prot

TP: 10

FP: 2

FN:

lyso

TP: 7

FP:

FN:

Podocyte [apoptosis:LYSO] is considered as the important element that promotes the development and progress of membranous nephropathy (MN). Unfortunately, the underlying mechanism of podocytes [apoptosis:LYSO] in MN remains elusive. We compared the renal expressions of miR-130a-5p and M-type [phospholipase:PROT] [A2:PROT] receptor ([PLA2R:PROT]) between MN patients (n = 30) and 30 controls by qRT-[PCR:PROT] and western blot, respectively. The podocyte damage model in vitro was established by [angiotensin II:PROT] ([Ang II:PROT], 100 nmol/L) exposure for 24 h. Interaction between miR-130a-5p and [PLA2R:PROT] was determined using dual-[luciferase reporter gene:PROT] assay. MN mice were induced by intravenous injection of cBSA. In this study, miR-130a-5p expression was significantly decreased both in the renal biopsy specimens from MN patients and podocyte cell line AB8/13 following stimulation of [Ang II:PROT]. Overexpressed miR-130a-5p in AB8/13 cells significantly attenuated the [Ang II:PROT] induced-[apoptosis:LYSO] in vitro. In contrast, down-regulated miR-130a-5p induced podocyte [apoptosis:LYSO]. [PLA2R:PROT] was identified as the target of miR-130a-5p in AB8/13 cells. And up-regulated or down-regulated [PLA2R:PROT] could obviously attenuate the effect of miR-130a-5p overexpression or knockdown on the [apoptosis:LYSO] of AB8/13 cells. Furthermore, it was also observed that overexpressed miR-130a-5p by miR-130a-5p agomir could obviously alleviate renal injury in MN mice. In conclusion, decreased miR-130a-5p was contributed to the pathological mechanism of MN through increasing [PLA2R:PROT] expression, which induced podocyte [apoptosis:LYSO].

prot

TP: 7

FP: 2

FN: 1

lyso

TP: 1

FP: 0

FN: 0

4. Rottlerin as a natural agent, which is isolated from Mallotus philippinensis, has been identified to play a critical role in tumor inhibition. However, the molecular mechanism of rottlerin-mediated anti -tumor activity is still ambiguous. It has been reported that [EZH2:PROT] exhibits oncogenic function s in a variety of human cancers. Therefore, inhibition of [EZH2:PROT] could be a promising strategy f or the treatment of human cancers. In this study, we aim to explore whether rottlerin could inhibit t umorigenesis via suppression of [EZH2:PROT] in prostate cancer cells. Multiple approaches such as F ACS, Transwell invasion assay, [RT:PROT]-[PCR:PROT], Western blotting, and transfection were perfo rmed to determine our aim. We found that rottlerin treatment led to inhibition of cell growth, migra tion and invasion, but induction of [apoptosis:LYSO] in prostate cancer cells. Importantly, we defined that rottlerin decreased the expression of [EZH2:PROT] and H3K27me3 in prostate cancer cells. Mor eover, overexpression of [EZH2:PROT] abrogated the rottlerin-induced inhibition of cell growth, migr ation, and invasion in prostate cancer cells. Consistently, down-regulation of [EZH2:PROT] enhanced rottlerin-triggered anti-tumor function. Collectively, our work demonstrated that rottlerin exerted its tumor suppressive function via inhibition of [EZH2:PROT] expression in prostate cancer cells. Our fin dings indicated that rottlerin might be a potential therapeutic compound for treating patients with p rostate cancer.

prot

TP: 3

FP: 0

FN: 4

lyso

TP: 7

FP: 0

FN: 3

6. Dendrites function as the primary sites for synaptic input and integration with impairments in dendritic arborization being associated with dysfunctional neuronal circuitry. Post-mitotic neurons require high levels of basal [autophagy:LYSO] to clear cytotoxic materials and autophagic dysfunction under native or cellular stress conditions has been linked to neuronal [cell death:LYSO] as well as axo-dendritic degeneration. However, relatively little is known regarding the developmental role of basal [autophagy:LYSO] in directing aspects of dendritic arborization or the mechanisms by which the autophagic machinery may be transcriptionally regulated to promote dendritic diversification. We demonstrate that [autophagy:LYSO]-related (Atg) genes are positively regulated by the [homeodomain transcription factor:PROT] Cut, and that basal [autophagy:LYSO] functions as a downstream effector pathway for Cut-mediated dendritic terminal branching in Drosophila multidendritic (md) sensory neurons. Further, loss of function analyses implicate Atg genes in promoting cell type-specific dendritic arborization and terminal branching, while gain of function studies suggest that excessive [autophagy:LYSO] leads to dramatic reductions in dendritic complexity. We demonstrate that the Atg1 initiator kinase interacts with the dual leucine zipper kinase ([DLK:PROT]) pathway by negatively regulating the [E3 ubiquitin ligase:PROT] Highwire and positively regulating the MAPKKK Wallenda. Finally, autophagic induction partially rescues dendritic

atrophy defects observed in a model of polyglutamine toxicity. Collectively, these studies implicate transcriptional control of basal [autophagy:LYSO] in directing dendritic terminal branching and demonstrate the importance of homeostatic control of autophagic levels for dendritic arbor complexity under native or cellular stress conditions.

prot

TP: 30

FP: 0

FN: 5

lyso

TP: 1

FP: 0

FN: 0

9. Mast cells and Kupffer cells secrete interleukin (IL)-1β, [interferon:PROT] ([IFN:PROT])-γ, and [tumor [necrosis:LYSO] factor:PROT] ([TNF:PROT])-α, which stimulate excess nitric oxide (NO) producing-inducible NO synthase ([iNOS:PROT]). Unlike Kupffer cells, [immunoglobulin E:PROT]-sensitized mast cells elicit sustained NO production. We investigated the participation of mast cell-released NO and cytokine-derived [iNOS:PROT] activation in type 1 allergy-suppressed hepatic cytochrome [P450:PROT] ([CYP:PROT]) metabolism. Aminoguanidine, a selective [iNOS:PROT] inhibitor, completely suppressed serum nitrate plus nitrite (NOx) concentrations after primary and secondary sensitization of ICR mice and markedly attenuated allergy-suppressed hepatic [CYP1A2:PROT], CYP2C, [CYP2E1:PROT], and CYP3A activities. In the liver, primary and secondary sensitization enhanced [iNOS:PROT]-stimulating [IFN:PROT]-γ (5-15-fold) and [TNF:PROT]-α (3-5-fold) mRNA levels more than [IL-1:PROT]β (2-fold) and [F4:PROT]/80-positive Kupffer cell (2-fold) mRNA levels. When mast cell-deficient (-/-) mice were sensitized, hepatic [CYP:PROT] activities were not suppressed. Serum NOx levels in the sensitized -/- mice were similar with those in saline-treated ICR and -/- mice. In the liver of -/- mice, secondary sensitization markedly enhanced mRNA expression of [iNOS:PROT] (20-fold), [IFN:PROT]-γ (15-fold), and [TNF:PROT]-α (3-fold). However, hepatic total [NOS:PROT] activities in -/- mice were not significantly different between saline treatment and sensitization. Similarly, primary and secondary ICR mice did not significantly enhance total [NOS:PROT] activities in the liver and hepatocytes. The total [NOS:PROT] activities observed did not relate to the high levels of [iNOS:PROT], [IFN:PROT]-γ, and [TNF:PROT]-α mRNA in the liver. Hepatic [c-kit:PROT]-positive mast cells in sensitized ICR mice were maintained at control levels. Therefore, our data suggest that mast cell-released NO participates in type 1 allergy-suppressed [CYP1A2:PROT], CYP2C, [CYP2E1:PROT], and CYP3A metabolism.