Targeted Therapy and Biomarker-Driven Treatments in Biliary Tract Cancer

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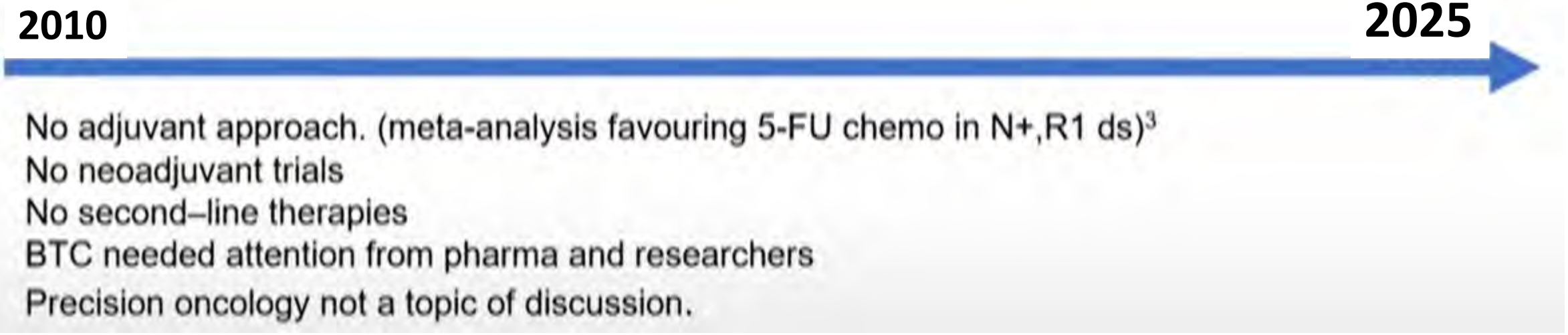
FLASCO, February 2025

THE UNIVERSITY OF TEXAS
MD Anderson Cancer Center
Making Cancer History®

BTC: Advancement in clinical science, for 15 years

ABC-02: GemCis improves OS in 1st line therapy (UK trial, 2010)

Standard of care for the 1st time in BTC

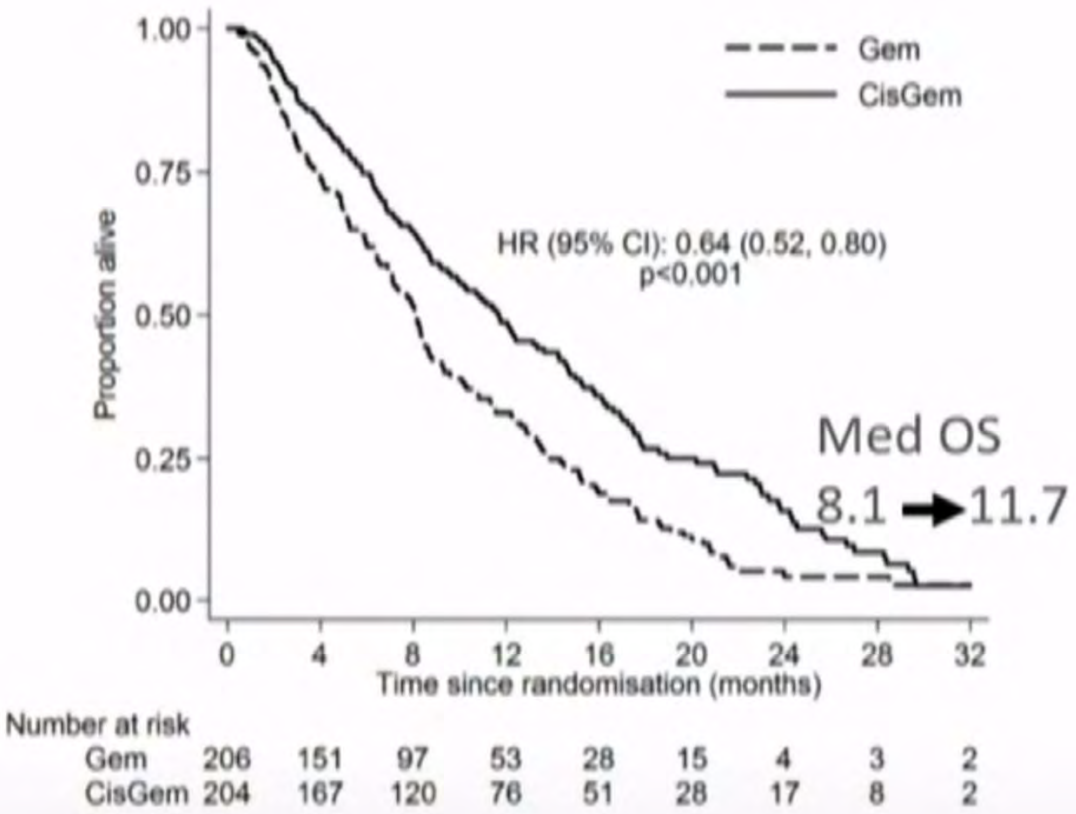


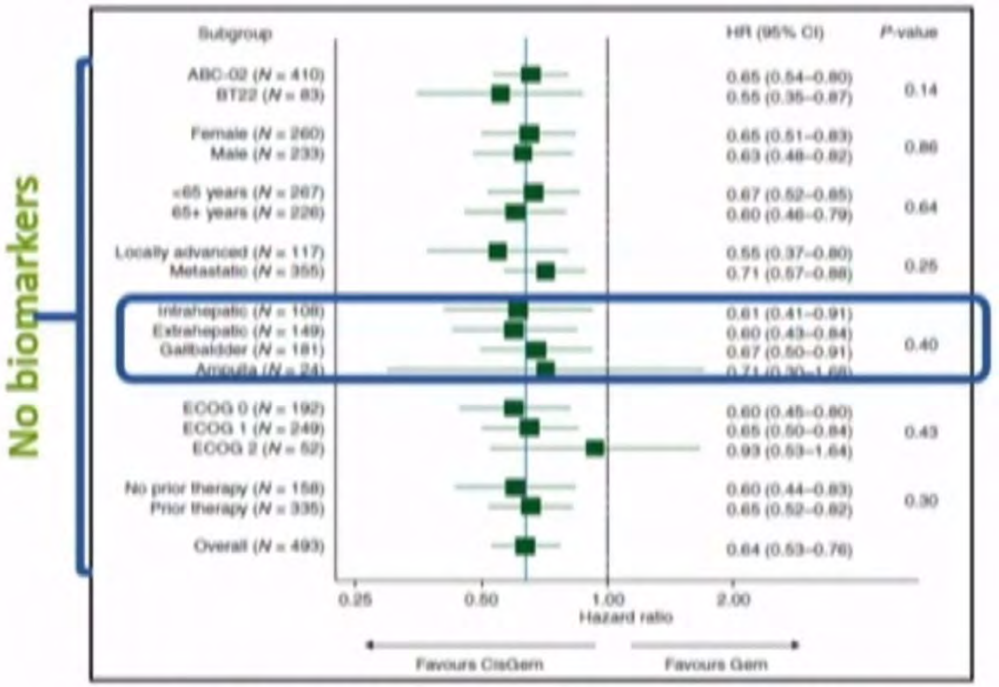
What was happening in oncology elsewhere?

2014 – nivolumab – 1st in class – anti-PD1, FDA approval for melanoma

2015 – RCC, SCC lung, NSCLC

ABC-02: OS of GemCis doublet over gem alone





Consistent benefit in sub-groups

No biomarker to select benefit

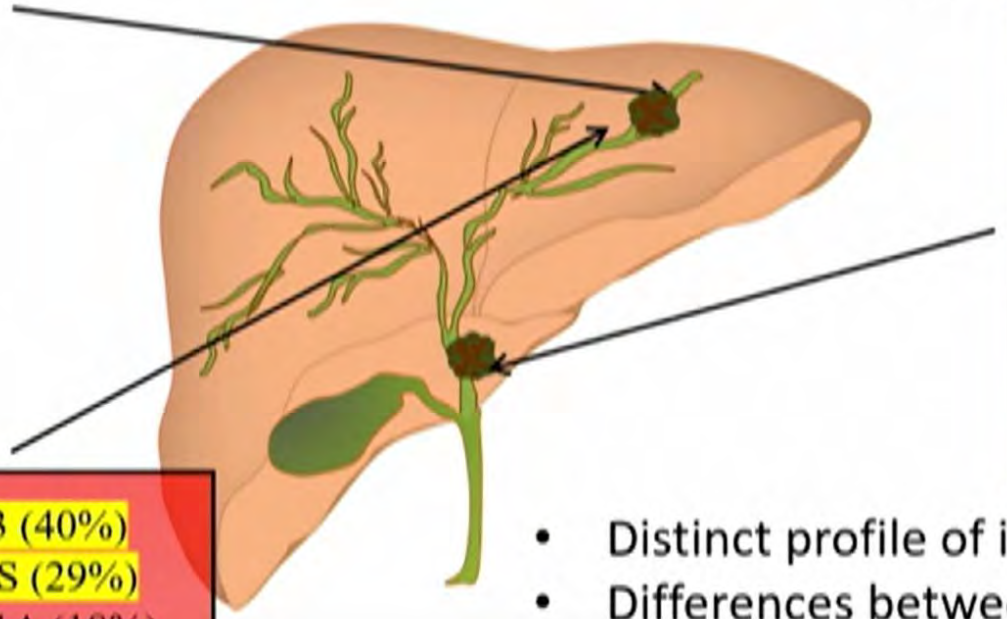
Multiple further studies..

- No benefit from adding EGFR inhibitors
- No benefit in adding MET TKI

2023: Heterogeneous Genomics of CCA

USA-Europe

CDK2A (30%)
IDH1/2 (23%)
TP53 (21%)
ARID1A (20%)
FGFR (18%)
BAP1 (17%)
KRAS (~16%)
PBRM1 (~11%)
PIK3A (9%)
BRAF (7%)



Extrahepatic Cholangiocarcinoma

TP53 (~45%)
KRAS (~36%)
ARID1A (~15%)
CDKN2A/B (~15%)
SMAD4 (~12%)
ERBB2 (~12%)
NF1 (~7%)
STK11 (~6%)
ERRB2 (~5%)

Intrahepatic Cholangiocarcinoma

Asia
Western (HBV)

TP53 (40%)
KRAS (29%)
ARID1A (18%)
CDKN2A (15%)
IDH1/2 (13%)
FGFR (13%)
PBRM1 (11%)
BAP1 (10%)
SMAD4 (10%)
PI3KCA (9%)
BRCA2 (9%)

- Distinct profile of iCCA vs eCCA
- Differences between iCCA in USA-Europe vs Asia
- Similar difference in USA-Europe in –HBV vs +HBV
- eCCA profiles resemble the Asia and +HBV groups

Intrahepatic Cholangiocarcinoma: Genomic Heterogeneity Between Eastern and Western Patients

Jingyu Cao, MD[1]; Jing Hu, PhD[2]; Siqin Liu, PhD[3]; Funda Meric-Bernstam, MD[4]; Reham Abdel-Wahab, MD[5]; Junjie Xu, MD[6]; Qiang Li, MD[7]; Maolin Yan, MD[8]; Yujie Feng, PhD[1]; Jianzhen Lin, MD[9]; Songhui Zhao, MSc[3]; Jian Wang, MS[3]; Lawrence N. Kwong, PhD[5]; Jinwei Hu, MD[3]; Fernando Carapeto, DMV[5]; Mitesh J. Borad, MD[10]; Kai Wang, MD, PhD[3]; Milind Javle, MD[4]; and Haitao Zhao, MD[9]

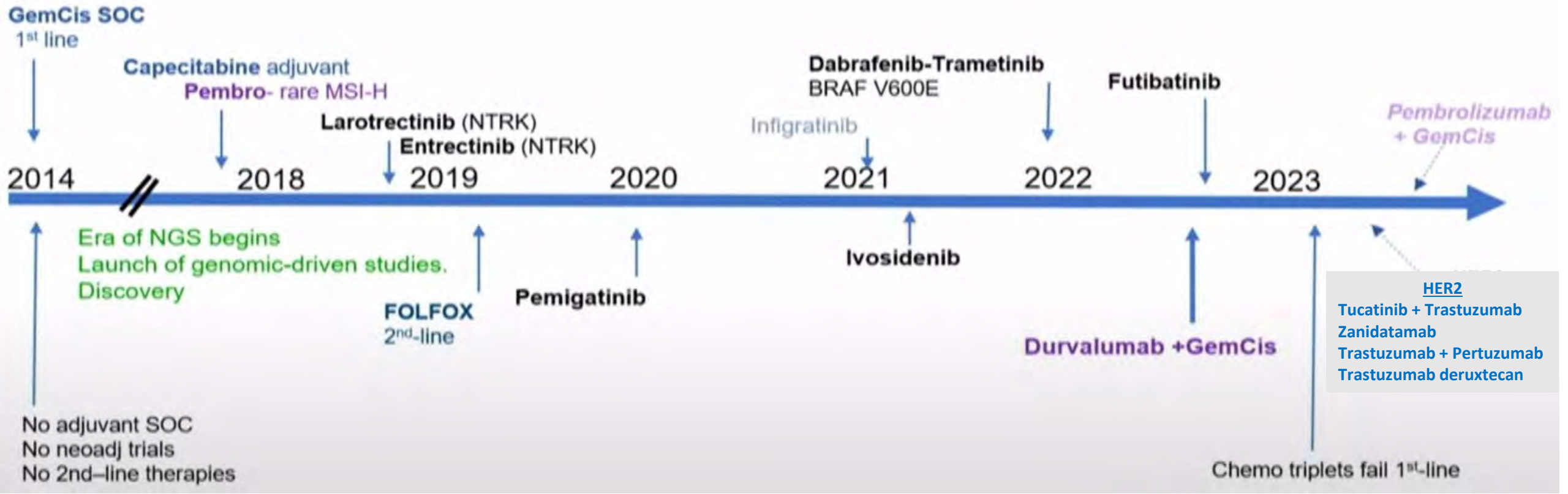
Foundation Series, n=554 BTC relative frequencies

Summary of Genomic Alterations

Genomic Findings	IHCCA N= 412	EHCCA N= 57	GBCA N= 85
Total Genomic alterations/pt	3.6	4.4	4.0
ERBB2 (HER-2) Amplification	4%	11%	**16%**
BRAF Substitutions	**5%**	3%	1%
KRAS Substitutions	22%	**42%**	11%
PIK3CA Substitution	5%	7%	14%
FGFR1-3 Fusions/Amplifications	**11%**	-	3%
CDKN2A/B Loss	27%	17%	19%
IDH 1/2 mutations	**20%**	-	-
ARID1A Alterations	**18%**	12%	13%
TP53	2-29%	40-45%	25-46%

Javle, Cancer 2016

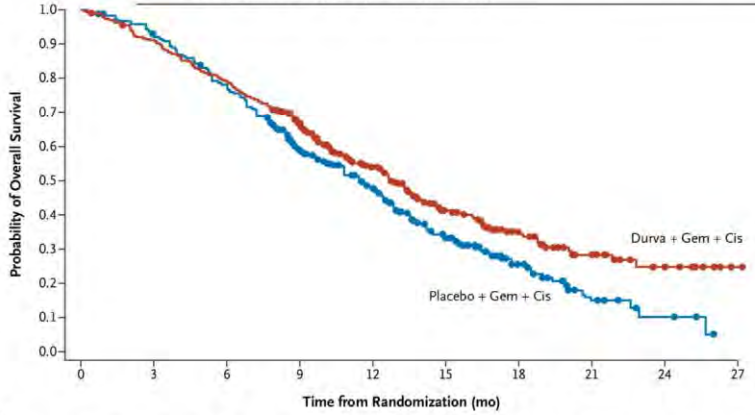
BTC Clinical Progress: 2014 - 2025



TOPAZ-1: adding durvalumab to SOC GemCis

A

	Median Overall Survival, mo (95% CI)	Hazard Ratio (95% CI)	Stratified Log-rank P Value
Durva + Gem + Cis (n=341)	12.8 (11.1–14.0)	0.80 (0.66–0.97)	0.021
Placebo + Gem + Cis (n=344)	11.5 (10.1–12.5)		

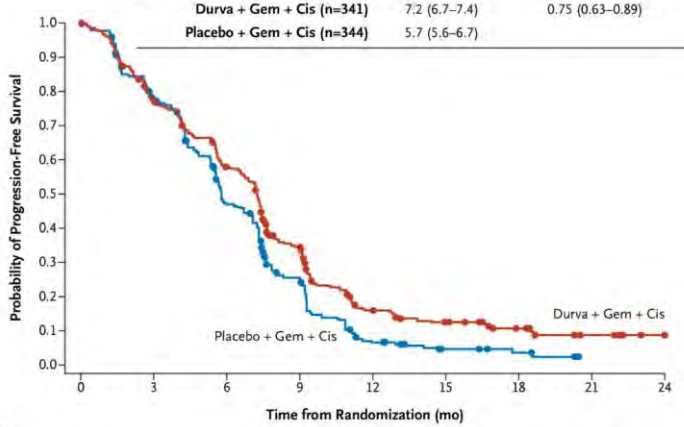


No. at Risk

Durva + Gem + Cis	341	331	324	309	294	278	268	252	238	208	174	151	135	118	93	79	74	57	49	39	29	24	15	12	9	8	4	1
Placebo + Gem + Cis	344	337	329	317	299	283	261	242	220	183	159	143	125	97	78	65	52	40	29	21	15	10	8	4	4	3	0	0

B

	Median Progression-free Survival, mo (95% CI)	Hazard Ratio (95% CI)	Stratified Log-rank P Value
Durva + Gem + Cis (n=341)	7.2 (6.7–7.4)	0.75 (0.63–0.89)	0.001
Placebo + Gem + Cis (n=344)	5.7 (5.6–6.7)		



No. at Risk

Durva + Gem + Cis	341	326	296	258	245	221	189	174	106	100	62	54	38	31	27	25	23	16	15	7	7	5	4	1	0
Placebo + Gem + Cis	344	327	280	255	237	197	149	137	80	71	39	31	17	14	11	7	7	5	4	2	2	0	0	0	0

N=685

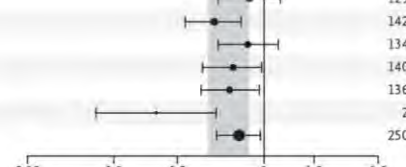
ORR
27% vs. 19%

A

Subgroup	Durva + Gem + Cis No. of events/Total no. (%)	Placebo + Gem + Cis No. of events/Total no. (%)	Hazard Ratio (95% CI)
All patients	198/341 (58.1%)	226/344 (65.7%)	0.80 (0.66–0.97)
Sex: female	99/172 (57.6%)	104/168 (61.9%)	0.82 (0.62–1.08)
Sex: male	99/169 (58.6%)	122/176 (69.3%)	0.78 (0.60–1.01)
Age at randomization: <65 yr	100/181 (55.2%)	116/184 (63.0%)	0.80 (0.61–1.04)
Age at randomization: ≥65 yr	98/160 (61.3%)	110/160 (68.8%)	0.79 (0.60–1.04)
PD-L1 expression: TAP ≥1%	120/197 (60.9%)	138/205 (67.3%)	0.79 (0.61–1.00)
PD-L1 expression: TAP <1%	57/103 (55.3%)	66/103 (64.1%)	0.86 (0.60–1.23)
Disease status at randomization: initially unresectable	176/274 (64.2%)	194/279 (69.5%)	0.84 (0.69–1.03)
Disease status at randomization: recurrent	22/67 (32.8%)	32/64 (50.0%)	0.56 (0.32–0.96)
Primary tumor location: intrahepatic cholangiocarcinoma	105/190 (55.3%)	126/193 (65.3%)	0.76 (0.58–0.98)
Primary tumor location: extrahepatic cholangiocarcinoma	38/66 (57.6%)	42/65 (64.6%)	0.76 (0.49–1.19)
Primary tumor location: gallbladder cancer	55/85 (64.7%)	58/86 (67.4%)	0.94 (0.65–1.37)
Race: Asian	107/185 (57.8%)	141/201 (70.1%)	0.73 (0.57–0.94)
Race: non-Asian	91/156 (58.3%)	85/143 (59.4%)	0.89 (0.66–1.19)
Region: Asia	103/178 (57.9%)	137/196 (69.9%)	0.72 (0.56–0.94)
Region: rest of the world	95/163 (58.3%)	89/148 (60.1%)	0.89 (0.66–1.19)
ECOG performance status at baseline: 0	95/173 (54.9%)	93/163 (57.1%)	0.90 (0.68–1.20)
ECOG performance status at baseline: 1	103/168 (61.3%)	133/181 (73.5%)	0.72 (0.56–0.94)
Biliary tract cancer: locally advanced	16/38 (42.1%)	36/57 (63.2%)	0.49 (0.26–0.88)
Biliary tract cancer: metastatic	182/303 (60.1%)	190/286 (66.4%)	0.83 (0.68–1.02)

B

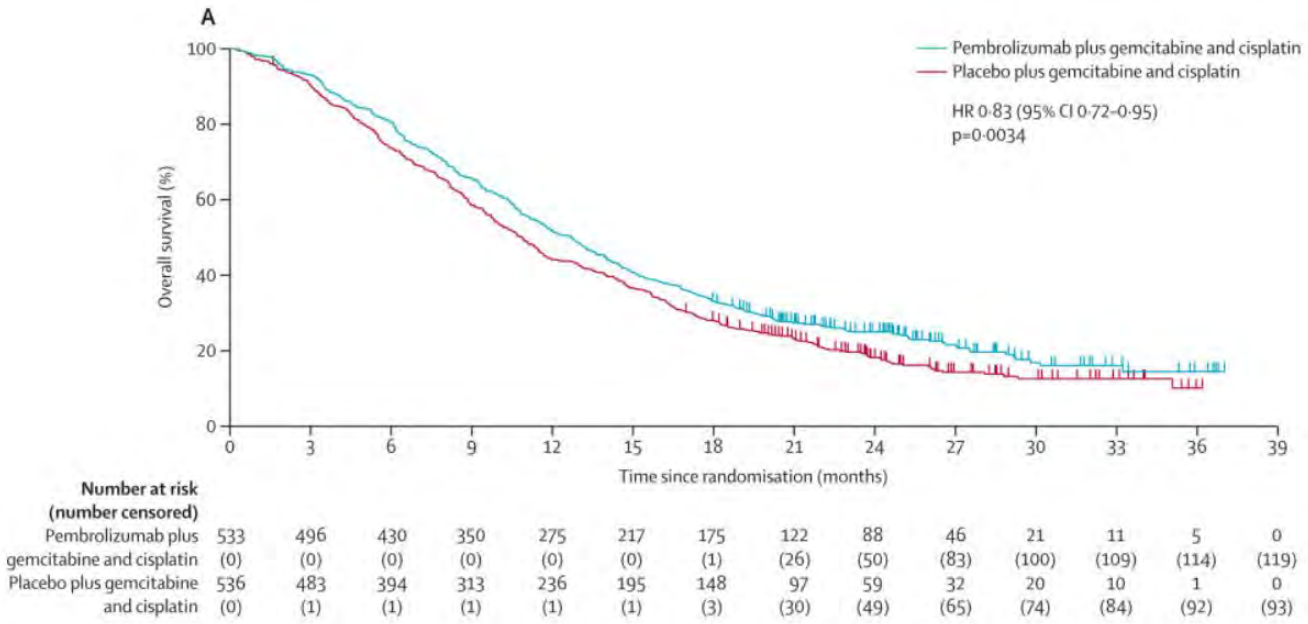
Subgroup	Durva + Gem + Cis No. of events/Total no. (%)	Placebo + Gem + Cis No. of events/Total no. (%)	Hazard Ratio (95% CI)
All patients	276/341 (80.9%)	297/344 (86.3%)	0.75 (0.63–0.89)
Sex: female	142/172 (82.6%)	146/168 (86.9%)	0.78 (0.62–0.99)
Sex: male	134/169 (79.3%)	151/176 (85.8%)	0.73 (0.58–0.93)
Age at randomization: <65 yr	144/181 (79.6%)	159/184 (86.4%)	0.68 (0.54–0.85)
Age at randomization: ≥65 yr	132/160 (82.5%)	138/160 (86.3%)	0.84 (0.66–1.07)
PD-L1 expression: TAP ≥1%	160/197 (81.2%)	179/205 (87.3%)	0.73 (0.59–0.91)
PD-L1 expression: TAP <1%	82/103 (79.6%)	87/103 (84.5%)	0.80 (0.59–1.09)
Disease status at randomization: initially unresectable	228/274 (83.2%)	247/279 (88.5%)	0.79 (0.66–0.95)
Disease status at randomization: recurrent	48/67 (71.6%)	50/64 (78.1%)	0.63 (0.42–0.94)
Primary tumor location: intrahepatic cholangiocarcinoma	154/190 (81.1%)	167/193 (86.5%)	0.79 (0.64–0.99)
Primary tumor location: extrahepatic cholangiocarcinoma	50/66 (75.8%)	55/65 (84.6%)	0.52 (0.35–0.78)
Primary tumor location: gallbladder cancer	72/85 (84.7%)	75/86 (87.2%)	0.90 (0.65–1.24)
Race: Asian	147/185 (79.5%)	179/201 (89.1%)	0.67 (0.54–0.83)
Race: non-Asian	129/156 (82.7%)	118/143 (82.5%)	0.88 (0.69–1.14)
Region: Asia	142/178 (79.8%)	174/196 (88.8%)	0.67 (0.53–0.83)
Region: rest of the world	134/163 (82.2%)	123/148 (83.1%)	0.87 (0.68–1.12)
ECOG performance status at baseline: 0	140/173 (80.9%)	140/163 (85.9%)	0.77 (0.61–0.98)
ECOG performance status at baseline: 1	136/168 (81.0%)	157/181 (86.7%)	0.76 (0.60–0.95)
Biliary tract cancer: locally advanced	26/38 (68.4%)	49/57 (86.0%)	0.42 (0.26–0.68)
Biliary tract cancer: metastatic	250/303 (82.5%)	248/286 (86.7%)	0.81 (0.68–0.97)

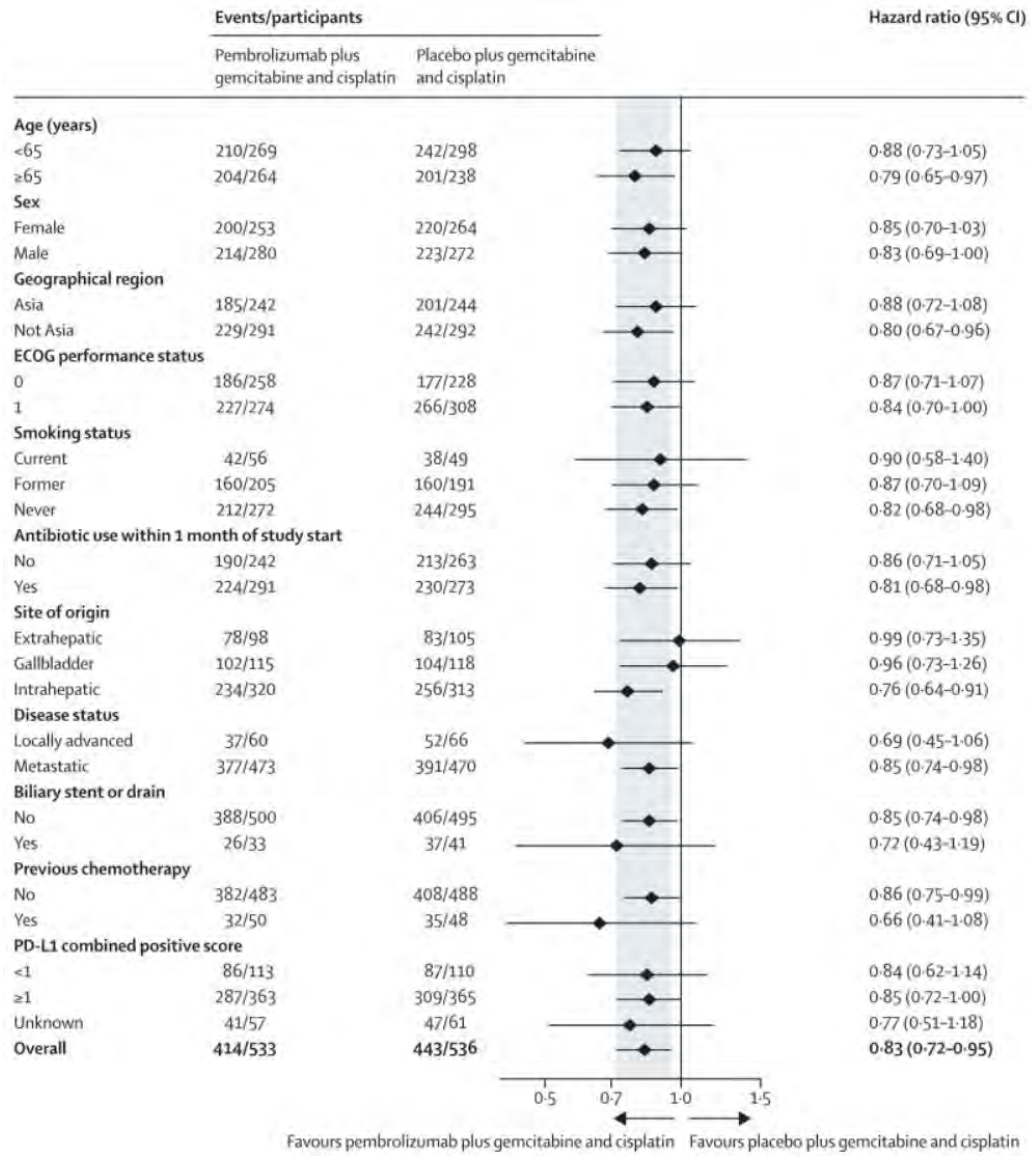


KEYNOTE-966, primary endpoint, OS

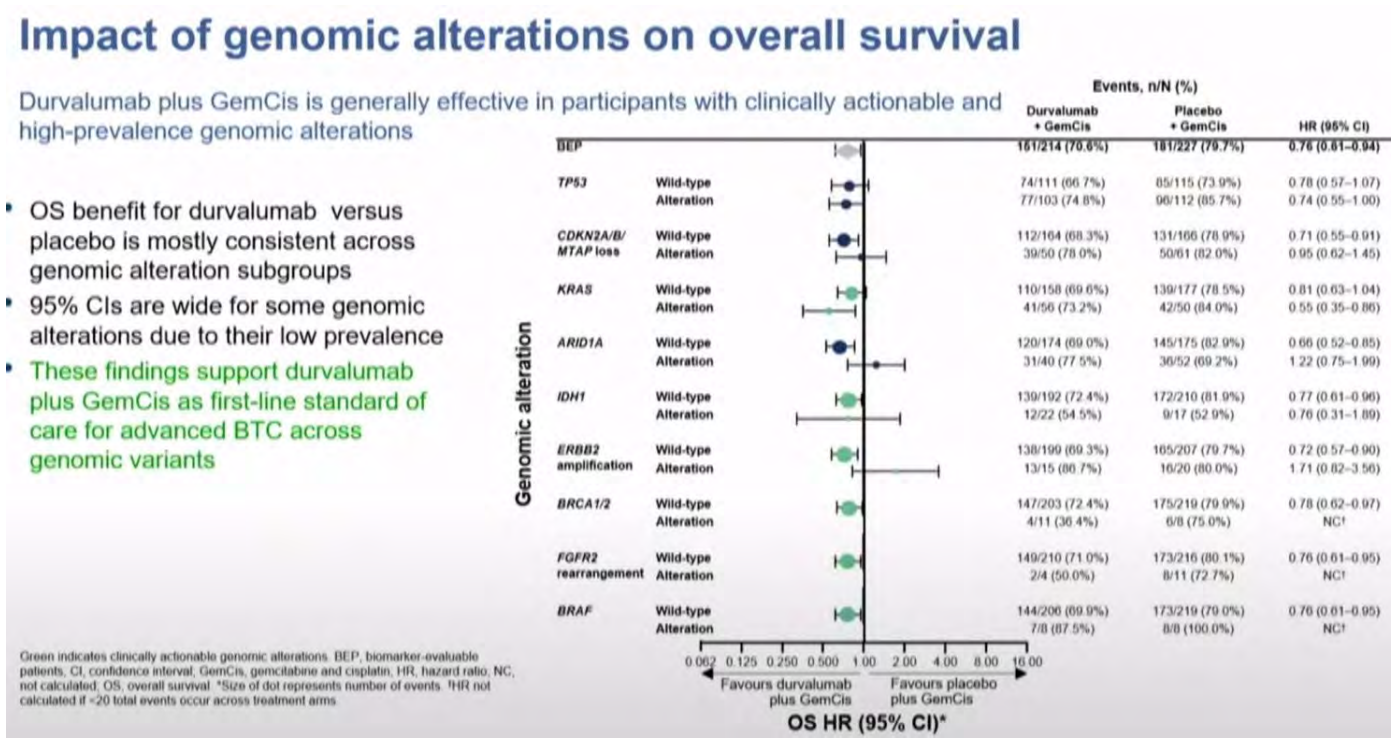
- Pembrolizumab + GemCis

  vs. Placebo + GemCis, advanced, 1st line





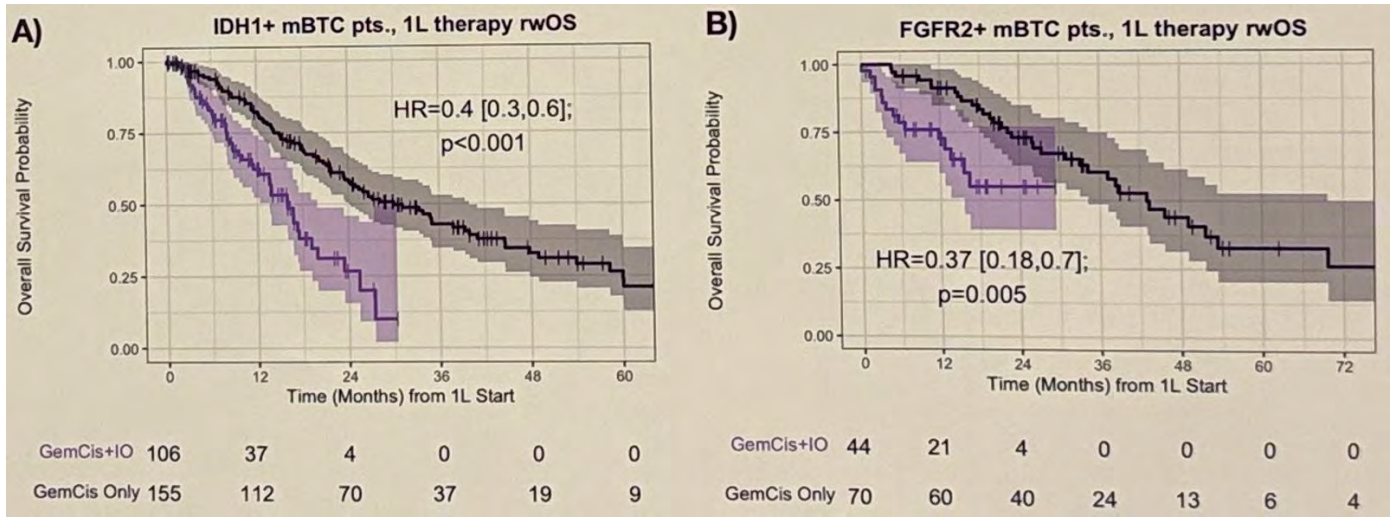
Pembrolizumab: continued until progression
Gemcitabine: continued until progression (different from TOPAZ1)
Cisplatin: given for a maximum of 8 cycles



Valle, Oh et al, ESMO Singapore 2023

Early data

- Immunotherapy may be beneficial across genomic alterations
- Data not mature

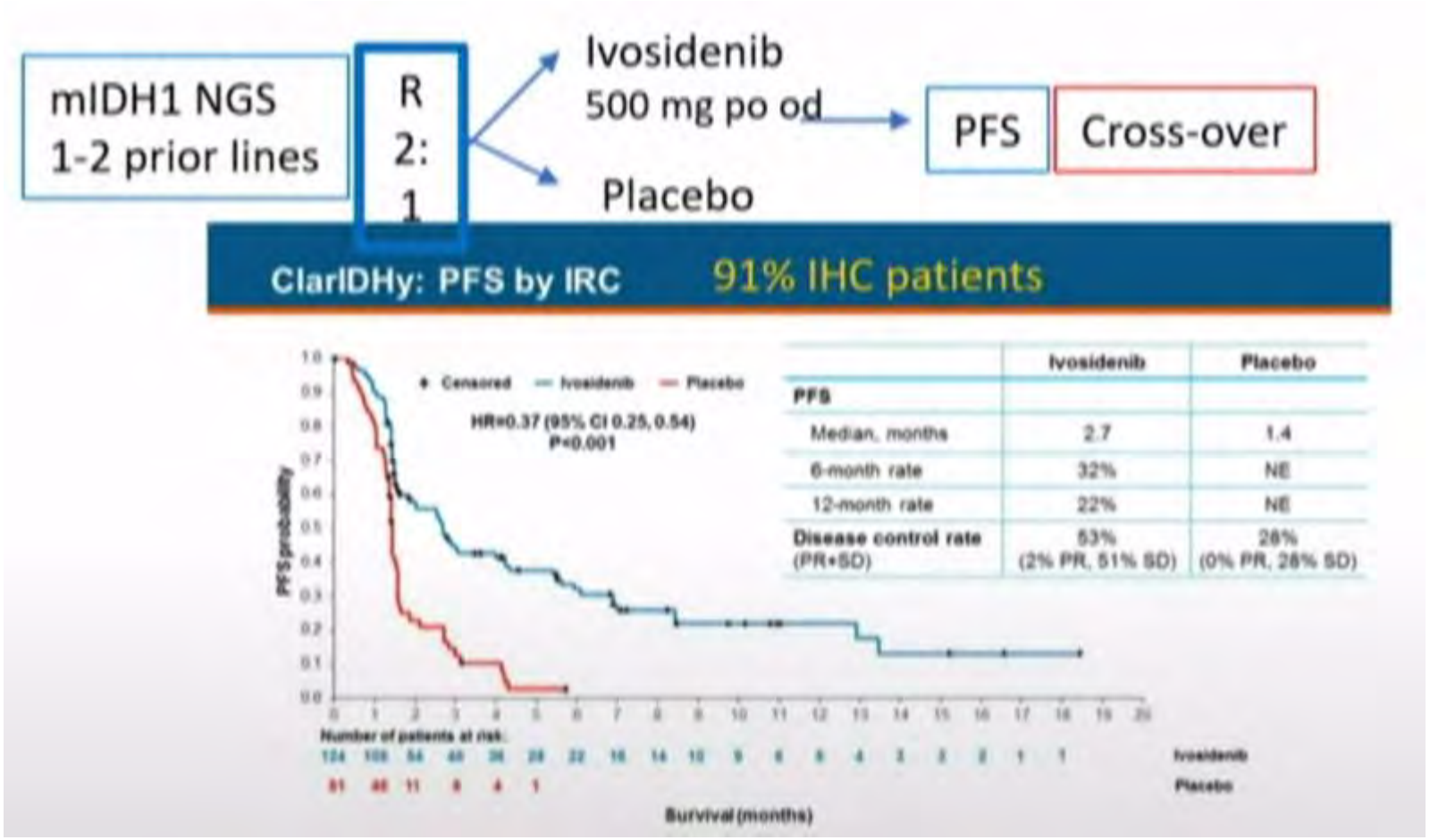


Kim R, et al, GI ASCO 2025

Conflicting data, more data accumulating

- Dr. Kim presented the data on GI ASCO 2025
- Patients who received immunotherapy may have a worse outcome, compared to those who did not, with IDH1 mutation or FGFR2 fusion

Targeting IDH1 population (ClarIDHy)



	Ivosidenib	Placebo
PFS		
Median, months	2.7	1.4
6-month rate	32%	NE
12-month rate	22%	NE
Disease control rate (PR+SD)	53% (2% PR, 51% SD)	28% (0% PR, 28% SD)

Pivotal trial of biomarker selected population in a rare cancer
Low response rate, there is a signal, but it needs a biomarker study

Abou-Alfa et al, NEJM 2022

Pemigatinib, 2nd line, FGFR fusions/rearrangements

Pemigatinib is a selective, potent, oral inhibitor of FGFR 1, 2, 3.

FDA approved late 2020- first drug ever in cholangiocarcinoma

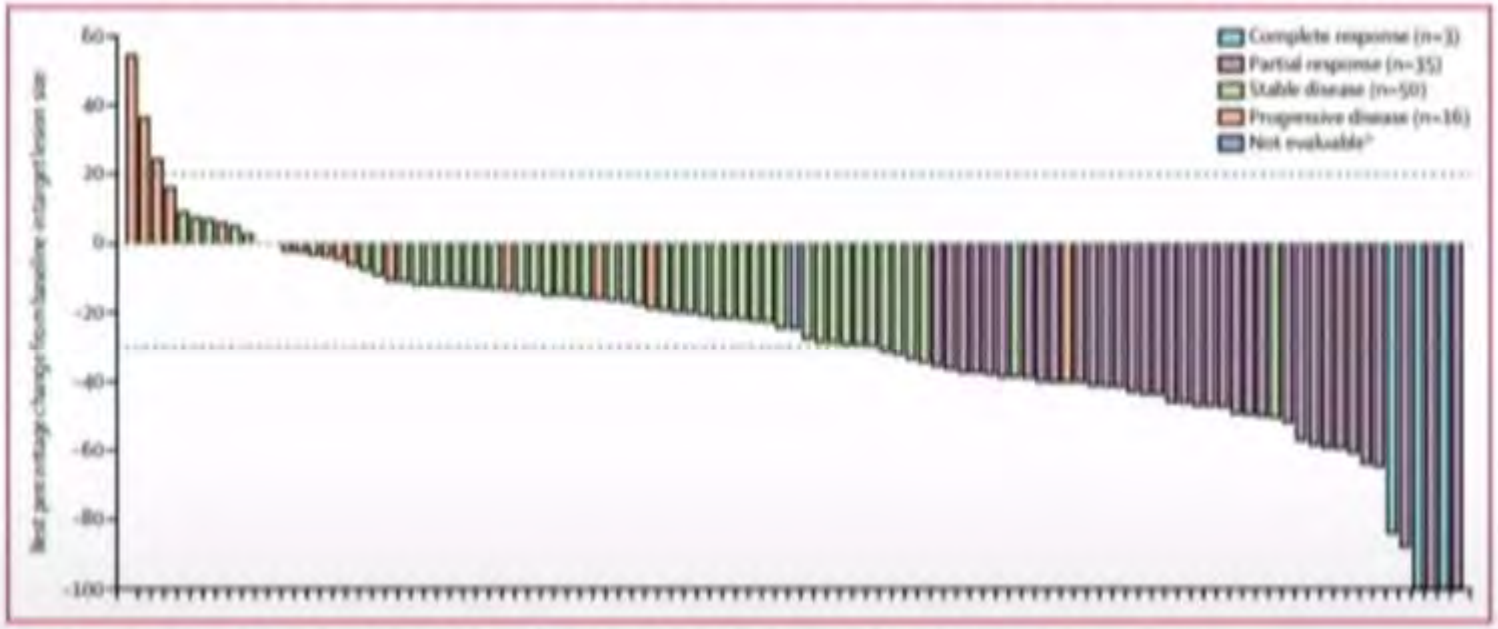


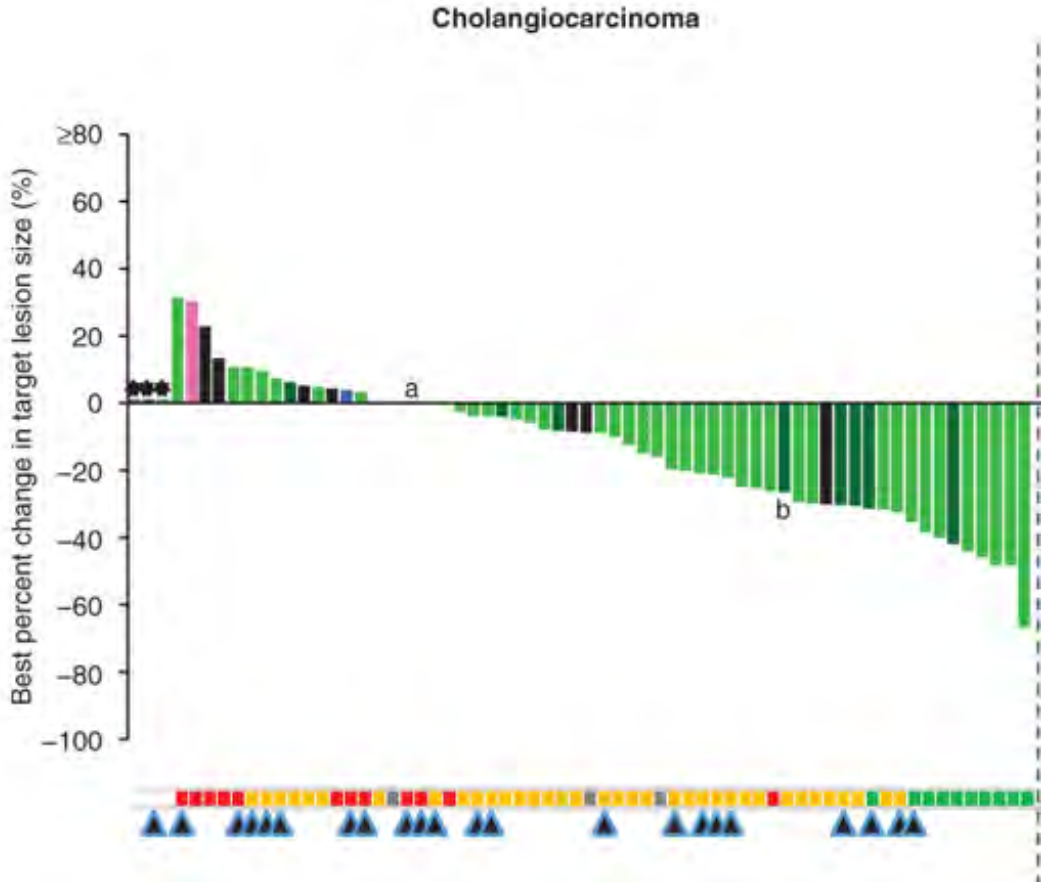
Figure 2: **Best percentage change from baseline in target lesion size for individual patients with *FGFR2* fusions or rearrangements**

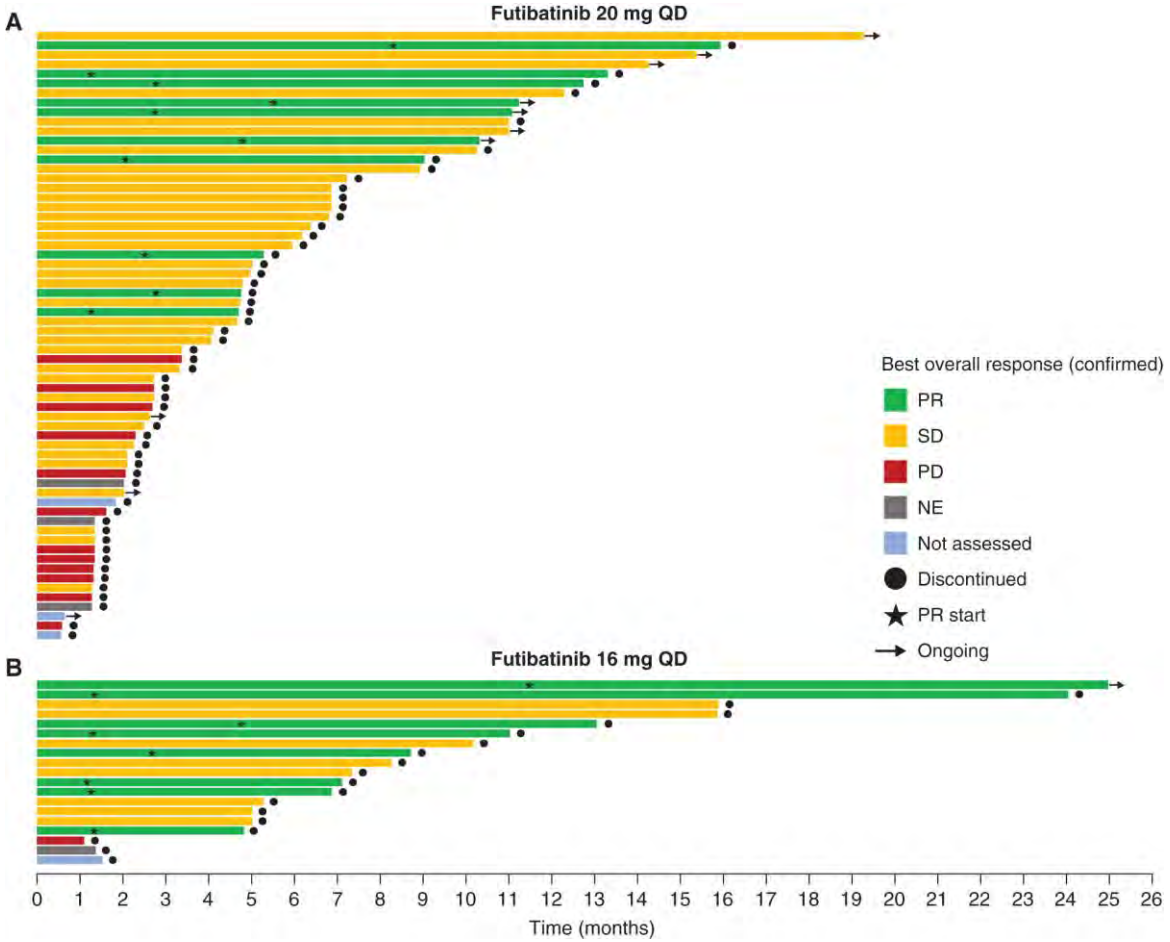
Coloured bars indicate confirmed responses assessed by RECIST 1·1. FGFR=fibroblast growth factor receptor. RECIST 1·1=Response Evaluation Criteria in Solid Tumors version 1.1. *Patient had a decrease in target lesion size but was not evaluable for response using RECIST.

ORR 36%, mPFS 7 m, mOS 21 m

Abou-Alfa et al, Lancet Oncol 2020

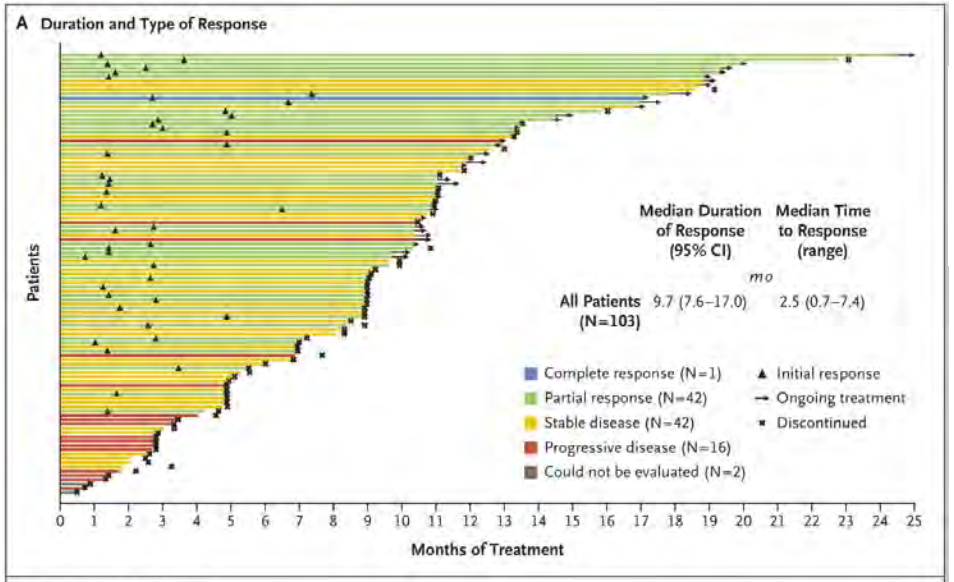
Next Gen FGFRi: futibatinib (covalent, FGFR1-4i)

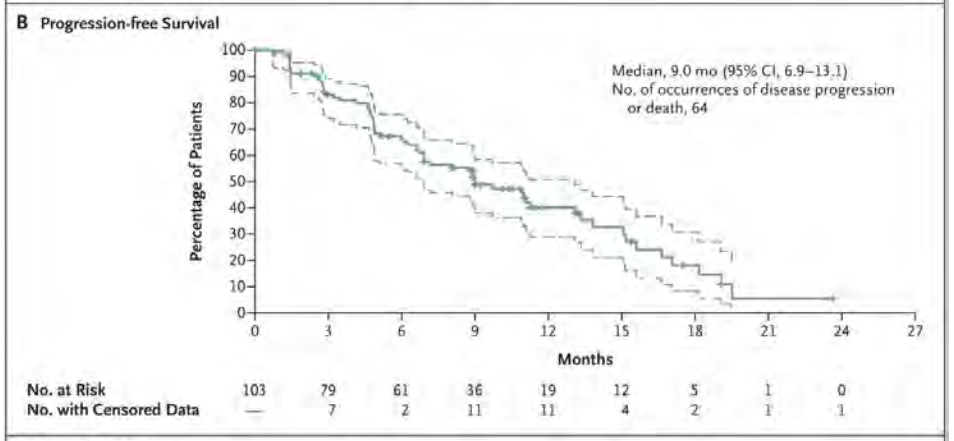


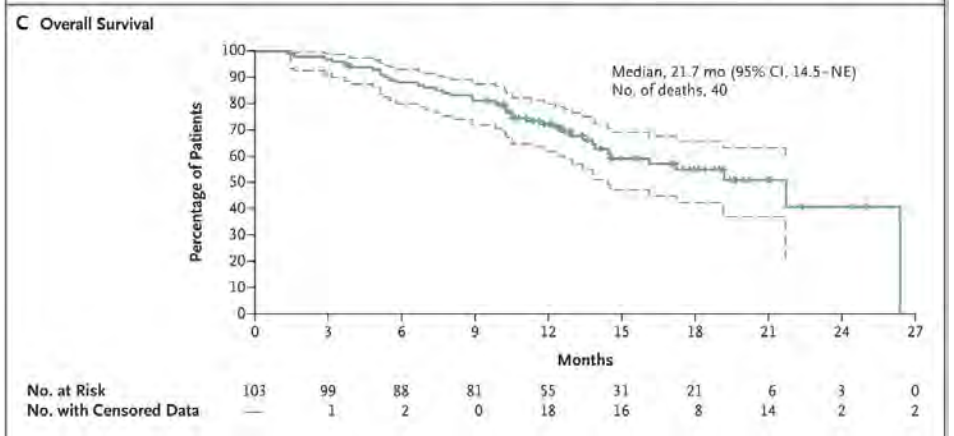


Activity in a broad range of cancers and against a broad variety of FGFR aberrations including patients who developed acquired resistance to a prior FGFRi.

Meric-Bernstam et al, Cancer Discov 2022







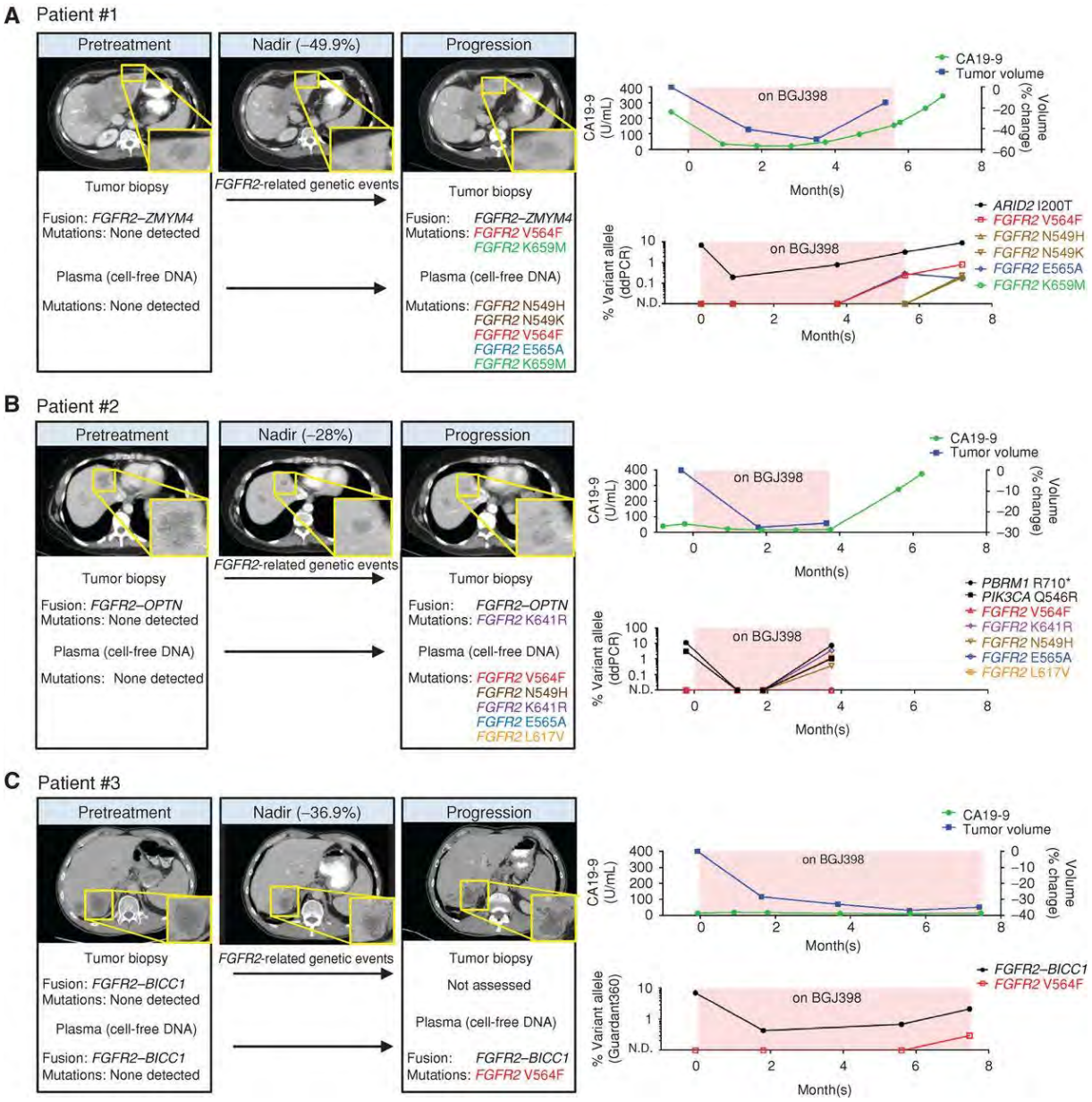
Futibatinib for *FGFR2*-Rearranged Intrahepatic Cholangiocarcinoma

Lipika Goyal, M.D., Funda Meric-Bernstam, M.D., Antoine Hollebecque, M.D., Juan W. Valle, M.D., Chigusa Morizane, M.D., Ph.D., Thomas B. Karasic, M.D., Thomas A. Abrams, M.D., Junji Furuse, M.D., Ph.D., Robin K. Kelley, M.D., Philippe A. Cassier, M.D., Heinz-Josef Klümpen, M.D., Ph.D., Heung-Moon Chang, M.D., et al., for the FOENIX-CCA2 Study Investigators*

Phase II data, non-randomized, 103 patients

- ORR>40%
- mPFS – 9 months
- Treatment-related AEs
  - Hyperphosphatemia, alopecia, dry mouth, diarrhea, fatigue
  - Well tolerated

Polyclonal Secondary Mutations in FGFR2 fusion



Goyal et al. Cancer Discov. 2017

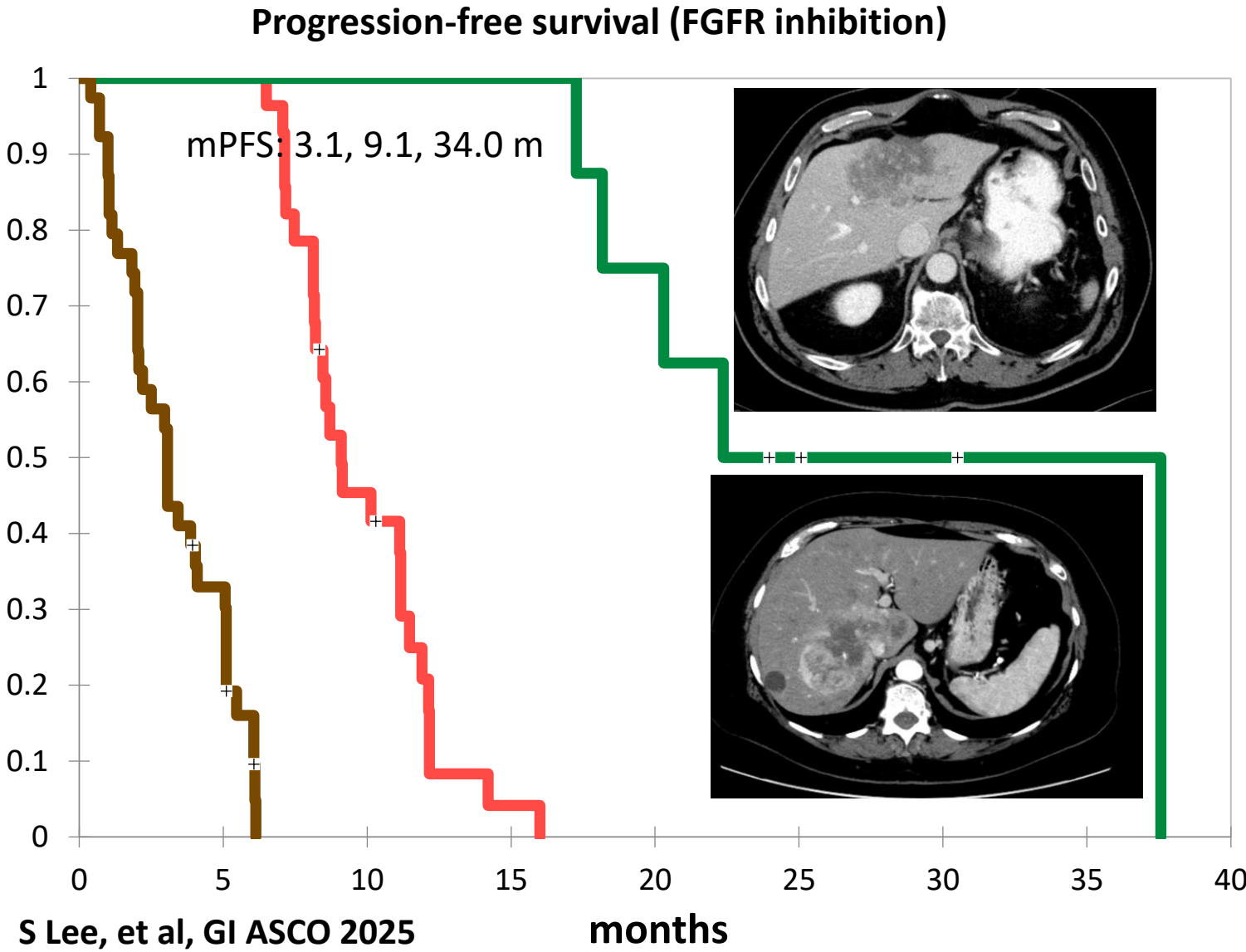
FGFRi, genomic alteration and vascularity

- Co-existing alterations with FGFR2 fusion/rearrangement
  - Resistant FGFR2 mutations
  - mTOR, TSC1/2 pathways
  - EGFR
  - KRAS

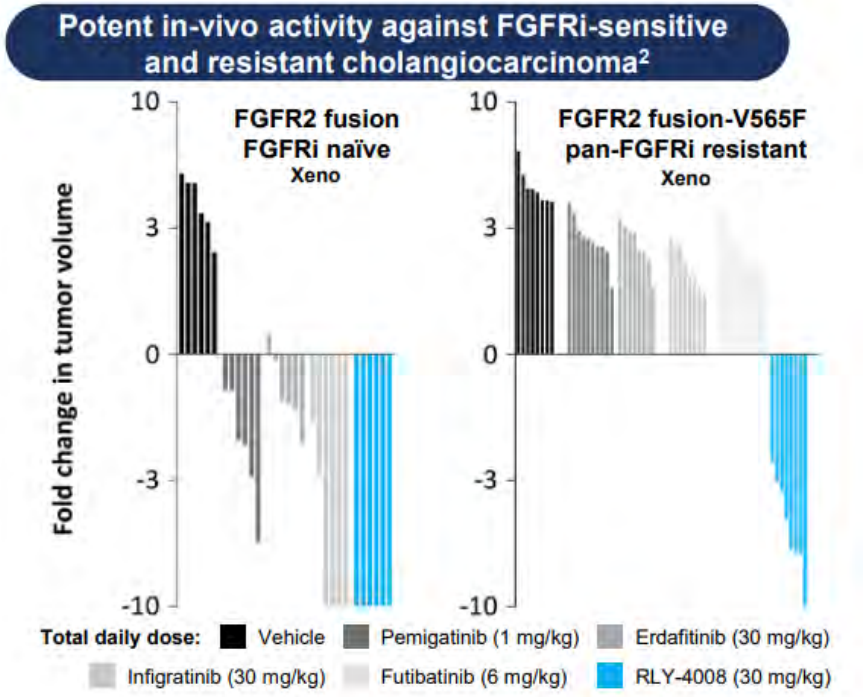
- Tumor vascularity significantly impacting response
- 3 groups based on tumor vascularity

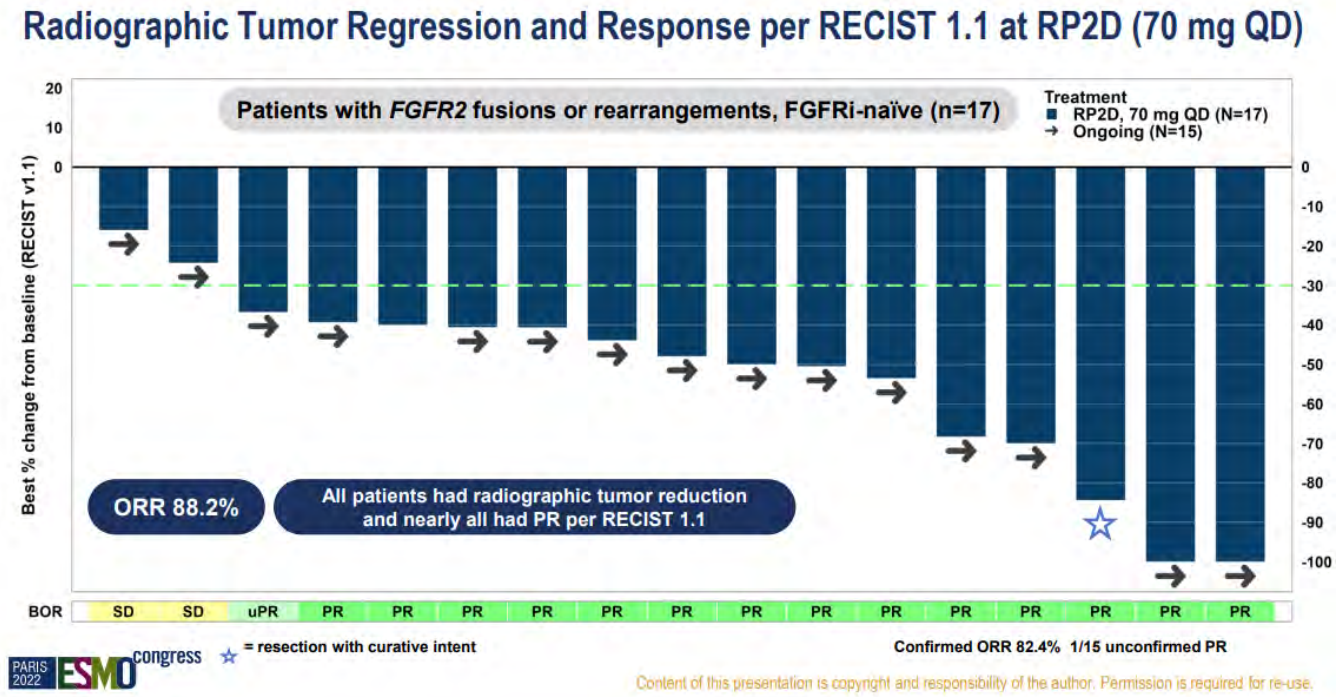
  - HU measurement

  - mRNA expression of genes related to VEGF(R)



NextGen FGFRi: RLY-4008 (lirafugratinib) Irreversible FGFR2i





- At the RP2D 70 mg QD, ORR is 88% (15/17, 15 with response ongoing)
- Across doses, ORR is 63%

Most AEs are low grade, largely reversible on-target AEs

Help from tumor agnostic trials

Targeting **BRAF V600E**

Efficacy and Safety of dabrafenib and trametinib in patients with BRAF V600E – ROAR trial

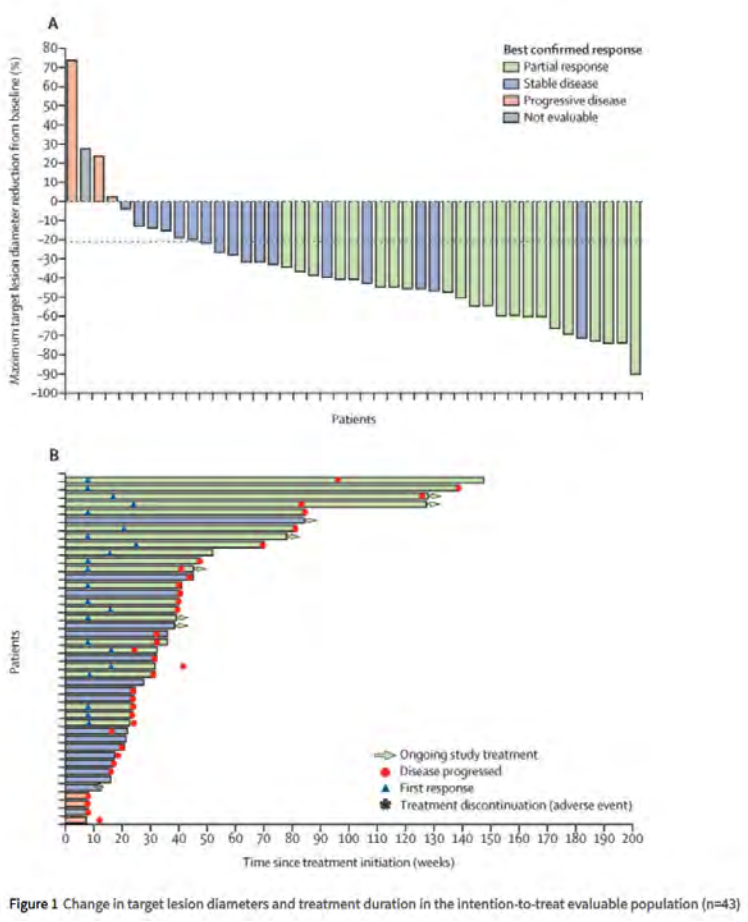


Figure 1 Change in target lesion diameters and treatment duration in the intention-to-treat evaluable population (n=43)

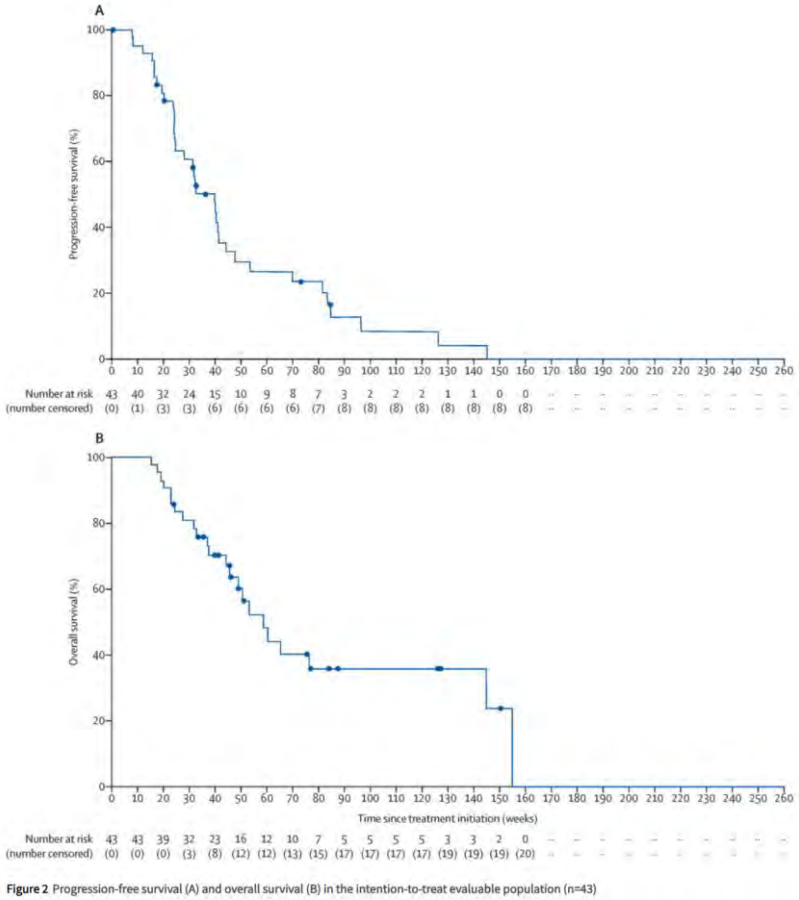


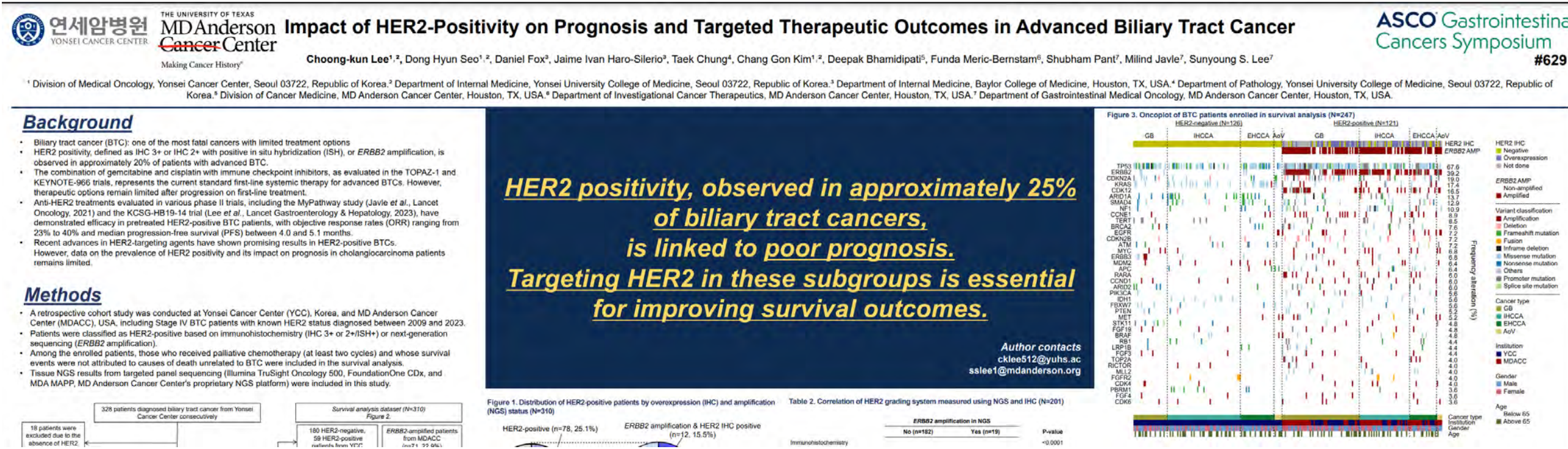
Figure 2 Progression-free survival (A) and overall survival (B) in the intention-to-treat evaluable population (n=43)

Confirmed partial responses occurred in 36%, mPFS 9.2 months, mOS 11.7 months

Subbiah et al, Lancet Oncology 2020

HER2 amplification in BTC

HER2 amplification or overexpression can be found in 5 - 10% of cholangiocarcinoma and in 15-20% of gallbladder cancer.



연세암병원 YONSEI CANCER CENTER
THE UNIVERSITY OF TEXAS MD Anderson Cancer Center Making Cancer History®

Impact of HER2-Positivity on Prognosis and Targeted Therapeutic Outcomes in Advanced Biliary Tract Cancer

ASCO Gastrointestinal Cancers Symposium #629

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Background

- Biliary tract cancer (BTC): one of the most fatal cancers with limited treatment options
- HER2 positivity, defined as IHC 3+ or IHC 2+ with positive in situ hybridization (ISH), or *ERBB2* amplification, is observed in approximately 20% of patients with advanced BTC.
- The combination of gemcitabine and cisplatin with immune checkpoint inhibitors, as evaluated in the TOPAZ-1 and KEYNOTE-966 trials, represents the current standard first-line systemic therapy for advanced BTCs. However, therapeutic options remain limited after progression on first-line treatment.
- Anti-HER2 treatments evaluated in various phase II trials, including the MyPathway study (Javle *et al.*, Lancet Oncology, 2021) and the KCSG-HB19-14 trial (Lee *et al.*, Lancet Gastroenterology & Hepatology, 2023), have demonstrated efficacy in pretreated HER2-positive BTC patients, with objective response rates (ORR) ranging from 23% to 40% and median progression-free survival (PFS) between 4.0 and 5.1 months.
- Recent advances in HER2-targeting agents have shown promising results in HER2-positive BTCs. However, data on the prevalence of HER2 positivity and its impact on prognosis in cholangiocarcinoma patients remains limited.

Methods

- A retrospective cohort study was conducted at Yonsei Cancer Center (YCC), Korea, and MD Anderson Cancer Center (MDACC), USA, including Stage IV BTC patients with known HER2 status diagnosed between 2009 and 2023.
- Patients were classified as HER2-positive based on immunohistochemistry (IHC 3+ or 2+/ISH+) or next-generation sequencing (*ERBB2* amplification).
- Among the enrolled patients, those who received palliative chemotherapy (at least two cycles) and whose survival events were not attributed to causes of death unrelated to BTC were included in the survival analysis.
- Tissue NGS results from targeted panel sequencing (Illumina TruSight Oncology 500, FoundationOne CDx, and MDA MAPP, MD Anderson Cancer Center's proprietary NGS platform) were included in this study.

328 patients diagnosed biliary tract cancer from Yonsei Cancer Center consecutively

18 patients were excluded due to the absence of HER2

Survival analysis dataset (N=310) *Figure 2.*

180 HER2-negative, 59 HER2-positive patients from YCC

ERBB2-amplified patients from MDACC

HER2 positivity, observed in approximately 25% of biliary tract cancers, is linked to poor prognosis. Targeting HER2 in these subgroups is essential for improving survival outcomes.

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Figure 1. Distribution of HER2-positive patients by overexpression (IHC) and amplification (NGS) status (N=310)

HER2-positive (n=78, 25.1%)

ERBB2 amplification & HER2 IHC positive (n=12, 15.5%)

Table 2. Correlation of HER2 grading system measured using NGS and IHC (N=201)

	ERBB2 amplification in NGS		
Immunohistochemistry	No (n=182)	Yes (n=19)	P-value
			<0.0001

Figure 3. Oncoplot of BTC patients enrolled in survival analysis (N=247)

HER2-negative (N=126) HER2-positive (N=121)

GB IHCA EHCCA AoV GB IHCA EHCCA AoV

HER2 IHC *ERBB2* AMP

TP53 ERBB2 CDKN2A KRAS CDK12 ARID1A SMAD4 NF1 CCNE1 TERT BRCA2 EGFR CDKN2B ATM MYC ERBB3 MDM2 APC RARA CCND1 ARID2 PIK3CA IDH1 FBXW7 PTEN MET STK11 FGF19 BRAF RB1 LRP1B FGF3 TOP2A RICTOR MLL2 FGFR2 CDK4 PBRM1 FGF4 CDK6

67.6 39.2 19.0 17.4 16.5 13.7 12.9 10.9 8.9 8.5 7.6 7.2 7.2 7.2 6.8 6.8 6.4 6.4 6.0 6.0 6.0 5.6 5.6 5.6 5.2 5.2 4.8 4.8 4.8 4.4 4.4 4.4 4.0 4.0 4.0 4.0 4.0 3.6 3.6 3.6

Frequency alteration (%)

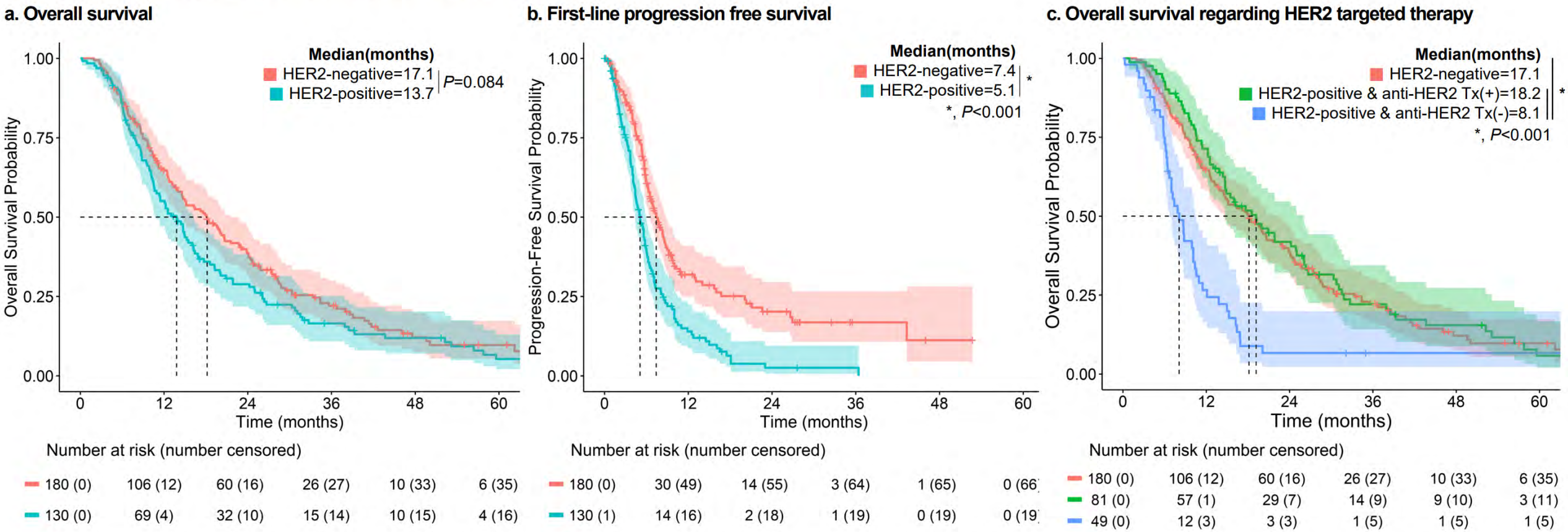
HER2 IHC: Negative, Overexpression, Not done
ERBB2 AMP: Non-amplified, Amplified
Variant classification: Amplification, Deletion, Frameshift mutation, Fusion, Inframe deletion, Missense mutation, Nonsense mutation, Others, Promoter mutation, Splice site mutation
Cancer type: GB, IHCCA, EHCCA, AoV
Institution: YCC, MDACC
Gender: Male, Female
Age: Below 65, Above 65

Cancer type Institution Gender Age

Lee, et al, GI ASCO 2025

HER2 amplification in BTC

Figure 2. Survivals according to HER2 status and HER2 targeted therapy (N=310)



Lee, et al, GI ASCO 2025, under review

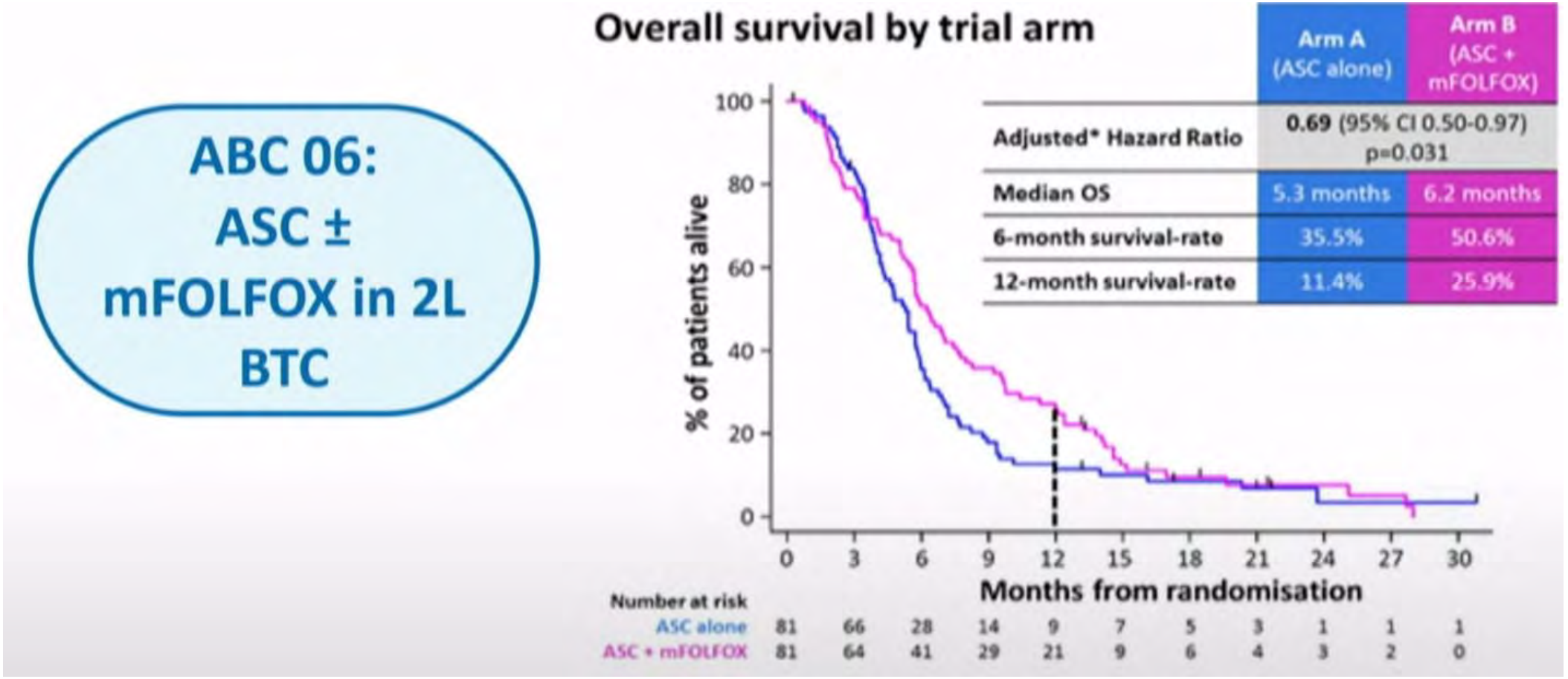
Targeting HER2 amplification

- HER2 amplification or overexpression can be found in 5 - 10% of cholangiocarcinoma and in 15-20% of gallbladder cancer.

Therapy	Progression-Free Survival (PFS)	Overall Response Rate (ORR)	Overall Survival (OS)	Key Notes
Trastuzumab + Pertuzumab	4.0 months	23%	10.9 months	NCCN guideline
Zanidatamab	6.0 months	41.3% overall; 51.6% (IHC3+), 5.6% (IHC2+)	15.5 months	FDA approved it a few months ago
Trastuzumab Deruxtecan	6.9 months	36%		FDA-approved for HER2-positive solid tumors
Trastuzumab + Tucatinib	5.5 months	46.7%	15.5 months	NCCN guideline

Second-Line Treatment – Chemo (FOLFOX)

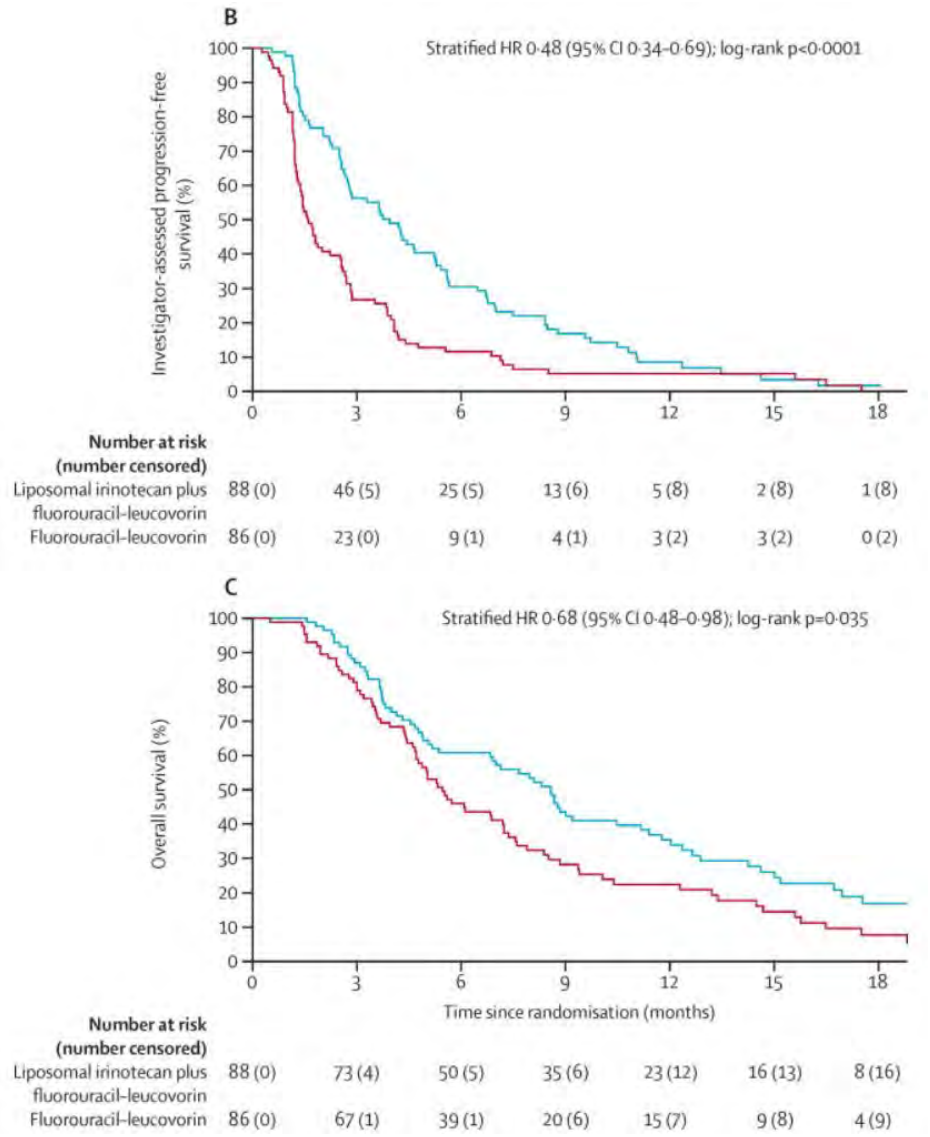
ABC 06:
ASC ± mFOLFOX in 2L BTC

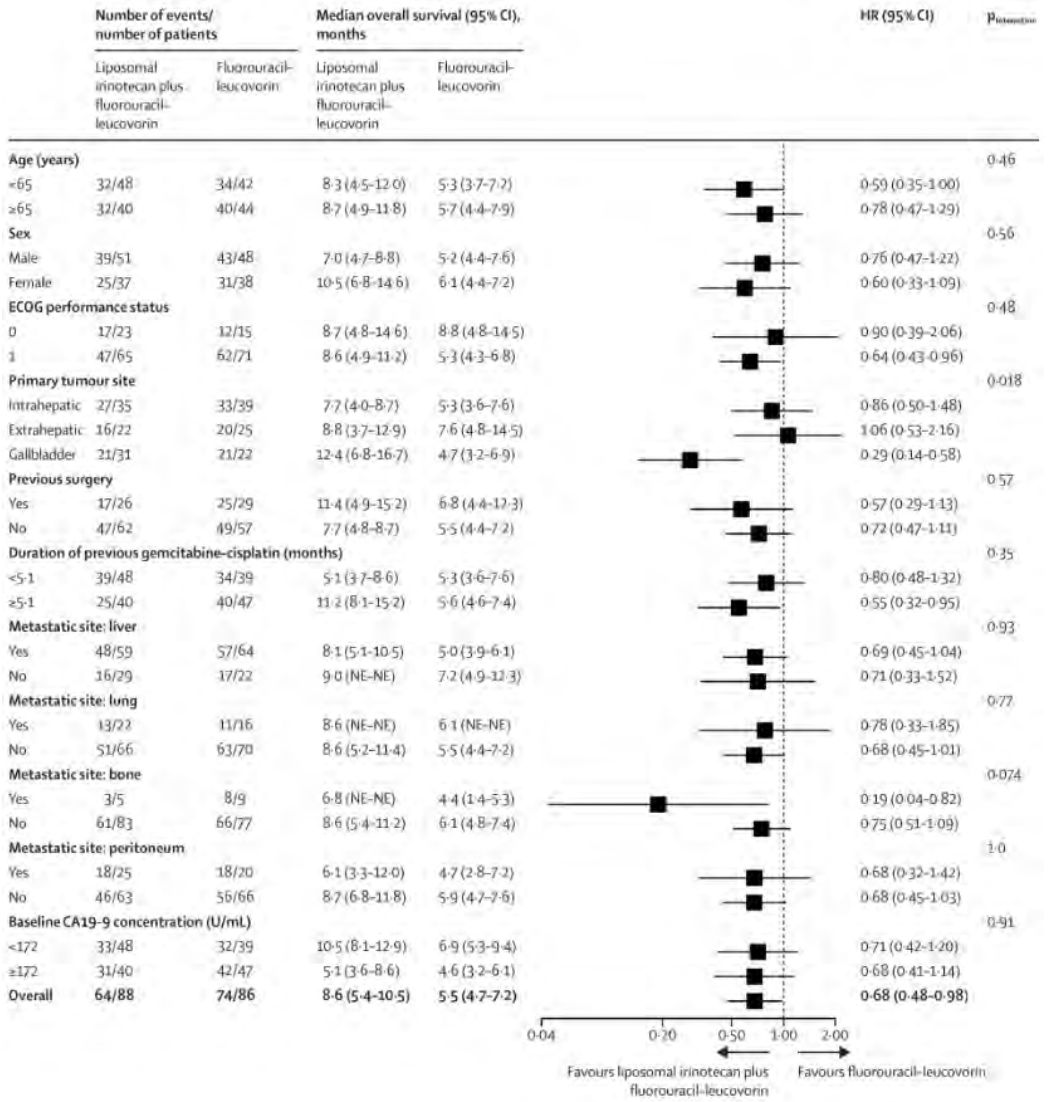


FDA approved.

Lamarca et al. ASCO 2019

Second-Line – Chemo (5-FU + liposomal irinotecan)



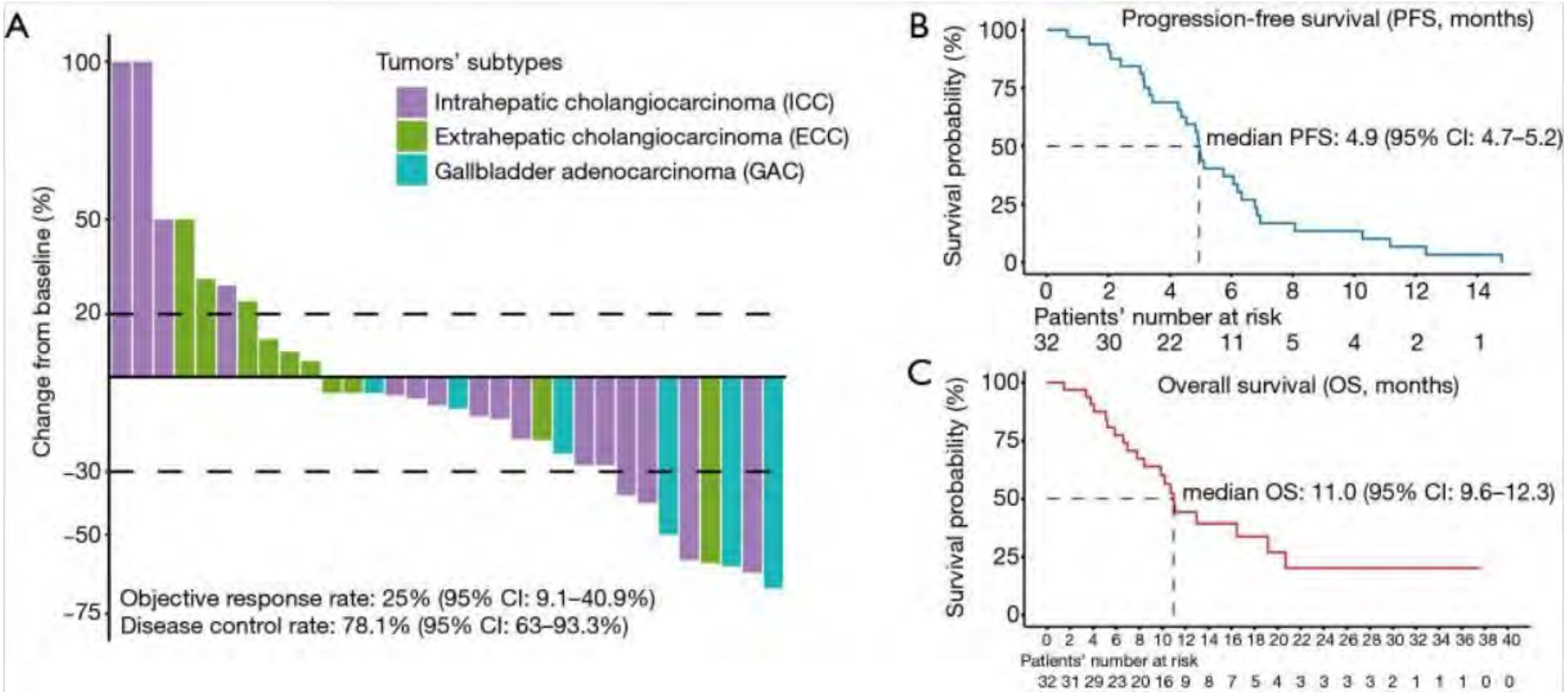


ORR: 17%, decent ORR in the 2nd line setting, OS 8.6 months

FDA not approved, but very commonly used in clinic.

Yoo et al, Lancet Onc 2021

Anti-VEGF(R): Lenvatinib + Pembrolizumab



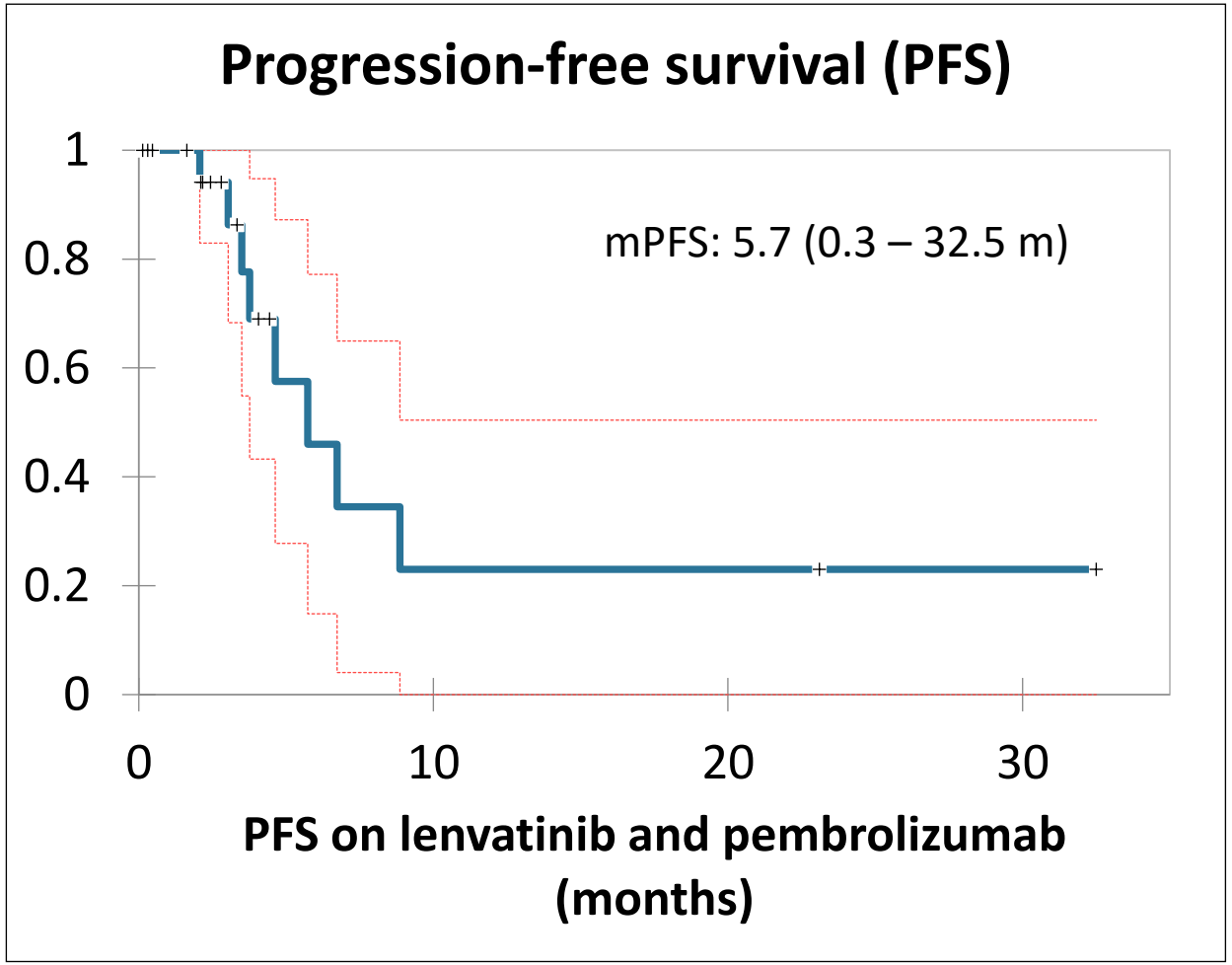
ORR: 25%, SD 53%, mPFS 4.9 m, mOS 11.0

FDA not approved, but commonly used in clinic → Recently removed from NCCN

Leap-5 trial, Lin et al. 2020

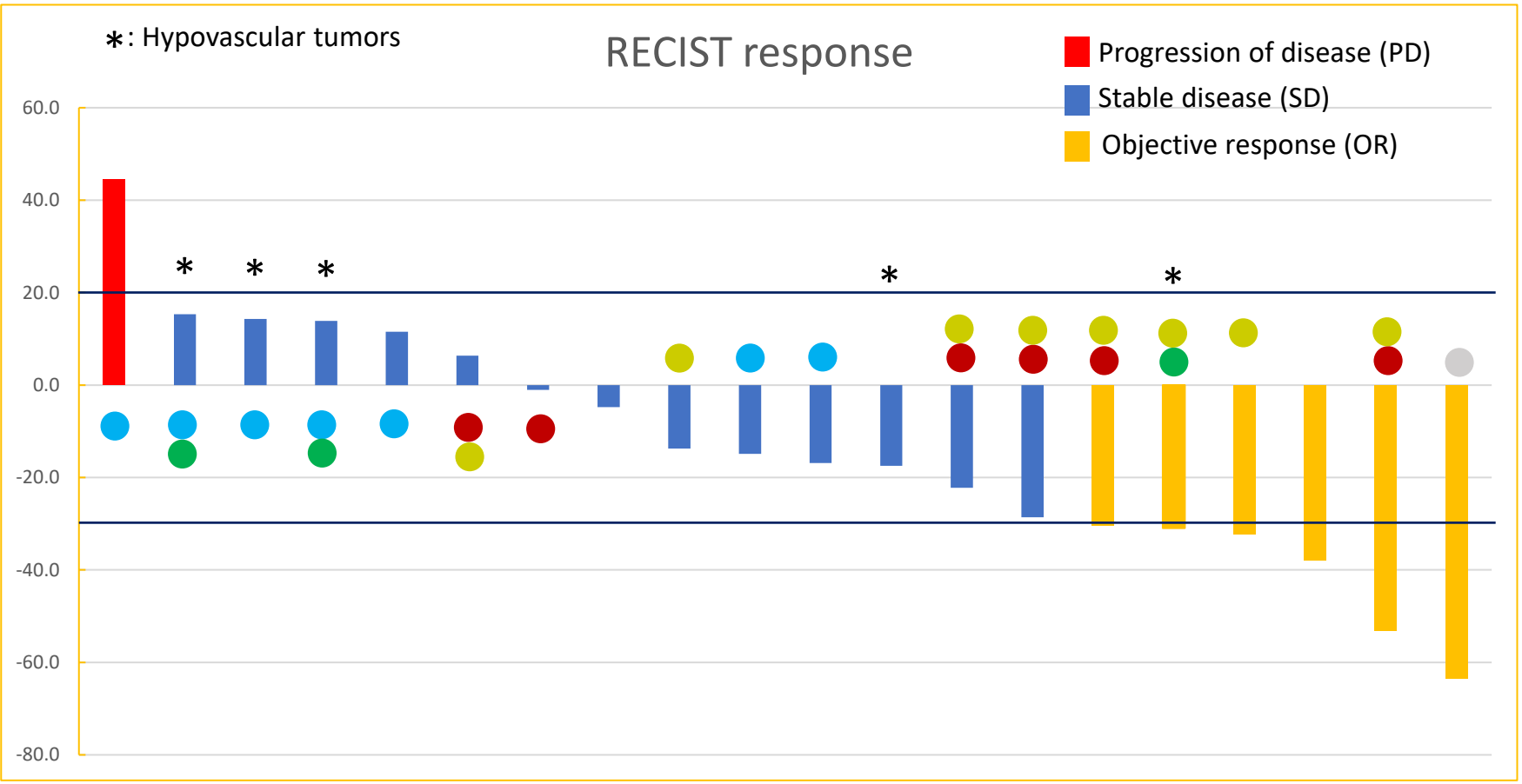
Lenvatinib + Pembrolizumab

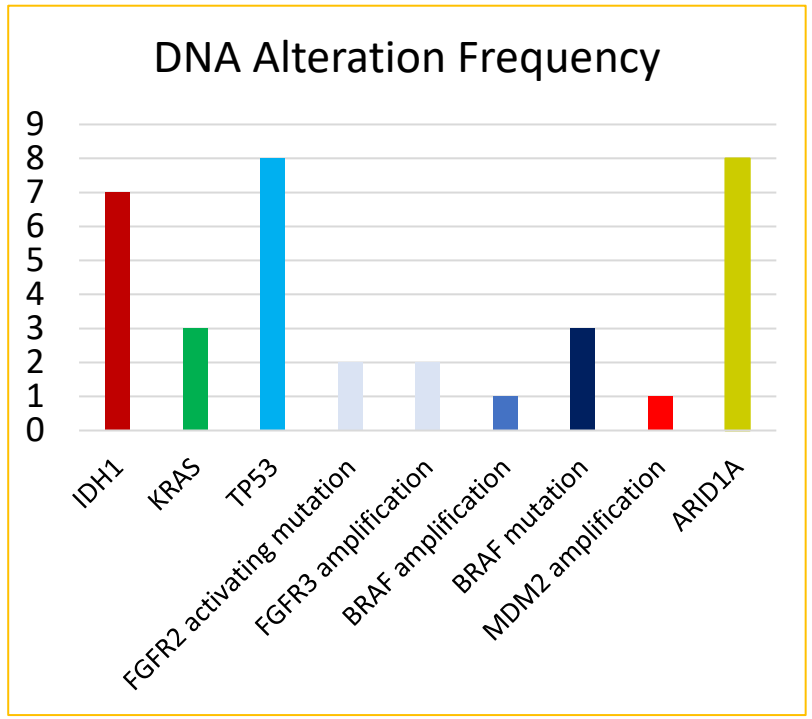
- MD Anderson study:
  - mPFS 5.7 month (0.3 – 32.5 months)
- 2 patients are on this over 23.1 and 32.5 m (still receiving them)
- Only 5 patients received this regimen in the 2nd line; all other patients received in 3+ lines.
- When lenvatinib was provided 4+ lines, patients did not tolerate it well.
- 4 patients with IDH1 mutation received ivosidenib without response, before receiving lenvatinib and pembrolizumab.



S Lee et al, 2025, under review

Overall Response Rate (ORR) / DNA Alterations





While # patients is not large:

1) TP53 mutations are associated with decreased response.
2) IDH1/ARID1A mutated tumors are hypervascular and associated with better response.

S Lee et al, under review

Progress in 10 years

- Biliary tract cancer firmly entered the era of precision medicine and immuno-oncology
  - Many problems still to solve
    - Biomarkers for selection and resistant pathways
    - Optimizing therapy – best sequencing and combinations
    - Improve access to profiling and precision drugs in routine care
- Inhibitors and clinical trials: KRAS, BRAF, MTAP loss, MDM2 amplification, PIK3CA, IDH2
- Antibody-drug conjugate (CDH6, FOLR1, TROP2, Nectin4)
- Anti-VEGF(R) including bevacizumab, CTX-009, lenvatinib, rivoceranib, ramucirumab
- Studies underway
  - Peri-operative and down-staging, transplant, radiotherapy approaches
  - More international collaborations

Thank you

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