XIAP-AS1: Long non-coding mRNA

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Abstract

XIAP-AS1 is located primarily in the nucleus. IncRNA XIAP-AS1 can regulate apoptosis in gastric cancer cells and might serve as a potential oncogene for colon cancer. Sp1 is a responsible transcription factor for transcription of the XIAP gene. XIAP-AS1 RNA interacts with Sp1 and thereby participates in XIAP transcription. XIAP-AS1 knockdown decreases the binding of Sp1 to the promoter region of XIAP. XIAP-AS1 knockdown blocks cell invasion of colon cancer cells by regulating the expression of EMT markers, such as E-cadherin, ZO-1, vimentin, and N-cadherin. Moreover, XIAP-AS1 knockdown significantly reduces STAT3 phosphorylation.

Introduction

XIAP-AS1 interacts with Sp1 and is involved in XIAP transcription. In gastric cancer cells, XIAP-AS1 is a potential target for TRAIL-induced apoptosis. Cancer is a complex disease associated with a variety of genetic mutations, epigenetic alterations, chromosomal translocations, deletions, and amplification. IncRNA is a group of non-coding RNAs that is more than 200 base pairs, generally do not code for proteins, and is associated with diverse functions, such as patient outcome, cell proliferation, cell apoptosis, cell metastasis and invasion, cell cycle, epithelial-mesenchymal transition (EMT), cancer stem cells (CSCs) and drug resistance.

Keywords: XIAP-AS1; epithelial-mesenchymal; cancer stem cells

Analysis

High HOTAIR expression is correlated tightly with the presence of liver metastasis, and is demonstrated in gastric cancer. ZEB1-AS1 overexpression facilitates cell growth by promoting p21-activated kinases 2 (PAK2) expression by sponging miR-455-3p in colon adenocarcinoma cells. Apoptosis is a very tightly programmed cell death with a number of enzyme-dependent biochemical processes.

Tumor necrosis factor-related apoptosis-inducing ligand (TRAIL; also known as Apo 2 ligand) is a member of the tumor necrosis factor family of ligands that can initiate apoptosis by a pathway triggered by its interaction with death receptors, sometimes referred to as the extrinsic pathway.

Apoptosis can be triggered by signals from within the cell (intrinsic or mitochondrial pathway), or by extrinsic signals (extrinsic or death receptor pathway). Defect in apoptosis can cause cancer or autoimmunity. Caspases (cysteine-aspartic proteases) are proteolytic enzymes largely known for their role in controlling cell death and inflammation. These are subdivided into the initiators and the effectors based on the presence or absence of specific-protein interaction domains toward the N-terminus. Initiator caspases comprise death effector domains (DED; caspase-8 and -10) or caspase-recruitment domains (CARD; caspase-2, -9, -1 and -11), which mediate their dimerization and/or recruitment into larger complexes to facilitate their activation.

Conclusion

Negative regulation of caspases function is achieved by IAP proteins family, they regulate both the intrinsic and extrinsic pathways. Two typical prosurvival NF-κB targets are Bcl-xL and XIAP, which can block apoptosis at multiple steps. Disrupted mitochondria also produce second mitochondria-derived activator of caspase (SMAC; also known as DIABLO), which releases caspase 3 from X-linked inhibitor of apoptosis (XIAP) mediated inhibition. XIAP is a direct inhibitor of caspase-3 and caspase-9 and modulates the Bax/cytochrome c pathway by inhibiting caspase-9. Down-regulation of XIAP is an important mechanism for caspase activation in response to various apoptotic stimuli.

Reference


