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# **Epithelial-Mesenchymal Transition (EMT) 2.0**

Guest Editors:

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Deadline for manuscript submissions:

closed (30 September 2020)

### **Message from the Guest Editors**

Epithelial–mesenchymal transition (EMT) is a biological process that allows an epithelial cell to assume a mesenchymal phenotype. It is initiated by different molecular processes, including activation of transcriptional factors, expression of specific cell-surface proteins, reorganization and expression of cytoskeletal proteins, production of ECM-degrading enzymes, and changes in microRNAs expression. There are both endogenous cell autonomous and exogenous noncell autonomous signals occurring in the process, including pathways orchestrated by TGF-b, Notch, Wnt, Hedgehog, and receptor tyrosine kinases, as well as the urokinase plasminogen activator system, the secretome of associated fibroblasts, macrophages, cancer stem cells and cancer cells, and exosomes with their cargo of microRNAs.

However, relatively little is known about how all these components are integrated and participate in the same process, and how the mesenchymal state is maintained. Deep knowledge of these aspects will help design potential therapeutic approaches that could exploit the plasticity of EMT to reverse the metastatic phenotype of many cancers. Papers related to any aspect of EMT will be considered.













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#### **Editor-in-Chief**

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## **Message from the Editor-in-Chief**

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