

The $\alpha 9$ -helix of GBP-1 mediates suppression of proliferation by the interaction with the Hippo transcription factor TEAD

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Background: Role of IFN γ in gastrointestinal diseases

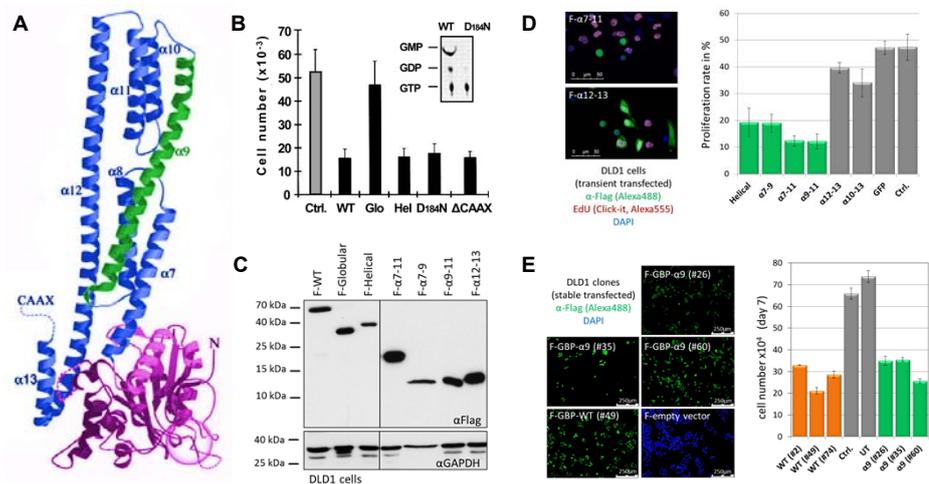
In Inflammatory Bowel Disease (IBD) interferon-gamma (IFN γ) is associated with an increased vessel permeability and a repressed angiogenesis (Haep et al., 2015). Also in Colorectal Cancer (CRC) IFN γ correlates with a repressed angiogenesis (Guenzi et al., 2003; Weinländer et al., 2008).

GBP-1 is among the major IFN γ induced factors. We have shown previously that GBP-1 is closely associated with an intratumoral Th1 tumor microenvironment in CRC and is an

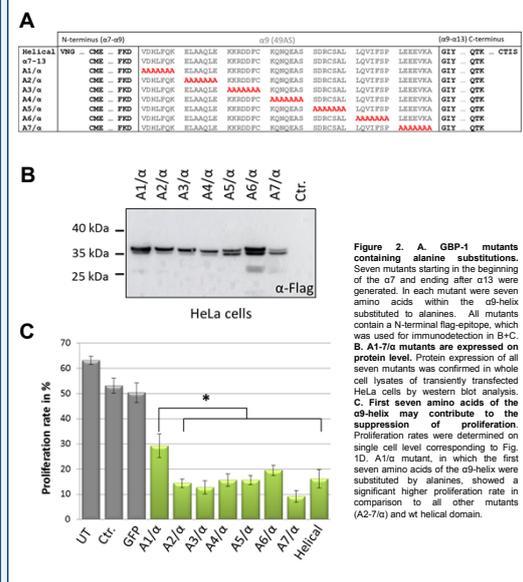
independent prognostic factor of increased cancer-related 5-year survival (Naschberger et al., 2008) in this disease.

This is due to the angiostatic activity of GBP-1. GBP-1 expression leads to a repressed proliferation in endothelial as well as tumor cells. Furthermore we demonstrated that GBP-1 acts as a tumor suppressor in CRC (Britzen-Laurent et al., 2013). However, the molecular mechanism of this suppression of proliferation remains to be elucidated.

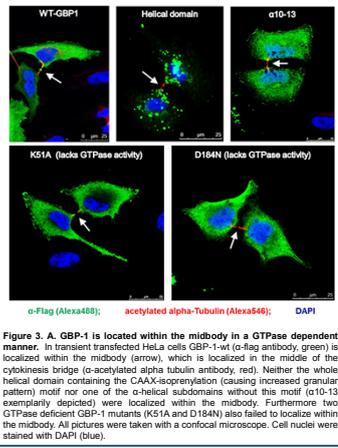
The $\alpha 9$ -helix of GBP-1 mediates suppression of proliferation



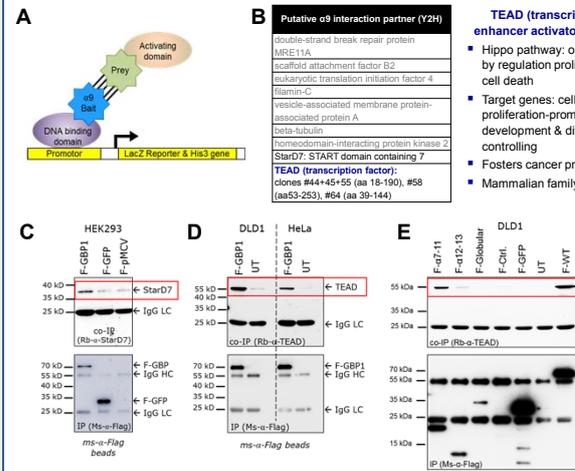
The first 7 amino acids of the $\alpha 9$ may contribute to the suppression of proliferation



GTPase dependent midbody localization



GBP-1 interacts with Hippo transcription factor TEAD



Outlook & Long Time Perspectives

