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Immunotargeting of Melanoma

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1. Introduction

The main task of the immune system is to defend the body against pathogens. The ability of the immune system to recognize and eliminate cancer cells is the basis for cancer immunotherapy. There is ample evidence of how important role plays the immune system to fight cancer: (i) spontaneous remission in patients with certain cancers; (ii) the presence of specific cytotoxic T lymphocytes in the environment of the tumor or regional lymph nodes, (iii) the presence of monocytes, lymphocytes and plasma cells infiltrating the tumor, (iv) increased incidence of some malignancies in immunosuppressed patients, (v) documented remissions of the disease after use of immunomodulators. Better understanding of the molecular and cellular mechanisms that control the immune system has enabled the development of many innovative and promising therapeutic strategies that modulate the immune response. It seems that in the next 5 to 10 years past surgery, radiotherapy and chemotherapy, immunotherapy will find a permanent position in the treatment of cancer modalities.

2. Anti-tumour immune response

Cancerogenesis is closely associated with non-lethal damage of genetic material. Such genetic damage (mutations) can stem from environmental factors, e.g. chemical, radioactive or viral, or can be hereditary. Neoplastic transformation occurs due to accumulation of mutations, predominantly in two gene classes: protooncogenes and suppressor genes. Mutated protooncogenes, termed oncogenes, promote autonomous cell proliferation. Proteins produced by the oncogenes transmit signals to cell nucleus and induce cell division. In contrast, mutated suppressor genes become inactivated and their protein products, deprived of their suppression properties, are not capable of controlling incorrect proliferation. Mutations in apoptosis-regulation genes via the synthesis of improper proteins develop mechanisms preventing programmed cell death.
Burnet's and Thomas' immune surveillance theory assumes that newly formed neoplastic cells are continually monitored by the human immune system which recognises and eliminates them. At some point, however, tumour cells escape immune surveillance, what may result in fully-fledged tumours. During the last twenty years, a number of mechanisms which enable tumour cells to evade effector immune mechanisms have been identified. They include: