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Preface

Ca^{2+} homeostasis and cancer

Cellular calcium (Ca^{2+}) homeostasis is regulated through a variety of extracellular and intracellular mechanisms. In tumors, a change in the cell microenvironment (e.g. pH, pO_2, mechanical stress) is generally associated with the activation of transmembrane receptors and Ca^{2+}-permeable channels that can alter Ca^{2+} homeostasis. Moreover, other ion channels specific for conducting Cl^-, K^+, Mg^{2+} and others are hijacked from their primary physiological function to alter Ca^{2+} homeostasis and contribute to tumorigenesis. In this special issue entitled "Ca^{2+} homeostasis and cancer", some of these mechanisms are presented. Cancer progression induces a remodeling of the microenvironment that becomes stiffer than the healthy tissue. This remodeling is typically associated with an acidification of the interstitial fluid and an increase of mechanical strain. In her review, Glitsch describes how the activation of mechano- and proton-sensing proteins contribute to the aberrant Ca^{2+} signals in cancer [1]. Petho *el al.* present an overview of mechanosensitive channels involved in cancer progression [2]. External Ca^{2+} can also contribute to the remodeling of the tumor microenvironment as discussed by Anract et al. focusing on the link between microcalcifications, Ca^{2+}-sensing receptor and prostate cancer progression [3]. Ca^{2+} channels can also interact with other plasma membrane components and form novel signaling complexes that contribute to cancer progression. In their extensive review, Crottès and Jan describe the multifaceted role of the Ca^{2+}-activated Cl^- channel TMEM16A in cancers [4] while Saberbaghi et al. focused on the role of Cl^- channels in glioma [5]. Ca^{2+} channels can also form functional complex with Ca^{2+}-activated K^+ channels to fuel membrane hyperpolarization and further enhance Ca^{2+} entry. The importance of lipids in Ca^{2+}/K^+ channel interaction in ovarian cancer is presented by Kouba et al. [6]. Moreover there are a variety of ion channel auxiliary subunits that can be dysregulated in cancer as extensively described by Haworth and Brackenbury [7]. Ca^{2+} homeostasis may be disrupted by dysregulation in magnesium (Mg^{2+}) homeostasis due to aberrant expression and function of Mg^{2+} transporters in cancer, as presented by Trapani and Wolf [8]. Finally, some of these ion channels can be targeted by toxins with anti-cancer activity as presented by Srairi-Abid et al. [9].

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