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MINIREVIEWS

Recent advancements in understanding of biological role of homeobox C9 in human cancers

Yong Zhang, Jing Li

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Abstract

Homeobox (HOX) C9, a member of the HOX family, is an important transcription factor, and it plays a significant role in various biological processes. This family of genes is highly valued for their essential roles in establishing and maintaining the body axis during embryonic development and adult tissues. Further, HOXC9 plays a central role in neuronal differentiation, angiogenesis, and adipose distribution, which are essential for the development of the nervous system, maturation of tissues and organs, and maintenance of energy balance and metabolic health. Recent research has found that abnormal HOXC9 expression is closely associated with the development and progression of various tumor types. The HOXC9 expression level can be an indicator of tumor prognosis. Therefore, elucidating the association between HOXC9 expression and its regulatory mechanisms and tumorigenesis can provide novel insights on the diagnosis and treatment of patients with cancer.

Key Words: Homeobox C9; Proliferation; Invasion; Malignant tumor; Molecular mechanism

Core Tip: This paper discusses the association between abnormal homeobox (HOX) C9 expression and the occurrence and progression of various tumors, the impact of HOXC9 on the clinical pathological characteristics and prognosis of cancer patients, the role of HOXC9 in central nervous system tumors, breast cancer, bladder cancer, gastric cancer, colon cancer, non-small cell lung cancer, and thyroid papillary carcinoma, and the molecular mechanisms underlying the biological functions of HOXC9, including its interaction with other proteins and its regulation through DNA methylation.

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INTRODUCTION

The homeobox (HOX) genes were first discovered in *Drosophila melanogaster*, where they are referred to as the HOM-C genes and are part of the Hox gene family[1-6]. In humans, the Hox genes are categorized into two subfamilies. The first subfamily, referred to as the anterior-posterior (A-P) type, is clustered in chromosomes and expressed in an A-P manner. The second subfamily comprises the non-A-P-type HOX genes, which are dispersed across different chromosomes. Based on sequence similarity, these genes form distinct groups including the Emx, Pax, Msx, and Otx families of the HOX genes [1].

Structurally, the HOX genes form a substantial part of the HOX gene family. These genes have a common 180-bp sequence referred to as the HOX, which encodes a conserved 60 amino acid region known as the homeodomain[7,8]. Functionally, proteins derived from the HOX genes can be transcription factors that, together with upstream signaling molecules and downstream target genes, contribute to complex regulatory networks *via* positive and negative feedback loops. Moreover, these proteins can interact with other transcription factors, playing roles in morphogenesis, cell adhesion and migration, and cell cycle regulation[9].

Previous studies have revealed that HOXC9 is a pivotal transcription factor in embryonic development. That is, it plays roles in essential biological processes such as cell cycle, differentiation, and apoptosis[10-14]. Some studies have revealed dysregulation in HOXC9 expression in various malignant tumors. Thus, HOXC9 has both oncogenic and tumor-suppressive effects[14-19]. Further, its expression is closely associated with the clinicopathological characteristics and prognosis of patients with cancer.

ASSOCIATION BETWEEN HOXC9 AND CENTRAL NERVOUS SYSTEM TUMORS

Neuroblastoma (NB) is an embryonic tumor of the sympathetic nervous system that originates from primitive neural crest cells and develops in infants and young children[20-24]. The inconspicuous onset and rapid progression of the tumor often lead to unsatisfactory clinical treatment outcomes, resulting in a poor prognosis in children with NB[25-28]. In addition, NB cells can differentiate to a sympathetic ganglion cell phenotype, indicating a disruption in the physiological molecular program governing neuroblast differentiation and growth control. In advanced-stage NBs, Mao *et al*[14] reported reduced HOXC9 expression, which is involved in cell cycle control and NB cell differentiation[14].

The differentiation state has a significant impact on the clinical outcomes of NB, with induced differentiation being utilized as a therapeutic method. An elevated HOXC9 expression level is associated with NB differentiation and thus a promising prognosis[19,29-33]. Growth arrest and neuronal differentiation are enhanced by HOXC9 by regulating genes associated with cell cycle progression and neuronal differentiation. Retinoic acid (RA) upregulates HOXC9 expression, and its downregulation results in resistance to RA-induced growth arrest and differentiation[34]. In addition, HOXC9 expression is epigenetically silenced in RA-resistant cells, and its overexpression inhibits cell proliferation and tumorigenesis[34]. HOXC9 expression was significantly decreased in the RA-resistant NB cell line SK-N-AS. Conversely, arsenic trioxide led to an increase in the quantity of neuronal synapses in SK-N-AS cells, thereby upregulating HOXC9 and HOXD8 levels while simultaneously downregulating PHOX2B and EZH2 levels[34].

Glioblastoma multiforme (GM), an exceedingly malignant primary neoplasm of the brain, is associated with a poor prognosis characterized by a 5-year survival rate of 5.8%[35-40]. High expression of HOXC9 in GM is a predictor of poor prognosis. Meanwhile, silencing HOXC9 inhibits the growth and invasion of GM cells. Mechanistically, HOXC9 directly inhibits *DAPK1* gene transcription, activates the DAPK1/Beclin1 signaling pathway, and suppresses autophagy[18].

ASSOCIATION BETWEEN HOXC9 AND BREAST CANCER

Breast cancer (BRCA) accounted for 24.2% of all female cancer cases in 2018. Therefore, it is an evident malignant neoplasm on a global scale[41-45]. Despite notable advancements in the diagnosis and management of patients aimed at improving both their quality of life and overall survival (OS), there are persistent obstacles in the form of tumor relapse

and distant spread of cancer cells, which ultimately lead to an increased mortality rate[46-48]. Hur *et al*[15] investigated differences in HOXC9 expression between BRCA and normal tissues by utilizing publicly available databases. They evaluated the association between HOXC9 Levels and both disease- and distant metastasis-free survival in patients with BRCA[15]. The results indicated increased HOXC9 expression in BRCA tissues, which was associated with a negative prognosis in patients with lymph node metastasis. Subsequent *in vitro* studies revealed that the upregulation of HOXC9 in BT474 and MCF7 BRCA cell lines enhanced cell invasion while inhibiting cell proliferation. This suggests that HOXC9 has a potential role in driving the phenotypic transition of BRCA cells from a proliferative to an invasive state.

ASSOCIATION BETWEEN HOXC9 AND BLADDER CANCER

Bladder cancer (BLCA), a prevalent urinary system malignancy, is the fifth most common cancer among men in developed countries[49-51]. Surgical intervention can effectively treat BLCA in its initial stages. Nevertheless, individuals diagnosed with late-stage BLCA who are not suitable candidates for surgery have a worse prognosis[52-56]. Moreover, chemoresistance poses a substantial challenge for cancer researchers and clinicians, contributing to chemotherapy failure in patients with advanced-stage BLCA[57-60]. miR-193a-3p significantly contributes to promoting multiple chemoresistance in BLCA. Lv *et al*[61] showed that the *HOXC9* gene is targeted directly by miR-193a-3p. The suppression of HOXC9 expression can possibly trigger the activation of DNA damage response and oxidative stress signaling pathways, thereby promoting resistance in cancer cells against chemotherapy[61].

ASSOCIATION BETWEEN HOXC9 AND GASTRIC CANCER

Gastric cancer (GC), a common malignant neoplasm of the gastrointestinal system, is associated with factors including *Helicobacter pylori* infection, gastroesophageal reflux disease, and Barrett's esophagus, which can increase susceptibility [41,62-65]. Diagnosing early-stage GC poses a significant challenge, frequently leading to delayed-stage diagnoses and an unfavorable prognosis. Previous studies have revealed that HOXC9 is overexpressed in GC tissues, thereby inhibiting immune response and the interferon gamma signaling pathway[66]. Mechanically, it elicits resistance to interferon gamma in GC cells by inhibiting DAPK1 and RIG1 expression, leading to the establishment of a tumor microenvironment characterized by immunological unresponsiveness. In addition, HOXC9 downregulation might be a potential indicator for the efficacy of programmed cell death protein 1 blockade in treating patients with GC.

In GC, HOXC9 expression is significantly correlated with the tumor metastasis ability and stem cell-like characteristics [17]. Previous studies have shown that HOXC9 can be a direct target of miR-26a[17]. The downregulation of miR-26a expression leads to the upregulation of HOXC9. Meanwhile, the restoration of miR-26a expression not only downregulates HOXC9 but also reverses the promoting effects of HOXC9 on metastasis and the stem cell-like phenotypes of GC cells.

ASSOCIATION BETWEEN HOXC9 AND COLON CANCER

Epidemiological investigations have revealed an increasing incidence of colorectal carcinoma in elderly individuals, particularly those aged ≥ 50 years[67-70]. The various risk factors associated with colon cancer include colon polyps, chronic colitis, and obesity[71]. The overexpression of HOXC9 in colon cancer tissues is strongly correlated with unfavorable clinical characteristics such as tumor-node-metastasis stage, distant metastasis, and venous invasion. Kaplan-Meier curve analysis showed that elevated HOXC9 levels are indicative of a worse OS and progression-free survival[72]. Further, gene set enrichment analysis revealed that HOXC9 overexpression in colon cancer significantly enriches pathways related to natural killer cell-mediated cytotoxicity, cell adhesion molecules, innate immune system, and interactions involving cytokine receptors.

ASSOCIATION BETWEEN HOXC9 AND NON-SMALL-CELL LUNG CANCER

Lung cancer is still a prominent contributor to cancer-related mortality worldwide, and non-small-cell lung cancer (NSCLC) accounts for approximately 85% of the cases[73-76]. Notably, 75% of diagnoses are made during advanced stages, consequently leading to a reduced 5-year survival rate[77,78]. In lung adenocarcinoma, HOXC9 overexpression is significantly correlated with OS and disease-free survival[79]. Bi *et al*[80] revealed that the circular RNA Hsa_circ_0020123 enhances the development of NSCLC *via* its function as a sponge for miR-495. In addition, upregulation of HOXC9 negates the suppressive effects on NSCLC cell proliferation and movement caused by miR-495[80]. Further, the methylation frequency of the *HOXC9* gene is significantly higher in stage I NSCLC tissues than in adjacent normal tissues [81].

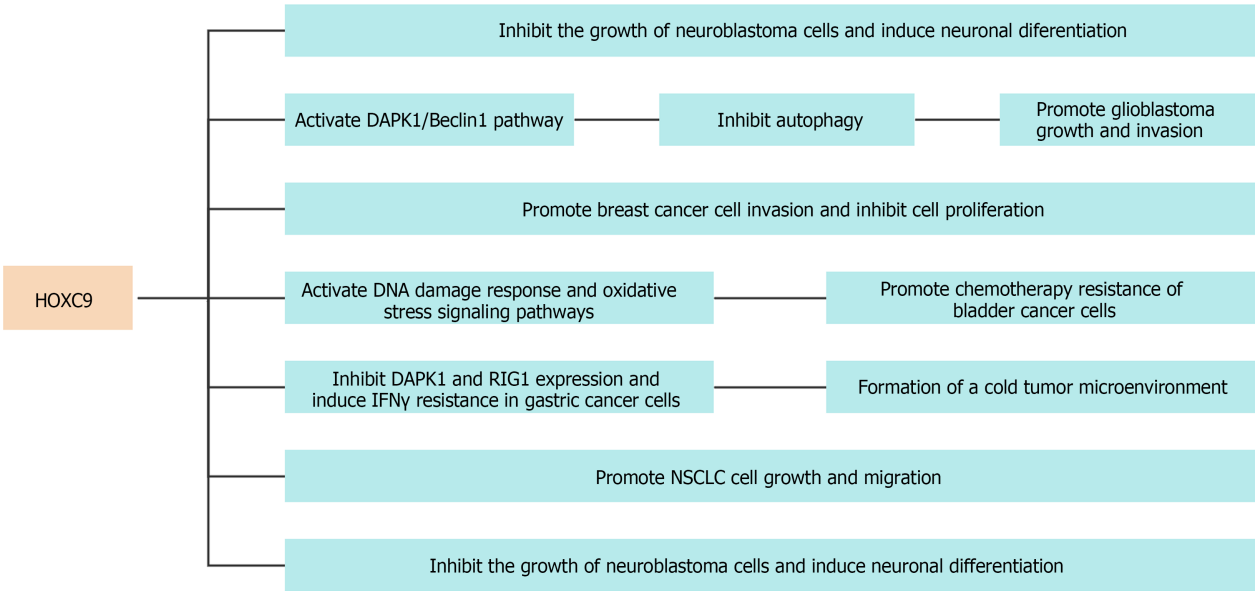

Figure 1 Biological role of homeobox C9 in human cancer. NSCLC: Non-small-cell lung cancer.

ASSOCIATION BETWEEN HOXC9 AND THYROID PAPILLARY CARCINOMA

Thyroid papillary carcinoma (TPC) is a relatively prevalent type of thyroid malignancy, predominantly affecting women aged 30-45 years[82-85]. The clinical remission rate is significantly high, which is associated with a favorable prognosis [86]. Previous studies have revealed a decrease in HOXC9 expression levels within TPC, exhibiting considerable variations in expression compared with that in adjacent normal tissues. Moreover, HOXC9 downregulation is associated with Hashimoto's thyroiditis and lymph node metastasis, thereby indicating its potential utility as a diagnostic and prognostic indicator of TPC[87].

MOLECULAR MECHANISMS UNDERLYING BIOLOGICAL FUNCTIONS OF THE *HOXC9* GENE

HOXC9, a pivotal embryonic development gene, effectively participates in different morphogenetic mechanisms including neuronal differentiation, vascular growth, and adipose tissue distribution[11-14] (Figure 1). Wang *et al*[10] performed a comprehensive analysis of the genome-wide impact of HOXC9 on neuronal differentiation[10], and identified an intricate network involving the transcriptional regulation of 2370 genes primarily associated with neuronal differentiation, cell cycle progression, and DNA damage response. Notably, HOXC9 was found to interact with the transcriptional repressor E2F6, thereby orchestrating its recruitment to cell cycle gene promoters to repress their expression. Figure 2 shows the protein-protein interaction network of HOXC9 based on data from the String database[88].

Previous studies have revealed the essential role of HOXC9 in regulating vascular morphology. HOXC9 overexpression or interleukin-8 knockout in zebrafish resulted in aberrant vascular development, marked by the loss or incompleteness of the dorsal aorta and intersegmental vessels[13]. This emphasizes the significance of HOXC9 as a transcriptional factor that promotes the quiescence of endothelial cells while suppressing angiogenesis, thereby influencing vascular morphogenesis *via* an interleukin-8-mediated process[13]. Moreover, HOXC9 plays an important role as a stimulator of Stab2, collectively contributing to the development of the thoracic duct. *In vitro* studies have validated the regulatory influence of Stab2 on endothelial cell movement and angiogenesis without affecting cell death.

Brune *et al*[89] performed an examination of *HOXC9* mRNA expression in abdominal subcutaneous and omental fat tissues collected from 636 individuals. The results showed significantly elevated *HOXC9* mRNA expression in the subcutaneous fat tissues compared with that in the omental fat tissues[89]. Moreover, in the subcutaneous adipose tissues, *HOXC9* mRNA expression was significantly negatively correlated with adipocyte volume, body mass, and fasting plasma insulin levels. Further studies showed that the *HOXC9* expression in the cells of the stromal vascular fraction was higher than that in adipocytes. Thus, *HOXC9* is an important developmental gene that regulates fat distribution.

DNA methylation is a common epigenetic modification that is typically associated with the suppression of gene expression[90-92]. Lin *et al*[81] revealed that the *HOXC9* gene is more frequently methylated in stage I NSCLC tissues than in adjacent normal tissues[81]. The methylation status of *HOXC9* is correlated with specific types of cancer and clinical characteristics. Thus, it can be a biomarker for the diagnosis, prognosis, and prediction of response to immunotherapy. Therefore, the association between HOXC9 and methylation emphasizes the significant role of epigenetic regulation in gene expression, cell development, and disease development.

In addition, the biological effects of mutations in the *HOXC9* gene are based on the type of mutation and its location within the gene. Suemori *et al*[93] presented mutations at the *HOXC9* gene locus in mice using gene targeting techniques, with an aim to elucidate the function of the *HOXC9* gene[93]. The results revealed that the *HOXC9* gene determines the

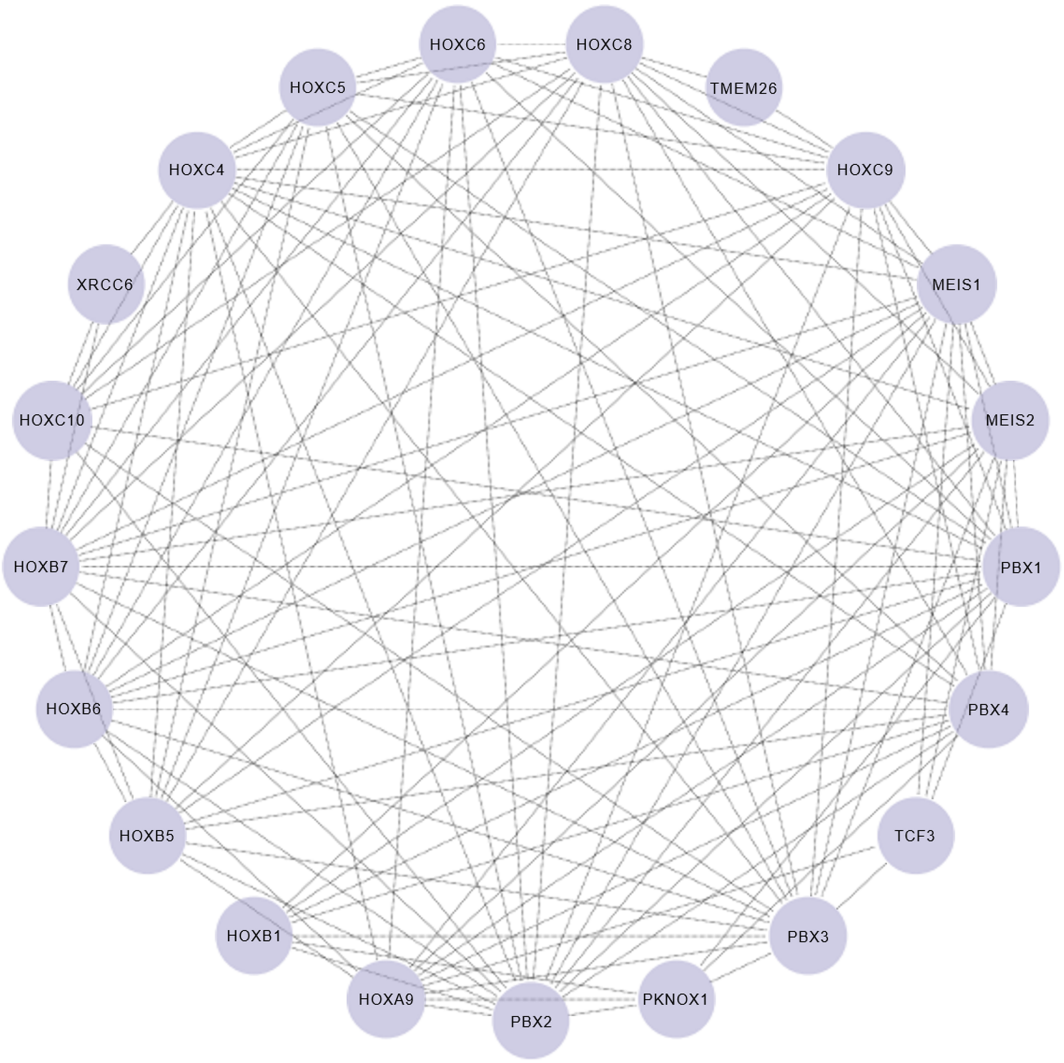

Figure 2 Protein–protein interaction network of homeobox C9 based on data from the String database.

anterior-posterior axis of the body by regulating the formation of various body parts. Further, the *HOXC9* gene can function by repressing *HOXC8* gene expression. This cross-regulatory mechanism is conserved among multiple species and is significantly important in understanding the establishment of the body axis in vertebrates. From a disease perspective, mutations in the *HOXC9* gene are associated with various developmental abnormalities and diseases, including congenital malformations and developmental delays. Therefore, understanding the normal function of the *HOXC9* gene and the effects of its mutations is important for not only facilitating fundamental biological research but also identifying potential therapeutic methods and intervention strategies.

CONCLUSION

The *HOXC9* gene plays an important role in embryonic development and cell differentiation, with recent research emphasizing its dual roles across various tumors. In NB, HOXC9 inhibits the cell cycle and promotes neuronal differentiation, and its elevated expression is significantly correlated with a better prognosis. Conversely, in BRCA, TPC, BLCA, GC, NSCLC, GM, and colon cancer, it functions as an oncogenic factor, and HOXC9 overexpression is significantly associated with a worse prognosis. In addition, DNA methylation is closely related to HOXC9 expression and can be a biomarker for the early diagnosis and prognosis of NSCLC. Elucidating the molecular mechanism of the *HOXC9* gene in malignant tumors may provide a novel reference for targeted therapy in patients with malignant tumors.

FOOTNOTES

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