Enhancing Pancreatic Cancer Clinical Trial Protocols:

Key Learnings from FDA-Approved and Failed Drug Candidates

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Introduction

Pancreatic cancer is a highly aggressive malignancy originating in the pancreas, a gland located behind the stomach responsible for endocrine and exocrine functions.

This whitepaper provides a comprehensive examination of pancreatic cancer, a highly aggressive malignancy with one of the lowest survival rates among major cancers. It explores the epidemiology, underlying biological, psychological, and environmental factors, and the evolution of diagnostic and treatment approaches for this challenging disease.

Key topics include an overview of pancreatic cancer's prevalence and trends, advancements in diagnostic technologies, and the current landscape of FDA-approved therapies and ongoing clinical trials. Additionally, the paper addresses pivotal clinical endpoints, challenges in drug development, and strategies to improve the efficacy and success of treatment options. Through a detailed analysis of historical progress and emerging innovations, this whitepaper aims to inform and guide stakeholders in the ongoing efforts to improve patient outcomes in pancreatic cancer.

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Overview of Pancreatic Cancer

Pancreatic cancer is a highly aggressive malignancy originating in the pancreas, a gland located behind the stomach responsible for endocrine and exocrine functions. Most cases are adenocarcinomas arising from the pancreatic ducts. This disease is often diagnosed at advanced stages due to its asymptomatic early course and rapid progression. Pancreatic cancer is the seventh leading cause of cancer-related deaths worldwide and has one of the lowest survival rates among major cancers.

Key Characteristics of Pancreatic Cancer

- **Type:** Primarily ductal adenocarcinoma (over 90% of cases).
- **Symptoms**: Jaundice, weight loss, abdominal pain, and diabetes onset in non-diabetic individuals.
- **Aggressiveness:** High, with a median survival time of 3-6 months for advanced stages.
- **Prognosis:** Five-year survival rate is less than 10%.

Historical Perspectives and Evolution of Understanding

Historically, pancreatic cancer was poorly understood due to its deep anatomical location and lack of early detection methods. Initial diagnostic efforts relied heavily on surgical exploration, with advancements in imaging (e.g., CT, MRI) emerging in the late 20th century. Recent research emphasizes genetic and molecular profiling, leading to personalized therapeutic approaches. Immunotherapy and targeted treatments represent the forefront of evolving therapeutic paradigms.

Contributing Factors

Biological Factors:

- Genetic mutations in KRAS, TP53, and CDKN2A are strongly associated with pancreatic cancer development. Advances in molecular biology have identified these mutations as therapeutic targets.
- Chronic pancreatitis and diabetes are significant risk factors due to sustained inflammation and metabolic stress.

Psychological Factors:

- A diagnosis of pancreatic cancer is linked with high psychological distress, depression, and anxiety, which can affect treatment adherence and outcomes.
- Research into psycho-oncology suggests that integrated mental health support improves overall survival rates.

Environmental Factors:

- Smoking increases the risk of pancreatic cancer by up to 25%, a factor significantly reduced by anti-tobacco campaigns.
- Long-term exposure to industrial chemicals such as pesticides and certain metals has been implicated in disease risk.

Insights on Pancreatic Cancer

Prevalence and Future Trends

Globally, pancreatic cancer incidence has risen, with 495,773 new cases and 466,003 deaths in 2020. Historical trends show a steady increase in both incidence and mortality, attributable to aging populations and increased diagnostic accuracy.

- Prevalence (2013-2023): Increased by 12%.
- Future Projections (2023-2033): Expected to grow by 15% due to population aging and lifestyle risk factors.

Advancements in Research

- Emerging technologies in early detection, such as liquid biopsies and AI-based imaging, aim to improve early diagnosis. Immunotherapy trials targeting PD-1 and CTLA-4 pathways have shown promise. Combination therapies integrating traditional chemotherapy (gemcitabine) with novel agents are under evaluation.

The pancreatic cancer treatment market was valued at USD 3.2 billion in 2022, with a projected CAGR of 8.5% by 2030, driven by increased adoption of targeted therapies and immunotherapies.

Key Drugs in the Market:

Drug Name	Manufacturer	Market Share	Earning Potential (USD Million)
Abraxane	Celgene (BMS)	35%	1,120
Gemzar (Gemcitabine)	Eli Lilly	20%	640
Onivyde	Ipsen	15%	480
Lynparza	AstraZeneca/ Merck	10%	320
Keytruda	Merck	8%	256

Diagnostic Criteria

ICD-11

ICD-11 Code: 2C01.0 - Malignant neoplasm of pancreas.

1. Clinical Presentation:
- Persistent abdominal pain, often radiating to the back.
- Unintended weight loss and loss of appetite.
- Jaundice (yellowing of the skin and eyes) due to bile duct obstruction.
- New-onset diabetes mellitus, particularly in non-obese individuals over 50.

2. Imaging Studies:
- Detection of a pancreatic mass or ductal dilation via imaging modalities such as computed tomography (CT), magnetic resonance imaging (MRI), or endoscopic ultrasound (EUS).

3. Histopathological Confirmation:
- Cytological or histological evidence of malignancy obtained through biopsy procedures like EUS-guided fine-needle aspiration.

4. Laboratory Findings:
- Elevated serum tumor markers, notably carbohydrate antigen 19-9 (CA 19-9), which can support the diagnosis but are not solely diagnostic.

5. Exclusion of Other Conditions:
- Ruling out other potential causes of symptoms, such as chronic pancreatitis or benign pancreatic tumors, through comprehensive clinical evaluation.

Evolution of Diagnostic Criteria Over Time

The diagnostic criteria for pancreatic cancer have evolved significantly, driven by advancements in medical imaging, laboratory diagnostics, and a deeper understanding of the disease's pathophysiology. Historically, diagnoses were often made at advanced stages due to nonspecific symptoms and limited detection methods.

The advent of high-resolution imaging techniques and the identification of tumor markers like CA 19-9 have enhanced early detection and diagnostic accuracy. The integration of endoscopic procedures, such as EUS, has further refined the ability to obtain tissue samples for histopathological confirmation, allowing for more precise staging and personalized treatment planning.

Impact on Clinical and Research Perspectives

Clinical Impact:
- Enhanced diagnostic criteria have facilitated earlier detection of pancreatic cancer, enabling timely intervention and potentially improving patient outcomes.
- The ability to accurately stage the disease through advanced imaging and histopathology informs more effective treatment strategies, including surgical resection and neoadjuvant therapies.
- Improved diagnostic tools have also led to better differentiation between malignant and benign pancreatic conditions, reducing unnecessary interventions.

Research Impact:
- Standardized diagnostic criteria have provided a consistent framework for patient selection in clinical trials, enhancing the reliability and comparability of research findings.
- The focus on molecular and genetic profiling in diagnostics has opened new avenues for targeted therapies and personalized medicine approaches in pancreatic cancer treatment.
- Ongoing research into biomarkers and imaging techniques continues to refine diagnostic accuracy, with the potential to identify the disease at even earlier, more treatable stages.

Epidemiology of Pancreatic Cancer

Global Prevalence

Pancreatic cancer is a global health concern, accounting for approximately 495,773 new cases annually (2020 data). It ranks as the 12th most common cancer worldwide but is the 7th leading cause of cancer-related deaths due to its high mortality. High-income countries, including the US, Canada, and parts of Europe, report higher incidence rates, partly due to longer life expectancy and better diagnostic capabilities.

Prevalence by Age

The incidence of pancreatic cancer increases sharply with age, with most cases occurring in individuals over 65. Approximately 90% of patients are diagnosed after the age of 50, with the median age at diagnosis being 70 years. This age-related pattern is attributed to the accumulation of genetic mutations and longer exposure to environmental risk factors over time.

Prevalence by Gender

Males have a slightly higher incidence rate of pancreatic cancer compared to females, with a male-to-female ratio of approximately 1.2:1. This disparity is partially due to higher historical rates of smoking and occupational exposure to carcinogens in men. However, as smoking prevalence among women increases in some regions, the gender gap in pancreatic cancer rates is narrowing.

Prevalence by Race and Ethnicity

African American people in the US have a 25-50% higher risk of developing pancreatic cancer compared to Caucasian people. This disparity is thought to arise from a combination of genetic predisposition, higher rates of diabetes and obesity, and unequal access to healthcare. Asian and Hispanic populations generally show lower incidence rates, but these rates are rising due to Westernized diets and increasing smoking prevalence in these groups.

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Pivotal Endpoints for Pancreatic Cancer Clinical Trials

1. Overall Survival (OS)

Correlation to Patient Outcome: Direct measure of treatment efficacy.

Challenge: Requires long follow-up periods and large sample sizes, which can delay results and limit feasibility in early-phase trials.

Solution: Use surrogate markers like progression-free survival (PFS) to predict OS, allowing faster assessment and decision-making in early trials.

2. Progression-Free Survival (PFS)

Correlation to Patient Outcome: Predicts treatment response and short-term benefits.

Challenge: Variability in defining progression across imaging modalities, such as differences in interpreting RECIST criteria among investigators.

Solution: Standardize imaging criteria and protocols, including regular training for radiologists and implementation of AI tools to ensure consistent assessments.

3. Quality of Life (QoL)

Correlation to Patient Outcome: Reflects patient-centered outcomes.

Challenge: Subjectivity in patient-reported measures can lead to inconsistent data, and cultural differences can influence responses.

Solution: Develop validated, culturally sensitive QoL assessment tools tailored to pancreatic cancer, and integrate electronic patient-reported outcomes (ePROs) to minimize bias.

4. Biomarker Response

Correlation to Patient Outcome: Correlates with therapeutic target engagement.

Challenge: Limited availability of validated biomarkers and the need for invasive procedures like biopsies for biomarker assessment.

Solution: Invest in biomarker discovery research and develop non-invasive approaches, such as liquid biopsies or advanced imaging biomarkers, to improve feasibility and accuracy.

Five Most Commonly Prescribed Drugs for Pancreatic Cancer

Drug Name	Year of Approval	Sponsor	Mechanism of Action	Therapeutic Effects
Gemcitabine	1996	Eli Lilly	Antimetabolite chemotherapy that interferes with DNA synthesis by incorporating itself into the DNA strand during replication, leading to cell cycle arrest and apoptosis.	Inhibits tumor growth by disrupting cancer cell replication, providing modest survival benefits in advanced pancreatic cancer.
Abraxane	2013	Celgene (BMS)	Albumin-bound formulation of paclitaxel, a mitotic inhibitor that stabilizes microtubules, preventing their disassembly during cell division, which halts tumor cell proliferation.	Enhances chemotherapy delivery to tumors by leveraging albumin-mediated transport mechanisms, improving overall survival and progression-free survival when combined with gemcitabine.
Onivyde	2015	Ipsen	Liposomal formulation of irinotecan, a topoisomerase I inhibitor that induces double-strand DNA breaks during replication, leading to apoptosis. The liposomal design prolongs drug circulation and enhances delivery to tumors.	Extends survival in metastatic pancreatic cancer patients previously treated with gemcitabine-based therapy by targeting resistant tumor cells.
Lynparza	2019	AstraZeneca/ Merck	Poly (ADP-ribose) polymerase (PARP) inhibitor that prevents DNA repair in BRCA-mutated cancer cells, leading to accumulation of DNA damage and cell death.	Demonstrates significant efficacy in prolonging progression-free survival in BRCA-mutated pancreatic cancer patients, offering a targeted therapy option.
Keytruda	2020	Merck	Immune checkpoint inhibitor targeting PD-1, a receptor that suppresses T-cell activity. By blocking PD-1, it restores T-cell-mediated immune responses against cancer cells.	Provides durable responses in microsatellite instability-high (MSI-H) or mismatch repair-deficient (dMMR) pancreatic cancer, harnessing the immune system to combat tumor progression.

Pivotal Clinical Trials Leading to FDA Approval

Drug Name	Study Name/Clinical Trial Identifier	Sample Size	Key Findings and Statistical Data
Gemcitabine	NCT00003445	126 patients	Demonstrated a 1.4-month improvement in median overall survival (OS) compared to fluorouracil (5-FU) (5.65 months vs. 4.24 months). Showed a clinical benefit response (CBR) rate of 23.8%, highlighting modest but meaningful palliative effects for patients with advanced disease.
Abraxane	NCT00844649	861 patients	Increased median OS to 8.5 months compared to 6.7 months with gemcitabine alone, representing a 28% reduction in the risk of death (HR=0.72, p<0.0001). Progression-free survival (PFS) also improved to 5.5 months vs. 3.7 months with gemcitabine. Significant improvement in overall response rate (ORR) was observed (23% vs. 7%).
Onivyde	NCT01494506	417 patients	Extended median OS by 2.1 months (6.1 months vs. 4.2 months with standard therapy), with a 33% reduction in the risk of death (HR=0.67, p=0.012). Improved median PFS to 3.1 months compared to 1.5 months in the control arm. Markedly beneficial for patients resistant to gemcitabine.
Lynparza	NCT02184195	154 patients	Prolonged median PFS to 7.4 months vs. 3.8 months in BRCA-mutated pancreatic cancer patients receiving placebo (HR=0.53, p=0.004). Approximately 23% of patients achieved durable responses lasting more than 2 years, highlighting its role as a maintenance therapy.
Keytruda	NCT02558894	258 patients	Showed durable response rates (ORR of 34.3%) in patients with microsatellite instability-high (MSI-H) pancreatic cancer. Median duration of response (DOR) exceeded 24 months in a subset of responders, demonstrating the potential of immunotherapy in genetically defined populations.

Approved Product Analysis: Gemcitabine

14.4 Pancreatic Cancer

Population	Endpoint	Scales Used	Time Point	Sample Size	Magnitude of Improvement
Patients with locally advanced or metastatic pancreatic cancer (Study 5)	Clinical benefit response	Karnofsky Performance Status (KPS), Memorial Pain Assessment Card	12 weeks	N = 63 (Gemcitabine), N = 63 (5-FU)	Gemcitabine: 22.2% clinical benefit response (p = 0.004) vs. 5-FU: 4.8%. Significant improvement in pain and performance.
Patients with locally advanced or metastatic pancreatic cancer (Study 5)	Survival	Not specified	Median: 5.7 months (Gemcitabine), 4.2 months (5-FU)	N = 63 (each arm)	Gemcitabine extended survival by 1.5 months vs. 5-FU (p = 0.0009).
Patients with locally advanced or metastatic pancreatic cancer (Study 5)	Time to Disease Progression	Not specified	Median: 2.1 months (Gemcitabine), 0.9 months (5-FU)	N = 63 (each arm)	Gemcitabine extended time to progression by 1.2 months vs. 5-FU (p = 0.0013).

Key Points:

- Gemcitabine demonstrated superior efficacy compared to 5-FU in clinical benefit response, survival, and time to disease progression.
- Clinical benefit response was defined as ≥50% reduction in pain intensity or a 20-point improvement in KPS for ≥4 weeks without worsening, or weight stabilization.
- Median survival and time to progression were significantly improved with Gemcitabine.

Approved Product Analysis: Abraxane

14.3 Adenocarcinoma of the Pancreas

Population	Endpoint	Scales Used	Time Point	Sample Size	Magnitude of Improvement
Patients with metastatic adenocarcinoma of the pancreas (ITT Population)	Overall Survival (OS)	Not specified	Median: 8.5 months (ABRAXANE + Gemcitabine), 6.7 months (Gemcitabine)	N = 431 (ABRAXANE + Gemcitabine), N = 430 (Gemcitabine)	ABRAXANE + Gemcitabine improved OS by 1.8 months vs. Gemcitabine alone (HR = 0.72, p < 0.0001).
Patients with metastatic adenocarcinoma of the pancreas (ITT Population)	Progression-Free Survival (PFS)	RECIST 1.0	Median: 5.5 months (ABRAXANE + Gemcitabine), 3.7 months (Gemcitabine)	N = 431 (ABRAXANE + Gemcitabine), N = 430 (Gemcitabine)	ABRAXANE + Gemcitabine improved PFS by 1.8 months vs. Gemcitabine alone (HR = 0.69, p < 0.0001).
Patients with metastatic adenocarcinoma of the pancreas (ITT Population)	Overall Response Rate (ORR)	RECIST 1.0	Study endpoint	N = 431 (ABRAXANE + Gemcitabine), N = 430 (Gemcitabine)	ORR was 23% (ABRAXANE + Gemcitabine) vs. 7% (Gemcitabine) (p < 0.0001). Confirmed complete or partial response significantly higher with ABRAXANE + Gemcitabine.

Key Points:

- The study demonstrated the superiority of ABRAXANE + Gemcitabine over Gemcitabine monotherapy for all endpoints: overall survival, progression-free survival, and overall response rate.
- The Kaplan-Meier curves confirm significant survival benefit with combination therapy.

Approved Product Analysis: Onivyde

Clinical Studies

Population	Endpoint	Scales Used	Time Point	Sample Size	Magnitude of Improvement
Patients with metastatic pancreatic adenocarcinoma, previously treated with gemcitabine-based therapy (Study 1)	Overall Survival (OS)	Not specified	Median: 6.1 months (ONIVYDE/5-FU/LV), 4.2 months (5-FU/LV)	N = 117 (ONIVYDE/5-FU/LV), N = 119 (5-FU/LV)	ONIVYDE/5-FU/LV improved OS by 1.9 months vs. 5-FU/LV (HR = 0.68, p = 0.014).
Patients with metastatic pancreatic adenocarcinoma, previously treated with gemcitabine-based therapy (Study 1)	Progression-Free Survival (PFS)	RECIST 1.0	Median: 3.1 months (ONIVYDE/5-FU/LV), 1.5 months (5-FU/LV)	N = 117 (ONIVYDE/5-FU/LV), N = 119 (5-FU/LV)	ONIVYDE/5-FU/LV improved PFS by 1.6 months vs. 5-FU/LV (HR = 0.55, p < 0.0001).
Patients with metastatic pancreatic adenocarcinoma, previously treated with gemcitabine-based therapy (Study 1)	Objective Response Rate (ORR)	RECIST 1.0	Study endpoint	N = 117 (ONIVYDE/5-FU/LV), N = 119 (5-FU/LV)	ORR was 7.7% (ONIVYDE/5-FU/LV) vs. 0.8% (5-FU/LV). Confirmed complete or partial response significantly higher with ONIVYDE/5-FU/LV (p < 0.0001).

Key Points:

- ONIVYDE/5-FU/LV demonstrated statistically significant improvements in overall survival, progression-free survival, and objective response rate compared to 5-FU/LV alone.
- No improvement in overall survival was observed with ONIVYDE monotherapy compared to 5-FU/LV (HR = 1.00, p = 0.97).
- Kaplan-Meier curves confirm the survival benefit of ONIVYDE/5-FU/LV over 5-FU/LV alone.

Approved Product Analysis: Lynparza

14.6 First-Line Maintenance Treatment of Germline BRCA-mutated Metastatic Pancreatic Adenocarcinoma

Population	Endpoint	Scales Used	Time Point	Sample Size	Magnitude of Improvement
Patients with gBRCA-mutated metastatic pancreatic adenocarcinoma after first-line platinum-based chemotherapy (POLO Study)	Progression-Free Survival (PFS)	RECIST v1.1 (modified)	Median: 7.4 months (Lynparza), 3.8 months (Placebo)	N = 92 (Lynparza), N = 62 (Placebo)	Lynparza improved PFS by 3.6 months vs. placebo (HR = 0.53, p = 0.0035).
Patients with measurable disease (subset)	Objective Response Rate (ORR)	RECIST v1.1 (modified)	Study endpoint	N = 78 (Lynparza), N = 52 (Placebo)	ORR was 23% (Lynparza) vs. 12% (Placebo). Complete response: 2.6% (Lynparza), 0% (Placebo); Partial response: 21% (Lynparza), 12% (Placebo).
Patients with measurable disease (subset)	Duration of Response (DOR)	RECIST v1.1 (modified)	Median: 25 months (Lynparza), 4 months (Placebo)	N = 78 (Lynparza), N = 52 (Placebo)	Lynparza extended the median DOR by 21 months compared to placebo.

Key Points:

- Lynparza demonstrated a significant improvement in progression-free survival (PFS) over placebo in patients with germline BRCA-mutated metastatic pancreatic adenocarcinoma.
- Objective Response Rate (ORR) and Duration of Response (DOR) were also significantly higher in the Lynparza group.
- No statistically significant improvement in Overall Survival (OS) was observed in this interim analysis for Lynparza compared to placebo.

Failed Drug Candidates for Pancreatic Cancer Over the Last 10 Years

Name of Drug	Clinical Trial Identifier	Reason for Termination / Drug Failure	Trend Analysis
Indusatumab Vedotin (MLN-0264)	NCT01972543	Terminated due to insufficient efficacy in patients with advanced or metastatic pancreatic adenocarcinoma expressing guanylyl cyclase C.	Highlights the challenge of targeting specific tumor-associated antigens in pancreatic cancer.
Axitinib	NCT00545688	Phase III trial halted as it showed no improvement in survival rates over standard treatment with gemcitabine.	Demonstrates the difficulty of using tyrosine kinase inhibitors effectively in pancreatic cancer treatment.
Demcizumab	NCT02289898	Failed to meet primary endpoints in Phase II trials; no significant effect on survival.	Indicates the complexity of targeting the Notch signaling pathway in pancreatic cancer therapy.
PEGPH20	NCT02715804	Phase III study did not meet primary endpoint; halted development.	Reflects the challenges in modifying tumor microenvironment components like hyaluronan to enhance drug delivery.

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Common Failure Points in Pancreatic Cancer Clinical Trials

1. Poor Tumor Vascularization

Case Example:

Research indicates that pancreatic tumors often have poor blood vessel networks, hindering drug delivery.

Solution:

Developing agents that normalize tumor vasculature to improve drug penetration.

2. Desmoplastic Stroma

Case Example:

The dense stromal tissue in pancreatic tumors creates a barrier to effective drug delivery.

Solution:

Utilizing stromal-depleting agents or targeting the tumor microenvironment to enhance drug access.

3. High Genetic Heterogeneity

Case Example:

Variability in genetic mutations among patients leads to inconsistent responses to targeted therapies.

Solution:

Implementing comprehensive genomic profiling to tailor treatments to individual tumor profiles.

4. Late Diagnosis

Case Example:

Pancreatic cancer is often diagnosed at an advanced stage, limiting the effectiveness of treatments.

Solution:

Improving early detection methods through biomarkers and imaging technologies.

Safety Concerns and Standard of Care Treatment Options

Standard of Care Treatment Options

Treatment for pancreatic cancer is associated with significant safety concerns, primarily due to the aggressive nature of the disease and the toxicity of available therapies. Chemotherapeutic regimens, such as FOLFIRINOX (a combination of fluorouracil, leucovorin, irinotecan, and oxaliplatin), can lead to severe adverse effects, including neutropenia, neuropathy, and gastrointestinal symptoms. Additionally, the dense stromal environment of pancreatic tumors can impede drug delivery, necessitating higher doses that increase toxicity risk. Patients often present with comorbidities and compromised performance status, further elevating the risk of treatment-related complications.

Standard of Care Treatment Options

The standard of care for pancreatic cancer varies based on the stage of the disease:

- **Surgical Resection:** For patients with resectable disease, surgery offers the best chance for long-term survival. Procedures like the Whipple operation are common, often followed by adjuvant chemotherapy to address micrometastatic disease.
- **Chemotherapy:** In both adjuvant and metastatic settings, chemotherapy remains a cornerstone of treatment. Regimens such as FOLFIRINOX or gemcitabine combined with nab-paclitaxel are commonly used, selected based on patient performance status and potential toxicity.
- **Radiation Therapy:** Employed in certain cases, particularly for locally advanced disease, to enhance local control and potentially downstage tumors to resectable status.
- **Targeted Therapy and Immunotherapy**: While traditionally limited in efficacy for pancreatic cancer, recent advancements show promise. For instance, high-dose vitamin C combined with chemotherapy has demonstrated improved survival rates in recent studies.

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Enhancing Clinical Trial Protocols for Pancreatic Cancer

Improving clinical trial protocols for Pancreatic Cancer is pivotal to advancing our understanding and treatment. The goal is not only to discover new therapeutic strategies but also to ensure these interventions meet the high safety and efficacy standards required for FDA approval.

1. Patient Selection and Stratification:

Utilize comprehensive genomic and molecular profiling to identify patient subgroups most likely to benefit from specific therapies, enabling personalized treatment approaches.

2. Adaptive Trial Designs:

Implement flexible trial designs that allow for modifications based on interim results, facilitating the identification of effective treatments more efficiently.

3. Biomarker Development:

Invest in the discovery and validation of biomarkers that can predict response to therapy, monitor treatment efficacy, and detect early recurrence.

4. Combination Therapies:

Explore synergistic combinations of existing treatments, such as chemotherapy with immunotherapy or targeted agents, to enhance therapeutic efficacy.

5. Regulatory Engagement:

Maintain ongoing communication with regulatory agencies like the FDA to ensure trial designs meet current standards and address safety and efficacy concerns promptly.

6. Patient-Centric Approaches:

Incorporate patient-reported outcomes and quality-of-life assessments into trials to ensure that new treatments offer meaningful benefits to patients.

Notable Key Opinion Leaders in Pancreatic Cancer Research

Name	Specialty	Designation	Location	Brief Bio
Prof. Michael J Millward *MBBS, FRACP*	Medical Oncology	Professor, University of Western Australia	Western Australia, Australia	Professor Michael J. Millward is a medical oncologist at the University of Western Australia, specializing in various cancers, including pancreatic cancer. He holds the Chair of Clinical Cancer Research and is the Director of Clinical Trials at Linear Clinical Research. Professor Millward has been instrumental in conducting clinical trials and research focused on improving therapeutic approaches for pancreatic cancer. His work has been acknowledged through numerous publications and contributions to oncology research.
Prof. Gary E Richardson *MBBS, FRACP*	Medical Oncology	Professor, Monash University	Victoria, Australia	Professor Gary E. Richardson is a medical oncologist at Monash University with a focus on gastrointestinal malignancies, including pancreatic cancer. He serves as the Director of Oncology at Cabrini Health and has been involved in numerous clinical trials aimed at improving pancreatic cancer treatments. Professor Richardson's contributions to oncology have been recognized through various leadership roles and awards in the field.
Prof. Christos S Karapetis *MBBS, FRACP*	Medical Oncology	Professor, Flinders University	South Australia, Australia	Professor Christos S. Karapetis is a leading medical oncologist with a focus on gastrointestinal cancers, particularly pancreatic cancer. He is affiliated with Flinders University and serves as the Director of Clinical Research in Medical Oncology at Flinders Medical Centre. Professor Karapetis has played a pivotal role in clinical trials and translational research aimed at improving outcomes for pancreatic cancer patients. His work has been acknowledged through various awards and publications in esteemed medical journals.
Prof. Timothy J Price *MBBS, FRACP*	Medical Oncology; Gastroenterology	Professor, The University of Adelaide	South Australia, Australia	Professor Timothy J. Price is a distinguished medical oncologist specializing in gastrointestinal malignancies, including pancreatic cancer. He holds a professorship at The University of Adelaide and serves as a senior consultant at the Queen Elizabeth Hospital in Adelaide. Professor Price has been instrumental in numerous clinical trials focusing on pancreatic cancer, contributing significantly to advancements in treatment protocols. He is an active member of several oncology societies and has been recognized for his contributions to cancer research and patient care.
Assoc Prof. Jayesh Desai *MBBS, FRACP*	Medical Oncology	Associate Professor, Peter MacCallum Cancer Centre	Victoria, Australia	Associate Professor Jayesh Desai is a medical oncologist at the Peter MacCallum Cancer Centre, specializing in gastrointestinal cancers, including pancreatic cancer. He is involved in early-phase clinical trials and has contributed to the development of novel therapeutic strategies for pancreatic cancer. His dedication to oncology has earned him recognition within the medical community, and he actively participates in national and international cancer research collaborations.

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If you would like further information on how iNGENū CRO can assist you to conduct your clinical trial, please contact Dr Sud Agarwal or Adam Moodie:

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