

Potential of Antineoplastons in Diseases of Old Age

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Rapid progress in cell biology has revealed common links in the pathogenesis of diseases previously thought to be unrelated. Recently, a large group of diseases of information processing, i.e. diseases caused by errors in the programme for cell differentiation and maturation, has become more visible. Diseases which are more common in the elderly, e.g. cancer, certain neurological and autoimmune disorders, and even atherosclerosis, have been found to have certain common molecular aspects. Aging seems to be a continuation of the process, beginning in the embryo, which depends upon activation of some genes and deactivation of others. The 'genetic clock' appears to involve telomeres and methylation of DNA.

Since the beginning of this century, several important classes of biochemical factors have been used in the treatment of diseases associated with aging and in an attempt to modulate the aging process itself. The emphasis has always been on stimulatory molecules, ignoring the obvious truth, known for centuries in the Orient, that in order to control biological processes, the harmonious use of stimulators along with inhibitors is necessary. The discovery of hormones, for example, was followed by the discovery of their antithesis – chalone. The discovery of growth factors and oncogenes was followed by the discovery of tumour suppressor genes and by the cancer growth-inhibiting substances we call antineoplastons.

1. Theory of Antineoplastons

1.1 Biochemical Defence System

According to my theory, the human body possesses a biochemical defence system, consisting of antineoplastons and protecting against the occurrence of abnormal cell growth.^[1-11] Chemical analysis reveals that antineoplastons, initially isolated from blood and urine, are peptides, amino acid derivatives, and carboxylic acids.^[4,10] The function of this biochemical defense system is to 'reprogramme' abnormally developing cells.^[2]

In the late 1960s it became known that cancer is a disease of information processing. Normal cells differentiate according to the programme encoded in the genes, eventually entering the stage of cellular senescence followed by apoptosis, or programmed cell death. Cancer cells, however, escape the fate of normal cells and instead of dying, multiply endlessly.^[9,12] Cellular proliferation is regulated by influences from both outside the cells (growth factors, antineoplastons) and from inside (oncogenes, tumour suppressor genes).

Adverse changes in the early stages of differentiation result in highly malignant neoplasms. Middle and late disruption of the process leads to less aggressive cancers and benign tumours. Errors in the final steps cause abnormal production of the proteins and may result in autoimmune and neurodegenerative disorders.