Special Issue

Molecular Mechanisms Underlying the Progression of Prostate Cancer

Message from the Guest Editors

Prostate cancer is the most diagnosed cancer among men in the US, except for skin cancer. Commonly used for patients with metastatic prostate cancer, androgen deprivation therapies (ADTs), which can be achieved by surgical or medical castration to lower androgen levels, have been initially effective. Unfortunately, a majority of prostate tumors invariably relapse and progress to become ADT-resistant, which is referred to as castration-resistant prostate cancer (CRPC). Approximately 20% of lethal metastatic CRPC have a neuroendocrine phenotype following the development of resistance to hormone therapy, and thus are called neuroendocrine prostate cancer (NEPC). NEPC is characterized by low androgen receptor signaling, castration resistance, and elevated levels of neuroendocrine markers. Unfortunately, without an effective therapy, most patients die within one year upon progression to NEPC. Mechanism by which prostate cancer progresses to CRPC and further progresses to treatment-emergent NEPC are largely unclear, dramatically hindering the therapeutic development for these lethal forms of the disease.

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