allied academies

11th International Conference on
CANCER STEM CELLS AND ONCOLOGY RESEARCH

June 11-13, 2018 | Dublin, Ireland

J Med Oncl Ther 2018, Volume 3

POLYPLOID GIANT CANCER CELLS MAY REPRESENT A SOMATIC EQUIVELANT OF BLASTOMERE

Jinsong Liu and **Na Niu**
The University of Texas, USA

It is now generally accepted that all mature somatic cells retain the capability to be reprogrammed (or dedifferentiated) to pluripotent state. However, it remains unclear how the endogenous developmental pathway is activated for such a reprogramming. We have recently shown that chemotherapy drug paclitaxel (PTX) can induce cancer cells undergo senescence and lead the formation of a big monster cells, refer as polyploid giant cancer cells (PGCCs). PGCCs bypass the spindle checkpoint and replicate the DNA without cell division. PGCCs show time- and space-dependent activation of expression of reprogramming factors OCT4, NANOG, and SOX2; lack expression of Xist; and are capable of de-differentiation. The parental cancer cells are reprogrammed via formation of PGCCs which can give a birth of diploid resistance cancer cells via budding. This division mode recapitulates that of blastomere-to-morula stage embryo and facilitates the dedifferentiation toward the blastomere stage embryonic stem cells. PGCCs use an evolutionarily conserved embryonic program used to reprogram zygote to new embryonic state for for disease relapse and thus represent a somatic equivalent of blastomere. Here, we provide a model on how PGCCs divide and how they achieve the dedifferention, named the blastomere model for cancer and disease relapse. This new conceptual paradigm, which integrates different tumors along bidirectional developmental hierarchy, should facilitate our understanding of cancer origin and to guide our efforts for therapeutic intervention.

jliu@mdanderson.org