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Biography

Dean G Tang, PhD, currently Professor and Chairman of Department of Pharmacology & Therapeutics at the Roswell Park Comprehensive Cancer Center (RPCCC) in Buffalo, NY, USA, was trained as an Oncological Pathologist and received his PhD at Wayne State University (Detroit, MI, USA) in 1994 Tang pursued a Burroughs-Wellcome senior post-doctoral fellowship with Dr. Martin Raff (MRC LMCB, UCL, London, UK) studying stem/progenitor cell development. Tang joined the University of Texas M.D Anderson Cancer Center (MDACC) as a faculty in 2000 In June of 2016, he moved to RPCCC to head the Department of Pharmacology & Therapeutics. Tang has made many contributions to cancer research, among which the most important is his pioneering work on identifying, characterizing, and targeting the prostate cancer stem cells (PCSCs). His laboratory, since 2002, has been applying normal stem cell biology knowledge to elucidate the fundamental biological principles that govern the generation of tumor cell heterogeneity via CSCs and epigenetic mechanisms. By focusing on prostate cancer (PCa), Tang and his colleagues have demonstrated the presence and revealed many unique biological, molecular, and tumorigenic properties of PCSCs. One line of Tang's laboratory studies is now undergoing a phase Ib/II clinical trial. Tang has published 170 papers with many in top-tier journals and a current h-index of 65 Tang has won many awards and is an elected fellow of AAAS. Tang is a passionate educator and has mentored about 20 graduate students and 40 postdoc fellows and junior faculty.

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CANCER STEM CELLS: ENGINE OF THERAPY RESISTANCE & SEEDS OF TUMOR RECURRENCE

Our lab has been studying basic principles governing generation of tumor cell heterogeneity via cancer stem cells (CSCs). By employing prostate cancer (PCa) as a model, we have demonstrated that PCa cells are not all equal with respect to their tumorigenic and metastatic potential. Rather, there exist stem cell-like PCa cells that are mostly undifferentiated (i.e., PSA-/lo), relatively quiescent, and resistant to clinical therapies including castration. These prostate CSCs (PCSCs) preferentially express stem cell genes and epigenetic landmarks, can undergo asymmetric cell division and regenerate differentiated (PSA+) cells, and become greatly enriched in treatment-failed tumors (Cell Stem Cell, 2012; Oncotarget, 2015, 2016; Clin Cancer Res, 2016). Several tumor-suppressive miRNAs, including miR-34a, miR-141, and miR-128, potently suppress the PCSC properties and PCa metastasis (Nat Med, 2011; Cancer Res, 2014; Nat Commun, 2017). Both PSA- normal human prostate basal/stem cells (Nat Commun, 2016) and PSA-/lo PCSCs express an intrinsic neurogenesis program that causally regulates their stem/progenitor cell activities. Of clinical relevance, PCa cell heterogeneity, in particular, AR heterogeneity, has a great impact on PCa response to current clinical therapeutics (Nat Commun, In revision). While we are uncovering novel therapeutics using organoids-based high throughput screening that can specifically target undifferentiated CSCs, we are already translating some of our laboratory findings to clinical trials.