

(Rodent Ulcer)**DEFINITION**

1. Cancer is a term which embraces a large number of different diseases, the common feature of which is a malignant tumour. This is a growth (neoplasm) which is not circumscribed but which infiltrates the surrounding tissues and metastasises (spreads to other sites in the body, thereby producing secondary deposits). Any tissue in the body may be affected.
2. Cancers are classified according to the tissue of origin. **Carcinoma** arises from epithelial tissue and **sarcoma** from connective tissue. The suffix - **blastoma** implies a tumour of embryonic origin.
3. **Basal Cell Carcinoma** is a malignant tumour of the skin. Primary skin cancers are classified according to their histology into epithelial, mesodermal and melanocytic tumours. Basal cell carcinoma arises from the basal area of the epidermis and its appendages and is composed of immature cells resembling the immature cells of these structures. There is a characteristic marginal palisade of tumour cells and surrounding well organised stroma.
4. The condition is a relatively benign form of cancer of remarkably slow growth which does not involve neighbouring lymph glands and very rarely gives rise to metastases. It is therefore described as being locally malignant.

CLINICAL MANIFESTATIONS

5. The majority of basal cell carcinomas arise on the head and neck with a predilection for the upper central part of the face about the cheek and nose, but they may arise in other parts of the body and are frequently multiple. The palms of the hands, the soles of the feet and the vermilion of the lips are seldom, if ever, involved.
6. Early tumours are small, translucent or pearly, raised and rounded areas covered by thin epidermis through which a few dilated vessels show. They may remain unchanged for years. The thin epithelium on the surface may periodically erode or scale. Ulceration is frequently initiated by minor trauma and may heal and break down several times before becoming permanent. The typical rodent ulcer is covered by a scab and has an indurated edge.

AETIOLOGY

7. Cancer is not one disease but a group of widely different diseases. While some aetiological factors may be common to a number of different types of cancer, each type should be recognised to be an individual disease with its own specific aetiology.

8. The common feature of all cancers is the loss of control over normal cell division and differentiation. Cell division proceeds by a complex sequence of events. For this to be maintained in a normal way it must be strictly controlled. It has been found that certain regions of the chromosomes are vital to this control. These regions are called oncogenes. Whilst the oncogenes perform normally, cell division and differentiation remain under control.
9. The process whereby oncogenes lose control of cell division and differentiation is known as activation. When this occurs cell division and differentiation become chaotic and neoplasia (carcinogenesis) ensues. The factors which activate oncogenes are numerous and varied, some being endogenous, others environmental. There is evidence that in most types of cancer a number of different factors play a part at different stages of the neoplastic process.
10. Some types of cancer are strongly genetically determined with a family history, for example retinoblastoma. In other types of cancer an external agent is the dominant factor, for example aniline dyes, which will cause carcinoma of the bladder in 100% of cases following sufficient exposure.
11. Some individuals are **genetically determined** to be more likely to develop cancer and there is a strong history of a certain type of cancer in their family of origin. Some cancers are more common in one sex than the other.
12. During life many **constitutional factors** are present which may activate oncogenes. These include humoral factors, immunological factors and the normal ageing process during which spontaneous changes affect the genes (somatic mutations).
13. For the most part, cancer is commoner at the extremes of life. This may be because the immune system is relatively less efficient in the young and the elderly. In addition, with increasing age, the summation of naturally occurring somatic mutations and any exposure to carcinogens may become sufficient to activate oncogenes.
14. **Environmental factors** play a part in the aetiology of some types of cancer. The following groups of factors have been identified:
 - 14.1. **Chemical**, for example aniline dyes and carcinoma of the bladder.
 - 14.2. **Physical** agents, for example asbestos and mesothelioma.
 - 14.3. **Ionising radiation** which, when a certain dose, is exceeded will cause cancer in some, but not all, tissues.
 - 14.4. **Ultraviolet radiation** which may cause cancer of the skin. Its tissue penetration is limited and so it does not cause cancer in the deeper tissues.
 - 14.5. Some specific **viruses** have been identified which play a part in the causation of particular types of cancer, for example hepatitis B and primary carcinoma of the liver.

- 14.6. It has been suggested that a wide variety of other environmental factors may cause certain types of cancer. Many of these suggestions have been based on animal studies, in vitro experiments or on epidemiological studies with small samples or inadequate controls. These contentions are still at the stage of speculation.
15. Of the above, both genetic and environmental factors may be important in the aetiology of cancer of the skin.
 16. Some genetically determined skin conditions predispose to skin cancer. There is evidence that the effect of a single carcinogen may differ from individual to individual. This is a function of genetic predisposition.
 17. Chemicals are potent skin carcinogens. They may be ingested, inhaled or absorbed into the body. Agents which have been incriminated include arsenic, cutting oils, crude paraffin, creosote and mineral oil.
 18. Ultraviolet light is an important environmental factor. The most damaging part of the spectrum is ultraviolet B light. Passage of the light through glass, or through air containing high concentrations of water, eliminates the carcinogenic potential. As a consequence, dry sunny climates are the most dangerous. As much as 50% of ultraviolet B light reaches the skin by scatter from the sky or by reflection from sun and sea. Sunlight effects are cumulative and there are well recognised premalignant states.
 19. Skin is susceptible to ionising radiation, although much less so than tissues such as thyroid and breast. Doses of ionising radiation used therapeutically may produce skin malignancy.
 20. Scar tissue is more susceptible to carcinogenic agents. Malignant transformations may occur in the borders of venous ulcers. Injury itself does not cause tumours in normal skin, although there is evidence that in skin previously exposed to a carcinogen an injury can localise and promote a tumour.
 21. With respect to basal cell carcinoma, it is the commonest malignant tumour of the skin in white races but it is extremely uncommon in black races. The disease is more common in males than in females and usually arises after the age of 40. People with a fair and freckled complexion are more likely to develop the condition. If they do, it may arise at an earlier age and multiple tumours are more likely.
 22. The incidence of basal cell carcinoma increases greatly with exposure to the sun. In Australia, where the light is very strong and the humidity is low, the disease is extremely common. In Great Britain it has been found to be more common in those whose occupation, such as farming, involves considerable and prolonged exposure to the sun.
 23. Basal cell carcinoma is not caused by other climatic extremes, trauma, physical or mental stress, or lowered resistance arising from hardship or other diseases. Its progress is independent of external factors other than medical treatment.

CONCLUSION

24. Basal cell carcinoma is a locally malignant tumour of the skin. Factors known to be associated with the development of the condition are discussed above. The course of the condition is unaffected by environmental factors other than those involved in its treatment.

REFERENCES

Harnden D G, Lorenzen J, Pusztai L and McGee J O'D. Carcinogenesis. In: Eds. McGee J O'D, Isaacson P G and Wright N A. Oxford Textbook of Pathology. 1992. Oxford. Oxford University Press. 633-678.

Doll R and Peto R. Epidemiology of Cancer. In: Eds. Weatherall D J, Ledingham J G G and Warrell D A. Oxford Textbook of Medicine. 2nd Ed. 1987. Oxford. Oxford University Press. 4.95-4.123.

Doll R. Epidemiology of Human Neoplasia. In: Eds. McGee J O'D, Isaacson P G and Wright N A. Oxford Textbook of Pathology. 1992. Oxford. Oxford University Press. 679-694.

Schimizu Y, Schull W J and Kato H. Cancer risk among Atomic Bomb Survivors: the RERF Life Span Study. JAMA 1990;264:601-604.

Boice J D. Studies of Atomic Bomb Survivors. JAMA 1990;264:622-623.

Darby, S C, Kendall G M, Fell T Petal. A summary of mortality and incidence of cancer in men from the United Kingdom who participated in the United Kingdom's atmospheric nuclear weapon tests and experimental programmes. BMJ 1990;296:332-338.

Mackie R M. Epidermal Skin Tumours - Basal cell carcinoma. In: Eds. Champion R H, Burton J L, and Ebling F J. Textbook of Dermatology, 5th Ed. 1992. Oxford. Blackwell Scientific Publications. 1488-1502.

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