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Calcium Signaling and Its Dysregulation in Cancer 2.0

Guest Editor:

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Deadline for manuscript submissions:
closed (30 June 2023)

Message from the Guest Editor

Dear Colleagues,

Calcium is the common currency of differentiation and homeostasis. It is stored primarily in the endoplasmic reticulum, rationed according to need, and replenished from the extracellular milieu via store-operated calcium entry (SOCE). This currency is disbursed by the inositol triphosphate (IP3) receptor in response to diverse extracellular signals. The rate of release is governed by regulators of metabolism and proliferation, differentiation, autophagy, survival, and programmed cell death, with different outcomes depending on the strength of the signal and context. This system is fundamentally tumor-suppressive, and cancer cells must find ways to subvert it in order to exploit its many growth-promoting effects.

This Special Issue invites both original manuscripts which describe novel findings and cutting-edge review articles which illustrate the many mechanisms by which cancer cells dysregulate SOCE, IP3, and ryanodine receptors; calcium transfer to mitochondria; and signaling to downstream effectors and targets to prevent cell death and enhance metabolism, mitogenesis, and metastasis.

Dr. Randolph C. Elble
Guest Editor

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Message from the Editor-in-Chief

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