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Zinc Finger Proteins in Gastric, Pancreatic, and Prostate Cancers: Molecular Functions, Biomarker Potential, and Therapeutic Opportunities

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ABSTRACT

Deciphering the molecular mechanisms that drive cancer progression is essential for the development of effective diagnostics and therapeutics. In this study, we systematically examine the structural and functional attributes of Zinc Finger Proteins (ZFPs) and delineate their mechanistic roles in gastric, pancreatic, and prostate cancers, with a particular focus on processes such as epithelial-mesenchymal transition (EMT), DNA damage response (DDR), stemness, and therapeutic resistance. As transcriptional regulators, ZFPs participate in critical cellular processes, including gene expression control, cell proliferation, differentiation, apoptosis, EMT, metastasis, and chemotherapy resistance. By evaluating these roles, the study explores the potential of ZFPs as diagnostic, prognostic, and therapeutic targets in cancers such as gastric, pancreatic, and prostate cancer. ZFPs contribute to key cellular functions, including transcriptional regulation, chromatin remodeling, and cellular differentiation. In oncogenic contexts, they modulate mechanisms like cell cycle control, EMT, metastasis, suppression of apoptosis, inflammation, and drug resistance. These diverse functions highlight ZFPs as promising molecular targets for advancing cancer diagnostics and therapeutics.

Keywords: Apoptosis, Biomarker, Cancer, Transcription factors, Zinc Finger Biology (ZFPs)

Gastrik, Pankreas ve Prostat Kanserlerinde Çinko Parmak Proteinleri: Moleküler İşlevleri, Biyobelirteç Potansiyeli ve Terapötik Olanakları

ÖZ

Kanser ilerlemesini yönlendiren moleküler mekanizmaların çözülmesi, etkili tanı ve tedavi stratejilerinin geliştirilmesi için hayati öneme sahiptir. Bu çalışmada, Çinko Parmak Proteinlerinin (ZFP'ler) yapısal ve fonksiyonel özellikleri sistematik olarak incelenmekte ve mide, pankreas ile prostat kanserlerindeki mekanistik rolleri, özellikle epiteliyal-mezenkimal geçiş (EMT), DNA hasar yanıtı (DDR), kök hücresel özellikler ve terapötik direnç süreçleri bağlamında açıklanmaktadır. ZFP'ler, transkripsiyonel düzenleyiciler olarak gen ekspresyonunun kontrolü, hücre çoğalması, farklılaşma, apoptoz, EMT, metastaz ve kemoterapi direnci gibi kritik hücresel süreçlerde görev almaktadır. Bu rollerin değerlendirilmesiyle, gastrik, pankreas ve prostat kanseri gibi bazı kanser türlerinde ZFP'lerin tanı, prognoz ve tedavi açısından potansiyel hedefler olarak taşıdığı önemin ortaya konması hedeflenmektedir. ZFP'ler transkripsiyonel düzenleme, kromatin yeniden yapılandırılması ve hücresel farklılaşma gibi temel hücresel işlevlere katkıda bulunurlar. Onkojenik bağlamda ise, hücre döngüsü kontrolü, EMT, metastaz, apoptozun baskılanması, inflamasyon ve ilaç direnci gibi mekanizmaları düzenlerler. Bu çeşitli işlevler, ZFP'leri kanser tanı ve tedavisini geliştirmede umut vadeden moleküler hedefler olarak öne çıkarmaktadır.

Anahtar Kelimeler: Apoptoz, Biyobelirteç, Kanser, Transkripsiyon Faktörleri, Çinko Parmak Proteinleri (ZFP'ler)

INTRODUCTION

Globally, cancer ranks as the second leading cause of death, with projections showing a considerable rise in its health burden soon. A large majority of cancer fatalities arise once the illness progresses to the metastatic phase (Kiri and Ryba, 2024). Globally, sex and geographic location have a significant impact on the incidence and death of cancer, according to GLOBOCAN 2020. Prostate cancer is the most frequently diagnosed cancer in men, followed by lung, colorectal, and liver cancers. Liver and prostate cancers are the next most common causes of cancer-related mortality in men, behind lung cancer. The two most common cancer forms in women are breast and cervical cancers. Although the most common causes of cancer-related fatalities in women are lung, breast, and cervical cancers, there is more variation in the overall

distribution of cancer-related deaths in women (Sung et al., 2021; GLOBOCAN, 2022).

Zinc finger proteins (ZFPs) form a diverse group within the transcription factor superfamily and are distinguished by the inclusion of at least one zinc finger (ZnF) domain in their structure. These proteins participate in various molecular interactions involving DNA, RNA, proteins, lipids, and poly-ADP-ribose, primarily functioning to modulate gene expression through DNA binding. Structural motifs like zinc finger domains and SCAN domains enable ZFPs to associate with a range of biomolecules, including RNA, membranes, lipids, and other proteins (Ngwa et al., 2021; Liu et al., 2022). Numerous essential cellular functions, such as transcriptional regulation, chromatin remodeling, protein homeostasis, signal transduction, and cellular proliferation and differentiation, are mediated by this protein family, of which more than 30 distinct types have been identified to date (Cassandri et al., 2017; Singh and Van Attikum, 2021). Furthermore, ZFPs are essential for a number of biological processes, including immune response, apoptosis, autophagy, stem cell maintenance, and cell differentiation, because of their capacity to identify particular DNA sequences (Liu et al., 2022). The ZnF motif, composed of approximately 30 amino acids, contains cysteine (C) and histidine (H) residues at specific positions. These amino acids tightly coordinate a central zinc ion, forming a characteristic three-dimensional finger-like structure within the protein chain (Ngwa et al., 2021). Zinc finger motifs were first identified in 1988 in the Xenopus oocyte transcription factor IIIA. Approximately 2% of human genes encode proteins that contain zinc finger domains, positioning this family as the most extensive group of specific transcription factors. Zinc finger domains that interact with zinc ions are responsible for maintaining the functionality and stability of the protein structure through autonomous folding (Liu et al., 2022; An et al., 2022). The ZFP family is categorized into 30 types and further subdivided into eight subclasses based on the structural characteristics of their motifs. Among these, the most common DNA-binding domain is the Cys_2His_2 (C_2H_2) type. Structural diversity supports the functional versatility of ZFPs. For instance, SET domains catalyze protein methylation, while BTB domains facilitate the binding of corepressors in the repression of target gene transcription. In this context, structural diversity enables ZFPs to perform diverse roles in various cellular processes (Del Rizzo et al., 2011; Jen et al., 2016; Liu et al., 2022). Zinc finger proteins are crucial regulators of transcription involved in diverse biological processes such as cellular differentiation and proliferation, notably contributing to muscle cell and keratinocyte maturation as well as the regulation of cancer stem cell populations. Recent studies have revealed that disruptions in the expression of these proteins are not limited to tumorigenesis and progression but may also play roles in the development of systemic pathologies such as diabetes, dermatological disorders, and neurodegenerative diseases. From the standpoint of oncology, ZFPs regulate important processes like the epithelial-to-mesenchymal transition (EMT), the cell growth cycle, the acquisition of invasive characteristics, the enhancement of metastatic potential, the suppression of mechanisms that cause programmed cell death, the reprogramming of inflammatory responses, the acquisition of invasive traits, and chemotherapy resistance (Hedayat-Evrigh et al., 2020; Liu et al., 2022). Interestingly, ZFPs significantly alter DNA methylation in cancer stem cells and a variety of malignancies, such as hepatocellular, colorectal, breast, gastric, and lung cancers (Rhie et al., 2018). ZFPs are intriguing candidates for diagnostic, prognostic, and therapeutic targets because of their diverse activities, especially in cancers such as breast, colorectal, liver, lung, gastric, and other cancers (Liu et al., 2022).

THE ROLE OF ZINC FINGER PROTEINS IN GASTRIC CANCER: DIAGNOSIS, PROGNOSIS, AND THERAPEUTIC POTENTIAL

Diagnosis and Prognosis of Gastric Cancer

According to global cancer statistics from 2020 released by the World Cancer Research Fund (WCRF), gastric cancer (GC) is the second greatest cause of cancer-related death and the fifth most frequent cancer globally (Qu et al., 2013; Li et al., 2014; Cao et al., 2018; Liu et al., 2023). It is more commonly seen in China, Japan, and South Korea (Li et al., 2014). In Japan, it is the most prevalent cancer among men, and in China, the number of newly diagnosed cases continues to surpass those in other countries. However, since World War II, the global incidence of gastric cancer has been declining. It is also known to be one of the least common cancers in North America (Ajani et al., 2010). Risk factors contributing to the development of gastric cancer include *Helicobacter pylori* (*H. pylori*) infection, smoking, alcohol consumption, excessive salt intake, aging, genetic mutations, and environmental influences, which play crucial roles in gastric cancer carcinogenesis and progression (Song et al., 2017; Smyth et al., 2020). Treatment options for patients include surgery, chemotherapy, radiotherapy, and neoadjuvant therapy. Despite advances in neoadjuvant therapy, early diagnosis rates remain low, with more than 70% of patients diagnosed at advanced stages. Surgical treatment is considered the only curative approach for gastric cancer; however, due to the absence of early symptoms and effective screening methods, most diagnoses occur at unresectable stages, making chemotherapy the preferred option. Therefore, it has become essential to improve detection methods and identify effective molecular targets for early screening of gastric cancer (Song et al., 2017; Tan, 2019; Huang et al., 2022; Zhu et al., 2022). The mechanisms of gastric cancer development remain unclear due to the complexity and multi-step nature of its pathogenesis (Li et al., 2014). Gain-of-function mutations in oncogenes and loss-of-function mutations in tumor suppressor genes are two examples of the genetic and epigenetic

changes involved in gastric carcinogenesis. Gastric cancer has been linked to genetic changes in p53, KRAS, PIK3CA, ARID1A, MLL3, and MLL, as well as gene amplifications in PIK3CA, C-MET, ERBB4, and CD44, suggesting their possible roles in carcinogenesis (Qu et al., 2013). *Helicobacter pylori* is classified as a class I carcinogen and is one of the most well-established risk factors for gastric cancer (Shi et al., 2017; Cao et al., 2024). Atrophic gastritis, intestinal metaplasia, and ultimately cancer are caused by a persistent *H. pylori* infection in the stomach mucosa. Additionally, it is linked to epigenetic and genetic processes that inhibit tumor suppressor genes and activate oncogenes (Cao et al., 2024). Although *H. pylori* infection is required, it is not enough on its own to cause stomach cancer. Only around 1% of *H. pylori* infections result in stomach cancer, even though the infection incidence is thought to be between 40 and 80% worldwide. Despite having equal environmental exposures, people with diverse genetic backgrounds may exhibit varied cancer susceptibilities. Changes in DNA, mRNA, and protein expression that are controlled by *H. pylori* control the migration, invasion, apoptosis, and proliferation of gastric cancer cells, hence having therapeutic effects on the disease's progression (Zhang et al., 2019).

Zinc Finger Protein-64 (ZFP64): A Transcriptional Regulator of Immune Response and Tumor Progression

Zinc finger proteins (ZFPs) represent a broad group of proteins that are extensively encoded throughout the human genome. They are involved in numerous biological processes such as controlling gene expression, contributing to the progression of gastric cancer, and mediating programmed cell death (Yu et al., 2013; Hao et al., 2015; Liu et al., 2023). The gene encoding ZFP64, alternatively named ZNF338, is positioned at the 20q13.2 locus and produces a protein that is classified within the C2H2-type zinc finger proteins, specifically within the Kruppel-like subgroup (Zhu et al., 2022; Sun et al., 2024; Huang et al., 2025). Initial studies identified ZFP64 as a co-activator of Notch1. It interacts with the Notch intracellular domain (NICD) and is involved in regulating Notch1 signaling. ZFP64 generally acts as a tumor-promoting factor. It is significantly upregulated in various human cancers, including gastric cancer, hepatocellular carcinoma (HCC), and esophageal cancer (He et al., 2023).

In lung adenocarcinoma, it supports cell viability, cell cycle progression, and migration. ZFP64 serves as a transcriptional regulator of GAL-1 and facilitates stem-like properties and immune suppression in gastric cancer (Qiu and Deng, 2022; Sun et al., 2024). It has also been shown to bind to the MLL promoter in leukemia, sustaining expression of the mixed lineage leukemia gene (Lu et al., 2018).

ZFP64 elevates programmed death-1 (PD-1) and cytotoxic T-lymphocyte antigen 4 (CTLA-4) levels, promoting esophageal cancer progression, and is significantly upregulated in hepatocellular carcinoma cases with vessel invasion, serving as a predictor of poor clinical outcome (Qiu and Deng, 2022; Wang et al., 2015).

Zinc Finger and BTB (Broad-Complex, Tramtrack, Bric-à-brac) Domain-Containing Protein: Regulator of Cell Growth and Apoptosis

Zinc finger and BTB domain-containing protein 20 (ZBTB20), located at chromosome region 3q13.31, is a transcription factor first discovered in human dendritic cells. This protein consists of 741 amino acids and features an N-terminal BTB domain along with one or more zinc finger domains at its C-terminus, responsible for DNA binding (Bai and Xiao, 2020; Liu and Zhang, 2024; Duan et al., 2024). Immune responses, tumor formation, and the production of α-fetoprotein in the adult liver are all significantly regulated by ZBTB20. ZBTB20 controls the invasion, migration, and proliferation of gastric cancer cells, according to studies (Bai and Xiao, 2020). It is a crucial transcriptional repressor in many physiological and pathological processes (Duan et al., 2024). Additionally, ZBTB20 stimulates NF-κB activation and hepatocyte proliferation during liver regeneration after partial hepatectomy (Zhang et al., 2019).

Studies on the Role and Clinical Relevance of Zinc Finger Proteins in Gastric Cancer in the Literature

Yuan et al. (2024) investigated the role of the long non-coding RNA FIRRE in driving malignancy and treatment resistance in gastric cancer. Using RNA sequencing, RNA immunoprecipitation, chromatin immunoprecipitation, and dual-luciferase reporter assays, they identified critical transcriptional regulators and downstream genetic targets influenced by FIRRE. Elevated FIRRE expression in gastric cancer was shown to promote tumor cell proliferation, motility, and invasiveness in both in vitro and in vivo models, correlating with poor clinical outcomes. Mechanistically, FIRRE was found to activate the promoter of the **TUBB3** gene, thereby enhancing its expression and contributing to tumor progression. Notably, ZFP64, acting as a transcription factor for TUBB3, binds exclusively to FIRRE. Through this interaction, FIRRE facilitates TUBB3 promoter activation and promotes cancer progression, a process that is disrupted when ZFP64 levels are reduced, demonstrating the critical role of the FIRRE–ZFP64–TUBB3 axis in gastric tumor growth.

Cao et al. (2018) showed that in 15 gastric cancer cell lines, promoter hypermethylation resulted in the silencing of ZNF471 mRNA expression. When comparing primary gastric cancer tissues to nearby normal tissues, they discovered significantly increased ZNF471 promoter methylation ($P<0.001$). In 120 individuals with gastric cancer, ZNF471 hypermethylation in the promoter CpG region was linked to a poor prognosis ($P=0.001$). ZNF471 ectopic expression in AGS, BGC823, and MKN74 cell lines

markedly reduced invasion, migration, and proliferation *in vitro*. It also caused apoptosis and prevented the development of xenograft tumors in nude mice. ZNF471 directly targets the TFAP2A and PLS3 genes, binding to their promoters to suppress their expression, as indicated by bioinformatics modeling and ChIP-PCR investigations. The KAP1 protein is drawn to the promoters by ZNF471, which enriches H3K9me3 histone modifications and further inhibits TFAP2A and PLS3. These results imply that ZNF471 suppresses tumors in gastric cancer, and KAP1 facilitates this suppressive action. As a result, in gastric cancer, ZNF471 promoter CpG hypermethylation is regarded as a separate prognostic indicator.

Bai and Xiao (2020) sought to ascertain if ZBTB20 single-nucleotide polymorphisms (SNPs) and the incidence of stomach cancer in Han Chinese individuals were related. In a case-control study, 507 healthy people and 509 patients with stomach cancer participated. Four SNPs were chosen from the ZBTB20 gene: rs9841504 G/C, rs9288999 G/A, 10934270 T/C, and rs73230612 C/T. The relationship between these SNPs and the risk of stomach cancer under different genetic models was examined using logistic regression. SNP-SNP interactions were investigated using multifactor dimensionality reduction (MDR), and variations in clinical traits between genotypes were assessed using univariate analysis and ANOVA. The research findings indicate that the ZBTB20 polymorphism rs9288999 exerts a protective effect against gastric cancer across multiple genetic models, with the most pronounced impact observed in individuals with the homozygous genotype (OR = 0.48, P = 0.0003). This variant also demonstrated a significant inverse association with cancer susceptibility in subgroups defined by tumor histology (adenocarcinoma), age, gender, body mass index, and lifestyle factors such as tobacco and alcohol use. Additionally, the rs9841504 and rs73230612 polymorphisms showed strong correlations with specific clinical biomarkers, namely platelet count and CA242 levels. Notably, the rs9841504 allele was linked to a higher gastric cancer risk in individuals with a BMI exceeding 24 kg/m². Collectively, these results suggest that rs9288999 in the ZBTB20 gene may contribute to reduced gastric cancer susceptibility within the Han Chinese population.

Zhu et al. (2022) aimed to elucidate the underlying mechanisms of gastric cancer resistance to albumin-bound paclitaxel (nab-paclitaxel) nanoparticles. Through RNA sequencing (RNA-seq) of samples from patients exhibiting either sensitivity or resistance to nab-paclitaxel, they identified zinc finger protein-64 (ZFP64) as a key mediator of drug resistance. The in vitro functional roles of ZFP64 were evaluated using sphere formation assays, TUNEL labeling, flow cytometry, and CCK8 assays, while in vivo effects were assessed via subcutaneous tumor models in both humanized and immunodeficient mice. Mechanistic insights were further elucidated using dual-luciferase reporter assays and chromatin immunoprecipitation sequencing (ChIP-seq). Their findings demonstrated that ZFP64 overexpression constitutes an independent prognostic factor in gastric cancer, correlating with aggressive tumor phenotypes and nab-paclitaxel resistance. Mechanistically, ZFP64 binds directly to the promoter region of Galectin-1 (GAL-1), enhancing its transcription. This activation promotes cancer cells with stem cell-like properties and establishes an immunosuppressive tumor microenvironment. Notably, the combination of nab-paclitaxel with GAL-1 inhibition markedly improved anti-tumor efficacy in preclinical models, particularly in humanized mice. Collectively, these results underscore the critical role of ZFP64 in driving gastric cancer progression by simultaneously promoting chemoresistance and immune evasion, suggesting that patients with specific gastric cancer profiles may benefit from combined nab-paclitaxel and GAL-1-targeted therapies.

THE ROLE OF ZINC FINGER PROTEINS IN PANCREATIC CANCER: DIAGNOSIS, PROGNOSIS, AND THERAPEUTIC POTENTIAL

Diagnosis and Prognosis of Pancreatic Cancer

The pancreas is a sponge-like, tube-shaped organ approximately 15 cm long, located in the upper abdominal region between the stomach and the spine. A healthy pancreas is composed of acinar cells that secrete digestive enzymes, ductal cells that produce bicarbonate, centroacinar cells that act as intermediates between the two, endocrine islets that synthesize hormones, and typically inactive pancreatic stellate cells. Pancreatic cancer is characterized by the uncontrolled proliferation of pancreatic cells due to DNA mutations at the cellular level, ultimately leading to the formation of a malignant tumor. Clinically, pancreatic cancer is considered one of the malignancies with the poorest prognosis and is known for its high mortality rates (Hu et al., 2021). Long non-coding RNAs (lncRNAs) are recognized as key players in the onset and advancement of cancer, contributing to poor clinical outcomes. Nevertheless, the roles of lncRNAs originating from pseudogenes, a specific subset, have not been extensively explored in oncological research. Within this scope, investigations targeting the ZFP91 pseudogene (ZFP91-P) have revealed its elevated expression levels in pancreatic cancer cell lines. Analyses using RT-qPCR revealed that ZFP91-P is most prominently expressed in the BXPC-3-H cell line. In these studies, BXPC-3-H pancreatic cancer cells were transfected with short hairpin RNA (shRNA) targeting ZFP91-P to investigate its biological function, with an appropriate plasmid vector used as a negative control. Western blot analysis was employed to assess epithelial-mesenchymal transition (EMT) markers such as vimentin and β-catenin, while Transwell and MTT assays were conducted to evaluate cell proliferation and migration, respectively (Huang et al., 2016). The results showed that knockdown of ZFP91-P significantly reduced pancreatic cancer cell proliferation and migratory capacity. Furthermore, β-catenin expression

was upregulated, whereas vimentin expression was downregulated. These results imply that ZFP91-P plays a role in the migration of pancreatic cancer cells and could be a useful biomarker for early diagnosis (Huang et al., 2016).

Zinc Finger E-box Binding Homeobox 1/2 (ZEB1/ZEB2): Invasive Phenotype and Poor Prognosis

High transcriptional activity of ZEB1 and ZEB2, members of the Zinc Finger E-box Binding Homeobox family, has been associated with poor clinical outcomes in pancreatic cancer, including reduced patient survival, increased tumor aggressiveness, lymph node involvement, and more advanced disease stages (Chen et al., 2017). These factors are therefore recognized as critical prognostic markers in this cancer type. On the other hand, miR-200 family microRNAs have demonstrated the ability to limit cancer cell invasiveness by targeting ZEB1/2 and suppressing EMT. The miR-200/ZEB/EMT axis may have a context-dependent role in pancreatic ductal adenocarcinoma, according to new research that suggests this regulatory mechanism may not operate consistently in this illness. A study aimed to clarify the involvement of the miR-200 family in pancreatic cancer by examining the expression profiles of miR-200a, miR-200b, miR-200c, miR-141, miR-429, and miR-205, along with ZEB1, ZEB2, and CDH1 genes, across pancreatic tumor tissues, adjacent non-tumorous parenchyma, and patient-derived xenograft models. The findings revealed that CDH1 expression was notably reduced in tumor samples, whereas miR-200a, miR-429, and miR-205 were frequently upregulated. Interestingly, ZEB1 and ZEB2 levels remained relatively unchanged. Further analysis indicated a positive association between CDH1 and members of the miR-200 family, while ZEB1 demonstrated an inverse correlation with miR-200c, miR-141, and miR-205 (Diaz-Riascos et al., 2019).

Zinc Finger Protein-521 (ZNF521): Cell Differentiation and Tumor Development

Many new networks of transcription factors and microRNAs (miRNAs) that control gene expression have been found as a result of the development of sophisticated bioinformatics techniques. The biological roles of miRNAs that target ZNF521 in pancreatic cancer and the ways that dysregulation of this pathway contributes to malignancy have received special interest in this context. Hematopoietic, neuronal, and mesenchymal stem cells are all regulated by ZNF521, a co-transcription factor linked to stem cells. Numerous malignancies, including gastric, breast, pancreatic cancer, hepatocellular, bladder transitional cell carcinomas, and ovarian cancers, have been found to exhibit aberrant expression of ZNF521 transcripts. MiRNA dysregulation is frequently linked to this aberrant expression. Additionally, investigations of miRNA expression profiles have shown a large number of miRNAs implicated in transcription and oncogene control. miRNA dysregulation is frequently linked to this aberrant expression. Furthermore, several miRNAs implicated in the control of transcription factors and oncogenes, including ZNF521, have been identified by miRNA expression profile analysis; they are thought to be promising biomarker candidates for the diagnosis and prognosis of cancer (Chiarella et al., 2021). Conventional chemotherapy's main method of killing quickly growing cancer cells is by causing DNA damage. Chemotherapy resistance is still one of the biggest obstacles to successful treatment, nevertheless. According to recent research, one important mechanism influencing how sensitive cancer cells are to chemotherapy is the epithelial-mesenchymal transition (EMT) (Drápela et al., 2020). An essential part of the gene network that controls EMT is the transcription factor ZEB1, which is involved in this process. Additionally, ZEB1 has been linked to cell differentiation, metastasis, and the DNA damage response (DDR). According to recent studies, elevated ZEB1 expression may function as a possible regulator of chemotherapy resistance. It is thought that cancer cells going through EMT develop traits in common with cancer stem cells (CSCs). These stem-like cells are more resistant to drugs, have improved repair and self-renewal capabilities, and respond more strongly to DNA damage. Even in the absence of additional EMT-related alterations, functional studies have shown that ZEB1 by itself can enhance chemotherapy resistance. It has been revealed that ZEB1 participates in the DDR process by direct interaction with ATM kinase, which is linked to radioresistance, and the ZEB1/p300/PCAF complex. Additionally, it has been demonstrated that ATM directly phosphorylates ZEB1, enhancing its stability, whereas ZEB1 reduction results in lower amounts of CHK1, a crucial effector kinase in DDR, which starts the degradation of ZEB1. ZEB1 is a key regulator of the DNA damage response and a major factor in the emergence of chemotherapy resistance, according to all these findings. ZEB1 is therefore seen as a possible therapeutic target, especially in cancers that are resistant to treatment, like pancreatic cancer (Drapela et al., 2020).

Studies on the Role and Clinical Relevance of Zinc Finger Proteins in Pancreatic Cancer in the Literature

Wang et al. (2021) reported that inflammatory dysregulation is a key feature of acute pancreatitis (AP) and highlighted the crucial role of non-coding RNAs (ncRNAs) in its pathogenesis. To investigate this, they employed an in vitro AP model using caerulein-stimulated rat pancreatic acinar AR42J cells. Within this framework, they examined the role of zinc finger protein 644 (circ_ZFP644), a circular RNA. Their results showed that caerulein treatment increased the release of inflammatory cytokines and amylase activity. Additionally, caerulein upregulated miR-106b expression while downregulating circ_ZFP644 and

Pias3. Functional assays revealed that inhibition of miR-106b or overexpression of circ_ZFP644 enhanced apoptosis and reduced inflammation, whereas suppression of Pias3 reversed these effects. Dual-luciferase reporter experiments confirmed that circ_ZFP644 binds miR-106b and upregulates Pias3 expression via this interaction. Collectively, these findings demonstrate that circ_ZFP644 exerts pro-apoptotic and anti-inflammatory effects in AP through the miR-106b/Pias3 axis. Zhang et al. (2012) examined the function of fructose-1,6-bisphosphatase (FBP1) in pancreatic β cells' glucose metabolism and insulin secretion, as well as the transcriptional regulation of this enzyme. In order to achieve this, they investigated how ZBTB20, a zinc finger transcription factor, affected FBP1. The work used chromatin immunoprecipitation (ChIP) experiments, RNA interference techniques (siRNA), genetic engineering (β-cell-specific deletion of ZBTB20 using Cre/LoxP technology), and gene expression analysis (real-time RT-PCR, immunoblotting, and immunohistochemistry). They showed that in normal mice, ZBTB20 is strongly expressed in the β cells of the pancreatic islets, but in diabetic db/db animals, this expression is decreased. In addition to normal cell formation, deletion of the ZBTB20 gene in β-cells resulted in glucose intolerance, hyperglycemia, hypoinsulinemia, and reduced glucose-stimulated insulin production. Insulin secretion, ATP synthesis, and glucose metabolism all decreased in isolated islets; however, KCl-stimulated insulin secretion stayed normal. Furthermore, the MIN6 β cell line's glucose-stimulated insulin production was also interfered with by siRNA-mediated suppression of ZBTB20 expression. An FBP1 inhibitor was able to reverse the functional deficits brought on by the increase in FBP1 expression in the absence of ZBTB20. ZBTB20 inhibits the transcription of the FBP1 gene by binding to its promoter region, according to chromatin immunoprecipitation investigations. To sum up, ZBTB20 is essential for β-cell activity and glucose homeostasis regulation. These results imply that ZBTB20 may be a viable therapeutic target for the management of type 2 diabetes.

The frequency of ZFP91 expression in pancreatic cancer and its inverse relationship with overall survival were examined by Tang et al. (2021) using data from the TCGA and GEO datasets. According to their Kaplan-Meier analysis, patients with pancreatic cancer who have high ZFP91 gene expression had a considerably lower overall survival rate. Furthermore, ZFP91 expression was much higher in tumor tissues than in nearby normal tissues, according to results from the GEO dataset. The majority of tumor tissues had higher ZFP91 expression than normal tissues, according to protein level detection in five human samples. We also looked into how ZFP91 silencing affected the invasion, migration, and growth of pancreatic cancer cells. ZFP91 expression was higher in the HuP-T3 and KP4 pancreatic cancer cell lines than in the other cell lines. Cell proliferation was significantly reduced when ZFP91 was silenced by lentiviral means, and colony formation experiments revealed a considerable drop in colony-forming efficiency in cells with silenced ZFP91. Additionally, transwell invasion studies demonstrated decreased invasive capacity in ZFP91-silenced KP4 cells, and wound healing assays demonstrated a delay in the migration rate of ZFP91-silenced cells. These results imply that ZFP91 is essential for the invasion, migration, and growth of pancreatic cancer cells. ZFP91 expression was linked to the invasiveness of pancreatic and several other malignancies, according to an evaluation of the association between ZFP91 and EMT using GSEA analysis. Western blot analysis of ZFP91-silenced KP4 cells revealed decreased active β-catenin expression, and gene sets linked to TGF-β and β-catenin were enriched in patients with high ZFP91 expression. ZFP91-silenced cells also showed a decrease in mesenchymal markers (Snail, Slug, Vimentin) and an increase in epithelial markers (E-cadherin, Claudin-1). These results confirm ZFP91's involvement in EMT and tumor growth through β-catenin signaling. Lastly, it was shown that in pancreatic cancer, ZFP91 silencing improves chemosensitivity.

According to Zheng et al. (2022), the Myc-associated zinc finger protein (MAZ) is a crucial transcription factor that is widely distributed in mammals. Their findings indicate that MAZ is abnormally overexpressed in pancreatic cancer and helps promote tumors through the epithelial-mesenchymal transition (EMT), autophagy, and the tumor microenvironment. These findings suggest that MAZ might be an oncogene in pancreatic cancer and could be targeted for diagnostic and therapy.

THE ROLE OF ZINC FINGER PROTEINS IN PROSTATE CANCER: DIAGNOSIS, PROGNOSIS, AND THERAPEUTIC POTENTIAL

Diagnosis and Prognosis of Prostate Cancer

Prostate cancer is a malignancy with high prevalence, particularly among middle-aged men aged 45–60, and it is the most frequently diagnosed cancer type among men after skin cancers. It remains one of the leading causes of cancer-related mortality worldwide, especially in Western countries (Chen et al., 2013; Kelly et al., 2018). Risk factors for prostate cancer include family history, ethnicity, age, obesity, and various environmental factors. This cancer type is heterogeneous both epidemiologically and genetically. The interaction between genetic factors and social or ecological determinants complicates the prediction of race-specific survival rates in prostate cancer. It contributes to the variations in prostate cancer epidemiology observed across different countries (Hjelmborg et al., 2014). Androgens encourage the proliferation of prostate cancer cells, and androgen deprivation therapy (ADT), which treats metastatic illness, is a key strategy for suppressing the androgen receptor signaling pathway (Paller et al., 2013). Researchers have examined the effects of androgens as well as the possible contribution of genetic differences

in androgen metabolism and production (Wen et al., 2015). Generally, gene mutations are among the primary causes of cancer development. Candidate genes associated with prostate cancer susceptibility are linked to the roles of testosterone in the androgen signaling pathway and its metabolism. The development of prostate epithelial cells and prostate cancer cells largely depends on androgen receptor-mediated signaling and the presence of testosterone (Bluemn et al., 2012). Prostate cancer can be classified as androgen-sensitive or androgen-insensitive based on its response to the stimulatory effect of testosterone. This classification is also an important indicator in determining potential treatment strategies (Takayama, 2019).

Zinc Finger Protein-185 (ZNF185): Invasion via Actin Dynamics and Cytoskeletal Regulation

The LIM domain protein family includes zinc finger protein-185 (ZNF185), which has an actin-targeting domain (ATD) at the NH2-terminal and a LIM zinc-binding domain at the COOH-terminal. The LIM domain is a double zinc finger motif that is rich in cysteines and histidines and is named after the three homeodomain proteins Lin-11, Isl-1, and Mec-3 (Karlsson et al., 1990; Vanaja et al., 2003; Zhang et al., 2007). Silencing ZNF185 has been demonstrated to affect alterations in the actin cytoskeleton and cell polarity in response to etoposide therapy, even though ZNF185 is not necessary for p53-dependent cell cycle arrest and apoptosis (Smirnov et al., 2018). Squamous cell carcinomas of the skin and head and neck region are among the epithelial malignancies that have significantly decreased ZNF185 expression at both the mRNA and protein levels. This downregulation implies that cancer cells' lack of ZNF185 may promote cell motility and spread (Smirnov et al., 2018). Targeting the protein to the actin cytoskeleton requires ZNF185's actin-interacting domain, which is found at its N-terminus. On the other hand, actin binding does not require the LIM domain at the C-terminus (Zhang et al., 2007). Many proteins involved in cytoskeletal dynamics have the structural motif known as the LIM domain, which promotes protein-protein interactions (Kadrmas et al., 2004). E-cadherin, a component of adherens junctions that are crucial for stratified epithelial tissues, especially in keratinocytes and the epidermis, has been demonstrated to physically interact with ZNF185. Additionally, in conditions of differentiation, it is substantially expressed (Smirnov et al., 2019). The p53-ZNF185 axis's discovery sheds light on how p53 regulates cell dispersion, namely in the actin cytoskeleton's remodeling, mechanical property determination, and tumor microenvironment. New anti-cancer tactics and treatments may be developed as a result of more research discovering new target genes and signaling pathways implicated in p53's regulation of cytoskeletal structure and cell polarity (Smirnov et al., 2018).

Zinc Finger and BTB Domain-Containing Protein-46 (ZBTB46): Remodeling the Tumor Microenvironment

ZBTB46 is a member of the BTB-ZF transcriptional repressor family and is considered a classical marker for dendritic cells (DCs). Although classical DCs, endothelial cells (ECs), and fibroblast/mesenchymal cells structurally express ZBTB46, the absence of this gene does not result in any apparent dysfunction in DCs or ECs (Satpathy et al., 2012). Under homeostatic conditions, ZBTB46 maintains DCs and ECs in a quiescent state. However, its role under pathological conditions has not been fully elucidated (Wang et al., 2019).

The Effect of Zinc Finger Proteins (ZFPs) on the Androgen Receptor Signaling Pathway

Prostate cancer growth and progression are significantly influenced by the androgen receptor (AR). Developing novel therapeutic approaches requires a thorough analysis of the AR signaling system (Locke et al., 2008). ARs are activated by androgens, especially dihydrotestosterone (DHT). ARs experience conformational changes upon activation, dimerizing, dissociating from heat shock proteins, and moving into the nucleus. The transcriptional activity of AR depends on this nuclear translocation. AR functions as a transcription factor inside the nucleus, controlling the expression of particular genes. Prostate-specific antigen (PSA) is one example of an androgen-responsive gene that AR can either activate or repress (Boustani et al., 2007). The main ligand for AR is testosterone, which has a variety of physiologic effects when it binds. The body transforms testosterone into the more powerful androgen DHT. DHT has greater biological activity than testosterone because of its greater binding affinity to AR. Furthermore, androstenedione and other androgen precursors can bind to AR and have comparable effects (Obinata et al., 2024).

Studies on the Role and Clinical Relevance of Zinc Finger Proteins in Prostate Cancer in the Literature

Jiang et al. (2012) investigated the role of the ZFX gene in prostate cancer. Using immunohistochemical labeling, they found ZFX positivity in 42.2% of prostate cancer tissues, compared to just 12.5% of tissues with benign prostatic hyperplasia (BPH) and 11.8% of surrounding normal tissues. ZFX expression was significantly elevated in prostate cancer tissues (χ^2=10.967, p=0.035). Western blot research revealed that ZFX was significantly expressed in the prostate cancer cell lines PC-3 and DU145. In gene silencing experiments using siRNA targeting ZFX, ZFX mRNA and protein levels were dramatically decreased in PC-3 and DU145 cells ($p<0.01$). These findings suggest that ZFX may play a key role in the development and metastasis of prostate cancer and could be a therapeutic target.

Montanaro et al. (2023) explored whether ZNF750 could serve as a prognostic biomarker in prostate cancer. The study examined 43 cases of prostate cancer and benign prostatic hyperplasia (BPH). In all BPH cases, ZNF750 showed strong nuclear and cytoplasmic positivity, particularly intense in the nucleus (>80% of cells with a 3+ score). In contrast, ZNF750 expression was markedly reduced in prostatic acinar adenocarcinoma cases. Only 6 of the 34 cancer cases (17.6%) exhibited cytoplasmic positivity, while 11 cases (34.4%) showed complete absence of nuclear staining. Metastases were identified in 17 patients (39.5%), most of whom had both lymph node and bone metastases, with bone-only metastasis observed in three cases. A significant association was found between loss of nuclear ZNF750 expression and the presence of metastasis ($p = 0.01$). Prostate cancer cell lines (PC3 and DU145) and the normal prostate cell line (RWPE1) were examined in cell culture research, and the cancer cells had considerably lower levels of ZNF750 mRNA and protein. Moreover, ZNF750 was primarily found in the nucleus of healthy cells. According to these results, ZNF750 may be a useful biomarker, especially for determining whether individuals with prostate cancer are at risk of metastases.

The biological activities and tumor-suppressive effect of zinc finger protein 403 (ZFP403) in prostate cancer (PCa) were investigated by Xu et al. (2020). ZFP403 was considerably downregulated in prostate cancer tissues as compared to normal tissues, according to analysis using the GEPIA2 database. Western blotting, RT-qPCR, and immunohistochemistry (IHC) were used to validate these results in clinical samples and cell lines. Migration, invasiveness, colony formation, and cell proliferation were all markedly enhanced in PC3 and DU145 cell lines with lentivirally inhibited ZFP403 expression. ZFP403-knockdown cells produced a significantly greater number of colonies in soft agar colony formation experiments. Analysis of the cell cycle revealed increased G2/M phase progression along with higher levels of the proteins Cdc2, cyclin B1, and Cdc25C. When ZFP403 was suppressed, transwell migration and invasion experiments showed increased cell motility, reduced E-cadherin levels, and increased expression of vimentin, β-catenin, heparanase, and MMP2. These findings suggest that ZFP403 deletion promotes metastasis and the epithelial-mesenchymal transition (EMT). Furthermore, ZFP403-knockdown cells used in *in vivo* xenograft assays showed markedly enhanced tumor growth in nude mice. All things considered, these results imply that ZFP403 is a tumor suppressor gene in prostate cancer and that tumor growth and metastasis may be linked to its decreased expression.

CONCLUSION

The largest transcription factor family, zinc finger proteins (ZFPs), are essential regulators of human physiology and disease. Cell proliferation, the epithelial-mesenchymal transition (EMT), liver metastasis, inflammatory responses, cell cycle regulation, cancer stem cell durability, and DNA methylation are just a few of the basic processes that ZFPs can affect. The emergence and spread of several cancer forms demonstrate these regulatory effects. Additionally, it has been demonstrated that ZFPs play a major role in the development of several solid tumors, most notably breast, colorectal, prostate, pancreatic, and gastric malignancies. ZFPs are therefore particularly noteworthy as possible biomarker candidates for both therapeutic and diagnostic objectives.

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