# **Special Issue**

# NF B in Cancer

## Message from the Guest Editor

NF-\( \B\) complexes, formed from a family of five NF-\( \B\) subunits, ReIA (p65), c-ReI, ReIB, NF-B1 (p105/p50) and NF-\( B2 \) (p100/p52) are generally held in an inactive form until induced by wide range of stimuli. In cancer, NF-\B activation only rarely results from direct mutation of the NF-\( \mathbb{B} \) or IKK subunits but most commonly arises either through mutation of upstream regulators leading to constitutive IKK activity or via effects of the tumour microenvironment. This special issue will include both NF-\(\times\)B's function as a driver of inflammation associated cancer and its function as an effector of oncogene induced malignancy. Also it will also cover the links between NF-\( \mathbb{N} \) B and tumour suppressors and how these can lead to altered NF-\B behaviour, the mechanisms leading to aberrant NF-\( \mathbb{N} \)B in cancer, the functions of the NF-\B subunits and consider, given the complexity of the pathway, the best strategies for targeting it to achieve new and improved cancer therapies.

### **Guest Editor**

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### Deadline for manuscript submissions

closed (30 June 2019)



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