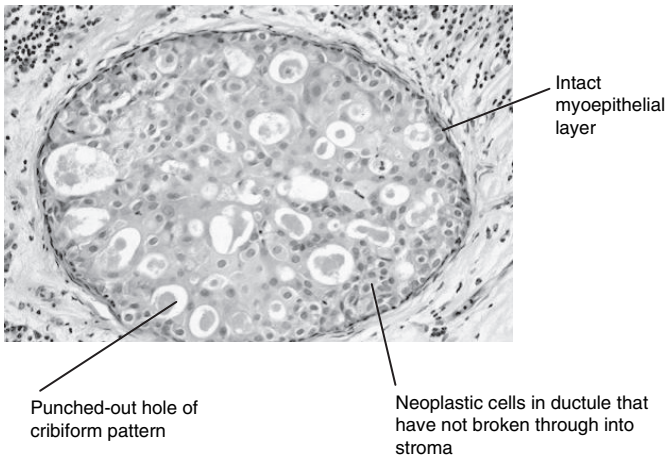


Figure 1.1 Histology of Intraductal carcinoma (Ductal carcinoma in situ—DCIS) of the breast, demonstrating neoplastic cells in breast ductule with intact myoepithelial layer. (See also colour plates between pages 120 and 121.)

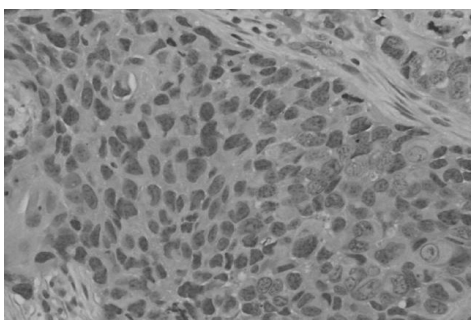


Figure 1.2 Histology of invasive ductal carcinoma of the breast with neoplastic cells invading breast stroma. (See also colour plates between pages 120 and 121.)

is malignant, the suffix *-carcinoma* (Greek for 'crab') is used for epithleial cancers or *-sarcoma* (Greek for 'flesh') for connective tissue cancers. The prefix is determined by the cells of origin of the tumour, e.g. *adeno-* for glandular epithelium, qualified by the tissue of origin, e.g. prostatic adenocarcinoma. There are numerous exceptions to this systematic nomenclature; for example, leukaemias and lymphomas are malignant tumours of bone marrow and lymphoid tissue, respectively. As a general rule, neoplasms are classified according to the type of normal tissue they most closely resemble. The four major categories are: epithelial, connective tissue, lymphoid and haemopoietic tissue, and germ cells (Tables 1.3–1.6). The latter arise in 'totipotential cells' and contain a variety of different mature and/or immature tissues from different germ layers, and these are given names with the root *terato-* (Greek for 'monster'). In addition, as with most fields of medicine where physicians try to leave their mark, there are a number of eponymous names. For example, Hodgkin's disease, used to describe seven cases in 1832 of the tumour that bears Hodgkin's name. Paradoxically re-examination of the specimens in 1926 revealed that the diagnosis was inaccurate in four of the seven cases.

Tumour grading

Tumours are graded according to the degree of tissue differentiation. Cancers that closely resemble their tissue of origin are graded as well-differentiated cancers, while cancers that retain little of their origin and have histological features of aggressive growth and high mitotic rates are graded as poorly differentiated cancers. The grade of the tumour is of prognostic significance.

In the case of breast cancer, the Scarff–Bloom–Richardson system is usually used to grade cancers based upon three features: the frequency of cell mitosis, tubule formation and nuclear pleomorphism. Each of these features is assigned a score ranging from 1 to 3 (1 indicating slower cell growth and 3 indicating faster cell growth). The scores of

Table 1.3 Nomenclature of epithelial tumours.

Epithelium	Benign tumour	Malignant tumour
Squamous	Squamous papilloma	Squamous carcinoma
Glandular	Adenoma	Adenocarcinoma
Transitional	Transitional papilloma	Transitional carcinoma
Liver	Hepatic adenoma	Hepatocellular carcinoma
Skin	Papilloma	Squamous cell carcinoma Basal cell carcinoma
Skin melanocyte	Naevus	Malignant melanoma

Table 1.4 Nomenclature of connective tissue tumours.

Tissue	Benign tumour	Malignant tumour
Bone	Osteoma	Osteosarcoma
Cartilage	Chondroma	Chondrosarcoma
Fat	Lipoma	Liposarcoma
Smooth muscle	Leiomyoma	Leiomyosarcoma
Striated muscle	Rhabdomyoma	Rhabdomyosarcoma
Blood vessel	Angioma	Angiosarcoma
Fibrous tissue	Fibroma	Fibrosarcoma

Table 1.5 Nomenclature of haematological tumours.

Tissue	Malignant tumour
Node lymphocyte	Lymphoma
Marrow lymphocyte	Lymphocytic leukaemia
Granulocyte	Myeloid leukaemia
Plasma cell	Myeloma

Table 1.6 Nomenclature of germ cell tumours.

Tissue	Benign tumour	Malignant tumour (male)	Malignant tumour (female)
Germ cell	Mature teratoma/dermoid cyst	Non-seminomatous germ cell tumour/malignant teratoma	Immature teratoma/embryonal carcinoma
	—	Seminoma	Dysgerminoma

each of the cells' features are then added together for a final sum that will range between 3 and 9. A tumour with a final sum of 3, 4 or 5 is considered a Grade 1 tumour (well differentiated). A sum of 6 or 7 is considered a Grade 2 tumour (moderately differentiated), and a sum of 8 or 9 is a Grade 3 tumour (poorly differentiated). The 5-year overall survival for grades 1, 2 and 3 is 95%, 75% and 50%, respectively.

Unknown primary identification (standard histological techniques)

Occasionally, patients may present with metastatic cancer without an obvious primary tumour site, and in addition to a careful clinical and radiological examination, the pathologist may provide a clue to the origins of the cancer. Most unknown primary cancers are adenocarcinoma (60%), and the remainder are poorly differentiated carcinomas (30%) and squamous cell carcinomas (5%). Light microscopy may provide pointers, such as the presence of melanin favours melanoma, while mucin production is common in gastrointestinal, breast and lung cancers but less common in ovarian cancers and rare in renal cell and thyroid cancers. Immunocytochemical staining of tissue samples can assist the pathologist in tissue identification.

Box 1.2: Cytokeratins

Cytokeratins (CKs) are intermediate filament proteins expressed in pairs comprising a type I (CK9–20) and a type II cytokeratin (CK1–8). Different tissues express different pairs, and immunocytochemical staining for cytokeratins can help identify the likely tissue origins of cancers cells. For example, in disseminated peritoneal metastases, CK7 expression favours an ovarian origin, whilst lack of CK7 is more common in colorectal cancer.

For example, the presence of oestrogen and progesterone receptors favours a diagnosis of breast cancer, while prostate-specific antigen and prostatic acid phosphatase staining points to prostatic adenocarcinoma; similarly, cytokeratin expression patterns may provide helpful hints (see Box 1.2). Cell-surface immunophenotyping is a sophistication of immunocytochemistry that is frequently applied to haematological malignancies. The pattern of immunoglobulin, T-cell receptor and cluster designation (CD) antigen expression on the surface of lymphomas is helpful in their diagnosis and classification. Immunophenotyping can be achieved by immunohistochemical staining, immunofluorescent staining or flow cytometry.

Unknown primary identification (special histological techniques)

The study of intracellular organelles by electron microscopy may identify the cellular origin of a tumour, such as the presence of melanosomes in melanomas and dense core neurosecretory granules in neuroendodermal tumours. Further laboratory techniques to aid diagnosis include molecular studies of DNA rearrangements that characterize malignancies. Monoclonal immunoglobulin gene rearrangements are present in B-cell malignancies, and rearrangements of T-cell receptors occur in T-cell tumours. In addition, a number of chromosomal translocations involving the immunoglobulin genes (heavy chain on chromosome 14q32, light chains on 2p12 and 22q11) and T-cell receptor genes (TCRα on 14q11, TCRβ on 7q35, TCRγ on 7p15, TCRδ on 14q11) occur in malignancies arising from these cell types. For instance, low-grade follicular lymphomas rearrange the Bcl-2 gene on 18q21, such as t(14;18)(q32;q21), most Burkitt lymphomas rearrange the Myc gene on 8q24, such as t(8;14)(q24;q32) and most mantle-cell lymphomas rearrange Bcl-1 on 11q13, such as t(11;14)(q13;q32). These rearrangements may be detected by karyotype analysis of mitotic chromosome preparations or by molecular techniques including Southern blotting and polymerase chain reaction (Box 1.3 and Table 1.7). Less commonly, these same methods may assist the diagnosis of solid tumours which are associated with specific chromosomal abnormalities, such as the i(12p) isochromosome found in germ-cell tumours and the t(11;22)(q24;q12) translocation seen in Ewing's sarcoma and peripheral neuroectodermal tumours. In addition to translocations, gene amplification may be detected and may have prognostic significance; for example, the amplification of the N-Myc oncogene in neuroblastoma is an adverse prognostic variable.

Tumour stage

In addition to the histological grade of a tumour, an important criterion in treatment decisions and the major determinant of outcome is the extent of spread or stage of a cancer. Staging a tumour is essentially an anatomical exercise that uses a combination of clinical examination and radiology. A uniform staging system is employed for most tumour sites, based upon the size of the primary Tumour, the presence of regional lymph Nodes and the presence of distant Metastases. The details of this TNM classification vary between different tumour sites. As always, there are exceptions including the staging system for lymphomas originally set out following a conference at Ann Arbour, in Michigan.

Radiology techniques

Staging depends to a large extent upon radiology, and this is the most commonly used tool to evaluate the response of cancers to therapies.

Box 1.3: The language of chromosomes—karyotype nomenclature

Each arm of a chromosome is divided into one to four major regions, depending on chromosomal length; each band, positively or negatively stained, is given a number, which rises as the distance from the centromere increases. The normal male is designated as 46,XY and the normal female as 46,XX (see Figure 1.3).

Polyploidy

Cell with more than one complete chromosome set or with multiples of the basic number of chromosomes characteristic of the species; in humans, this would be 69,92, etc.

Aneuploidy

Individual with one (or more) chromosome in addition to, or missing from, the complete chromosome set; for example, trisomy 21 (47XX +21).

Deletion

The loss of a chromosome segment from a normal chromosome.

Duplication

An extra piece of chromosome segment that may either be attached to the same or homologous chromosome, or be transposed to another chromosome in the genome.

Inversion

A change in linear sequence of the genes in a chromosome that results in the reverse order of genes in a chromosome segment. Inversions may be pericentric (two breaks on either side of the centromere) or paracentric (both breaks on the same arm).

Isochromosome

Breaks in one arm of a chromosome followed by duplication of the other arm of the chromosome to produce a chromosome with two arms that are both short (p) or both long arms (q).

Translocations

Translocations are the result of the reciprocal exchange of terminal segments of non-homologous chromosomes.

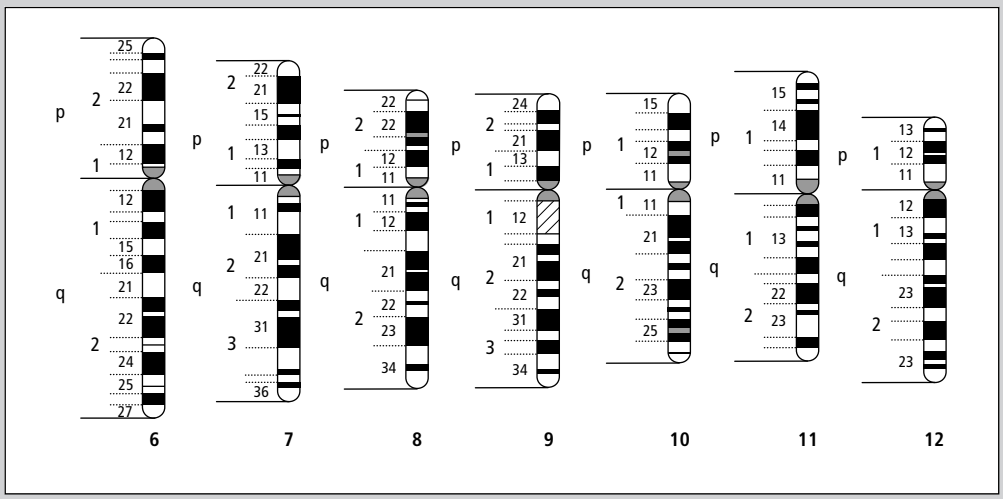


Figure 1.3 Karyotype nomenclature: For example, 11q23 designates the chromosome (11), the long arm (q), the second region distal to the centromere (2), and the third band (3) in that region.

Table 1.7 Examples of chromosomal abnormalities in cancers.

Chromosome defect	Karyotype	Tumour	Candidate gene
Monosomy	45, XY –22	Meningioma	NF2
Trisomy	47, XX +7	Papillary renal carcinoma	MET
Deletion	46, XY del (11) (p13)	Wilms tumour	WT1
Duplication	46, XX dup (2) (p23–24)	Neuroblastoma	N-myc
Inversion	46, XY inv (16) (p13q22)	AML (M4Eo)	MYH11/core binding factor b
Isochromosome	47, XX i (12p)	Testicular GCT	
Translocation	46, XX t (9;22) (q34;q11)	CML	bcr/abl

Table 1.8 Commonly used isotopes in nuclear imaging in oncology.

Isotope	Half life	Tracer	Oncological use
^{99}Tc	6 h	Methylene diphosphonate (MDP)	Bone scan
^{111}In	67 h	Octreotide	Neuroendocrine tumours
^{131}I	8 days	Sodium iodide	Thyroid cancer
^{131}I	8 days	Meta-iodobenzylguanidine (MIBG)	Phaeochromocytoma neuroblastoma
^{67}Ga	68 h	Gallium citrate	Lymphoma
^{18}F	110 min	Fluorodeoxyglucose (FDG)	Brain and soft tissue tumours

Anatomical imaging by plain films, computed tomography (CT), ultrasound and magnetic resonance imaging (MRI) are the standard methods. In addition, functional imaging using radiotracer isotopes to produce nuclear images is widely used in oncology (Table 1.8). The isotope- labelled tracers that are used diagnostically may also be used therapeutically (Fig. 1.4).

Performance status

In addition to the histological tumour grade and the stage or spread of a cancer, the patients' general status will determine how long they survive and may influence treatment decisions. Scales that measure the performance status or functional capacity of patients include the Eastern Cooperate Oncology Group (ECOG) grading system and Karnovsky scale (see Table 1.9). The performance status, however estimated, is an important prognostic indicator for almost all tumour types.

Prognosis—It's not cancer is it, doc?

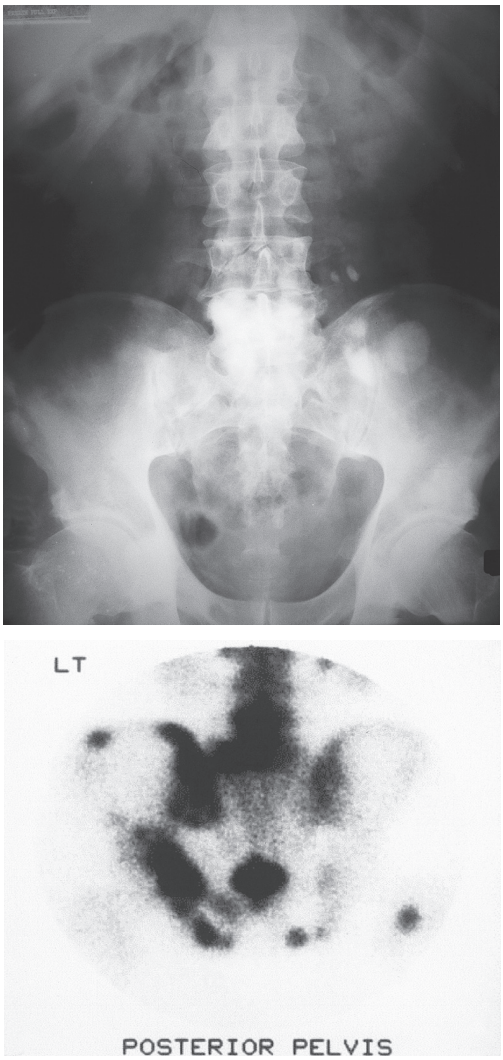
Although a very significant stigma is attached to the diagnosis of cancer, for most of the general population the fear outweighs the reality, and comparison with other more 'palatable' illnesses yields results that are not always expected (Table 1.10).

Cancer epidemiology

Epidemiology in the UK

Cancer is now the commonest cause of death in the UK (if cardiovascular and cerebrovascular diseases are classed separately):

- one in three people in the UK will develop a cancer (250 000/year)
- one in four die of cancer (180 000/year)
- by 2008, it is estimated that one in three in the UK will die of cancer



(a)

(b)

Figure 1.4 Plain pelvic radiograph (a) and corresponding area of Technetium pyrophosphate bone scan (b) of a patient with sclerotic bone metastases from prostate cancer.

The top 10 cancers diagnosed in the UK in 1998, excluding non-melanomatous skin cancers, are shown in Table 1.11.

Global epidemiology

The incidence of different cancers varies geographically with risk factors and the demographics of the local population (Fig. 1.5). There is, however, a general correlation between increasing wealth and

Table 1.9 Functional capacity grading (ECOG) and Karnovsky performance scales.

ECOG functional capacity grading	
0	Asymptomatic
1	Symptomatic but fully ambulant
2	Symptomatic, ambulant >50% waking hours
3	Symptomatic, confined to bed >50% waking hours
4	Symptomatic, bedfast
Karnovsky performance status score (%)	
100	Normal; no complaints; no evidence of disease
90	Able to carry on normal activity; minor signs or symptoms
80	Normal activity with effort; some signs or symptoms
70	Care for self; unable to carry on normal activity or do active work
60	Requires occasional assistance, but able to care for most of needs
50	Requires considerable assistance and frequent medical care
40	Disabled; requires special care and assistance
30	Severely disabled; hospitalization indicated but death not imminent
20	Very sick; hospitalization necessary; active supportive treatment necessary
10	Moribund; fatal processes progressing rapidly

Table 1.10 Survival rates for various diseases.

	Myocardial infarction	Hodgkin's disease	Heart failure (NYHA* III/IV)	Metastatic breast cancer
1 yr survival rate	75%	90%	50%	60%
5 yr survival rate	45%	85%	15%	20%

*NYHA: New York Heart Association grading scale

Table 1.11 Commonest cancers diagnosed in UK.

Tumour	As percentage of all cancers diagnosed	Lifetime risk (men)	Lifetime risk (women)
Breast	15		1 in 9
Lung	15	1 in 13	1 in 23
Colorectal	13	1 in 18	1 in 20
Prostate	9	1 in 12	
Bladder	5	1 in 30	1 in 79
Stomach	4	1 in 44	1 in 86
Non-Hodgkin's lymphoma	3	1 in 69	1 in 83
Head and neck	3	1 in 70	1 in 85
Oesophageal	3	1 in 75	1 in 95
Ovarian	3		1 in 48
Other	27		

increasing cancer incidence, which is attributable to tobacco use, diet and increased longevity in wealthy populations. There are intriguing exceptions; for example, the Gulf states of Kuwait, Qatar, Bahrain, United Arab Emirates and Saudi Arabia have lower cancer incidences than would be predicted from their per capita gross national product.

Cancer charities

The UK has 600 cancer charities. Their efforts increase awareness of cancer, improve diagnosis and treatment capability and provide care for patients with the disease. The total income generated by the cancer charities in 2004 was £500 m; and the average charitable efficiency was 65%, providing £320 m for spending on patients' care and on research. The two largest UK cancer charities, the Imperial Cancer Research Fund (ICRF) and the Cancer Research Campaign (CRC), merged to from Cancer Research UK (CRUK) in 2002. CRUK is the largest volunteer-supported cancer research organization in the world, with 3000 scientists and an annual scientific spend of more than £250 m—raised almost entirely through public donations.

Cancer hospitals

Philanthropists and social reformers during the 19th century tried to provide free medical care for the poor. William Marsden, a young surgeon, opened a dispensary for advice and medicines in 1828. His grandly named London General Institution for the Gratuitous Cure of Malignant Diseases—a simple four-storey house in one of the poorest parts of the city—was conceived as a hospital to which the only passport should be poverty and disease, and where treatment was provided free of charge. The demand for Marsden's free services was overwhelming, and by 1844, his dispensary, now called the Royal Free Hospital, was treating 30 000 patients a year. In 1846, when his wife died of cancer, Marsden opened a small house in Cannon Row, Westminster, for patients suffering from cancer. Within 10 years, the institution moved to Fulham Road and became known as The Cancer Hospital, of which Marsden was the senior surgeon. The Hospital was incorporated into the National Health Service in 1948 and renamed the Royal Marsden Hospital in 1954. Although other cancer hospitals have been established in Manchester (The Christie Hospital) and Glasgow (The Beatson Hospital), the Royal Marsden Hospital remains the most renown. With the recent emphasis on multidisciplinary approaches to cancer, single speciality hospitals are less *in vogue*, and the majority of cancer departments are in large teaching hospitals.

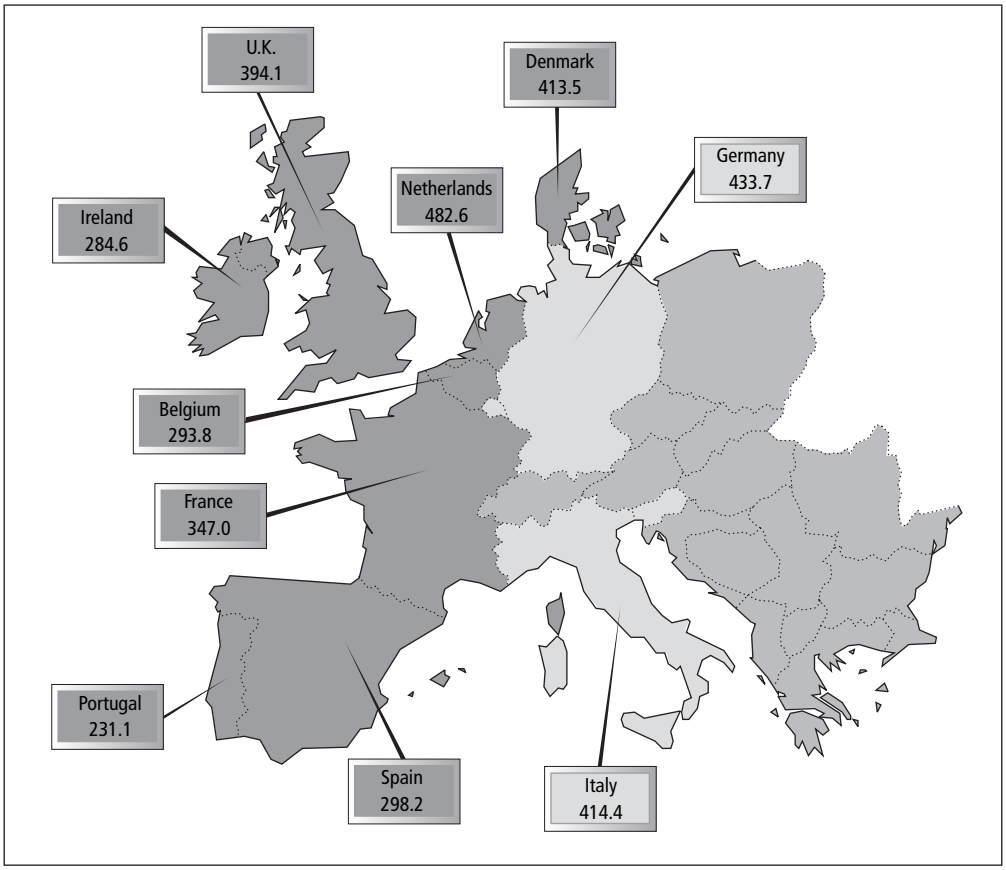


Figure 1.5 Map of cancer incidences in Europe (per 100 000 population).

Table 1.12 Rock star cancer deaths.

	Year of death	Age	Cause of death
George Harrison	2001	58	Non-small-cell lung cancer
Joey Ramone	2001	49	Non-Hodgkin's lymphoma
Ian Dury	2000	58	Colorectal cancer
Dusty Springfield	1999	60	Breast cancer
Carl Wilson (Beach Boys)	1998	52	Lung cancer
Linda McCartney	1998	57	Breast cancer
Frank Zappa	1993	53	Prostate cancer
Freddy Mercury	1991	45	Kaposi's sarcoma
Mel Appleby (Mel and Kim)	1990	24	Spinal tumour
Bob Marley	1981	36	Metastatic melanoma

Cancer celebrities

Celebrities influence public perceptions and behaviour inordinately, and this is true in oncology as elsewhere. Celebrities with cancer have contributed in three main ways; personal accounts bring patients' experiences into the limelight, reports of celebrity patients increase public awareness and may encourage health-seeking behaviour such as stopping smoking, and celebrity patients may support cancer charities and encourage donations. Prominent examples of patient's perspectives include John Diamond's account in *C: because cowards get cancer, too* and Ruth Picardie's *Before I say goodbye*, both moving accounts by accomplished journalists. Celebrity patients can influence the treatment choices that the public make. Following Nancy Reagan's mastectomy for localized breast cancer in 1987, there was a 25% fall in American women choosing breast-conserving surgery over mastectomy. Her husband's successful surgery for Dukes' B colon cancer while president in 1984 increased awareness and propelled the warning signs of colon cancer into the media. Successful cancer treatment is often most widely publicized, and no article describing Lance Armstrong's cycling victories seems complete without a mention of his treatment for metastatic non-seminomatous germ cell tumour, or of his two children conceived with stored sperm banked prior to chemotherapy. Other celebrity patients have used their wealth and fame to establish and support charitable projects to support cancer research and treatment, including Bob Champion, the steeple-chase jockey treated for testicular cancer in the 1970s, and Roy Castle, a lifelong non-smoker who was diagnosed with lung cancer in 1992. Of course, no one is immune to cancer, even rock stars whose deaths are traditionally associated with suicide and substance abuse (Table 1.12).