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The mTOR Targets 4E-BP1/2 Restrain Tumor Growth and Promote Hypoxia Tolerance in PTEN-driven Prostate Cancer
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Correction: Heparanase Promotes Glioma Progression and Is Inversely Correlated with Patient Survival
This Rapid Impact, by Andrieu and Denis (beginning on page 580), establishes that bromodomain and extraterminal (BET) proteins exert distinct and opposing transcriptional controls on EMT in breast cancer. The EMT is a developmental program that cancer cells often activate to acquire a highly plastic phenotype, notably eliciting metastasis, adaptation to the environment or chemoresistance. The cover image is an artistic rendering that shows epithelial breast cancer cells, exhibiting tight junctions enriched in the protein E-cadherin (staining). Cancer cells undergoing EMT lose their cuboidal shape, disassemble their tight junctions, hallmarks of their epithelial nature, to acquire a plastic mesenchymal phenotype. The study shows that BET proteins functionally oppose each other in the regulation of EMT. BRD2 positively controls several key EMT transcription programs, whereas BRD3 and BRD4 repress them. BET protein inhibitors obtained promising results for the treatment of some cancers, including breast cancer, yet little is known about the individual functions of each member of this particular family. To this end, more research is needed and will lead to the elaboration of improved strategies for cancer treatment.

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