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Hypoxia, epigenetics and estrogen receptor in breast cancer

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More than 1.7 million new cases of breast cancer occur every year, 70% of which are estrogen receptor alpha (ERα) positive. Anti-estrogen therapy to block ERα function is the most important approach in treatment of ERα positive patients. However, resistance eventually will develop for various reasons. Previous clinical studies suggest that *in vivo* tumor environment may play a role in tamoxifen resistance, as hypoxia-inducible factor 1 alpha (HIF-1α) protein expression is associated with tamoxifen resistance in neoadjuvant, primary therapy of ERα-positive breast cancers as well as resistance to chemo endocrine therapy. However, whether HIF-1α plays an autonomous role in modulating endocrine therapy efficacy such as tamoxifen resistance is unknown. It is also puzzling that increased HIF-1α is associated with ERα positivity in clinical samples, since ERα negative breast cancer is more hypoxic. These two important oncogenic transcriptional factors interact has not yet been defined. In this presentation, I will discuss a new signaling pathway between ERα and HIF-1α, providing evidence that HIF-1α may account for anti-hormone therapy. I will also discuss how HIF-1α and ERα cooperate to drive expression of histone demethylase KDM4B in breast cancer and demonstrate KDM4B as a potential therapeutic target.

Biography

Jun Yang completed his PhD from Institute of Cancer Research, Sutton, UK, and Post-doctoral studies from Oxford University and St Jude Children's Research Hospital. He is the Faculty in Department of Surgery, St Jude Children's Research Hospital. He has published more than 20 papers in reputed journals.

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