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Clinical Features and Prognostic Factors of Cutaneous and Extra Cutaneous Melanoma

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DESCRIPTION

Melanoma skin cancer originates in melanocytes in the skin and has a high risk of spreading metastases. The era of molecular genetics and next-generation sequencing has revealed the role of oncogenic mutations in many Melanomas, validated the role of UV-induced DNA mutations in Melanoma formation, and explored the molecular events occurring during Melanoma development. Revealed much of the Targeted therapy and immunotherapy have dramatically improved outcomes and allowed higher cure rates for metastatic Melanoma. This article provides an overview of Melanoma pathogenesis, the molecular events involved in Melanoma growth and metastasis, and the biology underlying resistance to Melanoma therapy.

The incidence of Melanoma is increasing more rapidly than any other malignancy. The development of Melanoma is multifactorial and results from the interplay between genetic susceptibility and environmental stress. It is believed that 60%-70% of Melanomas are caused by UV light. Most cutaneous Melanomas are at increased risk. Prevention strategies include mitigating environmental risk factors and identifying individuals with phenotypic risk factors for enhanced surveillance.

The incidence of Cutaneous Melanoma is still increasing in most Caucasian populations. The use of sunscreen is recommended for primary prevention of Melanoma. However, the use of sunscreen may increase the risk of Melanoma by increasing the amount of time spent in the sun when the user is willing to tan or stay in the sun for long periods of time. If the exposure is not associated with a desire to tan or stay in the sun for long periods of time, using sunscreen can prevent squamous cell carcinoma of the skin. Sun exposure should be reduced. Over the past two decades, tanning due to exposure to artificial UV sources has become common among Caucasian adolescents and young adults. There is increasing evidence that tanning bed use is associated with Melanoma if started before age 30.

Extracutaneous Melanoma (ECM) represents a heterogeneous group of Melanoma subtypes characterized by clinical and biological features distinct from Cutaneous Melanoma. These subtypes share an aggressive natural history with higher mortality compared to Nonaccrual Cutaneous Melanoma (NACM). Recent advances in NACM have significantly improved morbidity and mortality, but ECM continues to lag behind. As the etiology and molecular characteristics of these rare subtypes continue to emerge, therapeutic research has sought to fill in the gaps. Melanoma, a skin cancer that arises from pigment cells, has been intensively studied, especially with respect to immune responses to tumors, and has served as a model for the development of immunotherapies. This is due to the high mutational burden found in Melanoma, which increases both immunogenicity and immune cell infiltration into the tumor compared to other Cancers. The immune response to Melanoma involves complex components and interactions. As tumors develop, an increasing number of genetic and epigenetic alterations accumulate, some of which contribute to tumor cell immunogenicity and immune cell infiltration. However, tumor evolution also enables the development of resistance mechanisms, which in turn lead to tumor immune evasion. Understanding the interactions between Melanoma tumor cells and the immune system, and the evolving changes within Melanoma tumor cells, the immune system, and the microenvironment, is essential for the development of new Cancer therapies.

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CONFLICT OF INTEREST

The author's declared that they have no conflict of interest.

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