SCIENCE CHINA
Life Sciences

SPECIAL TOPIC: Non-resolving inflammation and cancer
• EDITORIAL •

June 2017 Vol.60 No.6: 561–562
doi: 10.1007/s11427-017-9096-4

Non-resolving inflammation and cancer

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Received May 21, 2017; published online May 28, 2017

Citation: Wang, H.Y. (2017). Non-resolving inflammation and cancer. Sci China Life Sci 60, 561−562. doi: 10.1007/s11427-017-9096-4

The discovery of the close relationship between inflammation and cancer has been traced back to the 17th century though back then little is known about cells, cytokines and physiological processes that are essential in inflammation and cancers.

Chronic-inflammation is a major pathogenic factor in certain types of tumors such as hepatocellular carcinoma, esophageal carcinoma, and gastric carcinoma. Likewise cancers can initiate and maintain local inflammatory processes that may facilitate the proliferation and infiltration of tumor cells. As a result, inflammatory pathways have been targeted in the therapeutic scheme of certain cancers. Inflammation is a central part of the innate immune system's response to tissue damage or infection, and also includes the recruitment of circulating cells and antibodies of the adaptive immune response to the tissue. Immune cells and cytokines of the innate immune response usually lead to a robust, but sometimes overly-conservative response, at the expense of specificity for the sake of preservation. Thus, when innate immunity goes wrong, it can have profound effect on normal physiological processes. How the innate and adaptive immune systems cooperate to eliminate pathogens and repair damaged tissues is still an ambiguous field. More importantly, what kind of contributions immune systems play in cancers which originate from normal 'mortal' cells that go through oncogenic transformation, has shed light on cancer therapeutic strategies. Consequently, the relationship between cancer and inflammation is now the forefront of clinical oncology.

In this special topic of 'Non-resolving inflammation and cancer', we provide 6 articles focusing on digging up the role inflammation might play in the pro/anti-tumorigenic process in inflammation-driven cancer.

In the article entitled 'APOBEC3B and IL-6 form a positive feedback loop in hepatocellular carcinoma cells', Yang et al. found that APOBEC3B could induce IL-6 expression through relocating HuR to enhance the IL-6 mRNA stability, in turn, IL-6 also increased the expression of A3B through JAK1/STAT3 signaling pathway, which formed a positive feedback to maintain the continuous expression of A3B and IL-6, and thereby promoted the prolonged non-resolving inflammation in hepatocellular carcinoma (Li et al., 2017).

In 'STING-Mediated DNA sensing in cancer immunotherapy', Jiang and his colleague pointed out that the STING pathway is an innate immune sensing mechanism driving type I interferon production in the tumor context. Better understandings of this pathway can guide further development of novel immunotherapeutic strategies in the treatment of cancer (Zhou et al., 2017).

In 'Gender disparity in hepatocellular carcinoma (HCC): multiple underlying mechanisms', Chen's review focused mainly on the molecular mechanisms behind the sex difference in HCC associated with HBV and other factors. Moreover, several potential treatment and therapeutic strat-

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egies for inflammation-driven HCC will be introduced in this review as well (Zheng et al., 2017).

In 'Targeting sphingosine-1-phosphate signaling for cancer therapy', Geng et al. mainly described recent advances in our understanding of S1P signaling which is identified as regulators of cell fate in cancer development (particularly in inflammation-associated cancer) as well as in innate and adaptive immunity, and they also discuss modulators of S1P signaling in cancer treatment (Xie et al., 2017).

In 'Prevention and treatment of cancer targeting chronic inflammation: research progress, potential agents, clinical studies and mechanisms', Jiang and his colleague summarized the relationship between chronic inflammation and cancer and describes some potentially useful agents including aspirin, meformin, statins, and some natural products for their cancer prevention and treatment targeting chronic inflammation (Zhang et al., 2017).

In 'Decoding early myelopoiesis from dynamics of core endogenous network', Zhu et al. established a core dynamic molecular endogenous network from well-documented gene regulation and signal transduction knowledge in myelopoiesis and further elucidate the existence of a transitional state between stable states in the process of myelopoiesis (Su et al., 2017).

I hope that these articles can help make a better understanding in the relationship between inflammation and cancer and hopefully in the future, the new findings in this area can shed light on novel therapeutic regime in cancers.

Compliance and ethics *The author(s) declare that they have no conflict of interest.*

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Biographical Sketch

Dr. Hong-Yang Wang, a professor, chief physician and doctoral supervisor, currently serves as the director of National Centre for Liver Cancer of China, the director of International Cooperation Laboratory on Signal Transduction in Eastern Hepatobiliary Surgery Hospital, and the chief of Department of Health of the National Natural Science Foundation of China (NSFC). She was selected as a member of Chinese Academy of Engineering in 2005 and as a member of the World Academy of Sciences in 2011. Dr. Hong-Yang Wang received her doctor degree at Ulm University, Germany (1992), and a post-doc training in Molecular Biology at Max-Planck Institute of Biochemistry, in 1995. She pioneered signal transduction research of liver in China. Dr. Hong-Yang Wang has authored more than 200 publications. Additionally, 14 patents of hers have been warranted, including 3 international authorizations. Dr. Hong-Yang Wang's research focuses on prevention, diagnosis and treatment of liver cancer in China. Her research team is interested in molecular mechanisms and translational medicine research of tumors. They researched and developed the Glypican-3 diagnostic kit to diagnose hepatocellular carcinoma, especially for alpha fetoprotein (AFP)-negative patients. The Kit was approved by China Food and Drug Administration (CFDA) in 2014.