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VIEW ON NEWS

Research highlights from ACACR members

Dr. Chuan He at The University of Chicago and his collaborator at the ShanghaiTech University found that N6-methyladenosine (m6A) facilitates hippocampus-dependent learning and memory through YTHDF1, a m6A binding protein that enhances protein synthesis in a neuronal-stimulus-dependent manner. https://www.nature.com/articles/s41586-018-0666-1.

Dr. Shirley Liu at Harvard University develop a novel bioinformatics method called TIDE to model two primary mechanisms of tumor immune evasion: the induction of T cell dysfunction in tumors with high infiltration of cytotoxic T lymphocytes (CTL) and the prevention of T cell infiltration in tumors with low CTL level. Using this computational method, her team identified Signatures of T cell dysfunction and exclusion that predict cancer immunotherapy response. https://www.nature.com/articles/s41591-018-0136-1.

Dr. Huiping Liu at Northwestern University identified a new mechanism of circulating tumor cell (CTC) cluster formation via cellular aggregation, dependent on CD44 homophilic interactions and subsequent CD44-PAK2 signaling. This work highlights the potential of $CD44^{+}$ CTC clusters serving as a poor prognosis biomarker as well as novel therapeutic targets of polyclonal metastasis. http://cancerdiscovery.aacrjournals.org/content/early/2018/10/18/2159-8290.CD-18-0065.

Dr. Wenliang Li at University of Texas Health Science Center at Houston identified a critical pathway that connects neuroendocrine differentiation with angiogenesis, two biological processes crucial for neuroendocrine prostate cancer (NEPC), which is aggressive, poorly understood with no effective treatment. https://www.nature.com/articles/s41467-018-06177-2.

Dr. Shuli Xia at Johns Hopkins University School of Medicine identified YAP and its downstream signaling pathway Notch mediating the cell growth-inhibiting effect of $IDH1^{R132H/WT}$. https://www.nature.com/articles/s41388-018-0334-9.

In another recently publication, her team found that KLF4 bound to methylated CpGs at the enhancer regions of the B-cell lymphocyte kinase (BLK) and Lim domain only protein 7 (LMO7) genes, and activated their expression via 3D chromatin loop formation with their promoter regions. https://www.tandfonline.com/doi/full/10.1080/15592294.2018.1504592.

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