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Omics sciences and precision medicine in pancreas cancer

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Abstract

Pancreatic cancer is a leading cause of death worldwide, associated with poor prognosis outcomes and late treatment interventions. The pathological nature and extreme tissue heterogeneity of this disease has hampered all efforts to correctly diagnose and treat it. Omics sciences and precision medicine have revolutionized our understanding of pancreatic cancer, providing a new hope for patients suffering from this devastating disease. By analyzing large-scale biological data sets and developing personalized treatment strategies, researchers and clinicians are working together to improve patient outcomes and ultimately find a cure for pancreatic cancer. ***Clin Ter 2023; 174 Suppl. 2 (6):85-94 doi: 10.7417/CT.2023.2475***

Key words: *Pancreas cancer, precision medicine, biomarker, genomics, metabolomics*

Introduction

Pancreatic cancer is a highly aggressive malignancy with a poor prognosis (1). It is the fourth leading cause of cancer-related deaths in the United States, with a five-year survival rate of less than 10% (2). It is often diagnosed at an advanced stage, making it difficult to treat (3). Recent advances in omics sciences and precision medicine have opened up new avenues for early detection and personalized treatment (4-6); but, despite the advances in medical technology and treatment options, the five-year survival rate for pancreatic cancer is still low (7). However, there is hope in the fight against this deadly disease, thanks to the emergence of precision medicine (8).

Precision medicine involves tailoring treatments to an individual's unique genetic makeup, thus allowing for more targeted and effective therapies (9). The pancreas is an extremely important part of the human body, for its digestive as well as regulatory roles: in fact, it produces enzymes and hormones through different cellular pathways. Pancreas contains acinar cells, duct cells, and the islets of Langerhans, which are involved in various functions by producing cell secretions. For instance, acinar cells produce inactive zymogen enzymes, which are activated upon exposure to the bicarbonate-rich pancreatic juice secreted by duct cells. Whereas, the islets of Langerhans are associated with the secretion of insulin and glucagon hormones, regulating glucose concentration in the body.

In this article, we will explore the role of genomics and proteomics in understanding the genetic basis of pancreatic cancer, identifying biomarkers for early diagnosis, and developing targeted therapies that can improve patient outcomes. We will also discuss the challenges and opportunities associated with translating these scientific discoveries into clinical practice. Despite advances in treatment, the disease remains difficult to diagnose and treat. Furthermore, we will explore the latest developments in precision medicine for pancreatic cancer, including targeted therapies and immunotherapies. We will also discuss specific precision medicines—such as Pamrevlumab, Herceptin®, Larotrectinib (LOXO-101), and Erbitux® (cetuximab)—that are showing promising results in clinical trials. By understanding these cutting-edge treatments, we can gain hope for a brighter future in the battle against pancreatic cancer. Moreover, recent developments in omics sciences and precision medicine have provided new hope for patients with pancreatic cancer.

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Prevalence of pancreatic cancer

Pancreatic cancer is one of the deadliest cancers, with a five-year survival rate under 10% (9) and an incidence that has been increasing over the past few decades, projected to become the second leading cause of cancer-related deaths by 2030 (10). The risk factors for pancreatic cancer include smoking, obesity, chronic pancreatitis, diabetes mellitus, and family history (11). Unfortunately, most cases are diagnosed at an advanced stage, when treatment options are limited (2, 12). Therefore, early detection and personalized treatment strategies are crucial for improving patient outcomes (13). Omics sciences such as genomics and proteomics have emerged as powerful tools for identifying novel biomarkers and therapeutic targets in pancreatic cancer (14).

What are omics sciences?

The name "omics sciences" refers to the study of large-scale biological data sets, including genomics, transcriptomics, proteomics, metabolomics, and epigenomics (15). These data sets provide a comprehensive view of the molecular changes that occur in cells and tissues during disease development and progression. By analyzing these data sets, researchers can identify new targets for therapy and develop personalized treatment strategies for patients.

Genetics of the pancreatic tumor

Pancreatic cancer is a highly aggressive and lethal malignancy, with poor prognosis. The genetic alterations that occur in pancreatic cancer are complex and heterogeneous, involving multiple genes and signaling pathways (16, 17). Several studies have identified specific genetic mutations associated with the development of pancreatic cancer, including KRAS, TP53, CDKN2A, SMAD4, and BRCA2. KRAS mutations are the most common genetic alteration found in pancreatic cancer, occurring in up to 95% of cases (18). These mutations lead to constitutive activation of the RAS signaling pathway, which promotes cell proliferation and survival (19). TP53 mutations are also frequently observed in pancreatic cancer and are associated with increased tumor aggressiveness and resistance to therapy (20). CDKN2A mutations result in loss of function of the p16INK4a protein, which regulates cell cycle progression and is involved in DNA repair mechanisms (21). SMAD4 mutations impair TGF-β signaling pathway activity, leading to increased cell proliferation and invasion (22). Finally, BRCA2 mutations have been linked to an increased risk of developing pancreatic cancer (23). Overall, understanding the genetic alterations that occur in pancreatic cancer is critical for developing targeted therapies that can improve patient outcomes. Advances in genomics technologies have enabled researchers to identify novel gene biomarkers that may be useful for early detection or predicting response to treatment.

Genomics of pancreatic tumor

Pancreatic cancer is a complex disease that arises from the accumulation of genetic and epigenetic alterations (24). Genomic studies have provided insights into the molecular mechanisms underlying pancreatic cancer development and progression. Whole-genome sequencing, whole-exome sequencing, and transcriptome analysis have identified numerous genetic alterations in pancreatic tumors, including mutations in KRAS, TP53, CDKN2A, SMAD4, and other genes (25-27).

The most frequently mutated gene in pancreatic cancer is KRAS, which is mutated in more than 90% of cases (25, 28). The KRAS mutation leads to constitutive activation of the RAS signaling pathway, which promotes cell proliferation and survival (29). Collins et al. studied that pancreatic cancer is associated with mutations in the KRAS gene, but it was not fully understood how these mutations promote cancer (30). Two mouse models were used to study pancreatic tumorigenesis, and it was found that KRAS mutations are required for tumor cell survival and promote the formation and maintenance of the fibroinflammatory stroma that plays a pivotal role in pancreatic cancer. Inhibiting KRAS mutations could provide a new approach for the treatment of pancreatic cancer.

Other commonly mutated genes include TP53 (50-75%), CDKN2A (30-40%), and SMAD4 (20-30%) (31). These mutations are associated with poor prognosis and resistance to therapy (32). For instance, Sinn et al. used next generation sequencing (NGS) in CONKO-001 trial to identify prognostic and predictive mutations in pancreatic adenocarcinoma patients (33). TP53 mutations were found to be a negative prognostic factor for disease-free survival in untreated patients but a positive predictive factor for gemcitabine efficacy in treated patients. Bartsch et al. evaluated the prevalence of mutations in the CDKN2A gene in familial pancreatic cancer (FPC) and determined their association with malignant melanoma (34). Germline mutations in p16 were found to be rare in FPC families, but they were identified in families with both pancreatic cancer and melanoma. No p14 germline mutations were found. These findings suggest a possible pancreatic cancer-melanoma syndrome associated with CDKN2A germline mutations affecting p16, and all members of such families should be screened for these mutations.

In addition to these recurrent mutations, genomic studies have identified other genetic alterations that may contribute to pancreatic cancer development and progression. For example, amplification of MYC or ERBB2 has been observed in a subset of pancreatic tumors (32, 35). Moreover, genomic profiling has revealed distinct subtypes of pancreatic cancer based on their molecular features. These subtypes may have different clinical outcomes and response to therapy. Overall, genomics studies have provided valuable insights into the molecular mechanisms underlying pancreatic cancer and may facilitate the development of precision medicine approaches for this deadly disease.

Table 1. Genes that are commonly associated with pancreatic cancer.

Gene	Function	Role in pancreatic cancer
KRAS	GTPase, regulates cell growth and differentiation	Activating mutations in KRAS are present in >90% of pancreatic ductal adenocarcinomas (PDACs) (36, 37)
TP53	Tumor suppressor gene, regulates cell cycle and apoptosis	TP53 mutations are present in >50% of PDACs (33)
CDKN2A	Tumor suppressor gene, regulates cell cycle	CDKN2A mutations are present in ~95% of familial pancreatic cancer cases and ~50% of sporadic cases (38)
SMAD4	Tumor suppressor gene, regulates TGF-β signaling pathway	SMAD4 mutations are present in ~55% of PDACs (39)
BRCA1/2	DNA repair genes	Germline mutations in BRCA1/2 increase the risk of developing pancreatic cancer (40)
ATM	DNA repair gene	Germline mutations in ATM increase the risk of developing pancreatic cancer (41)
PALB2	DNA repair gene	Germline mutations in PALB2 increase the risk of developing pancreatic cancer (42, 43)
STK11	Tumor suppressor gene, regulates cell polarity and metabolism	Germline mutations in STK11 increase the risk of developing pancreatic cancer (44)

Gene biomarkers of pancreatic cancer

Early detection and accurate diagnosis are crucial for improving patient outcomes. In recent years, significant progress has been made in identifying genetic biomarkers associated with pancreatic cancer (45). Several gene mutations have been identified in pancreatic cancer, including KRAS, TP53, CDKN2A, SMAD4, and BRCA2 (45). The KRAS gene is mutated in about 95% of pancreatic cancer cases, making it one of the most common genetic alterations in this disease. The TP53 gene, involved in regulating cell division and preventing the formation of tumors, has been found mutated in up to 70% of pancreatic cancers. The CDKN2A gene, which encodes a protein regulating cell cycle progression, has been found mutated in up to 95% of pancreatic cancer cases, while the SMAD4 gene, involved in regulating cell growth and division, has been found mutated in up to 55% of them.

Table 2. Some key gene biomarkers associated with prognosis and diagnosis of pancreatic cancer.

Gene	Phenotype MIM number	Pathology Correlation	**Gene/Locus MIM number**	**Inheritance**	**Role in pancreatic cancer**
BRCA2	600185	Increased risk of pancreatic cancer	260350	Autosomal dominant	Germline mutations in BRCA1/2 increase the risk of developing pancreatic cancer (40, 48)
CDKN2A	600160	Increased risk of pancreatic cancer	260350	Autosomal dominant	CDKN2A mutations are present in ~95% of familial pancreatic cancer cases and ~50% of sporadic cases (38, 49)
PALB2	610355	Increased risk of pancreatic cancer	260350	Autosomal recessive	Germline mutations in PALB2 increase the risk of developing pancreatic cancer (42, 43)
ATM	607585	Increased risk of pancreatic cancer	260350	Autosomal recessive	Germline mutations in ATM increase the risk of developing pancreatic cancer (41)
PRSS1	276000	Hereditary pancreatitis	167800	Autosomal dominant	Potential therapeutic target, as PRESS 1 is associated with poor prognosis (50)
STK11	602216	Peutz-Jeghers syndrome (PJS)	175200	Autosomal dominant	Tumor suppressor gene that regulates cell growth and division (51)
MLH1	120436	Lynch syndrome	120435	Autosomal dominant	DNA repair gene; mutations in MLH1 increase risk of developing pancreatic cancer (52)
MSH2	609309	Lynch syndrome	120435	Autosomal dominant	DNA repair gene; works in conjunction with MLH1 to help correct errors that occur during DNA replication (53)
MSH6	600678	Lynch syndrome	120435	Autosomal dominant	DNA repair gene; works in conjunction with MLH1 and MSH2 to help correct errors that occur during DNA replication (54)
PMS2	600259	Lynch syndrome	120435	Autosomal dominant	DNA repair gene; mutations in PMS2 have been associated with an increased risk of developing pancreatic cancer (55)

In short, mutations in these genes are associated with an increased risk of developing pancreatic cancer, which is why these genetic alterations can be used as biomarkers for early detection and prognosis prediction of pancreatic cancer. Furthermore, advances in NGS technologies have enabled the identification of novel genetic biomarkers associated with pancreatic cancer: for example, the GATA6 gene has been identified as a potential diagnostic biomarker for pancreatic ductal adenocarcinoma (PDAC) (46). Additionally, the ARID1A gene has been found to be frequently mutated in PDAC patients with a better prognosis (47).

In conclusion, identifying genetic biomarkers associated with pancreatic cancer can improve early detection and prognosis prediction.

Spatial gene analysis of pancreatic cancer

Spatial gene analysis is a relatively new approach that has been used to study the molecular characteristics of pancreatic cancer (56). This technique allows researchers to analyze the expression of genes in different regions of the tumor, providing valuable information about the heterogeneity of this disease (57). By studying the spatial distribution of genes, scientists have been able to identify specific subpopulations of cells within pancreatic tumors that may be responsible for driving tumor growth and metastasis. One example of spatial gene analysis in pancreatic cancer is single-cell RNA sequencing (scRNA-seq) (58). This technique allows researchers to sequence the RNA from individual cells within a tumor, providing a detailed picture of the gene expression patterns in each cell (59). Using scRNA-seq, scientists have identified distinct subpopulations of cells within pancreatic tumors that exhibit different gene expression profiles (60). These subpopulations may represent different stages of tumor progression or different cell types within the tumor microenvironment. Overall, spatial gene analysis has provided important insights into the molecular mechanisms underlying pancreatic cancer and may help guide the development of more effective treatments for this deadly disease.

Proteomics of pancreatic cancer

Proteomics is the study of proteins and their functions in a cell or organism. In pancreatic cancer, proteomics has been used to identify potential biomarkers for early detection, prognosis, and treatment response (61). Proteomic analysis has also provided insights into the molecular mechanisms underlying pancreatic cancer development and progression (62).

One of the major challenges in pancreatic cancer research is identifying new targets for therapy. Proteomic analysis can help identify novel proteins that are overexpressed or mutated in pancreatic cancer cells as compared to normal cells (62). These proteins can then be targeted with drugs to inhibit their function and prevent tumor growth (63). Furthermore, proteomic analysis can help predict which patients are likely to respond to certain treatments based on the protein expression patterns in their tumors. This information can be used to personalize treatment plans for individual patients, leading to better outcomes. Overall, proteomics is an important tool in understanding the complex biology of pancreatic cancer and developing more effective treatments for this deadly disease.

Pancreatic cancer protein biomarkers

Pancreatic cancer protein biomarkers play a crucial role in the diagnosis and prognosis of pancreatic cancer (64). These biomarkers are proteins that are found in the blood or tissues of patients with pancreatic cancer and can be used to detect the presence of the disease, monitor its progression, and predict patient outcomes (65). One such protein biomarker is CA 19-9, which is commonly used to monitor treatment response and recurrence of pancreatic cancer (66). However, this biomarker has limitations as it can also be elevated in other conditions, such as pancreatitis and liver disease. Other promising protein biomarkers include carcinoembryonic antigen (CEA), carbohydrate antigen 72-4 (CA 72-4), and mesothelin (67-69). These biomarkers have shown potential for early detection of pancreatic cancer and predicting patient outcomes.

For instance, Tempero et al. investigated the relationship between CA19-9 and Lewis antigens in pancreatic cancer patients (70). The researchers found that CA19-9 levels were elevated in patients with pancreatic cancer, and that the presence of Lewis antigen negative status was associated with higher levels of CA19-9. The authors thus suggest that CA19-9 may be a useful biomarker for pancreatic cancer diagnosis and monitoring. Furthermore, Berger et al. examined the use of post-resection CA19-9 levels as a prognostic marker for pancreatic cancer patients undergoing adjuvant chemoradiation (71). The researchers found that higher post-resection CA19-9 levels were associated with worse overall survival in these patients. The authors thus suggest that CA19-9 may be a useful biomarker for predicting outcomes in pancreatic cancer patients following surgery. On this matter, Tzeng et al. investigated racial disparities in CA19-9 levels and pancreatic cancer outcomes (72). The researchers found that Black patients with pancreatic cancer had lower levels of CA19-9 as compared to non-Black patients, and that this difference persisted after controlling for other factors. The authors suggest that these findings highlight the importance of considering race in the interpretation of CA19-9 levels for pancreatic cancer diagnosis and monitoring.

Metabolomics of pancreatic cancer

Metabolomics is the study of small molecules or metabolites present in biological systems. In pancreatic cancer, metabolomics has emerged as a promising tool for identifying biomarkers that can aid in early detection and diagnosis of the disease (86). Metabolomic profiling of pancreatic cancer tissues and biofluids has revealed significant alterations in metabolic pathways, such as glycolysis, amino acid metabolism, and lipid metabolism (86).

One of the key findings from metabolomic studies in pancreatic cancer is the identification of altered levels of certain metabolites, such as glucose, lactate, glutamine, and

Table 3. Some of the protein biomarkers used for prognosis of pancreatic cancer.

Protein Biomarker	**Function**	**Clinical Utility**	**References**
CA19-9	Glycosylated antigen	Diagnosis, Prognosis, Monitoring	(73, 74)
CEA	Glycoprotein	Diagnosis, Prognosis, Monitoring	(67)
MUC1	Glycoprotein	Diagnosis, Prognosis	(75, 76)
Survivin	Inhibitor of apoptosis protein	Diagnosis, Prognosis	(77, 78)
Mesothelin	Glycoprotein	Diagnosis, Prognosis	(79, 80)
Osteopontin	Glycoprotein	Diagnosis, Prognosis	(81, 82)
TIMP1	Protease inhibitor	Diagnosis, Prognosis	(83)
HE4	Glycoprotein	Diagnosis	(84, 85)

fatty acids (87). The researchers identified several metabolites that were significantly different between the two groups, including amino acids, bile acids, and lipids. They found that a combination of four metabolites (valine, leucine, lysine, and taurochenodeoxycholic acid) had high sensitivity and specificity for early detection of pancreatic cancer.

Shen et al. analyzed serum samples from patients with pancreatic cancer and healthy controls using gas chromatography-mass spectrometry (GC-MS) (88). The researchers identified several metabolites that were significantly different between the two groups, including amino acids, fatty acids, and sugars. They found that a combination of four metabolites (lysine, tyrosine, phenylalanine, and palmitic acid) had high diagnostic accuracy for pancreatic cancer. A study was conducted on Chinese cohorts to identify biomarkers for early detection of pancreatic cancer using untargeted metabolomics (89). The study found systematic metabolic network disorders before pancreatic cancer diagnosis, and a novel metabolite panel was identified that may have potential value in early detection of pancreatic cancer.

Another study identified potential metabolite biomarkers for early detection of stage-I pancreatic ductal adenocarcinoma (PDAC) (90). In this work, researchers analyzed the plasma samples of 22 stage-I PDAC patients and 22 healthy controls using gas chromatography-mass spectrometry (GC-MS) and liquid chromatography-tandem mass spectrometry (LC-MS/MS) (90). They identified 46 metabolites that were significantly different between the two groups, including amino acids, lipids, and carbohydrates. The researchers then selected a panel of four metabolites (cysteine, lysine, PC aa C34:3, and PC aa C36:2) as potential biomarkers for early detection of stage-I PDAC. The combination of these biomarkers showed high sensitivity and specificity in distinguishing stage-I PDAC patients from healthy controls. The study suggests that these metabolite biomarkers may have potential for early detection of stage-I PDAC, which could improve patient outcomes. These changes are thought to reflect the metabolic reprogramming that occurs in cancer cells to support their rapid growth and proliferation (91). Metabolomics has also been used to identify potential therapeutic targets for pancreatic cancer by revealing metabolic vulnerabilities that can be exploited for treatment (87).

Precision medicine in pancreatic cancer

Precision medicine is an approach to patient care that takes into account individual variability in genes, environment, and lifestyle (103). In the context of pancreatic cancer, precision medicine involves identifying the specific molecular alterations that drive tumor growth and developing targeted therapies to inhibit these alterations (8). This approach has shown promise in clinical trials, with several targeted therapies showing efficacy in patients with advanced pancreatic cancer.

Pancreatic cancer is a complex disease, requiring personalized treatment options (103). Precision medicine offers a promising approach to treating pancreatic cancer by tailoring therapies to the unique characteristics of each patient's tumor (104). This approach involves analyzing the genetic makeup of the tumor and identifying specific mutations or biomarkers that can be targeted with drugs (105). One example of

Table 3. Some of the commonly reported metabolomics biomarkers in pancreatic cancer.

Metabolite	**Biomarker Type**	**Potential Function**	**Reference**
Glutamate	Diagnostic	Energy production and biosynthesis	(92, 93)
Myo-inositol	Diagnostic	Cell signaling and osmoregulation	(94, 95)
Sphingomyelin	Diagnostic	Membrane structure and cell signaling	(96)
Lysophosphatidylcholine	Diagnostic	Membrane structure and cell signaling	(97)
Creatinine	Prognostic	Muscle metabolism and renal function	(98)
Lactate	Prognostic	Energy metabolism and acid-base balance	(99)
Choline	Prognostic	Membrane structure and cell signaling	(100)
Carnitine	Prognostic	Fatty acid metabolism and energy production	(101)
Glutathione	Prognostic	Antioxidant and detoxification	(102)

precision medicine in pancreatic cancer is the use of PARP inhibitors for patients with BRCA mutations (106). These drugs target a specific DNA repair pathway that is disrupted in tumors with BRCA mutations, leading to cell death (107). Another example is the use of immunotherapy for patients with tumors that express high levels of certain proteins, such as PD-L1 (108). Precision medicine also involves monitoring a patient's response to treatment using liquid biopsies, which can detect circulating tumor DNA in the blood (109). This allows doctors to adjust treatment plans as needed, based on changes in the tumor's genetic profile (110). Precision medicine holds great promise for improving outcomes for patients with pancreatic cancer by providing more targeted and effective treatments (111).

Traditional chemotherapy has been the mainstay of treatment for pancreatic cancer, but it often fails to provide significant benefits due to the heterogeneity of the disease. Precision medicine offers a promising approach to improve outcomes for patients with pancreatic cancer. Precision medicine involves using genomic and molecular information to tailor treatment to an individual patient's unique characteristics (112).

Precision medicine has revolutionized the way we approach cancer treatment, and pancreatic cancer is no exception. Pamrevlumab, Herceptin®, Larotrectinib (LOXO-101), and Erbitux® (cetuximab) are some of the precision medicines that have shown promising results in treating pancreatic cancer (113-115). For instance, Pamrevlumab is a monoclonal antibody that targets a protein called connective tissue growth factor (CTGF), which plays a crucial role in promoting tumor growth and metastasis in pancreatic cancer (113). Clinical trials have shown that combining Pamrevlumab with chemotherapy can significantly improve survival rates in patients with advanced pancreatic cancer (116). Herceptin® is another precision medicine that has been approved for the treatment of HER2-positive breast cancer (117). However, recent studies have shown that HER2 is also overexpressed in a subset of pancreatic cancers (118). This has led to clinical trials investigating the use of Herceptin® in combination with chemotherapy for the treatment of HER2-positive pancreatic cancer (119). Larotrectinib (LOXO-101) is a targeted therapy that inhibits TRK fusion proteins, which are found in a small percentage of pancreatic cancers (120). Clinical trials have shown that larotrectinib can induce durable responses in patients with TRK fusion-positive tumors (120). Erbitux® (cetuximab) is an immunotherapy drug that targets epidermal growth factor receptor (EGFR), which is overexpressed in many types of cancers, including pancreatic cancer (121). Clinical trials have shown that combining Erbitux® with chemotherapy can improve overall survival rates in patients with advanced pancreatic cancer (122).

Targeted therapies in pancreatic cancer

Targeted therapies have shown promising results in treating pancreatic cancer in recent years (110). These therapies work by targeting specific molecules or pathways that are involved in the growth and spread of cancer cells (123).

One example of a targeted therapy is erlotinib, which targets the epidermal growth factor receptor (EGFR) pathway (124). This pathway is often overactive in pancreatic cancer cells, leading to uncontrolled cell growth and division. Another targeted therapy that has shown promise in pancreatic cancer is nab-paclitaxel (125). This drug targets the protein albumin, which is found at high levels in the blood vessels surrounding pancreatic tumors. By binding to albumin, nab-paclitaxel can deliver chemotherapy drugs more effectively, directly to the tumor.

While targeted therapies have shown some success in treating pancreatic cancer, they are not effective for all patients. It is important for physicians to carefully evaluate each patient's individual case and determine which treatment approach will be most effective for them. Ongoing research into new targeted therapies and personalized treatment approaches will continue to improve outcomes for patients with pancreatic cancer.

Immunotherapies in pancreatic cancer

Immunotherapies have emerged as a promising approach in the treatment of pancreatic cancer (126). These therapies work by stimulating the patient's immune system to recognize and attack cancer cells. One type of immunotherapy that has shown promise in pancreatic cancer is checkpoint inhibitors, which block certain proteins on cancer cells that prevent the immune system from attacking them (127). Another type of immunotherapy being investigated for pancreatic cancer is adoptive cell transfer therapy, which involves removing T-cells from a patient's blood and genetically modifying them to target specific cancer cells before infusing them back into the patient's body (128). This approach has shown some success in clinical trials, but more research is needed to determine its effectiveness (129).

Overall, while immunotherapies are still being studied and refined for use in pancreatic cancer treatment, they offer hope for patients who may not respond well to traditional chemotherapy or radiation therapy. As precision medicine continues to advance, it is likely that in the future we will see even more effective immunotherapies, developed specifically for pancreatic cancer.

The future of omics sciences and precision medicine in pancreatic cancer

The future of omics sciences and precision medicine in pancreatic cancer is bright (130): advances in technology have made it possible to analyze large-scale data sets quickly and accurately, allowing for more precise diagnosis and treatment of the disease. In addition, collaborations between researchers and clinicians have led to the development of new clinical trials, incorporating omics sciences and precision medicine approaches (131).

Conclusions

In conclusion, the integration of omics sciences and precision medicine has provided promising approaches to the diagnosis and treatment of pancreatic cancer. Genomics,

spatial gene analysis, proteomics, and metabolomics data have enabled us to identify novel biomarkers and therapeutic targets for this deadly disease. Furthermore, precision medicine approaches—such as targeted therapies and immunotherapies—have shown great potential in improving patient outcomes. However, there is still much work to be done in order to fully understand the complex biology of pancreatic cancer and to develop treatments that are effective for all patients.

Continued research efforts in this field will undoubtedly lead to further advancements in our ability to diagnose and treat pancreatic cancer with greater accuracy and effectiveness. Precision medicine has emerged as a promising approach to treating pancreatic cancer: the ability to tailor treatments based on the unique genetic makeup of each patient has led to the development of targeted therapies and immunotherapies that offer new hope for people suffering from this disease. Pamrevlumab, Herceptin®, Larotrectinib (LOXO-101), and Erbitux® (cetuximab) are just a few examples of precision medicines that have shown promise in clinical trials. While there is still much work to be done in this field, the progress made so far is encouraging and offers hope for better outcomes for patients with pancreatic cancer in the future.

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Conflicts of interest statement

Authors declare no conflict of interest.

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