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Preface

Ca^{2+} channels in cancer

Calcium (Ca^{2+}) is a versatile second messenger which is involved in virtually all cellular processes [1,2]. Changes in cytosolic free Ca^{2+} concentration ($[Ca^{2+}]_{free}$) within specific cellular microdomains trigger signaling pathways that control a plethora of diverse cellular functions, including egg fertilization, muscle contraction, exocrine secretion, immune competence, learning and memory and programmed cell death. $[Ca^{2+}]_{free}$ is finely tuned by the activity of plasma membrane and organellar channels and transporters and by Ca^{2+} buffering proteins within the cell. The major regulators of cellular Ca^{2+} signaling are Ca^{2+}-conducting channels adorning the surface of the plasma membrane or membranes of intracellular organelles. These channels can allow Ca^{2+} influx into the cell from the extracellular space or Ca^{2+} release from internal organelles into the cytosol and as such can exquisitely regulate Ca^{2+} signaling in response to external stimuli. Altered Ca^{2+} signaling is a hallmark of numerous diseases including several types of cancer [3]. This special issue contains a collection of reviews focused on the emerging role of Ca^{2+} signaling in several malignancies. Lange et al. present a concise overview of Ca^{2+} signaling components that are deregulated in neuroblastoma that might provide promising targets for future therapies [4]. Ca^{2+} influx across the plasma membrane is mainly mediated by the ubiquitous store-operated Ca^{2+} entry (SOCE) pathway encoded by ORAI Ca^{2+} channels. ORAI channels are activated by depletion of inositol-1,4,5-trisphopshate (IP_3)-sensitive intracellular Ca^{2+} stores. Other store-independent Ca^{2+} entry (SICE) pathways have been reported in different cell types [5,6], including cancer cells and are encoded by different isoforms of ORAI or Transient Receptor Potential (TRP) channels. Cantonero et al. present an overview for SICE in cancer [7] while the review by Pierro et al. focuses on SOCE [8]. In particular, the importance of SOCE in colon cancer is extensively described by Villalobos et al. [9]. In another digestive cancer, the hepatocellular carcinoma, Ali et al. explain how lipids alter Ca^{2+} signaling to promote disease progression [10]. TRP channels are often upregulated in cancers, and the review by Sterea et al. summarizes how TRP channels regulate cancer cell fates specifically in gastric cancer [11]. TRP channels constitute a large family of Ca^{2+} channels that act as sensors for changes in the cell environment. TRP melastatin 2 (TRPM2) channels are particularly essential for cancer cell survival and protection against stress as described in the review of Miller [12]. Recently, the importance of cancer stem cells (CSCs) is an emerging area of experimental oncology. CSCs ability to self-renew and proliferate from a quiescent state is suspected to be responsible for resistance to standard chemotherapy and for tumor recurrence. The role of Ca^{2+} signaling in CSCs is nicely detailed by two highly complementary reviews from O'Reilly and Buchanan [13] and from Terrié et al. [14]. While this special issue focuses on the impact of remodeling Ca^{2+} channel expression and activity on cancer cell growth and progression, other equally important ion channels specific for conducting Cl^-, K^+, Mg^{2+} and others are also remodeled during cancer and can directly or indirectly alter cellular Ca^{2+} homeostasis through a variety of mechanisms. Some of these mechanisms are presented in an independent special issue entitled "Ion channels and Ca^{2+} homeostasis in cancer".

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