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Ewing sarcoma family of tumors: Translating biomarker research into patient treatment

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Ewing sarcoma family of tumors (ESFT) is an aggressive malignant tumor of bone and soft tissue in children and adolescents that may present with metastasis at diagnosis in 20-25% of cases. Modern treatment regimens for localized disease have resulted in significant improvement in survival. However, the presence of metastasis is associated with mortality in 80% of patients. New insights into the pathogenesis and proliferative mechanisms of this tumor have led to the identification of a number of specific translocation proteins and messenger molecules that are essential for tumorigenesis and are frequently used as biomarkers for pathologic diagnosis of this tumor. A chromosomal translocation involving EWS, a gene located in chromosome 22q12, and any of the ETS family transcription factors is considered a primary genetic event that drives the malignant transformation of ESFT. These fusion proteins act as transcription factors that bind DNA and cause altered expression of several down-stream genes which eventually lead to the activation and production of cell cycle proteins, growth factors and other proliferation molecules. Although research into targeting molecules that will directly inhibit the functions of EWS-FLI1 and other translocation proteins is lagging, there is a potential benefit of targeting downstream proliferation pathway molecules that will reduce tumor growth. Tumor inhibiting experiments on cell lines frequently yield promising results. However, there is a challenge is to generate a pharmacokinetically stable molecule that can be administered to patients for in vivo tumor growth control. Translating the research on inhibitory molecules into effective patient treatment often requires carefully controlled clinical trials that will also provide opportunities for personalized medicine.

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