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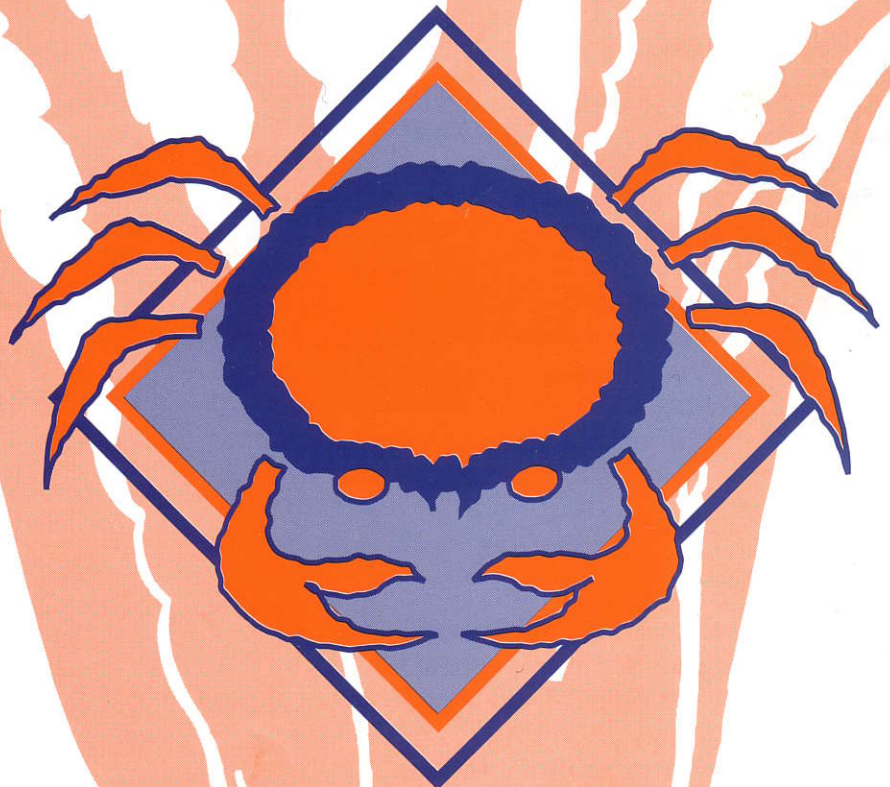
ALOE VERA

Aloe Vera and Cancer

By Dr. G. Lawrence Plaskett B.A., Ph.D., C.Chem., F.R.I.C.

Administration of Aloe Vera in various forms has been shown to inhibit the growth of animal cancers or to actually bring about shrinkage of already-grown tumours. From all the other knowledge we have about the actions of Aloe, it appears that the effects of Aloe upon tumours is mediated via the immune system.

This newsletter presents a general discussion of the formation and growth of cancers from the standpoint of Aloe and one other plant extract substance, bromelain, whose actions may well synergize usefully with those of Aloe.



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ALOE VERA AND CANCER

The Nature of Tumours, of Malignancy and of Tumour Cells

Malignant neoplasms or cancers have several distinguishing features that enable them to be characterized as abnormal. The commonest types of human cancers derive from epithelium, that is, the cells covering internal or external surfaces of the body. These cells have a supportive stroma of blood vessels and connective tissue. Malignant tumour tissues may resemble normal tissues; at least in the early phases of their growth and development. Cancer cells can develop in any tissue of the body that contains cells capable of cell division. Though they may grow fast or slowly, their growth rate frequently exceeds that of the surrounding normal tissue. This is not an invariant property, however, because the rate of cell renewal in a number of normal tissues (eg. gastrointestinal tract epithelium, bone marrow, and hair follicles) is as rapid as that of a rapidly growing tumour.

The term "neoplasm", meaning new growth, is often used interchangeably with the term "tumour" to signify a cancerous growth. It is important to keep in mind, however, that tumours are of two basic types: benign and malignant. The ability to distinguish between benign and malignant is crucial in determining the appropriate treatment and prognosis of a patient who has a tumour. The following are features that differentiate a malignant tumour from a benign tumour:

1. Malignant tumours invade and destroy adjacent normal tissue; benign tumours grow by expansion, are usually encapsulated, and do not invade surrounding tissue. Benign tumours may, however, push aside normal tissue and may become life-threatening if they press on nerves or blood vessels or if they secrete biologically active substances, such as hormones that alter normal homeostatic mechanisms.

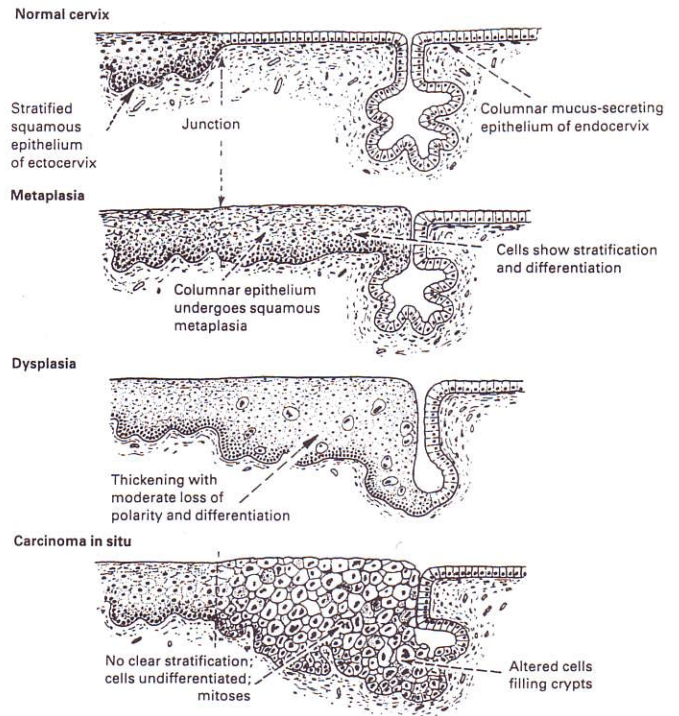
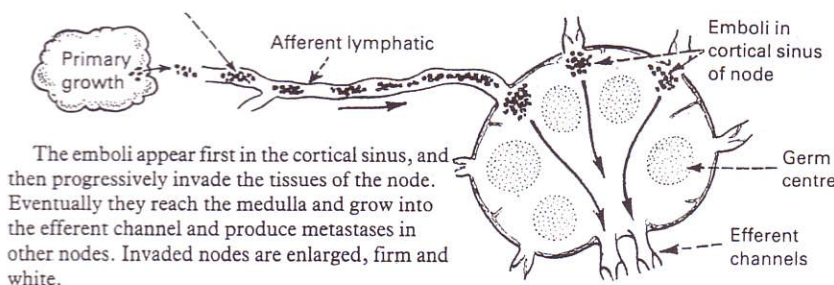


Figure 1 *Illustration of the way a malignant tumour grows out of control and with no functional purpose. The example shows cancer of the cervix (cervical carcinoma).*

2. Malignant tumours metastasize (a word which means breaking off pieces that are then transported to other parts of the body, where they lodge and resume growth) through lymphatic channels or blood vessels to lymph nodes, bones, lungs and other tissues in the body. Benign tumours remain localised and do not metastasize.

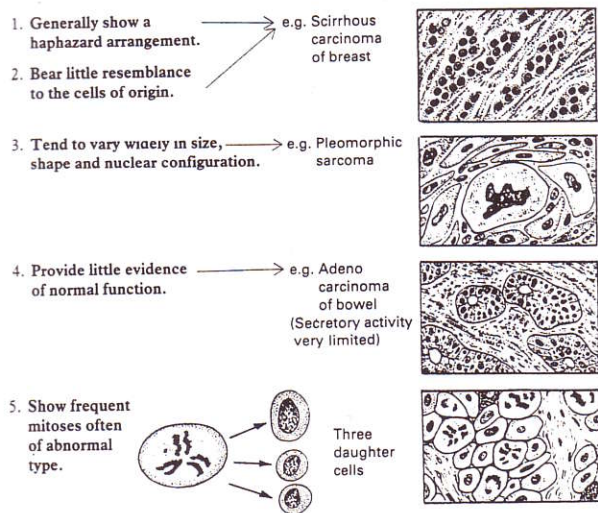
3. Malignant tumour cells tend to be "anaplastic" (a word which means less well differentiated than normal cells into recognisable cell types of the tissue in which they arise). Malignant cells may actually show varying degrees of "anaplasticity" or "undifferentiatedness". Correlations are often drawn between the degree of anaplasticity and the degree of aggressive invasiveness which a tumour displays. The more differentiated, the less invasive, the more anaplastic, the more invasive. Benign tumours usually resemble normal tissue more closely than malignant tumours do.



The emboli appear first in the cortical sinus, and then progressively invade the tissues of the node. Eventually they reach the medulla and grow into the efferent channel and produce metastases in other nodes. Invaded nodes are enlarged, firm and white.

Figure 2 *Illustration to show the invasiveness of cancer spreading by metastasis through the lymphatics. The emboli, having grown substantially in one lymph node (gland) create a position from which the spread to other lymph glands is possible.*

UNDIFFERENTIATED CELLS (MALIGNANT TUMOURS):



Lack of differentiation is often termed anaplasia, and anaplastic tumours are highly malignant. There is a spectrum of change in neoplasms from the very slowly growing, highly differentiated simple types to the rapidly growing, undifferentiated malignant examples.

Figure 3 *An Illustration of Undifferentiated (Anaplastic) Cell Types*

Some malignant neoplastic cells at first structurally and functionally resemble the normal tissue in which they arise. Later, as the malignant neoplasm progresses, it invades surrounding tissues, and metastasizes, and the malignant cells may then bear less resemblance to the normal cell of origin. The development of a less well differentiated malignant cell in a population of differentiated normal cells is sometimes called "dedifferentiation." This term is probably a misnomer for the process, because it implies that a differentiated cell goes backwards in its development process after carcinogenic insult. It is more likely that the anaplastic malignant cell type, arises from the progeny of a tissue "stem cell" (one that still has a capacity for renewal and is not yet fully differentiated), which has been blocked or diverted, in its usual pathway towards forming a fully differentiated functional cell.

The Process of the Generation and Growth of Cancers

At first it seems a little strange to have to admit that no-one actually knows just what happens when a cancer is first formed, because this seems to be such a basic thing that needs to be answered about the subject and, might one ask, where has all that cancer research money gone to if we do not know such a simple thing after so much research? But the fact is, that when a new human cancerous transformation takes place, no-one is looking. Nor are they even suspecting. Moreover, there is such a long gap between the time when first malignant change occurs and the time when the tumour makes itself felt by being physically noticed, or by

producing symptoms. Naturally, if the tumour is on the skin it is likely to be seen when quite small. If it is in your breast, you have a reasonable chance to palpate it when it is still reasonably small. But what do you do if it is in your liver, which you are not used to examining? In practice, nobody does anything except wait and see. Inevitably, liver cancers tend to be, on average, rather more advanced when first discovered.

The writer has heard a number of different estimates of this time interval, which elapses from the first malignant change, to the appearance of a diagnosable tumour. However, there is a widespread consensus that it is a long period. One authority has suggested 8 years but another suggested 20 to 30 years. One theory is that it depends to some extent upon the natural cell division rate in the particular tissue, which may be slower in one cell type and faster in another. Time factors of this sort partly explain why there is more cancer in older people.

Furthermore, it is clear that the process which produces a cancer is not a simple single-step event. Cancer production, or "carcinogenesis" involves a first step, called "initiation" followed by a process called "promotion". The first step requires the presence of a carcinogenic chemical, of the type referred to as an "initiator". Urethane is an example of such a substance. It seems that the initiator may only require an initial short exposure to produce its effect, which is then probably irreversible. There is probably a huge number of different possible "initiator" chemicals in the modern human environment. As they are numerous, and require only the shortest exposure to be effective, there is probably little practical hope for anyone to avoid exposure to this group of compounds, or to entirely avoid situations in which some of their tissues will be "initiated".

By contrast with the initiation, tumour "promotion" appears to be on a long time-scale. There are certain types of carcinogenic chemicals which have been found to be predominantly or solely "promoters". One of these, an archetype promoter, is the compound 12-O-tetradecanoylphorbol-13-acetate. Such a substance is extremely effective at promoting tumour generation from previously "initiated" cells, but if initiation has not previously taken place at all, then little, if any, cancer develops. The promotion phase appears to be itself a multi-stage process. From the evidence available, it looks as though, on the road towards cancer, one has many opportunities to take corrective action so as to remove the exposure to these "promoter" substances. The question of reversing the process is more problematic. Even if "promotion" is