Research Summary for Qianli Wang

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The tumor suppressor p53 activates an Epstein-Barr virus oncogenic protein

Epstein-Barr virus is known to be the cause of mononucleosis and is associated with multiple malignancies such as Burkitt's lymphoma and nasopharyngeal carcinoma. A key reason for this is EBV's ability to transform cells which could lead to tumor formation. For EBV induced transformation to occur, the viral latent membrane protein 1 (LMP1) expression is required. The human body also has a series of methods to prevent cancer. Among these, DNA damage response (DDR) and p53, an important anti-tumor gene, are major players. The p53 gene is capable of inducing apoptosis to prevent cancer formation. It is known that DDR activates p53 activity and LMP1 suppresses P53 functions. However, the relationships between DDR and LMP1 and the effects of p53 on LMP1 are not known. We found that DDR can increase LMP1 expression through p53. Also, we have evidence suggesting that IRF5 is one possible intermediate linking p53 and LMP1 expression. To our knowledge, this is the first case where DDR, through the activity of an anti-tumor gene, increases a viral oncoprotein expression.

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