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Review Report Open Access

Article Title: Cells Signal Before Driving the Cycle: Metabolic Signaling Regulates Cell Division

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Review Report

This article reviewed recent findings that obesity as a result of hyper nutrition or hormonal imbalance induce an chronic inflammation, which would link to cancer by virtue of its altered hormonal and inflammatory climates. The key roles of obesity promoting cancerogenesis include polarization of adipose-sepcific macrophages into a pro-inflammatory M1 population that secretes pro-inflammatory cytokines. Macrophages are known for their contribution to obesity by FFA activating stress MAPK kinase signaling, specifically JNK, which in turn stimulates production of cytokines.

After that, the article deeply discusses the developments in the field of cell metabolism between metabolic disorder and the cell cycle. Nutrients like glucose and saturated fats activates stress-related signaling cascade of MAPK, increase insulin secretion. Secreted insulin activates the PI3K/Akt/PKB signaling pathway, which induces the change of cell proliferation in T and B lymphocytes. Asymmetrically distribution of PI3K/Akt/mTOR cascade will favor plasma cell differentiation into an antibody-secreting cell, while daughter cells that inherited weaker PI3K/Akt/mTOR signaling displayed a plasma cell fate during B lymphocyte division. The authors raised a speculative hypothesis that obesity-induced PI3K has a potential to alter cell fate and differentiation potential. In macrophages, saturated FFAs promote activation of MLK and JNK MAPK kinase, which will implicated polarization. In beta cells, the majority are arrested in G0 stage of the cell cycle by the cyclin dependent kinase Ink4 family inhibitors and CIP/KIP family inhibitors. Much recent work has focused on understanding the G0 arrest and striving to overcome it by stimulating known replicative pathways even with only limited success.

Finally, this article talks about metabolic pathways mediating cell centrosome physiology. Inflammatory stimuli activate MLK pathway and induce centrosome maturation in interphase. But it is not clear, weather such Plk1-independent centrosome maturation prevents cells from entering mitosis. Recent advances indicate the role of metabolism-induced signaling pathways, and at the same time, more researches are needed to establish the mechanistic and functional connection between metabolic disorder and the cell cycle.

I have read this submission. I believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard, however I have significant reservations, as outlined above.