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Update on chronic pancreatitis and pancreatic cancer - A review

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ABSTRACT

Background: Chronic pancreatitis (CP) is a pathological fibro-inflammatory disorder. Pancreatic cancer (PC) is known to be one of the most deadly types of cancer, and its early detection remains difficult. The most common PC is pancreatic ductal adenocarcinoma (PDAC). Patients with CP have a higher risk of PDAC. Differentiation of CP from PDAC can be challenging. Objectives: To summarize recent findings on the CP-PC relationship, risk factors, biomarkers, and potential therapies. Materials and methods: Articles from the PubMed database, primarily published from 2024/5/1 to 2025/5/31. Chronic pancreatitis and cancer: Pitavastatin suppresses cancer-prone chronic inflammation, reducing the risk of CP and PC. Nuclear factor of activated T cells 5 (NFAT5) may be a potential druggable target in PDAC resistant to KRAS therapy. Human rhomboid family-1 (RHBDF1) could be a viable target for the diagnosis and treatment of early-stage PC. Leptin may serve as a biomarker to distinguish PC from CP. Liquid assays using the in situ-proven Ex-miR-4516 may have the potential for detecting relatively early-stage PDAC and monitoring its clinical course. An autoantibody (AAb) panel (CEACAM1-DPPA2-DPPA3-MAGEA4-SRC-TPBG-XAGE3) may potentially serve as a PDAC diagnostic test. Conclusion: This review offers essential insights into the relationship between PDAC and CP.

Keywords: chronic pancreatitis, pancreatic cancer, pancreatic ductal adenocarcinoma, potential biomarkers

1. INTRODUCTION

Background

Chronic pancreatitis (CP) is recognised as a pathological fibro-inflammatory disorder of the pancreas. It develops in individuals with genetic, environmental, and/or other risk factors who develop ongoing pathological responses to parenchymal injury or stress (Whitcomb et al., 2016). This disease is characterised by ongoing inflammation of the pancreas, which leads to progressive loss of the endocrine and exocrine compartments in consequence of atrophy and/or replacement with fibrotic tissue (Kleeff et al., 2017). Some of the environmental risk factors associated with significant increases in risk for pancreatic diseases are:

current tobacco use, obesity, and heavy use of alcohol (Alsamarrai et al., 2014). CP, due to the symptoms associated with it, considerably affects patients' lives. The three major clinical features of this disease are pain, maldigestion, and diabetes (Drewes, 2013).

Pancreatic cancer (PC) may result from hereditary germline or somatic acquired mutations and is one of the most deadly types of cancer, because it is tough to detect early (Goral, 2015). In 2012, PC held the position of the eleventh most common cancer globally and the seventh leading cause of cancer-related death across both sexes (Ilic & Ilic, 2016). A minority of patients are diagnosed with PC at an early stage, which allows for surgical resection of the tumour - approximately 80 to 85% of patients present with advanced, unresectable disease. Additionally, pancreatic cancer responds poorly to most chemotherapeutic agents (Vincent et al., 2011). Median survival of patients with PC is approximately 4 months with a 5-year survival of 13% (Stoop et al., 2025). PDAC is the most common malignant neoplasm of the pancreas. The conventional type of PDAC is a tubular adenocarcinoma (Dhillon & Betancourt, 2020). Patients which are diagnosed with CP have an highier risk of PDAC. In some studies, researchers state that the incidence rates of PDAC increase with the duration of CP disease (Gandhi et al., 2022).

On the other hand, some sources say that the risk of developing PC is indeed higher in patients with CP, but the association diminishes with long-term follow-up. Five years after diagnosis, CP patients have a nearly eight-fold increased risk of PC (Kirkegård et al., 2017). Moreover, CP is one of several conditions that can mimic PDAC. Differentiation of it from PDAC can be challenging due to overlapping CT and MRI features (Miller et al., 2023).

Objectives

Since CP significantly increases the risk of developing PC, and cancers in this area often have a poor prognosis because detection usually occurs at an advanced stage, it is crucial to focus on understanding the relationship between carcinogenesis and CP, as well as to conduct activities aimed at reducing this risk, searching for potential biomarkers that could be useful in screening patients at risk, as well as looking for potential possible methods of treating patients. This work aims to summarize the essential information from articles published in the PubMed database during the past year (May 2024 - May 2025), regarding the association of PC and CP. Hopefully, this review can serve as an update of information and perhaps a starting point for conducting further research in this area, which may be beneficial for patients.

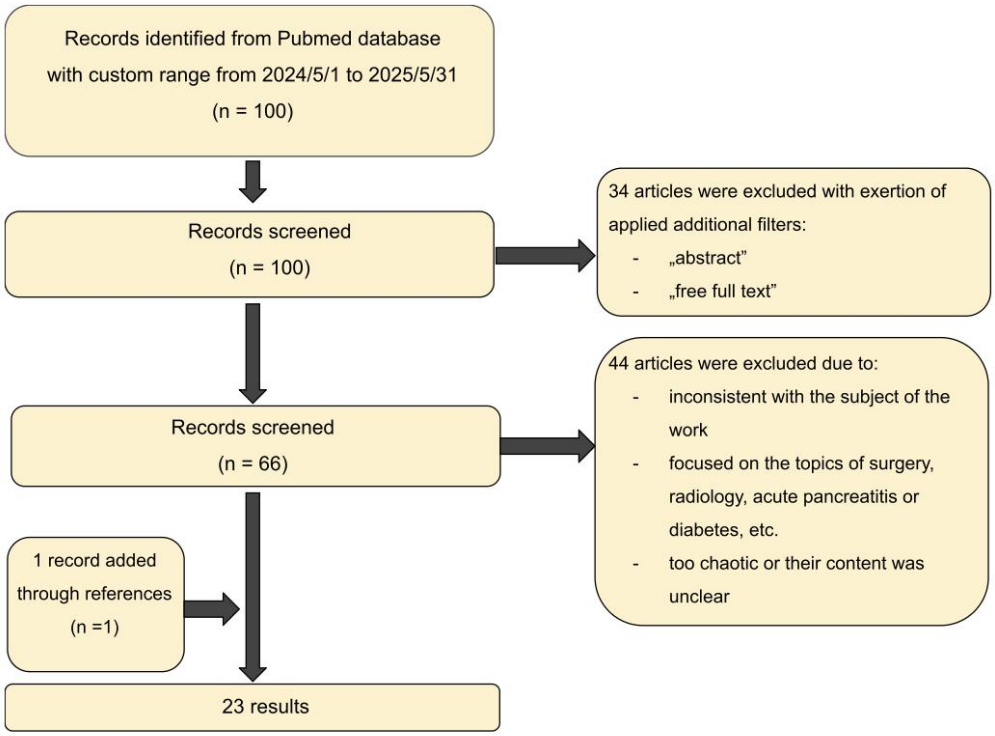

Figure 1. Graphic representation of the methodology for carrying out the central part of the work.

2. REVIEW METHODS

Twelve sources of information form the basis of the "introduction" section. A literature search was performed within the PubMed bibliographic database, using the following keywords: chronic pancreatitis, pancreatic disease, pancreatic cancer, risk, risk factor, PDAC and symptoms.

To create the central part of the article, a literature search was conducted in June 2025 using the PubMed database with keywords: chronic pancreatitis, cancer, neoplasm in the title or abstract of published articles, with a custom range from 2024/5/1 to 2025/5/31. Case reports or reviews were excluded. One hundred results were displayed. Applying the filters "abstract" and "free full text" eliminated thirty-four articles without an abstract or free access to the full text. Sixty-six results appeared. Then, the abstracts of the articles were read and assessed for their relevance to the paper's topic. The authors focused particularly on articles discussing biomarkers of PC and differentiation from CP, potential treatments, and risk factors. Articles that focused on the surgery, radiology, acute pancreatitis, or diabetes, etc., were not considered for writing this paper. The authors also excluded articles that were too confusing or unclear in content. Twenty-two articles constitute the central section of the work, and one additional article, cited in reference number eighteen, was added through references, resulting in a total of twenty-three sources used to formulate the central part of the text. Figure 1 shows the methodology used to develop the central part of the work.

3. RESULTS & DISCUSSION

3.1. Risk factors and protective factors

According to a study conducted recently, lipophilic statins suppress cancer-prone chronic inflammation by blocking the TANK-binding kinase 1 - interferon regulatory factor 3 - Interleukin-33 (TBK1-IRF3-IL-33) signaling axis. Pitavastatin inhibits the mevalonate pathway-mediated TBK1 binding to the membrane, and by doing so, it inhibits IL-33 expression. By inhibiting IL-33 expression, Pitavastatin blocks the cytokine and nuclear functions of IL-33 in chronic inflammation. Pivastatin effectively reduces the risk of CP and PC in mice and humans. This effect is independent of the cholesterol levels (Park et al., 2024).

People who smoke tabaco have higher risk for CP and PC. It is possible to significantly lower the risk for these pancreatic diseases by smoking cessation. Some studies showed that those who quit smoking had similar risk of the diseases as those who never smoked (Han et al., 2024). In their work, scholars state that men, black individuals, people aged 40-59 years, and those with a body mass index (BMI) of 25-29.9 have an increased risk for CP. Researchers also point out that older age (> 40 years) and BMI (between 25 and 29.9) are important risk factors for the development of PDAC in CP patients (Das et al., 2024).

CP is a known risk factor for PC. However, according to researchers from the United Kingdom, there is also a link between CP and extra-pancreatic (EP) cancers. Patients with CP exhibit a higher incidence of developing EP cancers compared to those without CP (in particular, lung and liver cancer). Quammie et al., (2025) in their work suggest that this correlation can result from sharing common risk factors such as alcohol and smoking.

3.2. Genetics, diagnosis, and treatment possibilities

Deng et al., (2024) in their study mentioned that pancreatitis promotes resistance to KRAS*-targeted therapy in PDAC through TGFβ signalling. It also identifies the nuclear factor of activated T cells 5 (NFAT5) as a target in PDAC that is resistant to KRAS therapy. NFAT5 forms a transcriptional regulatory complex with SMAD3 and SMAD4 to mediate TGFβ-driven KRAS* bypass and sustain EMT-associated escaper tumor growth. Inhibiting NFAT5 suppresses S100 Calcium Binding Protein A4 (S100A4) expression in both tumor cells and macrophages, which leads to attenuation of pancreatitis-induced resistance to KRAS inhibition and extends mouse survival.

Scientist, in their paper, state that human rhomboid family-1 (RHBDF1) is upregulated in PDAC tumor samples relative to para-tumor and CP tissues. Ma et al., (2025) in their work, they highlighted that its expression is increased in correlation with the progression of pancreatitis to cancer. RHBDF1 promotes the progression of PC through the SRC-YAP signaling pathway. Scientists point out that RHBDF1 can be a promising target for the diagnosis and treatment of early-stage PC.

Brusatol (BRT) is a plant-derived natural quassinoid isolated from Fructus Bruceae (dried fruit of the Brucea Javanica plant used in traditional Chinese medicine) (Lau et al., 2008). A group of scientists from China conducted a study on genetically engineered Krastm4Tyj Trp53tm1Brn Tg (Pdx1-cre/Esr1*) #Dam/J (KPC) mice and a cerulein-induced mouse model of CP in KPC mice to investigate the therapeutic effects of BRT. The researchers observed a decrease in the expression of the ductal marker CK19 and an attenuated expression of Ki-67 following BRT treatment. Zhang et al. in their work suggests that treatment with BRT can alleviate CP-mediated PC tumorigenesis, and observed that it reduces the production of inflammatory cytokines such as IL-6, IL-1β, and TNF-α.

Additionally, they discovered that, in genetically engineered KPC mice, treatment with BRT may enhance the therapeutic effects of Gemcitabine. They also noticed that, after treatment with BRT, the elevated serum lipase and amylase levels induced by cerulein lowered significantly (Zhang et al., 2024).

Furthermore, they assessed the serum levels of AST, ALT, and Cre in KPC mice to evaluate any potential liver and kidney toxicity. They indicate that the treatment with BRT at the concentrations used in their study did not exhibit overt toxicity to the KPC mice. They observed that BRT has an effect on impeding pancreatic carcinogenesis through targeting NLRP3 inflammasome and suggest BRT has good potential as a prospective agent for the treatment of PC (Zhang et al., 2024).

Researchers from Florida and Kansas, in their study, demonstrate that cyclic adenosine monophosphate response element binding protein 1 (CREB) and Kras G12D/+ (Kras*) promote irreversible acinar-to-ductal metaplasia (ADM), accelerating PC progression with alcoholic chronic pancreatitis (ACP). In their paper, they mention that ACP induction promotes PC progression via irreversible ADM reprogramming with Kras* cooperativity. They highlighted that these ductal lesions originating from acinar cells display continuous CREB hyperactivation. They state that attenuating high-grade pancreatic intraepithelial neoplasia (PanIN) formation and tumor progression through acinar-specific loss of CREB provides foundational insights for future studies on CREB's role in PC progression in the ACP with Kras*. They also mention that targeting CREB may offer a hopeful strategy for effective treatments for inflammation-driven PC (Srinivasan et al., 2025).

An article published in 2025 showed that deubiquitinating enzyme BRCA1 BRCA1-associated protein-1 (BAP1) deletion leads to the overactivation of the nuclear factor-κB (NF-κB) signalling in PDAC, which promotes the proliferation, migration, and invasion of PDAC. The researchers discovered that dual-target inhibitors of IRAK1/4 exhibited significant inhibitory effects on BAP1-deficient tumors in PDAC models. In their paper, they explain in detail the mechanism by which BAP1 inhibits the NF-κB signalling and highlight that their work presents a promising strategy for the targeted treatment of BAP1-deficient PC (Zhao et al., 2025).

Scholars conducted a study on an immunocompetent PDAC-bearing mouse model - KPC PDAC syngeneic C57Bl/6 mouse. They observed that the combination of anti-PD-1 antibody and TLY012 prevented PDAC growth in an immunocompetent mouse model while increasing tumour-infiltrating CD8+ T cells, decreasing circulating T-regulatory cells, and altering plasma cytokine expression of CCL5, interferon-gamma, and IL-3 to promote proinflammatory, antitumour effects. The researchers report that combining TLY012 and anti-mouse PD-1 modifies immune cell and cytokine levels to foster a more proinflammatory immune environment that leads to decreased PDAC tumour growth. Their results support the further exploration and clinical testing of TLY012 alone and in combination with anti-PD1 therapy in PC and other malignancies (Louie et al., 2025).

3.3. Potential biomarkers

Belfrage et al., (2024) published an article whose topic concerned necroptosis and its association with the pathogenesis of pancreatitis and its potential progression to PDAC. They conducted a study on patients with CP, PDAC, and healthy controls (HC). Scientists from Finland compared plasma levels of necroptosis-related markers, such as mixed lineage kinase domain-like protein (MLKL), IL-33, and IL-33 soluble receptor (sST2). They found that elevated levels of sST2 are associated with pancreatic diseases. Moreover, they observed that circulating levels of MLKL are lower in patients with CP than in those with PDAC. They suggest that monitoring for an increase in MLKL levels may help in distinguishing transformation from CP to PDAC, probably most effective when combined with other reliable markers. They also declared that further research is required to determine whether MLKL and sST2 could serve as valuable biomarkers in evaluating pancreatic diseases.

Scientists from the Czech Republic observed a significant decrease in retinol serum concentration in PDAC compared to type-2 diabetes mellitus (T2DM), CP, and HC. The levels of retinol decreased stepwise, with the highest levels observed in HC and the lowest in the PDAC group. (HC → DM2 → CHP →PDAC). They also discovered a significant decrease in the active metabolite of retinol, all-trans retinoic acid (ATRA), levels in PDAC compared to T2DM and HC. Both ATRA and retinol levels were significantly lower in stage III and IV of PDAC compared to HC and DM2. In their work, they suggest that ATRA and retinol are related to advanced PDAC or disease progression. Although the findings may seem interesting, neither ATRA nor retinol is suitable for detecting early PDAC, as the differences between early stages (I+II) of PDAC and non-carcinoma groups were not significant (Hrabak et al., 2024).

Scientists from the United States and Singapore were analysing circulating extracellular vesicles (EVs) to search for potential biomarkers to differentiate between patients with PDAC from those with benign pancreatic diseases such as CP and intraductal papillary mucinous neoplasm (IPMN). They used the EVtrap method to isolate EVs from plasma and performed proteomics analysis on samples from 124 individuals. The researchers observed that EVs containing high levels of PDCD6IP, SERPINA12, and RUVBL2

were associated with PDAC compared to benign diseases. EVs with PSMB4, RUVBL2, and ANKAR were associated with metastasis, whereas those with CRP, RALB, and CD55 were correlated with poor clinical prognosis. They propose that these findings could assist in developing biomarkers (Bockorny et al., 2024).

The study conducted in 2024 aimed to identify potential proteomic biomarkers that could be useful in detecting PC among patients with CP. Researchers, using quantitative ELISA techniques, found that the median leptin concentration in the plasma of patients with PC is higher than in those with CP. They also observed that the statistical significance remained consistent regardless of other variables such as BMI or gender. In their work, they emphasised that the detection of increased serum levels of leptin in patients with CP should raise suspicion of malignant transformation and prompt further diagnostic management. The scientist suggests that leptin could serve as a biomarker to help in distinguishing patients with PC from those with CP. Nevertheless, it is essential to note that this study focused on patients with advanced PC. Because of this, it remains uncertain and requires further investigation to determine whether leptin can or cannot act as a possible biomarker for early detection of this condition (Gheorghe et al., 2024).

Scholars aimed to identify blood protein biomarkers for PC diagnosis and differential diagnosis of CP using a high-throughput multiplex proteomic assay and machine learning-based analysis. They collected serum samples from four groups of patients: HC and those with CP, stage I/II PC (PC1), and stage III/IV PC (PC2). They observed that mucin-16 and interleukin-6 proteins were expressed at higher levels in the PC (PC1 and PC2) groups than in the CP and HC groups. Additionally, they identified some new biomarkers for PC diagnosis, including C1QA and CDHR2. They also state that pro-neuropeptide Y (NPY) seems to be a promising biomarker to help distinguish between CP and other diseases (Kim et al., 2025).

The results of a study conducted by Xu et al., (2024) showed that the plasma level of insulin-like growth factor binding protein 2 (IGFPB-2) is significantly higher in a group of patients with PDAC than in those with CP and HC. This elevation of IGFPB-2 and its extent are associated with tumour progression, glucose metabolism, and prognosis. The researchers point out that, at a cut-off value of 333.9 ng/mL, the specificity and sensitivity were 78.08 and 65.33%, respectively. They evaluated that IGFBP-2 alone did not outperform carbohydrate antigen 19-9 (CA 19-9) in diagnostic power. However, combining IGFBP-2 and CA19-9 proved more accurate in detecting PDAC than CA19-9 alone. On the other hand, IGFBP-2 was more precise than the other in discriminating between CP and PDAC. IGFBP-2 might serve as a supplementary biomarker for the diagnosis and prognostic prediction of PC.

Piwi-interacting RNAs (piRNAs) constitute a class of non-coding RNAs. The scientists conducted sequencing of circulating plasma small RNAs from patients with PC and CP. Researchers compared the differentially expressed piRNAs with those in tissues. They found that piRNAs hsa-piR-23246, hsa-piR-32858, and hsa-piR-9137 may play a key role in PC development, since their expression levels showed a correlation in both plasma and tumour tissue. Furthermore, they identified a total of nineteen deregulated piRNAs in plasma samples from patients with CP, which are linked to genes associated with chronic inflammation. The researchers state that their study offers a description of piRNA changes in pancreatic malignancy and inflammation that, in the future, may help in searching for potential biomarkers (Saha et al., 2024).

The researchers from Lithuania discovered that concentrations of interleukin 8 (IL-8) are statistically significantly higher in the PDAC group compared to the CP and control groups. According to them, a combination of IL-8, CEACAM6, and CA19-9 reached the highest AUC values for distinguishing between the group with PDAC and the control group. Combining IL-8 and CA19-9 resulted in the highest classification score between the PDAC group and the control group with CP patients. According to Bukys et al., IL-8 is a promising biomarker for diagnosing PDAC. However, the scientist highlighted that only the combination of IL-8, CA19-9, and CEACAM6 provides sufficient diagnostic power (Bukys et al., 2024).

An article published in 2024 showed that the expression levels of IFN-γ, 4-1BBL, CD3, CD8, and CD56 in patients with stages I-II and III-IV cancer were lower than those in patients with CP. Moreover, the expression of IL-17 in patients with stages I-II and III-IV cancer was higher than in patients with CP. Furthermore, among patients with PC, survival time was longer in those with higher expression levels of CD3, CD8, CD56, IFN-γ, and 4-1BBL. In contrast, higher expression of IL-17 was connected to shorter survival times. The expression levels of CD56, CD8, CD3, the costimulatory molecule 4-1BBL, and cytokines IL-17 and IFN-γ showed associations with the following aspects: degree of differentiation, Tumour-Node-Metastasis staging, and the prognosis of PC. They can be considered as novel immunological indicators for assessing the condition and treatment effectiveness in patients with PC (Liu et al., 2024).

The scholars from Japan and Ohio were aiming to identify PDAC-specific exosomal microRNAs (Ex-miRs) from pancreatic juice and assess their diagnostic potential. They declared that in serum samples, the sensitivity, specificity, and accuracy of Ex-miR-4516

were 97.5%, 34.3%, and 68%, respectively. In their paper, they concluded that liquid assays using the in situ-proven Ex-miR-4516 may have a high potential for detecting relatively early-stage PDAC and monitoring its clinical course (Sakaue et al., 2024).

The researchers declare that the apolipoprotein A2 isoform (APOA2-i) Index, combined with CA 19-9, may improve early-stage PC detection, mainly in demanding cases and for surveillance of high-risk patients. According to Shionoya et al., APOA2, compared to CA 19-9, showed lower accuracy for detecting advanced PC (stages II-IV), but it provided superior accuracy for early-stage (stages zero and I). The sensitivity was 33.3% vs. 22.2%, and the specificity was 66.7% vs. 59.4% (Shionoya et al., 2025).

Scientists used high-throughput, custom cancer antigen microarrays to identify a clinically relevant autoantibody (AAb) biomarker combination able to detect PDAC differentially. They identified the most effective biomarker combination to be CEACAM1-DPPA2-DPPA3-MAGEA4-SRC-TPBG-XAGE3 with an AUC = 85·0% (SE = 0·828, SP = 0·684). Later they, validated the specificity of this 11-biomarker panel against other cancers (PDAC vs other PC: AUC = 70·3%; PDAC vs colorectal cancer: AUC = 84·3%; PDAC vs prostate cancers: AUC = 80·2%) and HC (PDAC vs HC: AUC = 80·9%), confirming that this novel AAb biomarker panel is capable of selectively detecting PDAC. Mowoe et al., (2025) suggested that this AAb panel can be a useful tool and has the potential to form the basis of a diagnostic test for PDAC.

This review, despite the authors' pursuit to obtain the best results, still has several limitations, including a limited search scope, which is restricted to one year. While it does not provide a comprehensive understanding of the relationship between CP and PC, it allows one to concentrate on recent information on the topic. Another limitation is that this review, in the central part of the work, only included articles available in free full-text format. The review adopts this approach to summarise new information for all interested in the topic, and to provide unrestricted access to sources of interest. However, this approach has limitations, because the review excluded some interesting and potentially useful information by not considering abstracts of articles without free full-text versions during the content search. The summary of this article is shown in the form of a table 1.

Table 1. Update on chronic pancreatitis and pancreatic cancer

Paragraph in the text	Reference	Information
Risk factors and protective factors	(Park et al., 2024)	Pitavastatin suppresses cancer-prone chronic inflammation, effectively reducing the risk of CP and PC in mice and humans.
	(Han et al., 2024)	Tobacco smokers have higher odds for CP and PC.
	(Das et al., 2024)	Older age (> 40 years) and a BMI of 25 - 29.9 are significant risk factors for developing PDAC in CP patients.
	(Quammie et al., 2025)	Patients with CP have an increased risk of developing EP cancers, particularly lung and liver cancer.
Genetics, diagnosis, and treatment possibilities	(Deng et al., 2024)	NFAT5 may be a potential druggable target in PDAC resistant to KRAS therapy.
	(Ma et al., 2025)	RHBDF1 promotes the progression of PC through the SRC-YAP signalling pathway, and could be a viable target for the diagnosis and treatment of early-stage PC.
	(Zhang et al., 2024)	BRT affects impending pancreatic carcinogenesis through targeting NLRP3 inflammasome and has good potential as a prospective agent for the treatment of PC.
	(Srinivasan et al., 2025)	Targeting CREB may offer a hopeful strategy for effective therapies for inflammation-driven PC. BAP1 deletion causes the overactivation of NF-κB signalling in PDAC, which encourages the proliferation, migration, and invasion of PDAC.
	(Louie et al., 2025)	Combining TLY012 with anti-mouse PD-1 alters immune cell and cytokine levels to create a more proinflammatory immune environment that contributes to decreased PDAC tumour growth.
Potential biomarkers	(Belfrage et al., 2024)	Monitoring for an increase in MLKL levels may be helpful to discern transformation from CP to PDAC, likely in combination with other markers.
	(Bockorny et al., 2024)	EVs containing high levels of PDCD6IP, SERPINA12, and RUVBL2 were associated with PDAC. EVs with PSMB4, RUVBL2, and ANKAR are linked to metastasis, while those with CRP, RALB, and CD55 are associated with poor clinical prognosis.
	(Gheorghe et al.,	Leptin appears to be a biomarker that can help differentiate patients with PC from

	2024).	patients with CP.
	(Kim et al., 2025)	Mucin-16 and interleukin-6 proteins showed higher expression levels in the PC group than in the non-PC group. C1QA and CDHR2 can be new biomarkers for PC diagnosis. NPY seems to be a promising biomarker for the differential diagnosis of CP.
	(Xu et al., 2024)	IGFBP-2 may be considered a supplementary biomarker for the diagnosis and prognostic prediction of PC.
	(Saha et al., 2024)	piRNAs hsa-piR-23246, hsa-piR-32858, and hsa-piR-9137 may serve a key role in PC development, since their expression levels showed a correlation in both plasma and tumour tissue.
	(Bukys et al., 2024)	A combination of IL-8, CEACAM6, and CA19-9 may help differentiate patients with PDAC from those with CP and other diseases.
	(Liu et al., 2024)	The expression levels of CD3, CD8, CD56, cytokines IL-17 and IFN-γ, and the costimulatory molecule 4-1BBL were found to correlate with the degree of differentiation, Tumour-Node-Metastasis staging, and the prognosis of PC.
	(Sakaue et al., 2024)	Liquid assays using the in situ-proven Ex-miR-4516 may have a high potential for detecting relatively early-stage PDAC and monitoring its clinical course.
	(Shionoya et al., 2025)	APOA2-i Index, when combined with CA 19-9, may enhance early detection of PC.
	(Mowoe et al., 2025)	Autoantibody (AAb) panel, with the biomarker combination CEACAM1-DPPA2-DPPA3-MAGEA4-SRC-TPBG-XAGE3, may have the potential to serve as the basis for a diagnostic test for PDAC.

4. CONCLUSION

Despite these limitations, this work provides relevant insights into the association between PDAC and CP, and it includes information that may serve as an update for anyone interested in the topic. This review may also serve as an indication of the direction in which further research can be conducted regarding the search for potential biomarkers, methods of treatment, reducing the risk of PDAC in patients with CP, and identifying which group of patients, in particular, needs screening for cancer. Further research on potential PC markers and possible therapeutic targets is necessary.

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Author's Contribution

The contribution of each author to the entire work was equal.
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Conflict of interest
The authors declare that there is no conflict of interest.

Data and materials availability
All data sets collected during this study are available upon reasonable request from the corresponding author.

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