OMICS Group Conferences
Accelerating Scientific Discovery

3rd World Congress on
Cell Science & Stem Cell Research
November 20-22, 2013 DoubleTree by Hilton Baltimore-BWI Airport, MD, USA

miR-302 contributes to tumorigenicity through promoting G1 to S transition and maintaining OCT4 overexpression by inhibiting AKT1

Shihua Wang and Robert Chunhua Zhao
Peking Union Medical College Hospital, China

The tumorigenicity of human pluripotent stem cells (hPSCs) has been considered to be a major hurdle for their clinical applications. Here, we found that downregulation of miR-302 suppresses the tumorigenicity of hNT-2 cells (a human embryonal carcinoma pluripotent stem cell line). By gain- and loss-of-function approaches, we demonstrated that miR-302 promotes the proliferation and tumorigenicity of hPSCs through the dominant regulation of a set of cell cycle inhibitors and subsequent acceleration of the G1 to S transition. Importantly, we also found that downregulation of miR-302 damages the self-renewal and pluripotency, and promotes differentiation of hPSCs. The underlying mechanism is that high endogeneous expression of miR-302 suppresses the expression of Akt1 by directly targeting its 3'UTR, and subsequently maintains pluripotent factor OCT4 at high level in hPSCs. However, overexpression of miR-302 in human mesenchymal stem/stromal cells (hMSCs) dose not leads to tumor formation *in vitro* or *in vivo*. These findings indicated that in human pluripotent stem cells miR-302 is not a causal factor, but just a contributing factor for the tumorigenicity through promoting G1 to S transition and maintaining OCT4 at high level by directly inhibiting AKT1.

wshawp@163.com, zhaochunhua@vip.163.com