

CHEMOTHERAPY STRATEGIES IN METASTATIC PANCREATIC DUCTAL ADENOCARCINOMA (mPDAC): SECOND-LINE CHEMOTHERAPY OPTIONS

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DEVELOPED BY GI CONNECT

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EDUCATIONAL OBJECTIVES

1. Be able to differentiate the **efficacy and safety profiles** of chemotherapies for mPDAC and understand how **mode of delivery** plays a role in this
2. Be able to recognise the **cause of toxicities** and have an awareness of strategies that can be used to improve tolerability and manage side effects whilst maintaining optimal efficacy

CLINICAL TAKEAWAYS

- Pancreatic ductal adenocarcinoma (PDAC) is usually diagnosed at an advanced, incurable stage due to non-specific symptoms and has an extremely poor prognosis
- Systemic chemotherapy is the standard treatment for metastatic PDAC but molecularly targeted treatments and immunotherapies may have a role for specific patients
- Treatment selection depends on several factors, including patients' performance status and co-morbidities. These should be considered alongside the efficacy and safety profiles of the different chemotherapy regimens
- Treatment strategies can be implemented to manage toxicities associated with the different chemotherapy regimens to enable a patient to stay on treatment for optimal efficacy

OVERVIEW OF TREATMENT FOR mPDAC

- Chemotherapy is the mainstay of treatment for mPDAC patients
- Enrolment on clinical trials should always be encouraged

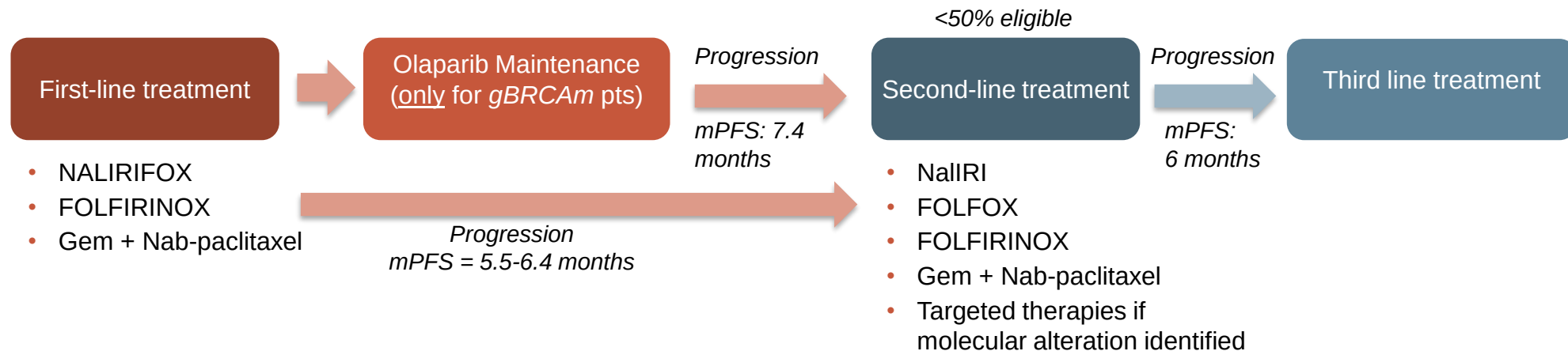


Figure adapted from Casolino 2022

FOLFIRINOX, folinic acid (leucovorin calcium), fluorouracil, irinotecan, and oxaliplatin; FOLFOX, folinic acid (leucovorin calcium), fluorouracil, and oxaliplatin; gBRCAm, germline BReast CAncer (BRCA) gene mutation; gem, gemcitabine; mPDAC, metastatic pancreatic ductal adenocarcinoma; mPFS, median progression-free survival; Nab, nanoparticle albumin-bound; Nal-IRI, nanoliposomal irinotecan; NALIRIFOX; Nal-IRI, fluorouracil/folinic acid (leucovorin calcium), and oxaliplatin; mPDAC, metastatic pancreatic adenocarcinoma; mPFS, median progression-free survival

KEY STUDIES OF 2L SYSTEMIC THERAPY FOR MPDAC

Study setting	Study	Study type	Arm (N)	Primary endpoint	Primary endpoint		Secondary endpoint	Secondary endpoint		ORR (%)	Notable adverse events
					Months	HR (95% CI)		Months	HR (95% CI)		
Second-line	CONKO-003 ¹ (2014)	RCT, phase 3	OFF (77)	OS	5.9	0.66 (0.48-0.91)	PFS	2.9	0.68 (0.50-0.94)	–	Rates of adverse events were similar between treatment arms, with the exception of grades 1 to 2 neurotoxicity 38.2% and 7.1% in the OFF and FF groups
			FF (91)		3.3			2.0		–	
Second-line	PANCREOX ² (2016)	RCT, phase 3	mFOLFOX (54)	PFS	3.0	1.00 (0.66-1.53)	OS	6.1	1.78 (1.08-2.93)	13.2	Increased toxicity was observed with the addition of oxaliplatin, with grade 3/4 adverse events occurring in 63.0% of patients who received mFOLFOX6 and 11.0% of those who received FU/LV.
			5FU/LV (54)		2.8			9.9		8.5	
Second-line	NAPOLI-1 ³ (2016)	RCT, phase 3	Nal-IRI + 5-FU/LV (117)	OS	6.1	0.67 (0.49 to 0.92)	PFS	3.1	0.56 (0.41 to 0.75)	16.2	Most frequent grade 3 or 4 AEs for Nal-IRI + 5-FU/LV vs 5-FU/LV: neutropenia 27.0 vs 1.0%, diarrhoea 13.0 vs 4.0%, vomiting 11.0 vs 3.0%, and fatigue 14.0 vs 4.0%
			5-FU/LV (119)		4.2			1.5		0.8	

5-FU, fluorouracil; AE, adverse event; CI, confidence interval; FF, folinic acid (leucovorin calcium) and fluorouracil; HR, hazard ratio; LV, leucovorin calcium (folinic acid); mFOLFOX, modified FOLFOX: folinic acid (leucovorin calcium), fluorouracil, and oxaliplatin; mPDAC, metastatic pancreatic ductal adenocarcinoma; Nal-IRI, nanoliposomal irinotecan; OFF, oxaliplatin and FF; ORR, objective response rate; OS, overall survival; PFS, progression-free survival; RCT, randomised controlled trial

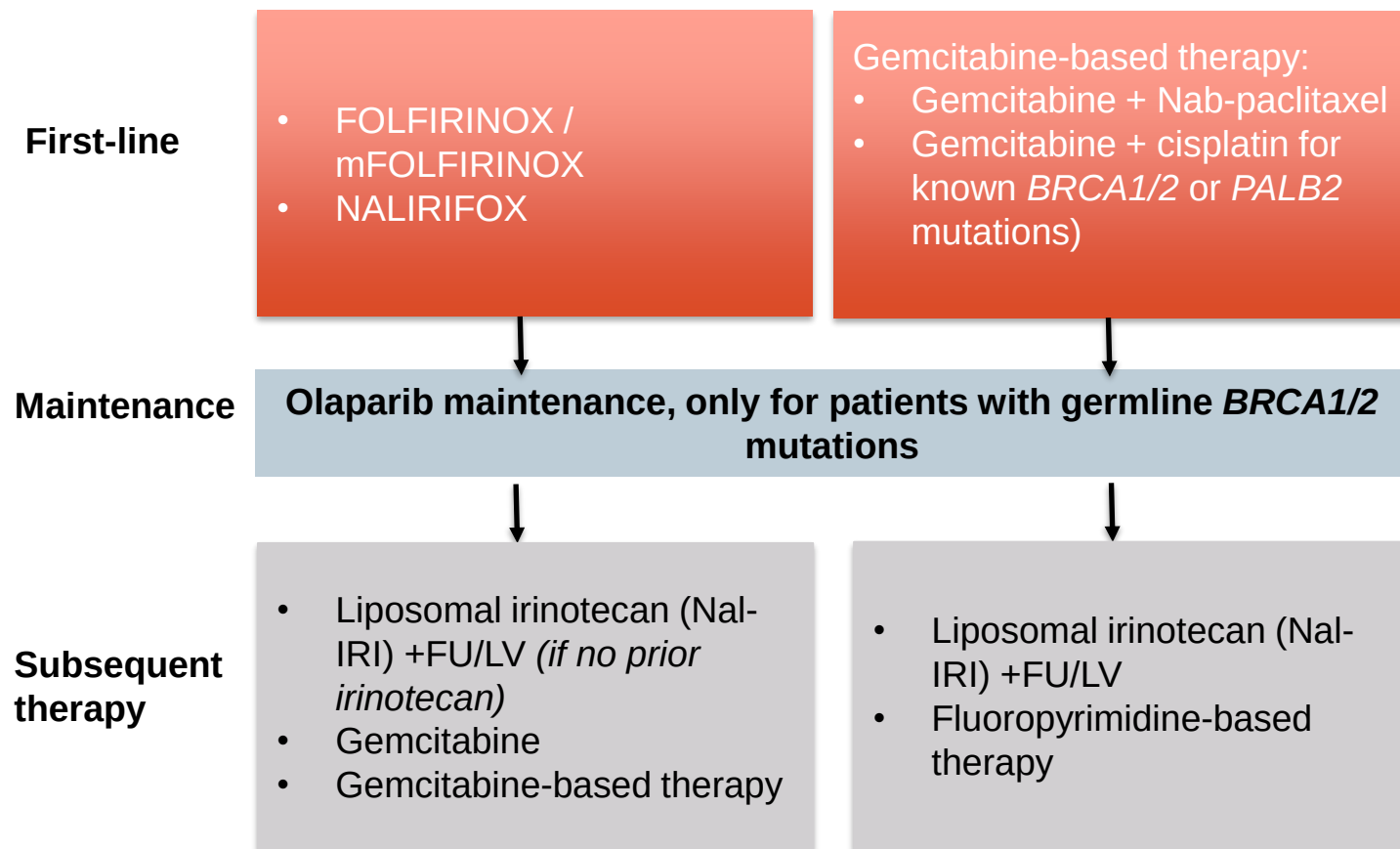
1. Oettle H, et al. J Clin Oncol. 2014; 32: 2423-9; 2. Gill S, et al. J Clin Oncol. 2016;10;3914-20; 3. Wang-Gillam A, et al. Lancet 2016;387:545-57

CONSIDERATIONS FOR TREATMENT SELECTION

- Prior treatments
- Patient performance status
- Age and frailty
- Co-morbidities
- Residual side effects from prior treatments
- Molecular profile
- Patient preference
- Supportive system

SYSTEMIC THERAPIES FOR GOOD PS 0-1

BASED ON NCCN GUIDELINES



Targeted therapies Useful in certain circumstances

- Entrectinib, Larotrectinib, Repotrectinib (*NTRK* fusion)
- Dabrafenib + trametinib (*BRAF* V600E mutation)
- Selpercatinib (*RET* fusion)
- Pembrolizumab (MSI-H, dMMR, or TMB-H)

Chemotherapy is recommended as front-line therapy for mPDAC patients, but targeted therapies may be useful in certain circumstances (e.g. if a patient can no longer tolerate chemotherapy)

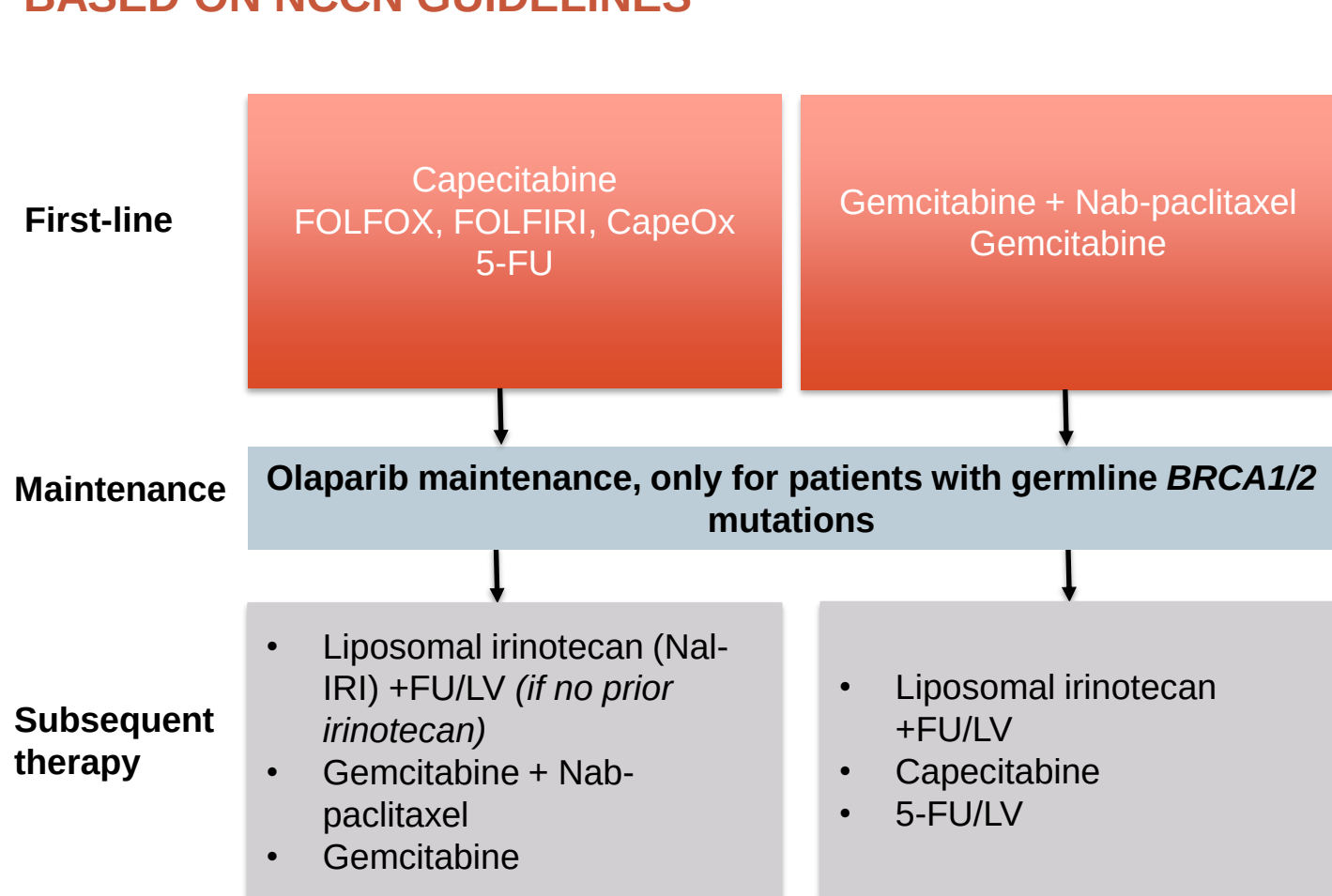
- Entrectinib, Larotrectinib, Repotrectinib (*NTRK* fusion)
 - Dabrafenib + trametinib (*BRAF* V600E mutation)
 - Selpercatinib (*RET* fusion)
 - Adagrasib, sotorasib (*KRAS* G12C)
 - Trastuzumab deruxtecan (*HER2*)
- If no prior immunotherapy:**
- Pembrolizumab (MSI-H, dMMR, or TMB-H)
 - Dostarlimab (MSI-H or dMMR)
 - Nivolumab + ipilimumab (TMB-H)

5-FU, fluorouracil; BRAF, B-Raf proto-oncogene serine/threonine kinase; *BRCA1/2*, BReast Cancer 1/2 gene; dMMR, deficient DNA mismatch repair; *HER2*, human epidermal growth factor receptor 2; *KRAS*, Kirsten rat sarcoma viral oncogene homolog; LV, leucovorin calcium (folinic acid); (m)FOLFIRINOX, (modified) FOLFIRINOX, folinic acid (leucovorin calcium), fluorouracil, irinotecan, and oxaliplatin; MSI-H, microsatellite instability-high; Nab, nanoparticle albumin-bound; Nal-IRI, nanoliposomal irinotecan; NALIRIFOX; Nal-IRI, fluorouracil/folinic acid (leucovorin calcium), and oxaliplatin; NCCN, National Comprehensive Cancer Network; NRG1, neuregulin-1; NTRK, neurotrophic tyrosine receptor kinase; *PALB2*, partner and localiser of *BRCA2*; PS, performance status; RET, rearranged during transfection; TMB-H, tumour mutational burden-high

NCCN Guidelines Version 3.2024. Available at: <https://www.nccn.org/guidelines/guidelines-detail?category=1&id=1455>. Accessed October 2024 NCCN guidelines for pancreatic adenocarcinoma, Version 3.2024: [pancreatic.pdf \(nccn.org\)](#)

SYSTEMIC THERAPIES FOR INTERMEDIATE PS 2 OR HIGHER

BASED ON NCCN GUIDELINES



Targeted therapies Useful in certain circumstances

- Entrectinib, Larotrectinib, Repotrectinib (*NTRK* fusion)
- Dabrafenib + trametinib (*BRAF* V600E mutation)
- Selpercatinib (*RET* fusion) [ECOG PS 2 only for first-line]
- Pembrolizumab (MSI-H, dMMR, or TMB-H)

Chemotherapy is recommended as front-line therapy for mPDAC patients, but targeted therapies may be useful in certain circumstances (e.g. if a patient can no longer tolerate chemotherapy)

- Entrectinib, Larotrectinib, Repotrectinib (*NTRK* fusion)
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 - Selpercatinib (*RET* fusion) [ECOG PS 2 only for first-line]
 - Adagrasib, sotorasib (*KRAS* G12C)
- If no prior immunotherapy:**
- Pembrolizumab (MSI-H, dMMR, or TMB-H)
 - Dostarlimab (MSI-H or dMMR)
 - Nivolumab + ipilimumab (TMB-H)

5-FU, fluorouracil; BRAF, B-Raf proto-oncogene serine/threonine kinase; BRCA1/2, BReast CAncer 1/2 gene; CapeOX, capecitabine and oxaliplatin; dMMR, deficient DNA mismatch repair; FOLFOX, folinic acid (leucovorin calcium), fluorouracil, and oxaliplatin; FOLFIRI, folinic acid (leucovorin calcium), fluorouracil and irinotecan; KRAS, Kirsten rat sarcoma viral oncogene homolog; LV, leucovorin calcium (folinic acid); MSI-H, microsatellite instability-high; Nab, nanoparticle albumin-bound; NCCN, National Comprehensive Cancer Network; NRG1, neuregulin-1; NTRK, neurotrophic tyrosine receptor kinase; PALB2, partner and localiser of BRCA2; PS, performance status; RET, rearranged during transfection; TMB-H, tumour mutational burden-high

PATIENTS WITH POOR PERFORMANCE STATUS AND PROGRESSIVE DISEASE

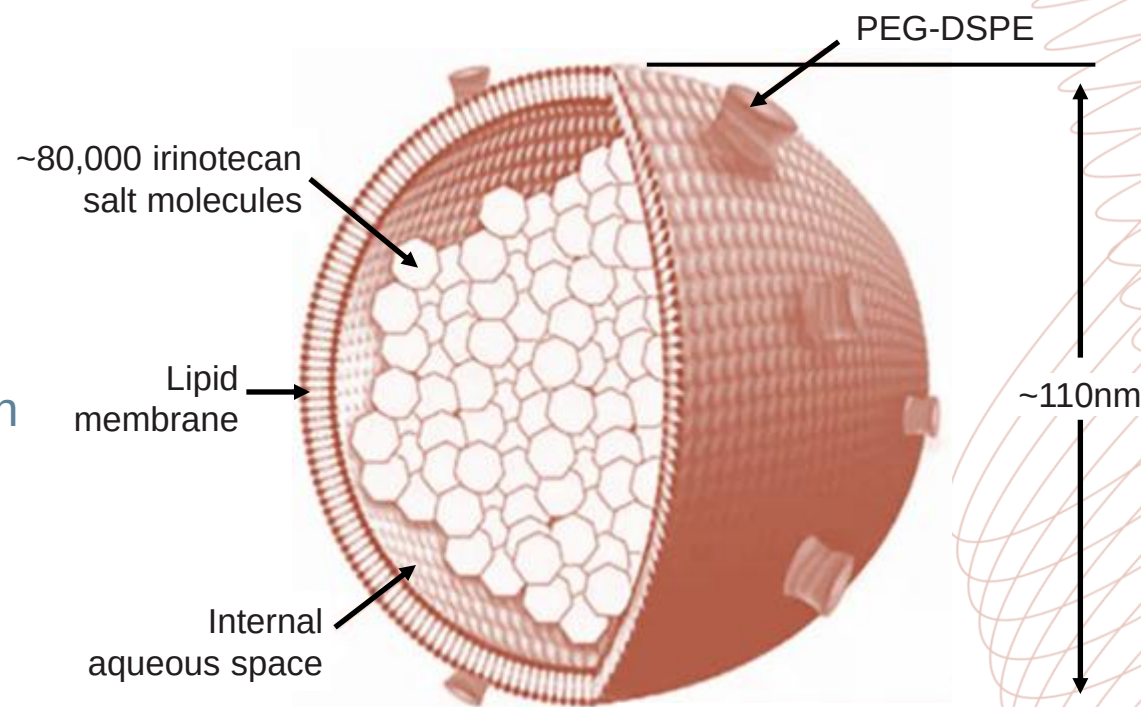
PALLIATIVE CARE

- **Single agent chemotherapy** (*gemcitabine*)
- **Targeted therapy** (*based on molecular profiling and as clinically indicated*)
- **Palliative radiotherapy**
 - To relieve pain, bleeding and/or local obstructive symptoms

HOW IS NANOLIPOSOMAL IRINOTECAN (NaI-IRI) DIFFERENT TO IRINOTECAN?

- **Nanoliposomal irinotecan:** irinotecan encapsulated in liposome nanoparticles¹
- Liposome shelters irinotecan from conversion to its active metabolite (SN-38) thereby remaining in the circulation for longer than free (unencapsulated) irinotecan¹⁻³
- Leads to increases and prolonged intratumoural levels of both irinotecan and SN-38 compared with free irinotecan¹
- Median OS of 5.2 months for NaI-IRI in a phase 2 study of gemcitabine-refractory metastatic pancreatic cancer^{1,4}

Liposomal irinotecan⁵



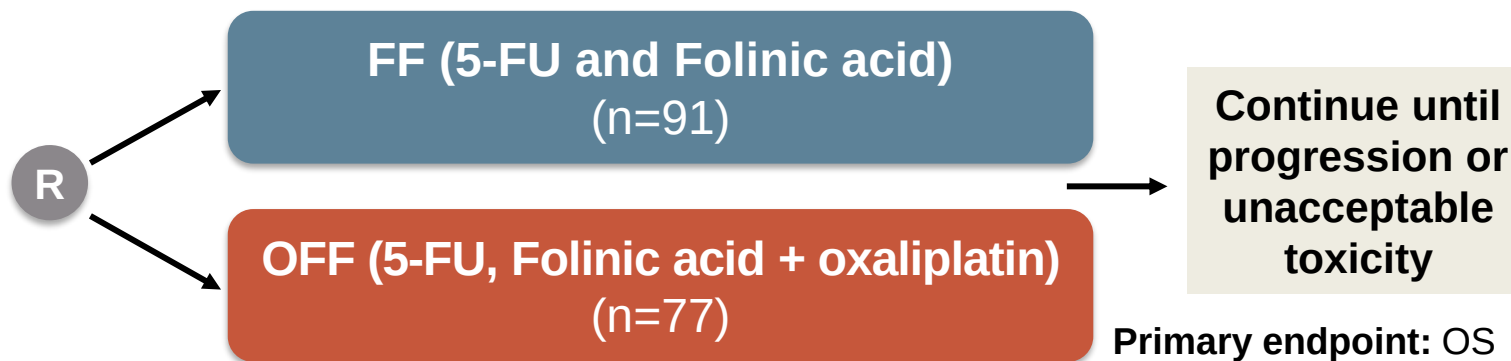
NaI-IRI, nanoliposomal irinotecan; OS, overall survival; PEG-DSPE, polyethylene glycol-distearoylphosphatidylethanolamine

1. Wang-Gillam A, et al. Lancet 2016;387:545-57; 2. Kalra AV, et al. Cancer Res. 2014;74:7003-13; 3. Roy AC, et al. Ann Oncol. 2013;24: 1567-73; 4. Ko AH, et al. Br J Cancer. 2013;109:920-25; 5. Image: Camptothecin & Its Derivatives for Cancer Therapy | Biopharma PEG. Available at: <https://www.biochempeg.com/article/310.html>. Accessed July 2024

OXALIPLATIN PHASE 3 SECOND-LINE STUDIES

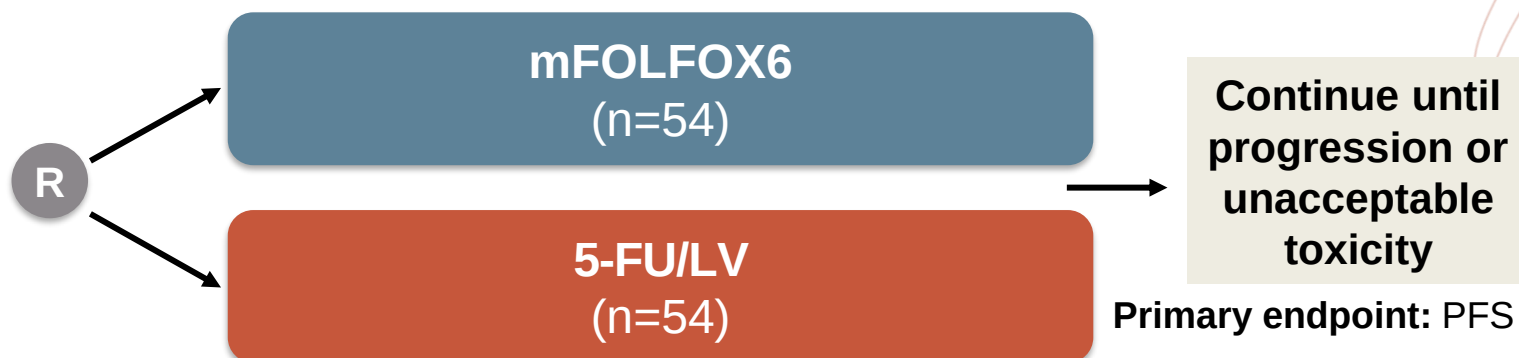
CONKO-003¹

Patients with advanced pancreatic cancer previously treated with gemcitabine, with a KPS $\geq$ 70%
N=168



PANCREOX²

Patients with advanced pancreatic cancer previously treated with gemcitabine, with an ECOG PS 0-2
N=108



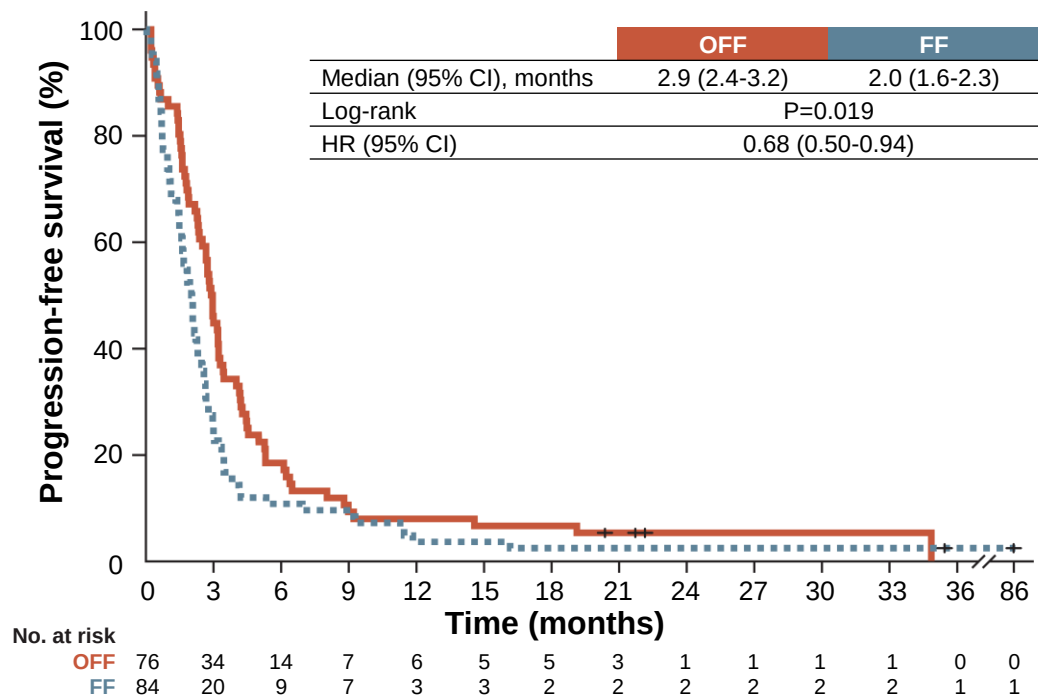
5-FU, fluorouracil; ECOG PS, Eastern Cooperative Oncology Group performance status; FF, folinic acid (leucovorin calcium) and fluorouracil; KPS, Karnofsky performance status; LV, leucovorin calcium (folinic acid); mFOLFOX6, modified FOLFOX6: folinic acid (leucovorin calcium), fluorouracil, and oxaliplatin; OFF, oxaliplatin and FF; OS, overall survival; PFS, progression-free survival

1. Oettle H, et al. J Clin Oncol. 2014 Aug 10;32:2423-9; 2. Gill S, et al. J Clin Oncol. 2016;10;3914-20

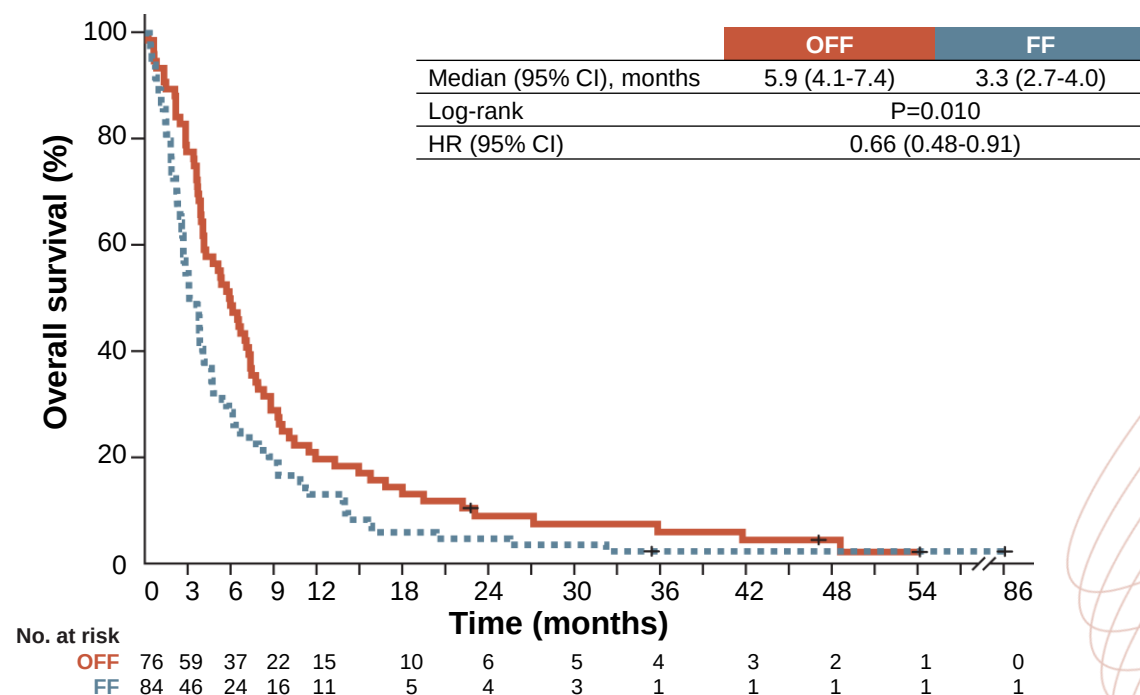
CONKO-003: 5FU+ FOLINIC ACID +/- OXALIPLATIN (OFF)

EFFICACY

PROGRESSION-FREE SURVIVAL



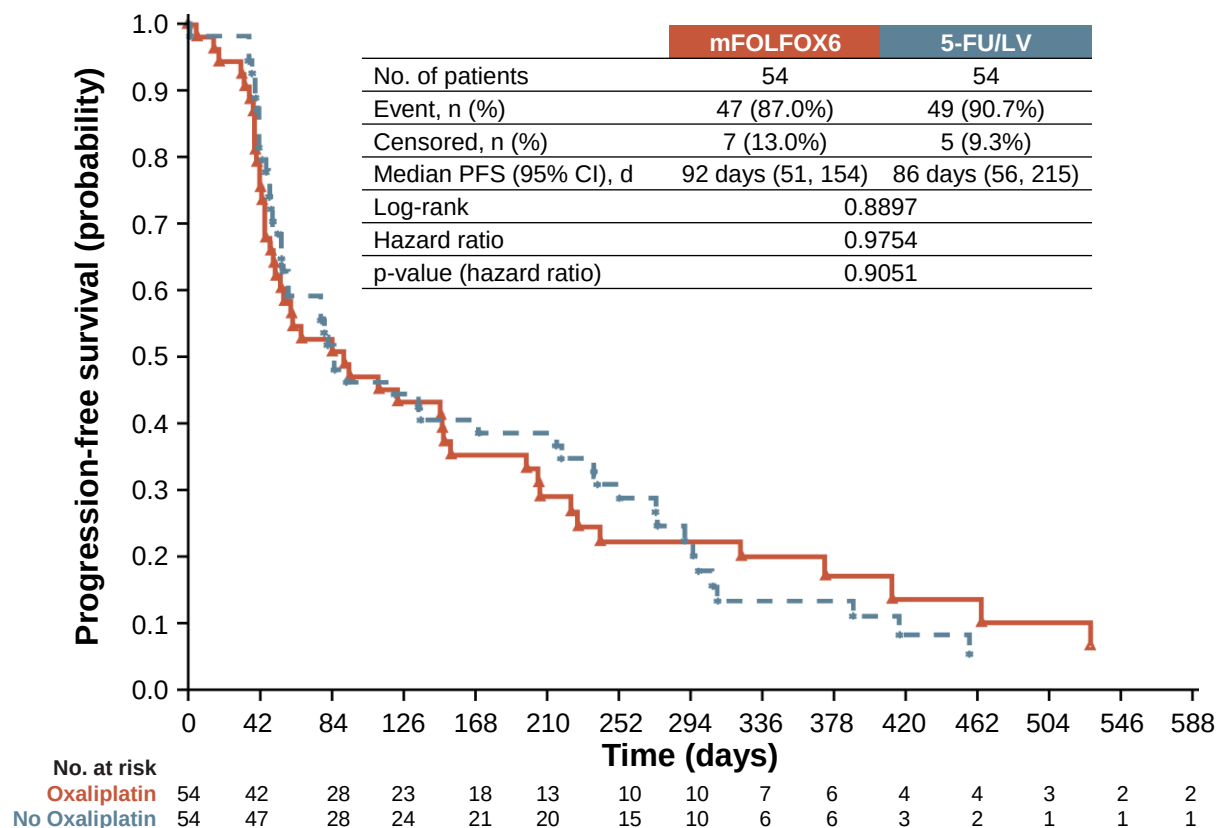
OVERALL SURVIVAL



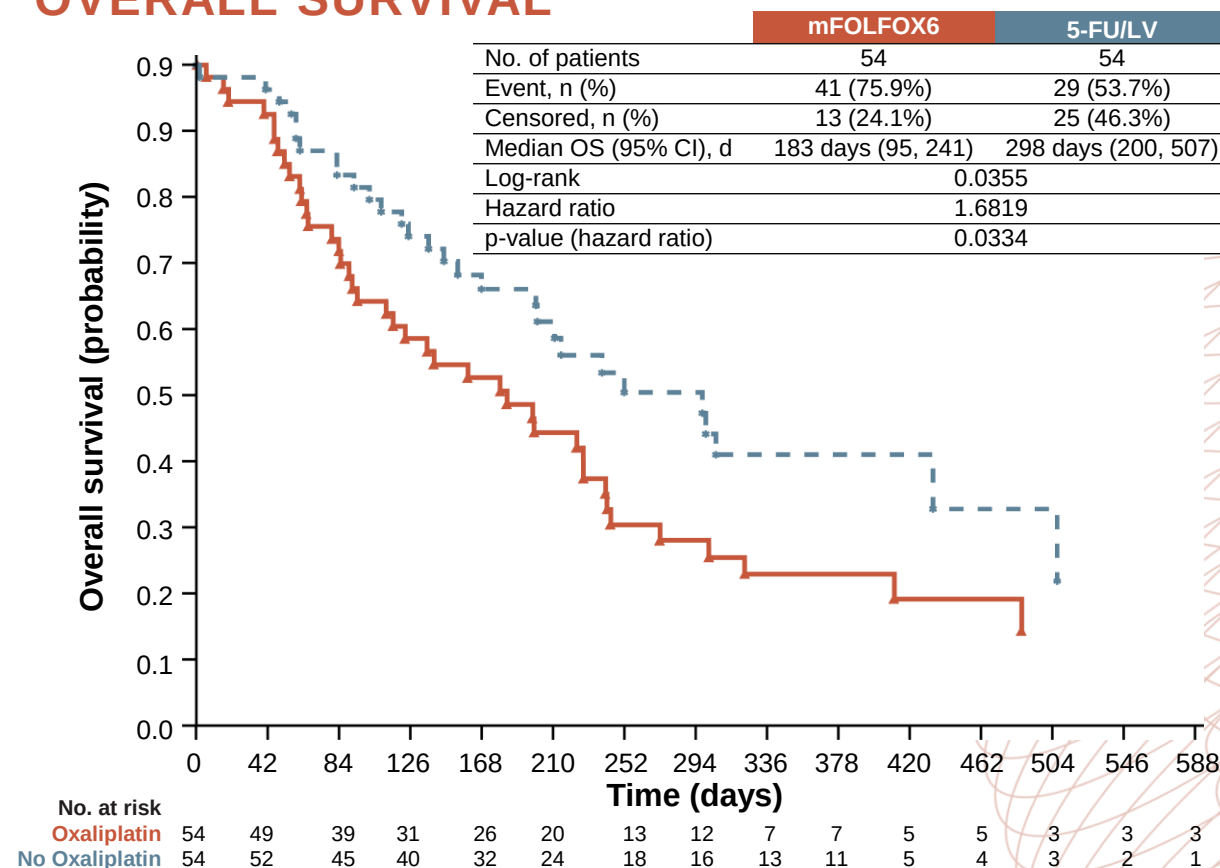
- Rates of adverse events were similar between treatment arms, except for grades 1 to 2 neurotoxicity, which were reported in 29 patients (38.2%) and six patients (7.1%) in the OFF and FF groups, respectively (P<0.001)

PANCREOX: ADDITION OF OXALIPLATIN TO 5-FU IN 2L WAS DETRIMENTAL

PROGRESSION-FREE SURVIVAL



OVERALL SURVIVAL



- **Grade 3 or 4 toxicity:** 63% on mFOLFOX6; 11% on 5-FU/LV

2L, second-line; 5-FU, fluorouracil; CI, confidence interval; d, days; LV, leucovorin calcium (folinic acid); mFOLFOX6, modified infusional fluorouracil, leucovorin (folinic acid), and oxaliplatin; PFS, progression-free survival

PHASE 3 EXPERIENCE IN 2L WITH OXALIPLATIN

CONTRADICTING RESULTS OBSERVED WITH SECOND-LINE OXALIPLATIN REGIMENS IN THE CONKO-003 AND PANCREOX TRIALS

	CONKO-003 ¹ N=160 ^a		PANCREOX ² N=108	
Treatment	OFF	5-FU/LV	mFOLFOX6	5-FU/LV
Median OS, mo	5.9	3.3	6.1	9.9
HR	0.66, P=0.01		1.78, P=0.02	
Median PFS, mo	2.9	2.0	3.0	2.8
HR	0.68, P=0.02		0.98, P=0.91	

Data presented for information purposes. Cross-trial comparison is not intended

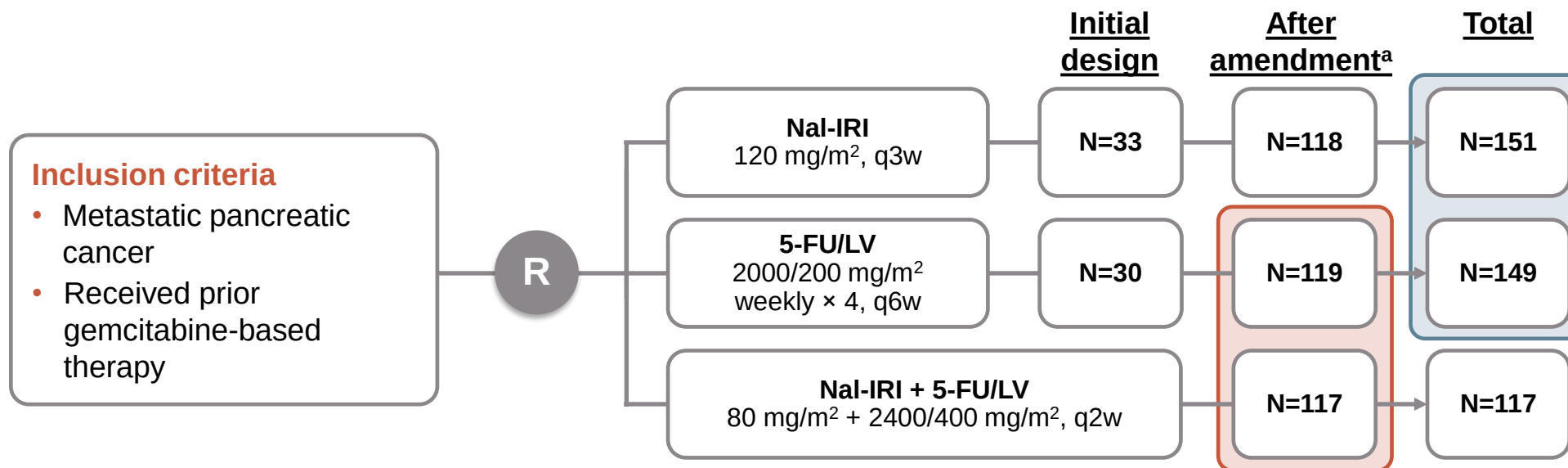
^aPatients eligible for primary analysis

2L, second-line; 5-FU, fluorouracil; HR, hazard ratio; LV, leucovorin calcium (folinic acid); mFOLFOX6, modified infusional fluorouracil, leucovorin, and oxaliplatin; mo, months; OS, overall survival; PFS, progression-free survival; OFF, folinic acid (leucovorin calcium), fluorouracil and oxaliplatin

1. Oettle H, et al. J Clin Oncol. 2014;32: doi.org/10.1200/JCO.2013.53.6995; 2. Gill S, et al. J Clin Oncol. 2016;10; 3914-20

NAPOLI-1: STUDY DESIGN

NAL-IRI ALONE AND IN COMBINATION WITH 5-FU/LV AS 2L THERAPY



Stratification factors: Albumin, KPS and ethnicity

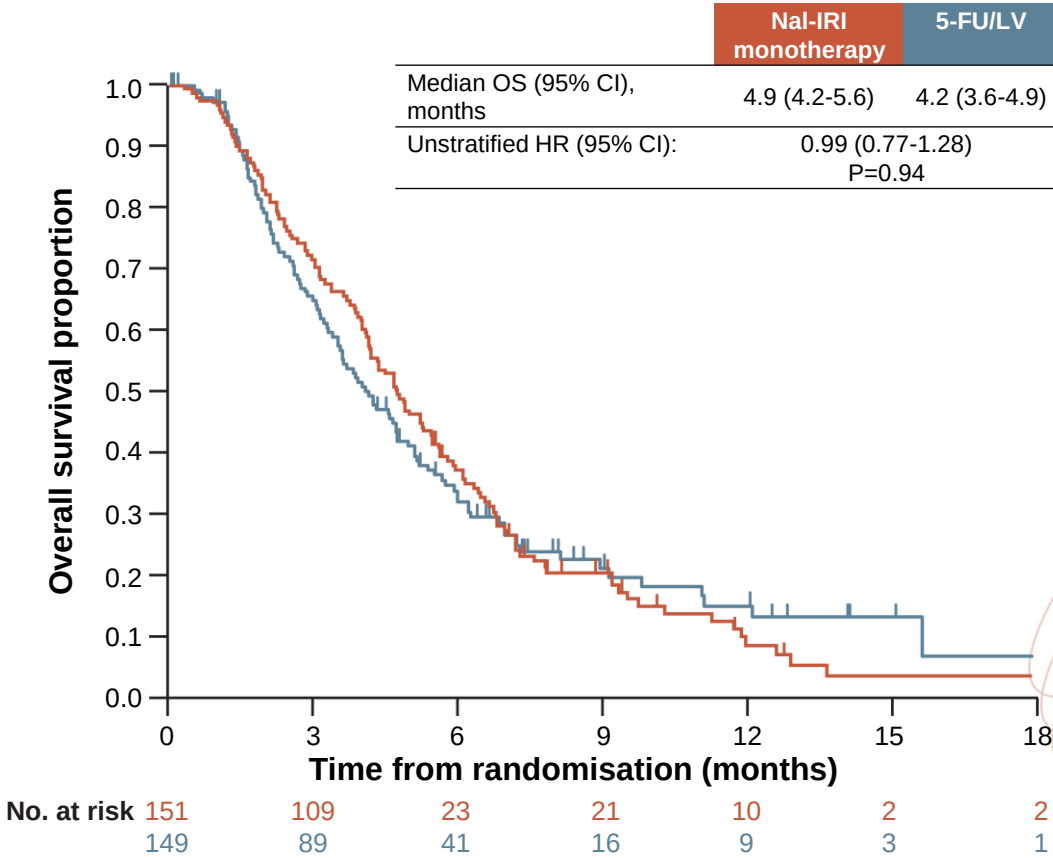
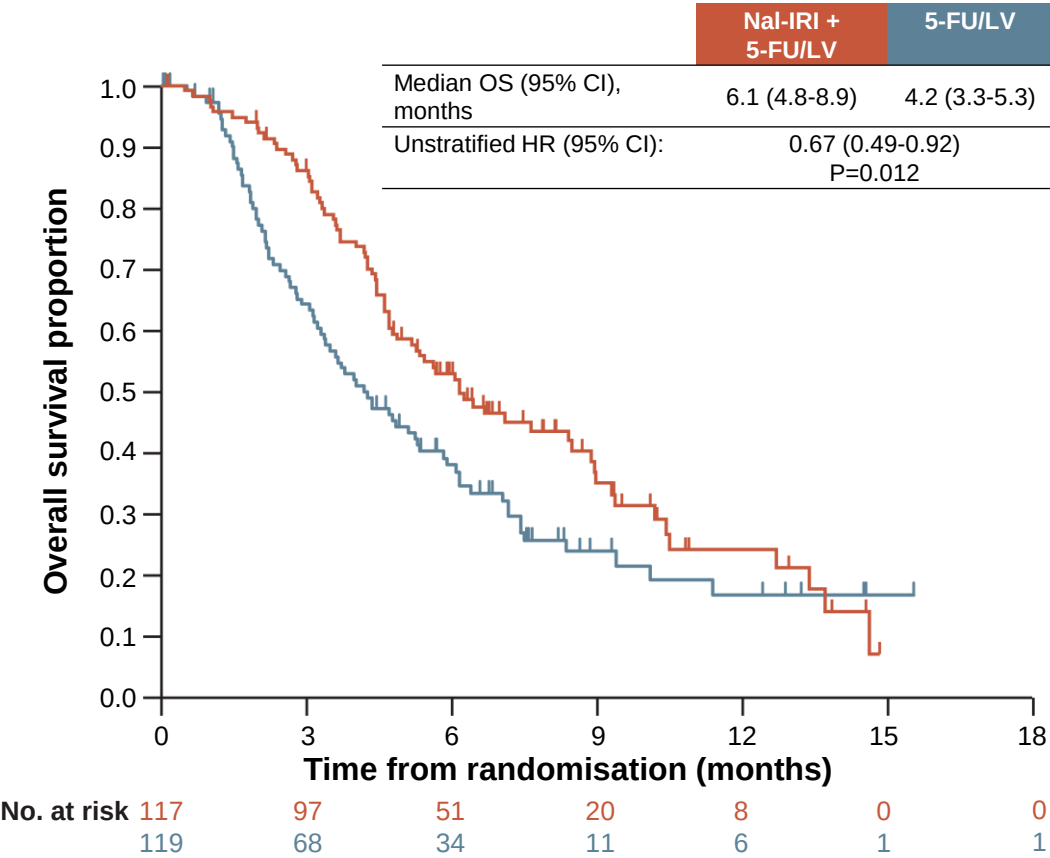
Primary endpoint: Overall survival

Secondary endpoints: PFS, ORR, TTTF, CA19-9 response, safety

^a Study was amended to add the Nal-IRI + 5-FU/LV arm once safety data on the combination became available. Only those patients enrolled in the 5FU/LV arm after the amendment (N=119), were used as the control for the combination arm

2L, second-line; 5-FU, fluorouracil; CA19-9; carbohydrate antigen 19-9; KPS, Karnofsky performance status; LV, leucovorin calcium (folinic acid); Nal-IRI, nanoliposomal irinotecan; ORR, overall response rate; PFS, progression-free survival; q2w/q3w/q6w, every 2/3/6 weeks; R, randomised; TTTF, time to treatment failure

NAPOLI-1: OVERALL SURVIVAL (ITT)



Protocol-defined primary analysis data cut (14 February 2014, after 305 events)

5-FU, fluorouracil; CI, confidence interval; HR, hazard ratio; ITT, intention-to-treat; LV, leucovorin calcium (folinic acid); Nal-IRI, nanoliposomal irinotecan; OS, overall survival

Wang-Gillam A, et al. Lancet. 2016;387:545-57

NAPOLI-1: SAFETY

Adverse event, n (%)	Nal-IRI + 5-FU/LV (N=117)		Nal-IRI monotherapy (N=147)		5-FU/LV (N=134)	
	Any grade	Grades 3-4	Any grade	Grades 3-4	Any grade	Grades 3-4
Diarrhoea	69 (59%)	15 (13%)	103 (70%)	31 (21%)	35 (26%)	6 (4%)
Vomiting	61 (52%)	13 (11%)	80 (54%)	20 (14%)	25 (26%)	4 (3%)
Nausea	60 (51%)	9 (8%)	89 (61%)	8 (5%)	46 (34%)	4 (3%)
Decreased appetite	52 (44%)	5 (4%)	72 (49%)	13 (19%)	43 (32%)	3 (2%)
Fatigue	47 (40%)	16 (14%)	54 (37%)	9 (6%)	37 (28%)	5 (4%)
Neutropenia ^a	46 (39%)	32 (27%)	37 (25%)	22 (15%)	7 (5%)	2 (1%)
Anaemia	44 (38%)	11 (9%)	48 (33%)	16 (11%)	31 (23%)	9 (7%)
Hypokalaemia	14 (12%)	4 (3%)	32 (22%)	17 (12%)	12 (9%)	3 (2%)

Data are number of patients (%). The table shows grade 3 and 4 adverse events reported in $\geq 5\%$ of patients whose treatment included nanoliposomal irinotecan with $\geq 2\%$ incidence versus fluorouracil and folinic acid. ^a includes agranulocytosis, febrile neutropenia, granulocytopenia, neutropenia, neutropenic sepsis, decreased neutrophil count, and pancytopenia

NAPOLI-1: DOSE MODIFICATIONS OF NAL-IRI + 5-FU/LV

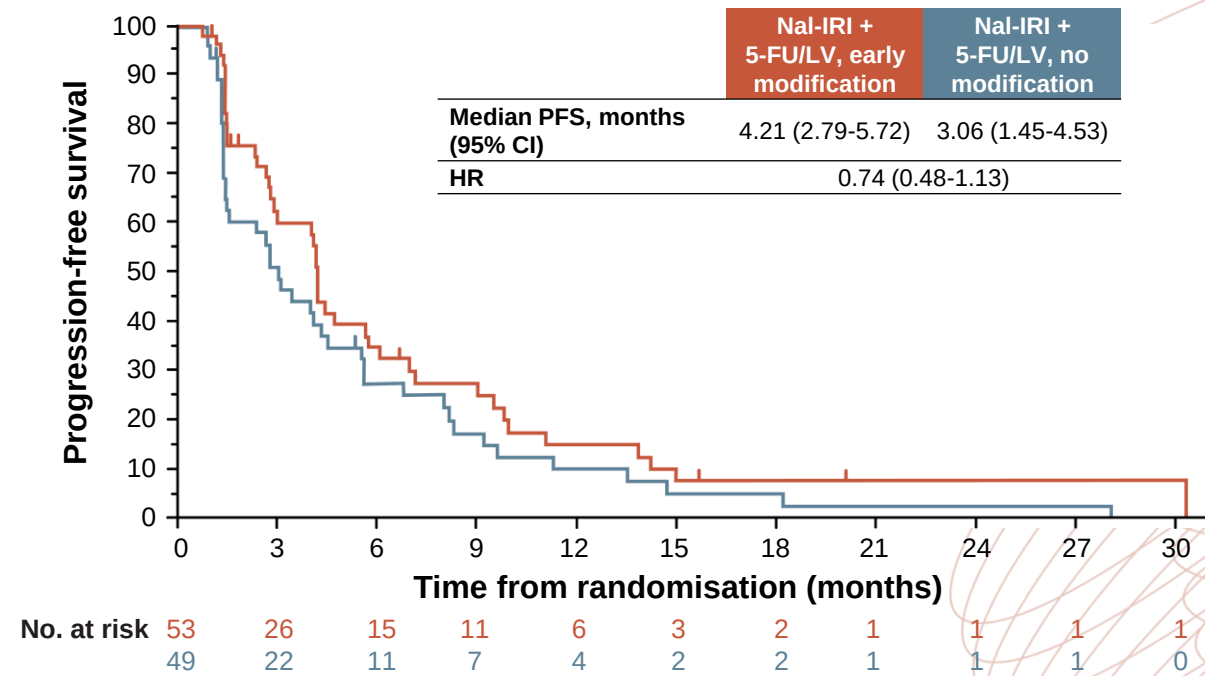
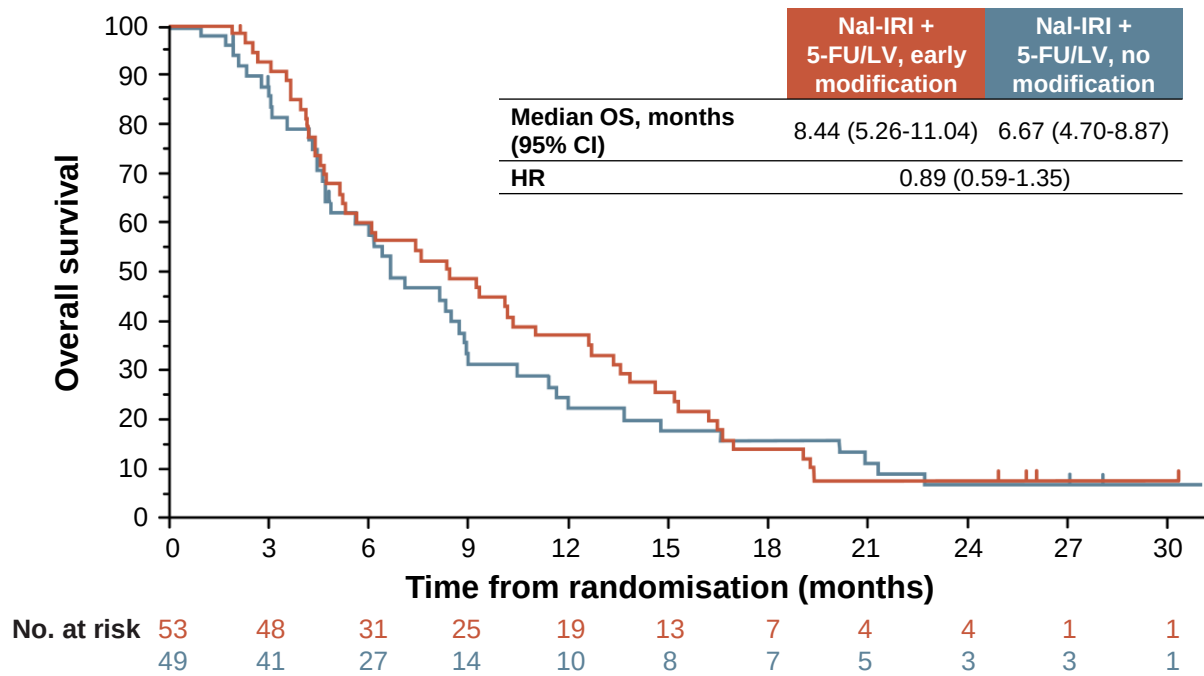
POST HOC ANALYSIS: IMPACT OF DOSE MODIFICATIONS OR DELAYS ON EFFICACY

	Nal-IRI + 5-FU/LV	5-FU/LV
	Nal-IRI dose delay	
Median overall survival, mos	8.4 (N=49)	4.2 (N=105)
Hazard ratio (95% CI)	0.66 (0.46, 0.94)	
	Nal-IRI dose reduction	
Median overall survival, mos	9.4 (N=34)	4.2 (N=105)
Hazard ratio (95% CI)	0.58 (0.38, 0.88)	

5-FU, fluorouracil; CI, confidence interval; LV, leucovorin calcium (folinic acid); mos, months; Nal-IRI, nanoliposomal irinotecan; OS, overall survival
Wang-Gillam A, et al. J. Clin. Oncol. 2018; 36(4_suppl):388

NAPOLI-1: DOSE MODIFICATIONS OF NAL-IRI + 5-FU/LV

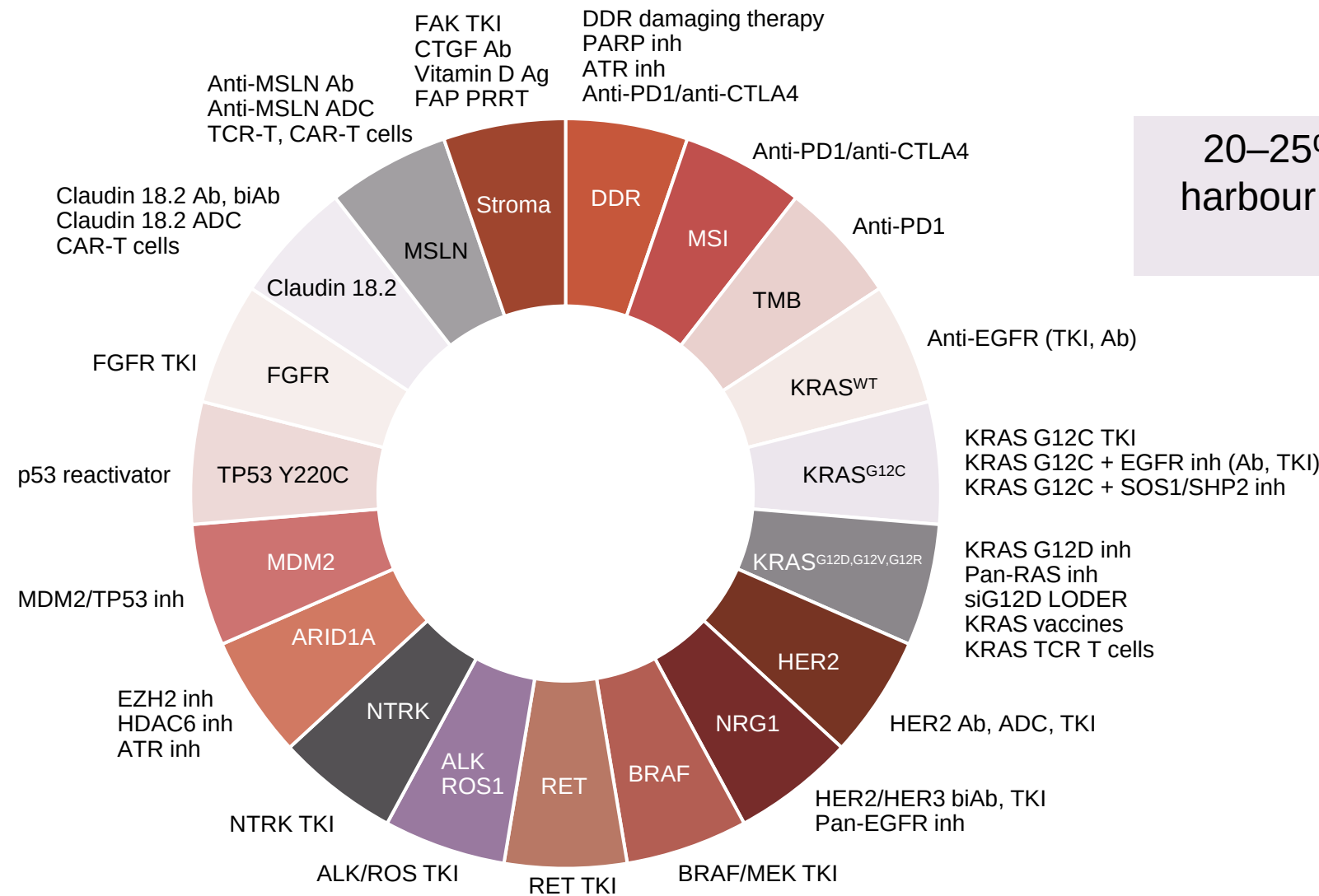
POST HOC ANALYSIS: IMPACT ON EFFICACY



- Tolerability-guided dose modification of liposomal irinotecan does not adversely affect efficacy outcomes

5-FU, fluorouracil; CI, confidence interval; HR, hazard ratio; LV, leucovorin calcium (folinic acid); Nal-IRI, nanoliposomal irinotecan; OS, overall survival; PFS, progression-free survival

POTENTIAL MOLECULAR TARGETS IN PDAC



20–25% of PDAC patients harbour targetable molecular alterations

ADC, antibody-drug conjugate; ALK, anaplastic lymphoma kinase; ARID1A, AT-rich interaction domain 1A; ATR, ataxia telangiectasia and Rad3-related protein; (bi)Ab, (bi-specific) antibody; BRAF, B-Raf proto-oncogene serine/threonine kinase; CAR-T, chimeric antigen receptor T-cell therapy; CTLA-4, cytotoxic T-lymphocyte-associated protein 4; DDR, DNA damage repair; EGFR, epidermal growth factor receptor; FAK, focal adhesion kinase; FAP, fibroblast activation protein; FGFR, fibroblast growth factor receptor; HDAC, histone deacetylase 6; inh, inhibitor; HER2/3, human epidermal growth factor receptor 2/3; KRAS, Kirsten rat sarcoma viral oncogene homolog; MEK, mitogen-activated protein kinase kinase; MSI, microsatellite instability; MSLN, mesothelin; NRG1, neuregulin-1; NTRK, neurotrophic tyrosine receptor kinase; PARP, poly(ADP-ribose) polymerase; PD-1, programmed cell death protein 1; PDAC, pancreatic ductal adenocarcinoma; PRRT, peptide receptor radionuclide therapy; RAS, rat sarcoma; RET, rearranged during transfection; ROS, ROS proto-oncogene receptor tyrosine kinase; siG12D LODER, small interfering RNA G12D Local Drug Eluter; TCR, T-cell receptor; TKI, tyrosine kinase inhibitor; TMB, tumour mutational burden; TP53, tumour protein p53 gene; WT, wild-type

TARGETED THERAPY FOR PANCREATIC ADENOCARCINOMA

Molecular Target	Targeted Therapy	NCCN panel recommendations
<i>NTRK</i> gene fusions	Larotrectinib	1 st line and subsequent treatment options for pts with <i>NTRK</i> gene fusion-positive locally advanced or metastatic pancreatic adenocarcinoma and for recurrent disease
	Entrectinib	
	Repotrectinib	Category 2B recommendation as 1 st line for patients with metastatic disease (PS 3) and subsequent therapy or therapy for recurrent disease for patients with intermediate/poor PS (PS 2-3)
<i>RET</i> gene fusions	Selpercatinib	1 st line: pts with locally advanced/metastatic disease (PS 0–2) and as subsequent therapy for pts with good PS (0–1)
<i>NRG1</i> gene fusions	Zenocutuzumab-zbco	FDA approved for advanced, unresectable, or metastatic pancreatic adenocarcinoma harboring a <i>NRG1</i> gene fusion with disease progression on or after prior systemic therapy. Awaiting incorporation into the NCCN guidelines
<i>KRAS</i> G12C mutations	Adagrasib	Subsequent therapy options for patients with any PS (category 2B for poor PS)
	Sotorasib	
<i>BRAF</i> V600E mutations	Dabrafenib/trametinib	1 st line: metastatic disease (category 2B) and as subsequent line options (category 2A) for pts with good/poor PS and <i>BRAF</i> V600E mutations
HER2-positive	Fam-trastuzumab-deruxtecan-nxki	As a subsequent therapy option only for patients with good PS and HER2 IHC 3+ expression
MSI-H/TMB-H/dMMR	Pembrolizumab	In the advanced disease setting for first-line and subsequent treatment (if no prior immunotherapy)
	Dostarlimab-gxly	As a subsequent treatment option (if no prior immunotherapy) for patients with MSI-H or dMMR locally advanced, metastatic, or recurrent pancreatic adenocarcinoma and any PS
	Nivolumab/Ipilimumab	Category 2B, subsequent therapy option for patients with good or intermediate PS and those who did not receive prior immunotherapy

BRAF, B-Raf proto-oncogene serine/threonine kinase; dMMR, deficient DNA mismatch repair; HER2, human epidermal growth factor receptor 2; *KRAS*, Kirsten rat sarcoma viral oncogene homolog; MSI-H, microsatellite instability-high; *NTRK*, neurotrophic tyrosine receptor kinase; PDAC, pancreatic ductal adenocarcinoma; PS, performance status; *RAS*, rat sarcoma; *RET*, rearranged during transfection; TMB-H, tumour mutational burden-high

NCCN Guidelines Version 3.2024. Available at: <https://www.nccn.org/guidelines/guidelines-detail?category=1&id=1455>. Accessed October 2024 NCCN guidelines for pancreatic adenocarcinoma, Version 3.2024: [pancreatic.pdf \(nccn.org\)](#)

SUMMARY

- Cytotoxic chemotherapy remains the cornerstone of systemic therapy for advanced or metastatic pancreatic cancer
- The choice of second-line therapy depends on which treatments the patient has received in the first-line setting and therefore which options are left available to them
- Treatment selection depends on several other factors, including patients' performance status and co-morbidities. These should be considered alongside the efficacy and safety profiles of the different chemotherapy regimens
- Treatment strategies can be implemented to manage toxicities associated with the different chemotherapy regimens to enable a patient to stay on treatment for optimal efficacy



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