Investigation of the action of phosphatase of regenerating liver on PTEN using murine models

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Abstract:

The addition and removal of phosphate groups is a key regulatory mechanism for many cellular processes. The balance between phosphorylation and dephosphorylation is delicate and must be maintained in order for proper cell functions to be carried out. Protein kinases and phosphatases are the keepers of this balance with kinases adding phosphate groups and phosphatases removing them. As such, mutation and/or altered regulation of these proteins can be the driving factor in disease. Phosphatase of Regenerating Liver (PRL) is a family novel of three dual specificity phosphatases (DSPs) first discovered in the regenerating liver tissue of rats. PRLs have also been shown to act as oncogenes in cell culture and in animal models. However, the physiological substrate and mechanisms of the PRLs are not yet known. Recently, our lab has

developed a PRL 2 knockout mouse and found several striking phenotypes all of which correspond to a significant increase in PTEN. We also found that PRL 2 is targetable by small molecular inhibitors that can potentially be used to disrupt tumor growth and spermatogenesis. Furthermore, a PTEN heterozygous mouse model crossed into our PRL 2 knockout line was generated to investigate the relevance of PRL interaction with PTEN in cancer.

Description:

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