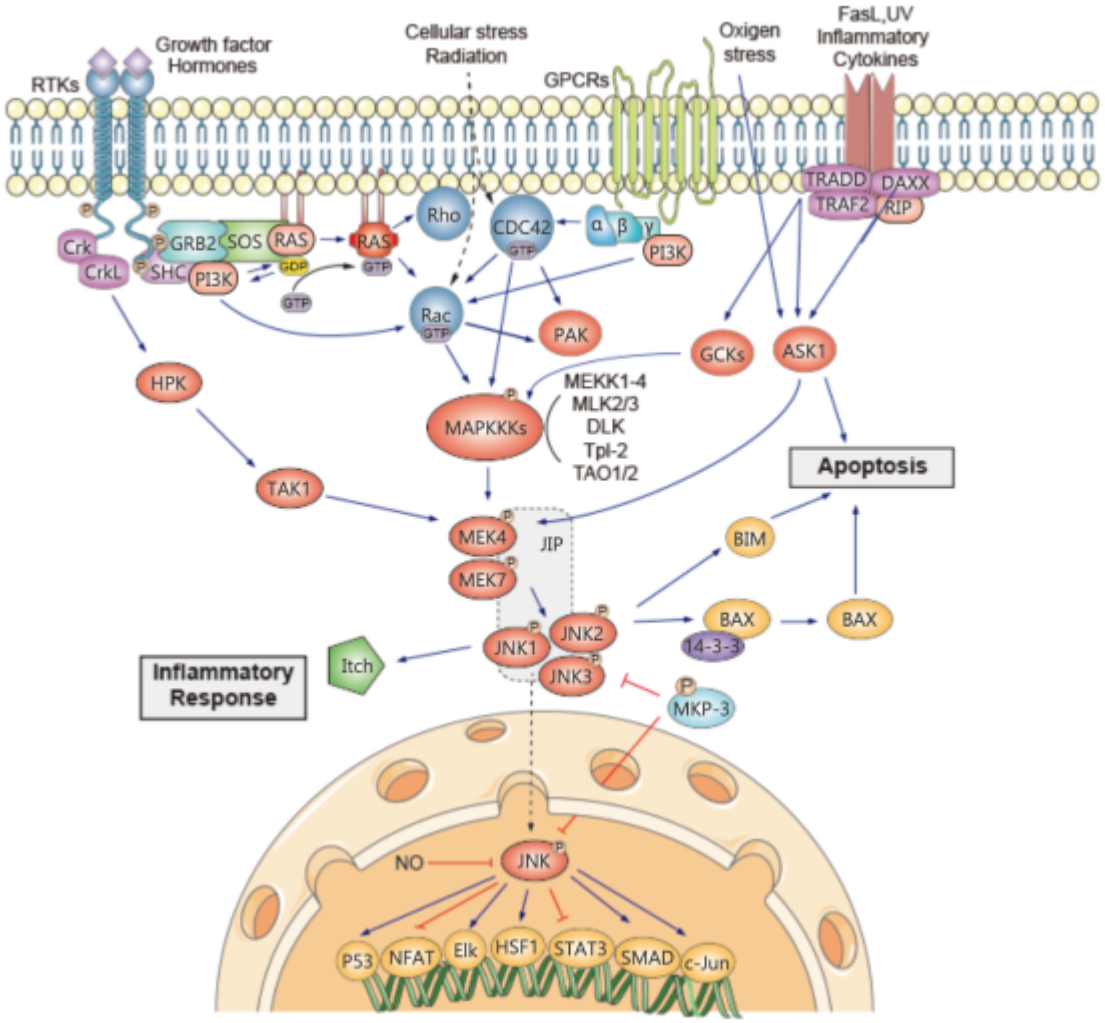
JNK signaling pathway

The c-Jun N-terminal kinase (JNK) pathway is one of the major signaling cassettes of the mitogen-activated protein kinase (MAPK) signaling pathway. It functions in the control of a number of cellular processes, including proliferation, embryonic development and apoptosis. The pathway takes its name from the c-Jun N-terminal kinases 1–3 (JNK1–JNK3), which are the MAPKs that interact with the final effectors. The JNK pathway is activated by a bewildering number of mechanisms. This complexity is evident by the fact that there are 13 MAPK kinase kinases (MAPKKKs) responsible for feeding information into the JNK pathway. The JNK pathway can also be activated through G protein-coupled receptors (GPCRs) using G proteins such as G12/13.



The expression of MAP2K4 was negatively correlated with miR-363-3p while positively related to HNF1A-AS1 in glioma tissues. Bi et al. found the regulatory effect of HNF1A-AS1 on the MAP2K4-dependent JNK signaling pathway. All findings indicated that HNF1A-AS1 induces the upregulation of MAP2K4 to activate the JNK signaling pathway to promote glioma cell growth by acting as a miR-363-3p sponge [1].

1)
Bi Y, Mao Y, Su Z, Du J, Ye L, Xu F. Long non-coding RNA HNF1A-AS1 regulates proliferation and apoptosis of glioma through activation of the JNK signaling pathway via miR-363-3p/MAP2K4 [published online ahead of print, 2020 Aug 10]. J Cell Physiol. 2020;10.1002/jcp.29916. doi:10.1002/jcp.29916