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11th International Conference on
CANCER STEM CELLS AND ONCOLOGY RESEARCH

June 11-13, 2018 | Dublin, Ireland

Wei Qiang Gao, J Med Oncl Ther 2018, Volume 3

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Biography

Wei Qiang Gao received his PhD from Columbia University in 1989 and did his post-doctoral research at Columbia University and Rockefeller University. From 1993-2010, he was a Scientist and Senior Scientist at Genentech, Inc.. He then relocated to China to initiate his endowed chair professorship in Shanghai. He has made important contributions to the fields of neuroscience, stem cells and tumorigenesis. More recently, his group focuses on "cancer research and cancer stem cells". Dr. Gao has published more than 80 papers as either corresponding or the first author, including Nature, Cell, Science, Neuron, Nature Neuroscience, Nature Communications, Gastroenterology, PNAS, J. Neurosci., Stem Cell Reports, etc. and has been granted 48 US patents. He is a scholar of national "Thousand-Talents Program", the Chief Scientist of 2 program projects from the Ministry of Science and Technology of China and 2 key grants from the National Natural Science Foundation of China. He has served as a reviewer for grant proposals of Wellcome Trust in UK, NIH in US, and NSFC and 36 journals including Nature, Nature Medicine, Nature Cell Biology, Nature

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KEY REGULATORS OF SYMMETRICAL AND ASYMMETRICAL DIVISION OF EPITHELIAL CELLS IN PROSTATE DEVELOPMENT AND TUMORIGENESIS

Although symmetrical and asymmetrical divisions of stem cells are extensively studied in invertebrate and mammalian neural epithelia, their role remains largely unknown in mammalian non-neural epithelial development, regeneration and tumorigenesis. Using basal and luminal cell-specific markers and cell lineage tracing transgenic mice, we report that in developing prostatic epithelia, basal and luminal cells exhibit distinct division modes. While basal cells display both symmetric and asymmetric divisions leading to different cell fates, luminal cells only exhibit symmetrical divisions, producing two luminal cells. Examination of cell division modes in prostate-specific Pten mull mice indicates that while transformed luminal cells can independently produce tumors composed of exclusive luminal cells via symmetrical divisions, transformed basal cells appear to generate cancer through the daughter luminal cells derived from asymmetrical divisions. Cell polarity and correct mitotic spindle positioning are essential for the proper prostate epithelial cell division mode, and disruption of the two biological features occurs at early stages in prostate tumorigenesis. However, whether and how these two epithelial attributes are coordinated *in vivo* is largely unknown. We report that conditional genetic deletion of E-cadherin, a key component of adherens junctions, in a mouse model results in loss of prostate luminal cell polarity and randomization of spindle orientations. Critically, E-cadherin ablation causes prostatic hyperplasia which progresses to invasive adenocarcinoma. Mechanistically, E-cadherin forms a complex with the cell polarity protein SCRIB and the spindle positioning determinant LGN to link cell polarity and cell division orientation. Collectively, these findings provide direct evidence for the existence of a hierarchy of epithelial cell lineages during prostate development and tumorigenesis and a novel mechanism by which E-cadherin acts an anchor to maintain prostate epithelial division orientation and to prevent tumorigenesis *in vivo*.