

事例検討⑨ 資料

HRD signatureについて

アカデミア・アセンブリ 全体会議 検討日
2024年8月22日 (木)

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HRD signatureについて

FoundationOne® CDx がんゲノムプロファイル 解析結果レポートの機能変更のお知らせ



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このたび疾病診断用プログラム 高度管理医療機器「FoundationOne® CDx がんゲノムプロファイル (F1CDx)」につきまして、
以下の通り、解析結果レポートの記載方法を一部変更いたします。

変更予定の内容（詳細は次頁以降参照）

#	変更箇所	変更内容	変更対象製品	
			F1CDx	F1LiquidCDx
①	Professional service page Summary Box	LOHスコアとHRDステータスの削除（卵巣がん、卵管がん、腹膜がん） HRD signature の追加（全ての固形がん）	○	—
②	Professional service page Summary Table	LOHスコアとHRDステータスの削除（卵巣がん、卵管がん、腹膜がん） HRD signature の追加（全ての固形がん）	○	—
③	Professional service page Biomarker Findings page	HRD signature スコアの追加（HRDsig Positiveまたは HRDsig Negativeの場合）	○	—
④	AppendixGenes Assayed in FoundationOne®CDx	LOHスコアとHRDステータスの削除（卵巣がん、卵管がん、腹膜がん）	○	—

FoundationOne® CDx がんゲノムプロファイル 解析結果レポートの機能変更のお知らせ

遺伝子変異解析プログラム(がんゲノムプロファイリング検査用)

体細胞遺伝子変異解析プログラム(抗悪性腫瘍薬適応判定用)



【変更箇所①】 Professional service page Summary Box

【変更内容①】 LOHスコアとHRDステータスの削除（卵巣がん、卵管がん、腹膜がん）、HRD signature の追加（全ての固形がん）

変更前

Biomarker Findings

Homologous Recombination status - HRD Positive

Loss of Heterozygosity score - 8.8%

Microsatellite status - MS-Stable

Tumor Mutational Burden - 0 Muts/Mb

Genomic Findings

For a complete list of the genes assayed, please refer to the Appendix.

ERBB2 amplification

TP53 M237I

BRCA1 splice site 594-2A>C - subclonal[†]

RAD21 amplification

変更後

Biomarker Findings

HRD signature - HRDsig Positive

Microsatellite status - MS-Stable

Tumor Mutational Burden - 6 Muts/Mb

Genomic Findings

For a complete list of the genes assayed, please refer to the Appendix.

TP53 R249S

PMS2 splice site 538-2A>G

- ・ 卵巣がん、卵管がん、腹膜がんにおいて、HRDステータスとLOHスコアは報告されなくなります。
- ・ 全ての固形がんにおいて、HRD signatureが報告されます。
- ・ HRD signatureは、Positive/Negative/Cannot Be Determinedの3種類で報告されます。

※本画像は、変更箇所①のサンプルです。実際の解析結果レポートの表示は変更となる可能性があります。

<解析結果レポートご確認の際にご注意いただきたいこと>

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遺伝子変異解析プログラム(がんゲノムプロファイリング検査用)

体細胞遺伝子変異解析プログラム(抗悪性腫瘍薬適応判定用)



【変更箇所②】 Professional service page Summary Table

【変更内容②】 LOHスコアとHRDステータスの削除（卵巣がん、卵管がん、腹膜がん）、HRD signature の追加（全ての固形がん）

変更前

BIOMARKER FINDINGS
Homologous Recombination status - HRD Positive
Loss of Heterozygosity score - 8.8%



変更後

BIOMARKER FINDINGS
HRD signature - HRDsig Positive
10 Trials see p. 11

- ・ 卵巣がん、卵管がん、腹膜がんにおいて、HRDステータスとLOHスコアは報告されなくなります。
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【変更箇所③】 Professional service page Biomarker Findings page

【変更内容③】 HRD signature スコアの追加 (HRDsig Positiveまたは HRDsig Negativeの場合)



- ・ 全ての固形がんにおいて、HRD signatureが報告されます。
- ・ HRD signatureは、Positive/Negative/Cannot Be Determinedの3種類で報告されます。
- ・ HRD signatureがPositiveまたはNegativeの場合、スコアが報告されます。

※本画像は、変更箇所③のサンプルです。実際の解析結果レポートの表示は変更となる可能性があります。

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PARP阻害薬

薬剤	適応
オラパリブ (リムパーザ®)	・ 白金系抗悪性腫瘍剤感受性の再発卵巣癌における維持療法 ・ BRCA遺伝子変異陽性の卵巣癌における初回化学療法後の維持療法 ・ 相同組換え修復欠損を有する卵巣癌におけるベバシズマブ（遺伝子組換え）を含む初回化学療法後の維持療法 ・ がん化学療法歴のあるBRCA遺伝子変異陽性かつHER2陰性の手術不能又は再発乳癌 ・ BRCA遺伝子変異陽性かつHER2陰性で再発高リスクの乳癌における術後薬物療法 ・ BRCA遺伝子変異陽性の遠隔転移を有する去勢抵抗性前立腺癌 ・ BRCA遺伝子変異陽性の治癒切除不能な膀胱癌における白金系抗悪性腫瘍剤を含む化学療法後の維持療法
ニラパリブ (ゼジューラ®)	・ 卵巣癌における初回化学療法後の維持療法 ・ 白金系抗悪性腫瘍剤感受性の再発卵巣癌における維持療法 ・ 白金系抗悪性腫瘍剤感受性の相同組換え修復欠損を有する再発卵巣癌
タラゾパリブ (ターゼナ®)	〈ターゼナカプセル0.1mg〉 ・ BRCA遺伝子変異陽性の遠隔転移を有する去勢抵抗性前立腺癌 〈ターゼナカプセル0.25mg〉 ・ BRCA遺伝子変異陽性の遠隔転移を有する去勢抵抗性前立腺癌 〈ターゼナカプセル1mg〉 ・ がん化学療法歴のあるBRCA遺伝子変異陽性かつHER2陰性の手術不能又は再発乳癌

コンパニオン診断 (2024年8月5日版)

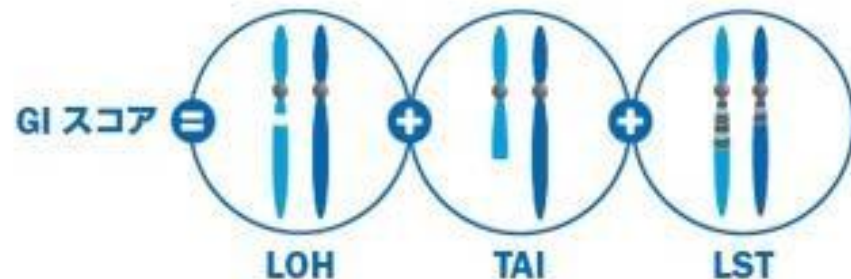
販売名	成分名	適応	適応判定に利用可能な体外診断用医薬品又は医療機器	検査項目
リムパーザ錠100 mg 同錠150 mg	オラパリブ	乳癌	BRACAnalysis診断システム	BRCA1/2遺伝子変異
		卵巣癌	BRACAnalysis診断システム	
			FoundationOne CDx がんゲノムプロファイル	
			myChoice診断システム	相同組換え修復欠損
				BRCA1/2遺伝子変異
		前立腺癌	FoundationOne CDx がんゲノムプロファイル	BRCA1/2遺伝子変異
			BRACAnalysis診断システム	
			FoundationOne Liquid CDx がんゲノムプロファイル	
ゼジューラカプセル100 mg 同錠100 mg	ニラパリブ トシル酸塩水和物	卵巣癌	myChoice診断システム	相同組換え修復欠損
ターゼナカプセル0.1mg 同カプセル0.25mg 同カプセル1mg	タラゾパリブ トシル酸塩	前立腺癌	FoundationOne CDx がんゲノムプロファイル	BRCA1/2遺伝子変異
		乳癌	BRACAnalysis診断システム	

MyChoice[®]診断システム

HRD Companion Diagnostic Test

① 腫瘍ゲノム不安定性の状態 (GIS) | LOH + TAI + LST
ヘテロ接合性の消失 + テロメアアレル不均衡 + 大規模な状態遷移

● ゲノム全体に渡るLOH、TAI、LSTを包括的に評価



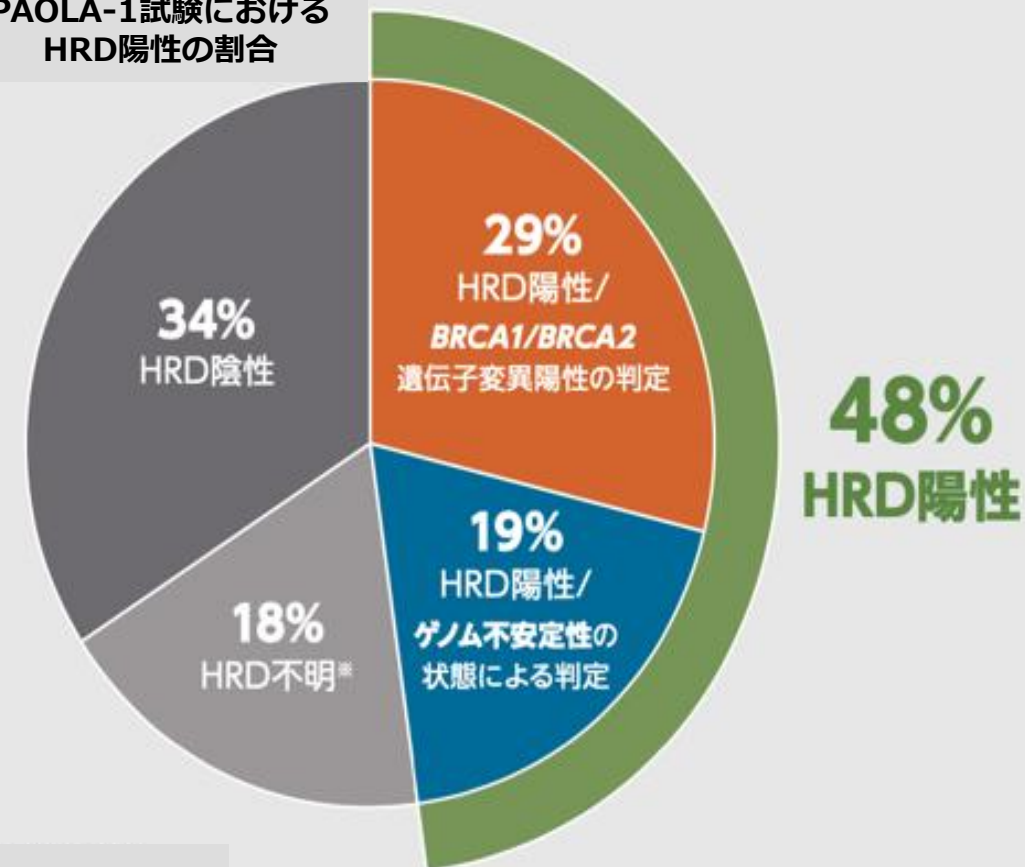
② BRCA1/BRCA2 バリエーション状態 | シーケンスバリエーション + 大規模遺伝子再構成

● 腫瘍組織から抽出したゲノムDNA中のBRCA1/BRCA2の生殖細胞系列および体細胞変異を検出・分類

留意事項: 生殖細胞系列の変異と、体細胞の変異の区別はできません。

③ LOH、TAIおよびLSTの集計量解析に基づいて、ゲノム不安定性 (GI) スコアが算出されます。バリデートされたカットオフ値により、検査報告書にはGISが陽性 (42以上) または陰性として報告されます。

PAOLA-1試験における
HRD陽性の割合



MyChoice[®] 診断システム

HRD Companion Diagnostic Test

myChoice[®] CDx biomarker definitions



LOH: Presence of a single allele



TAI: A discrepancy in the 1:1 allele ratio at the end of the chromosome (telomere)



LST: Transition points between regions of abnormal and normal DNA or between two different regions of abnormality

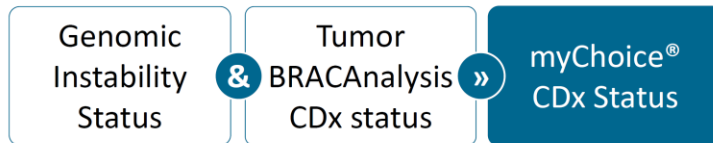
Univariate Analysis of Dichotomous Scores (SCOTROC4 cohort)

	PFS		OS	
High/Low	P Value	HR	P Value	HR
HRD	2×10^{-6}	0.66	1×10^{-8}	0.55
LOH	5×10^{-5}	0.70	8×10^{-6}	0.64
TAI	9×10^{-5}	0.71	6×10^{-7}	0.61
LST	1×10^{-5}	0.68	3×10^{-7}	0.60

MyChoice[®] 診断システム

HRD Companion Diagnostic Test

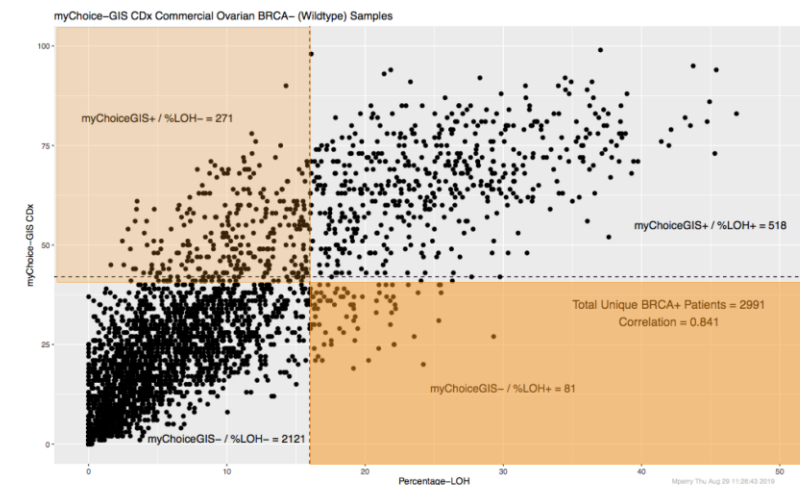
How Tumor BRACAnalysis CDx status and Genomic Instability Status factor into final myChoice[®] CDx status



Genomic Instability Status	Tumor BRACAnalysis CDx status	Final myChoice [®] CDx status
Positive	Negative	Positive
Positive	Positive	Positive
Negative	Positive	Positive
Negative	Negative	Negative

myChoice[®] CDx vs. %LOH (BRCA wild type samples)

myChoice[®] HRD+/%LOH- = 271
(9% of total/34% of myChoice[®] HRD+)



MyChoice[®] 診断システム

HRD Companion Diagnostic Test

myChoice[®] CDx vs. %LOH (BRCA wild type, N = 2991)

		%LOH	
		Positive	Negative
myChoice [®] CDx	Positive	518	271
	Negative	81	2121

- PPA: 65.7% (false negative rate from %LOH = 34.3%)
- NPA: 96.3% (false positive rate from %LOH = 3.7%)

myChoice[®] CDx vs. 11 Gene HRR Panel

- Performed variant and large rearrangement detection on genes from TumorNext-HRD panel on 187 samples from the SCOTROC4 cohort
- Assumed no differences in assay sensitivity or variant classification between the Myriad and other company's processes

		TumorNext-HRD Gene Panel	
		Positive	Negative
myChoice [®] CDx	Positive	26	23
	Negative	9	129

- PPA: 53.1% (false negative rate from 11 gene panel = 46.9%)
- NPA: 93.5% (false positive rate from 11 gene panel = 6.5%)

Summary

- These data show that tests used to evaluate HR deficiency in published and ongoing clinical trials are not equivalent, and they should not be considered interchangeable in predicting PARP inhibitor response in clinical practice
- %LOH may miss up to one third of patients who are appropriate candidates for PARP inhibitors
- An 11 HRR gene panel failed to identify nearly half of all myChoice[®] positive tumors in a HGSOC cohort

Summary

- The myChoice® CDx GI score is comprised of 3 independent measures of genomic instability
- The myChoice® CDx GI score shows improved sensitivity to identify patients that derive clinical benefit from DNA damaging chemotherapy than any of the individual biomarkers
- myChoice® CDx is being, or has been, used in over 30 PARP inhibitor clinical trials
- myChoice® CDx is the only HRD test to have been approved as a companion diagnostic by the US FDA for PARPi treatment in ovarian cancer, and is the only FDA approved HRD CDx which includes a genomic instability score

Expand Your Clinical Trial Population with **Our HRD Signature** (HRDsig)

—
HRDsig can identify more patients, including those **with or without relevant HRR gene mutations**, that may respond to therapy.

2023 PUBLICATION IN OVARIAN CANCER¹



39%

of patients were **HRDsig+** compared to
18% of patients who were **BRCA1/2+**.



Mutation+



Wild-Type with BRCAness

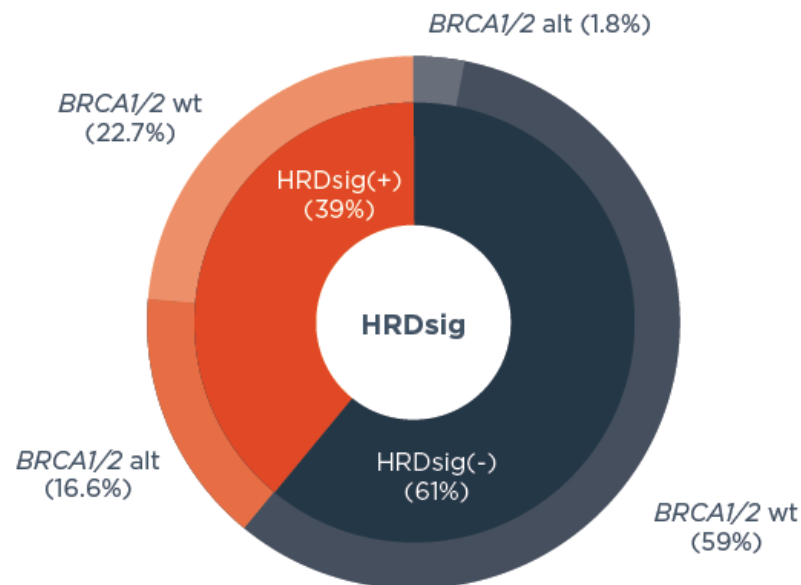


HRDsig has **validation across tumor types** that can support **clinical trials**
and future **CDx development** for tumor-specific or pan-tumor applications.

HRD signature



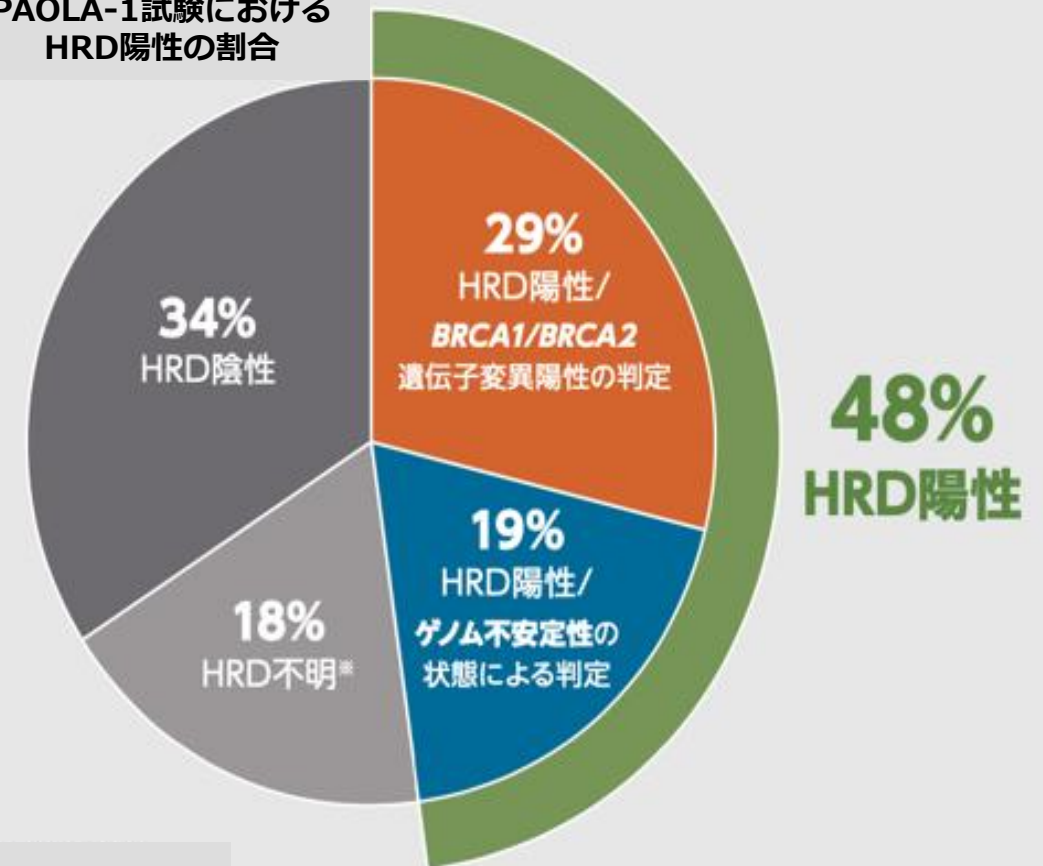
DATA EXAMPLE: OVARIAN CANCER¹



HRDsig+ is detected in 39% of patients overall, with 58% of these patients being BRCA1/2wt

MyChoice

PAOLA-1試験における HRD陽性の割合



HRDsig is a comprehensive, scar-based signature with diverse potential use cases for therapy development.

- 1 **Better performance than gLOH** (sensitivity and specificity)² and designed for use **across tumor types**
- 2 **Captures non-genomic mechanisms** of HRD, like *BRCA1* promoter methylation
- 3 Detects different HRD phenotypes and **can identify monoallelic HRR passenger mutations as HRDsig-**



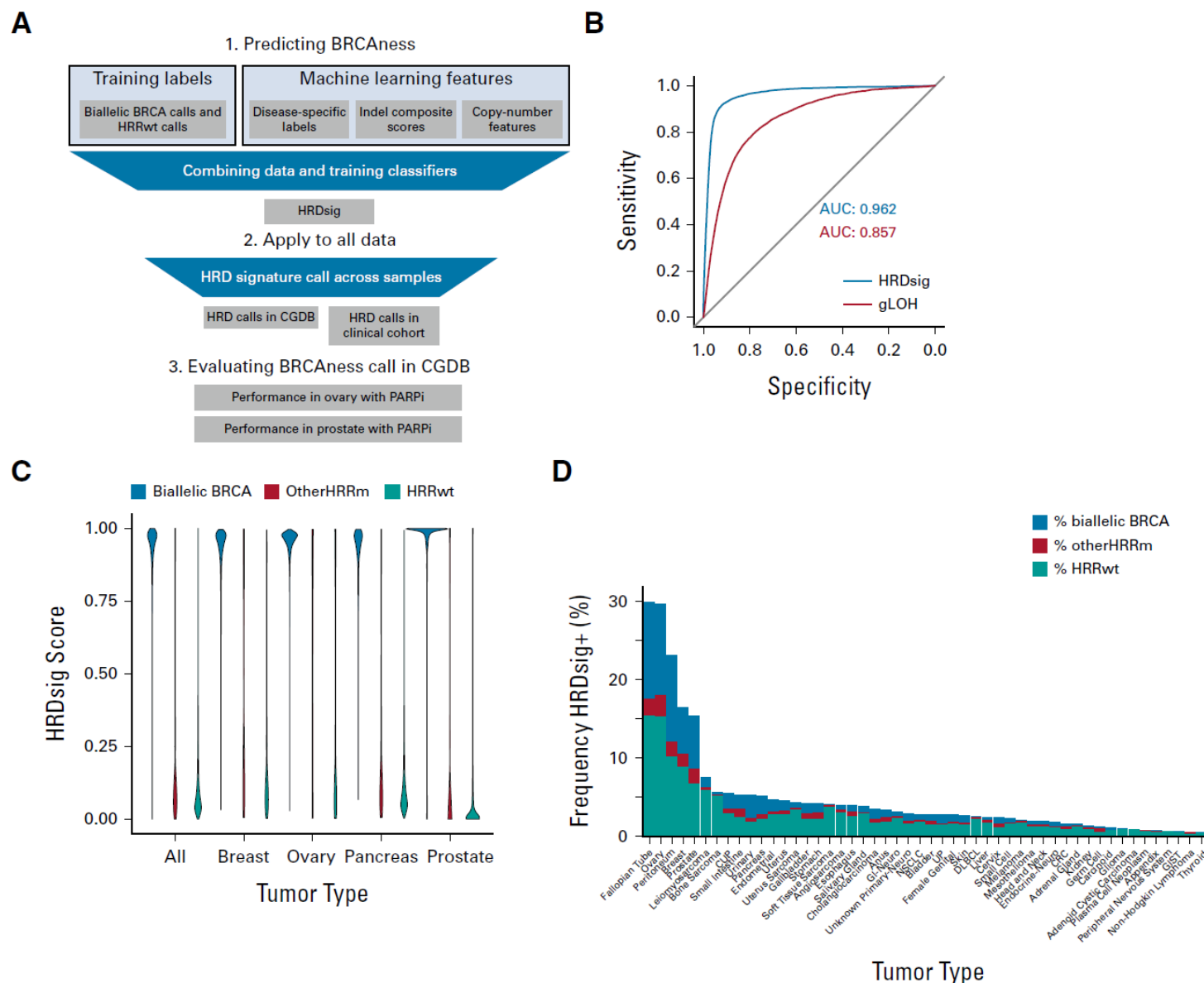
HRDsig was developed with the use of machine-learning methods and further explored using our FoundationCore[®] database, which contains comprehensive, de-identified genomic data from >600,000 patient samples.³



HRDsig can also be explored for research collaborations using blood or plasma samples—please contact us for more details.



HRD signature (HRDsig)



Using a large training data set, we trained an XGB machine learning classifier to detect HRD positivity (HRDsig).

We analyzed the landscape of CN features in 260,333 pan-cancer samples. We examined the association of 10 signatures with genomic alterations and clinical characteristics and trained a machine learning classifier using CN and insertion and deletion features to detect homologous recombination deficiency signature (HRDsig) positivity.

BIOMARKER

HRD signature

RESULT

HRDsig Positive

SCORE

POTENTIAL TREATMENT STRATEGIES

— Targeted Therapies —

On the basis of clinical data from real-world clinicogenomic database analyses, HRD signature (HRDsig) Positive status is associated with sensitivity to PARP inhibitors. Multiple studies of patients with either metastatic castration-resistant prostate cancer or metastatic breast cancer treated with PARP inhibitors observed more favorable OS, PFS, and time to treatment discontinuation for patients with HRDsig Positive status compared to patients with HRDsig Negative status¹⁻³. Patients

with HRDsig Positive platinum-sensitive advanced ovarian cancer who received PARP inhibitor maintenance therapy had more favorable PFS than patients who received no maintenance therapy or patients with HRDsig Negative status⁴. Additionally, in a real-world dataset, HRDsig Positive status was associated with improved outcomes on a platinum chemotherapy-containing regimen in pancreatic cancer⁵.

FREQUENCY & PROGNOSIS

HRD signature (HRDsig) Positive status has been identified in multiple solid tumor types, including prostate (43%), ovarian (39%), breast (21%), and pancreas (9%)²⁻⁵. A pan-cancer analysis of a large real-world dataset found a strong association between HRDsig Positive status and biallelic inactivation of BRCA1/2 in ovarian, prostate, and pancreatic cancers³. Published data investigating the prognostic implications of HRDsig Positive status in solid tumors are limited (PubMed, Jul 2024).

FINDING SUMMARY

Homologous recombination deficiency (HRD) produces characteristic genome-wide changes that accumulate as genomic scars because of incorrect DNA double-strand break repair. HRD signature (HRDsig) is a biomarker developed and validated to classify HRD status in pan-cancer samples using hundreds of copy number (CN) features to capture these genomic changes. HRDsig identifies both genomic and non-genomic mechanisms of HRD and has high sensitivity to detect samples with biallelic genomic alterations in homologous recombination repair (HRR) genes such as BRCA1/2. This sample has a status of HRD signature (HRDsig) Positive, defined as a model score of ≥ 0.7 . In real-world clinico-genomic database analyses, HRDsig Positive status was associated with sensitivity to PARP inhibitors in breast, ovarian, and prostate cancers¹⁻⁴ and a platinum chemotherapy-containing regimen in pancreatic cancer⁵.

Pan-Cancer Analysis of Copy-Number Features Identifies Recurrent Signatures and a Homologous Recombination Deficiency Biomarker to Predict Poly (ADP-Ribose) Polymerase Inhibitor Response

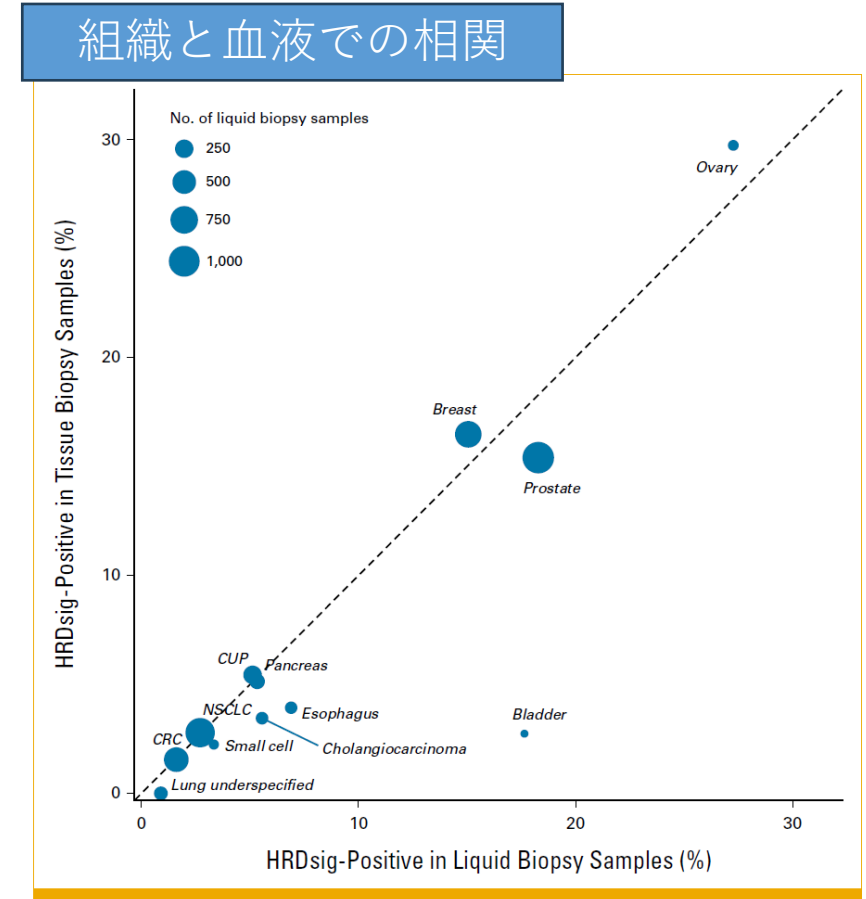
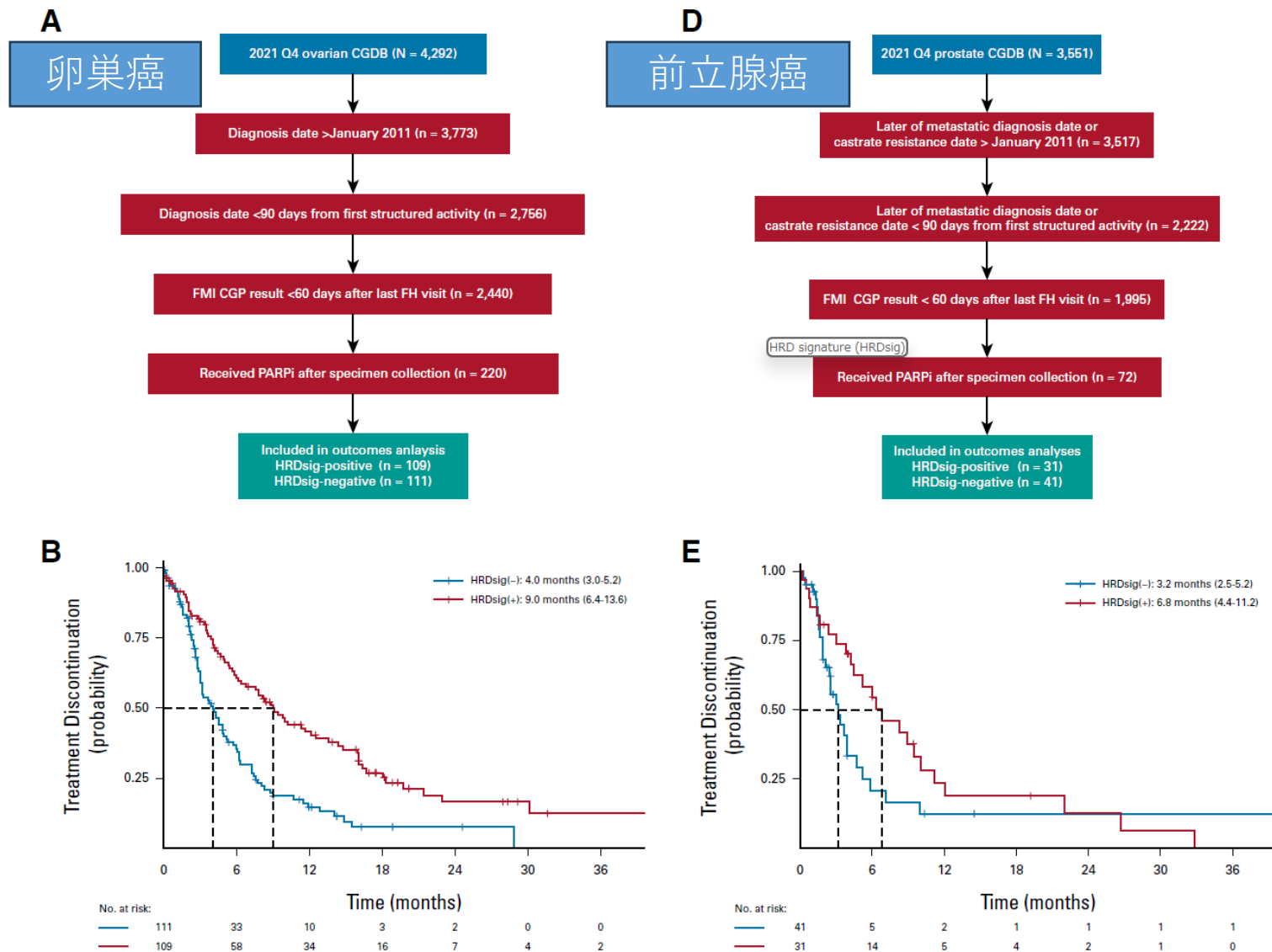


FIG A5. HRDsig is called at significant rates in liquid biopsy. Scatterplot show the prevalence of HRDsig-positive calls in tissue biopsy and prevalence of HRDsig-positive calls in liquid biopsy samples. A cutoff of >50 assessable liquid biopsy samples was used. Size of the points is the number of assessable liquid biopsies in each tumor type. CRC, colorectal cancer; CUP, carcinoma of unknown primary; HRDsig, homologous recombination deficiency signature; NSCLC, non-small-cell lung cancer.

Effectiveness of PARP inhibitor main 1 tenance therapy in ovarian cancer by BRCA1/2 and a scar-based HRD signature in real-world practice

- Patients with OC receiving 1st-line platinum-based chemotherapy and either mPARPi or no maintenance were included.
- Data was obtained from the US-wide Flatiron Health and Foundation Medicine clinicogenomic database (CGDB).
- Retrospective de-identified longitudinal clinical data were derived from electronic health records (EHR) from approximately 280 US cancer clinics (~800 sites of care) between January 2015 and March 2023 and comprises patient-level structured and unstructured data, curated via technology-enabled abstraction of clinical notes and radiology/pathology reports.
- A total of 673 OC patients were included in the study.

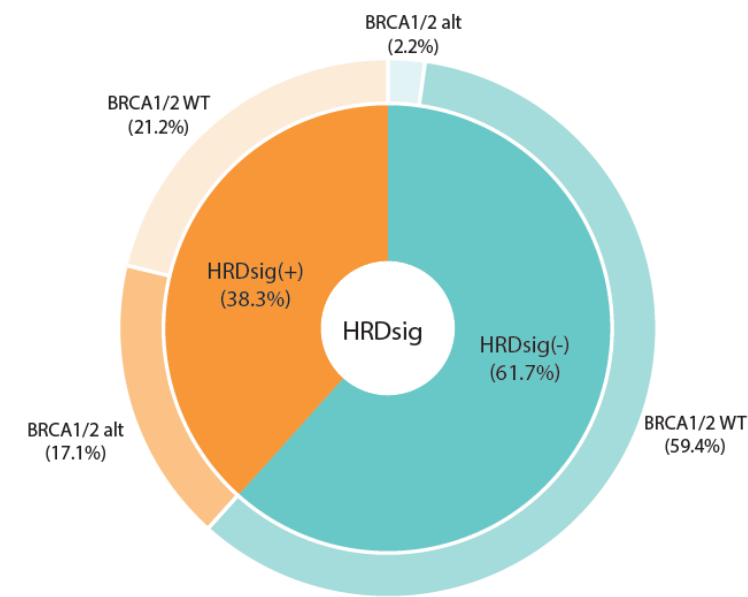
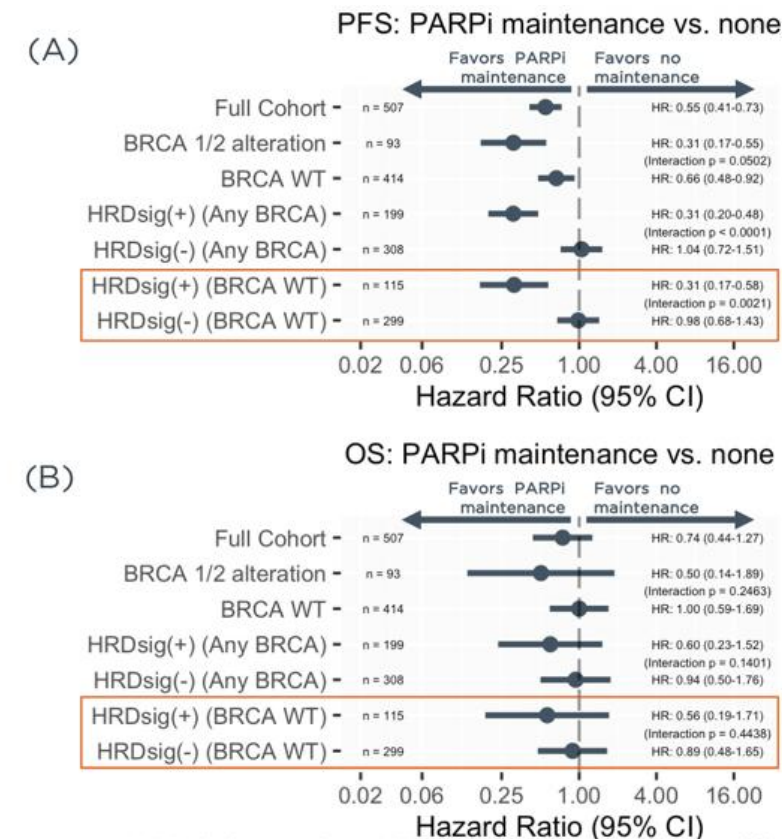
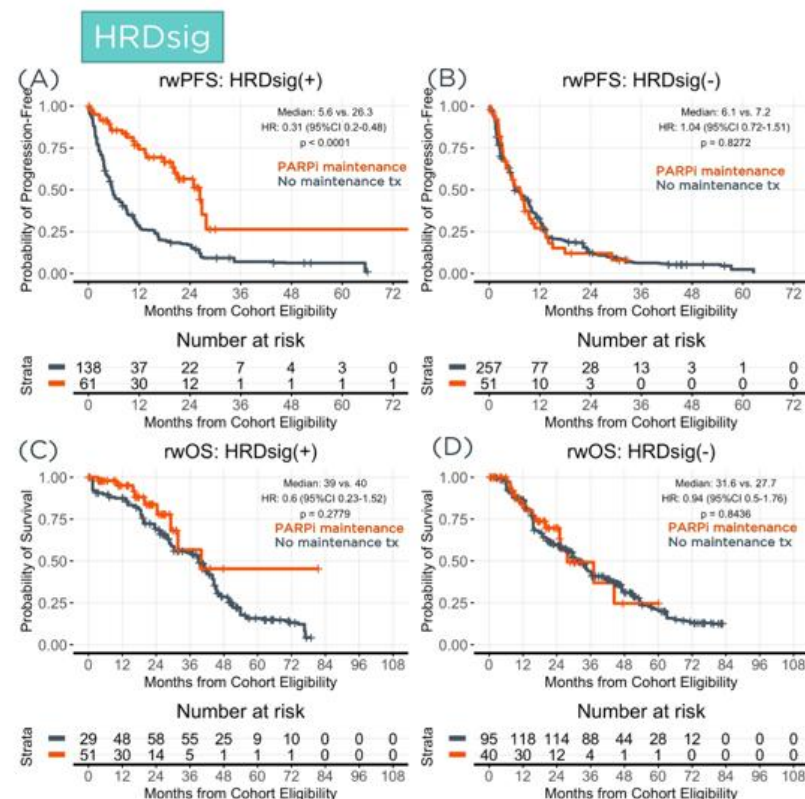
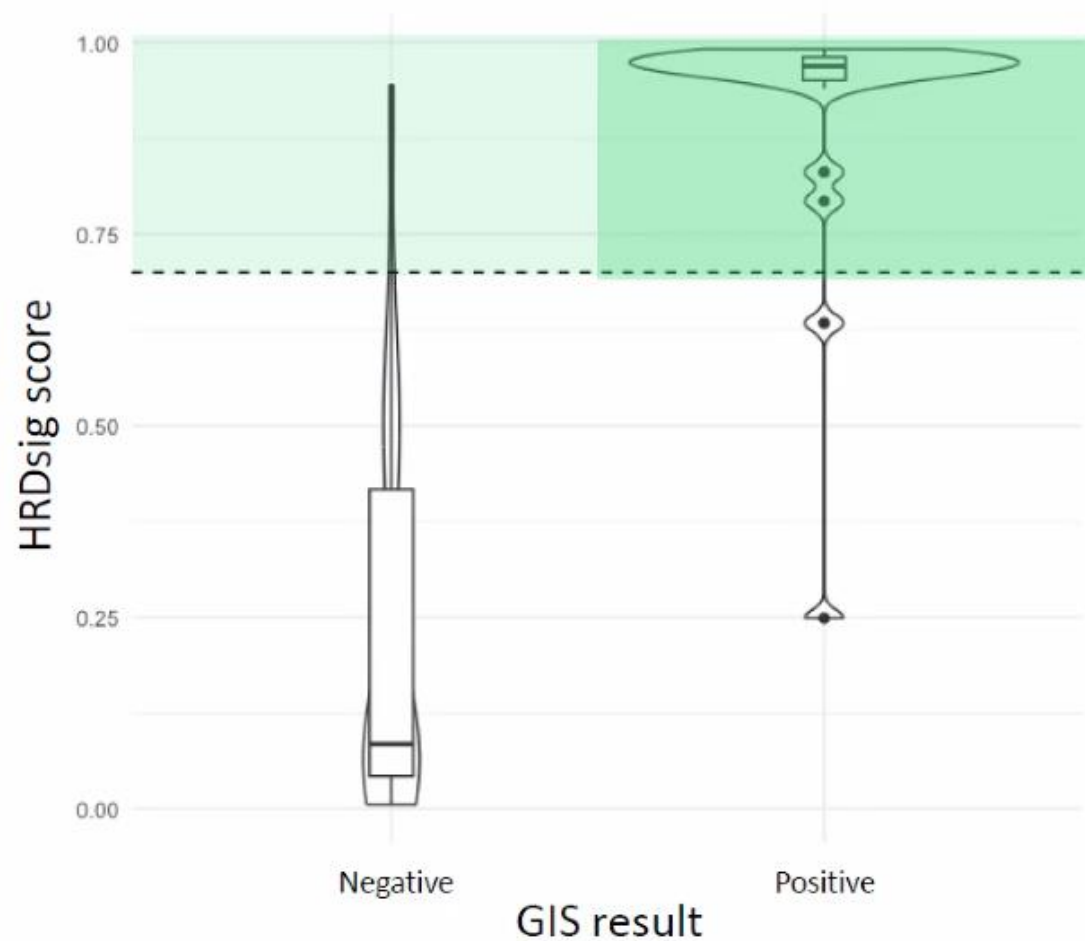


FIGURE 4: Patients receiving maintenance PARPi had more favorable outcomes when HRDsig(+) but not HRDsig(-).

FIGURE 6: HRDsig is superior to BRCA for enrichment of favorable PFS and OS.

(A)



(B)

		GIS	
		Negative	Positive
HRDsig	Negative	63	2
	Positive	4	19

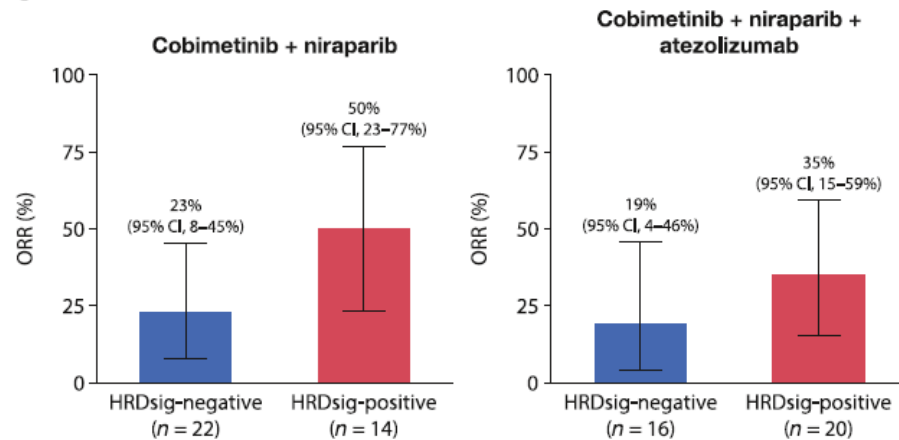
Agreement=0.93

Supplemental Figure S14. Comparison between HRDsig and Myriad MyChoice genomic instability score (GIS) results. (A) HRDsig score distribution by GIS result. The dashed line represents the 0.7 cut-off for HRDsig(+) (B) Agreement for HRDsig and GIS results.

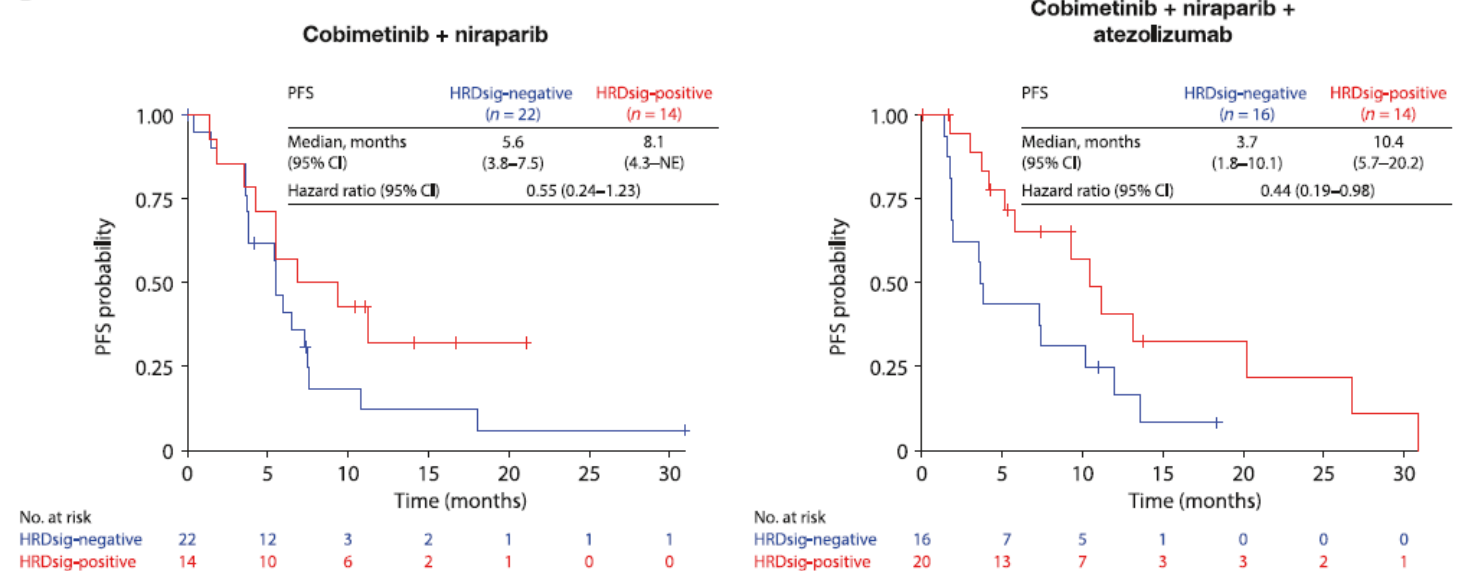
Primary results and characterization of patients with exceptional outcomes in a phase 1b study combining PARP and MEK inhibition, with or without anti-PD-L1, for BRCA wild-type, platinum-sensitive, recurrent ovarian cancer

- This phase 1b study (ClinicalTrials.gov identifier NCT03695380) evaluated regimens combining PARP and MEK inhibition, with or without PD-L1 inhibition, for BRCA wild-type, platinum-sensitive, recurrent ovarian cancer (PSROC).
- Patients with PSROC who had received one or two prior treatment lines were treated with 28-day cycles of cobimetinib 60 mg daily (days 1–21) plus niraparib 200 mg daily (days 1–28) with or without atezolizumab 840 mg (days 1 and 15).

C

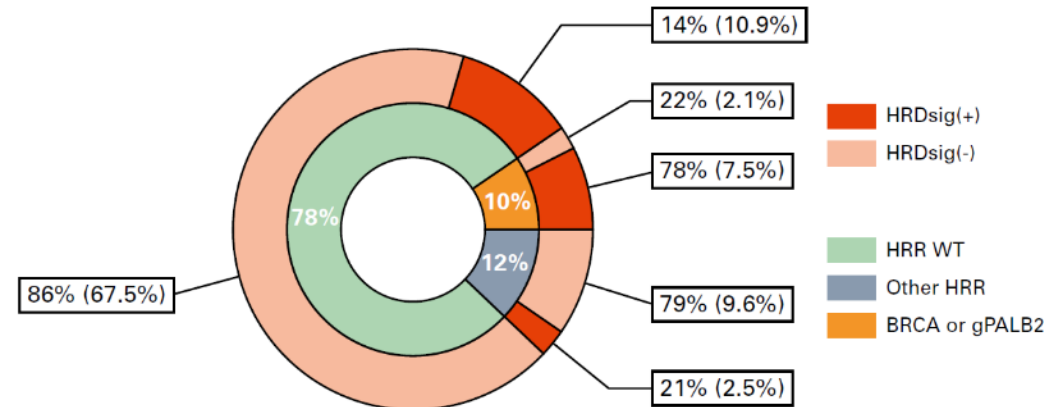
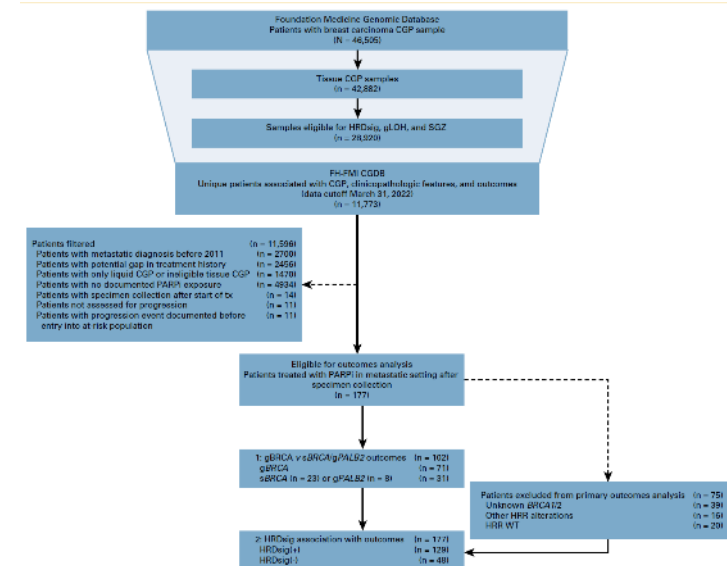
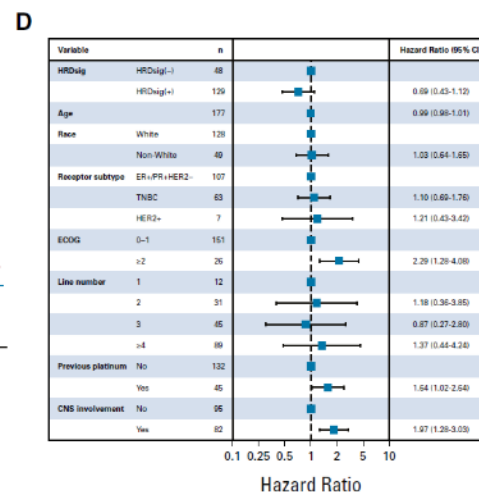
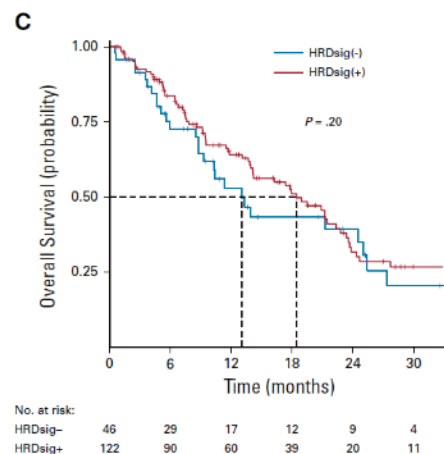
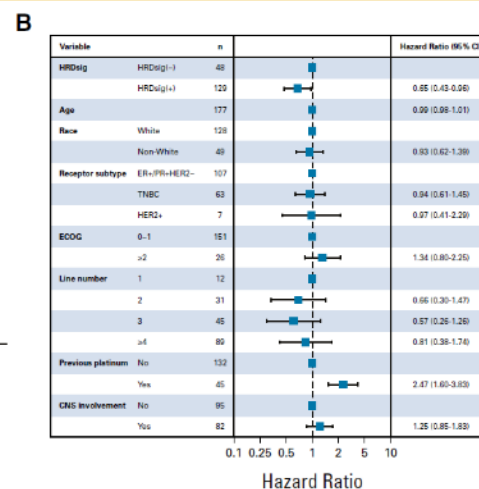
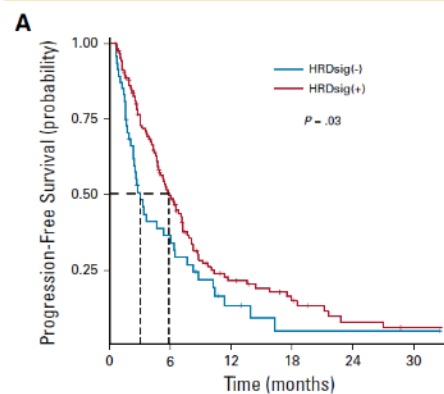


D



Homologous Recombination Deficiency Landscape of Breast Cancers and Real-World Effectiveness of Poly ADP-Ribose Polymerase Inhibitors in Patients With Somatic BRCA1/2, Germline PALB2, or Homologous Recombination Deficiency Signature

- A real-world (RW) clinico-genomic database (CGDB) of comprehensive genomic profiling (CGP) linked to deidentified, electronic health record-derived clinical data was used. CGP was analyzed for HRR genes and HRDsig.
- The CGDB enabled cohort characterization and outcomes analyses of 177 patients exposed to PARPi.



A Novel HRD Signature Is Predictive of FOLFIRINOX Benefit in Metastatic Pancreatic Cancer

- This study used the nationwide (US-based, ~280 US cancer clinics) de-identified Flatiron Health-Foundation Medicine PC clinico-genomic database profiled from August 2014 to September 2021.
- Comprehensive genomic profiling was performed on 8358 Pancreatic cancer (PC) patients. Outcomes were available for 1149 metastatic PC patients treated with 1L FOLFIRINOX or gemcitabine and paclitaxel.

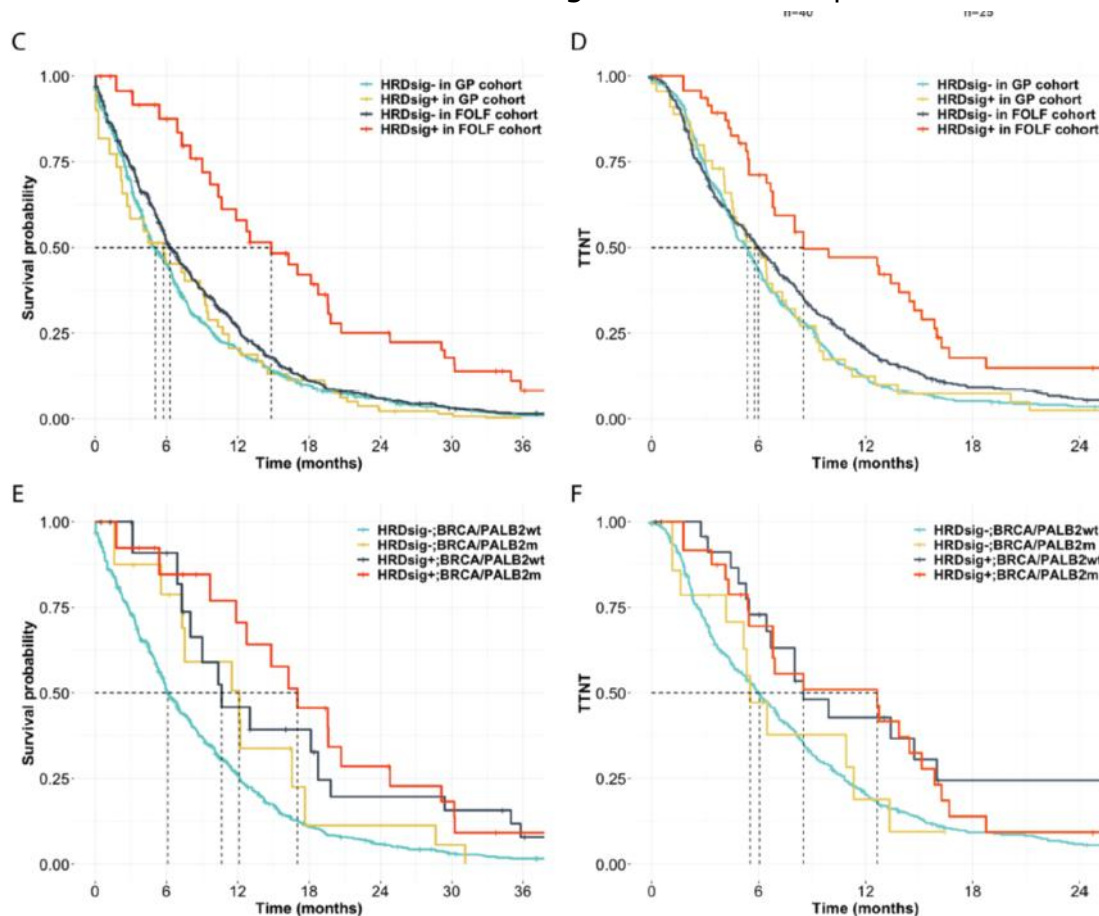


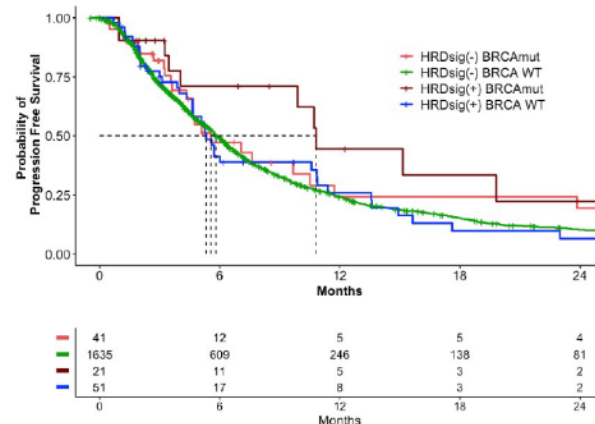
Figure 3. HRDsig is associated with FOLFIRINOX benefit in a clinical cohort. (A) Overlap of HRDsig with mutations in *BRCA1/2* or *PALB2* in the CGDB cohort. (B) Allelic status for *BRCA1/2/PALB2* mutant samples based on overlap with HRDsig. Kaplan-Meier plots for rOS (C) and TTNT (D) in HRDsig(+) and HRDsig(-) patients treated with FOLFIRINOX or GP. rOS (E) and TTNT (F) for FOLFIRINOX or GP-treated patients based on HRDsig and *BRCA1/2/PALB2* mutation status.

Homologous recombination deficiency (HRD) and genomic associations in non-small cell lung cancer (NSCLC) using a novel HRD signature (HRDsig)

- This study used the nationwide (US-based) de-identified Flatiron Health-Foundation Medicine NSCLC clinico-genomic database (FH-FMI CGDB)
- Targeted next generation sequencing was performed on 48,344 NSCLC specimens.
- Patients included in outcomes analysis received FMI tissue CGP with specimen collection prior to first line therapy and were treated with cisplatin or carboplatin combined alone or combined with any of the following therapies: paclitaxel, pemetrexed, docetaxel, gemcitabine and bevacizumab.

HRDsig(+) BRCAmut NSCLC HAVE PROLONGED BENEFIT ON PLATINUM CHEMO

	HRDsig(+) (n = 72)	HRDsig(-) (n = 1,679)
Histology:		
Non-Squamous, n (%)	35 (49)	1140 (68)
Squamous, n (%)	32 (44)	462 (28)
NOS, n (%)	5 (7)	77 (5)
Female, n (%)	27 (38)	798 (48)
Race:		
White	49 (68)	1178 (70)
Non-White	17 (24)	365 (22)
Unknown	6 (8)	136 (8)
Age, median [IQR]	68 [61-74]	68 [60-75]
Former/Current Smoker, n (%)	66 (92)	1157 (92)
PD-L1 Status		
Negative, n (%)	11 (15)	243 (15)
Low Positive, n (%)	6 (6)	208 (12)
High Positive, n (%)	6 (8)	166 (10)
Unknown	51 (71)	1062 (64)
ECOG		
0-1	53 (74)	1212 (72)
2+	12 (17)	302 (18)
Unknown	7 (10)	165 (10)
CNS Mets, n (%)	10 (14)	308 (18)
Liver Mets, n (%)	5 (7)	167 (10)
KRAS Status		
G12C, n (%)	5 (7)	219 (13)
Other, n (%)	5 (7)	330 (20)
WT, n (%)	62 (86)	1130 (67)
BRCA altered, n (%)	21 (29)	41 (2)



	BRCA WT		BRCA Altered	
	HRDsig(-) [ref]	HRDsig(+)	HRDsig(-) [ref]	HRDsig(+)
Number of Events, n (%)	1190/1638 (73%)	38/51 (75%)	24/41 (59%)	11/21 (52%)
Median rwPFS months [95% CI]	5.8 [5.6-6.3]	5.3 [4.6-10.9]	5.6 [4.7-11.7]	10.8 [9.9-NR]
Hazard ratio [95% CI]	1.04 [0.75-1.44]		0.69 [0.34-1.41]	

HRDsig(+) NSCLC ARE MORE FREQUENTLY DRIVER NEGATIVE

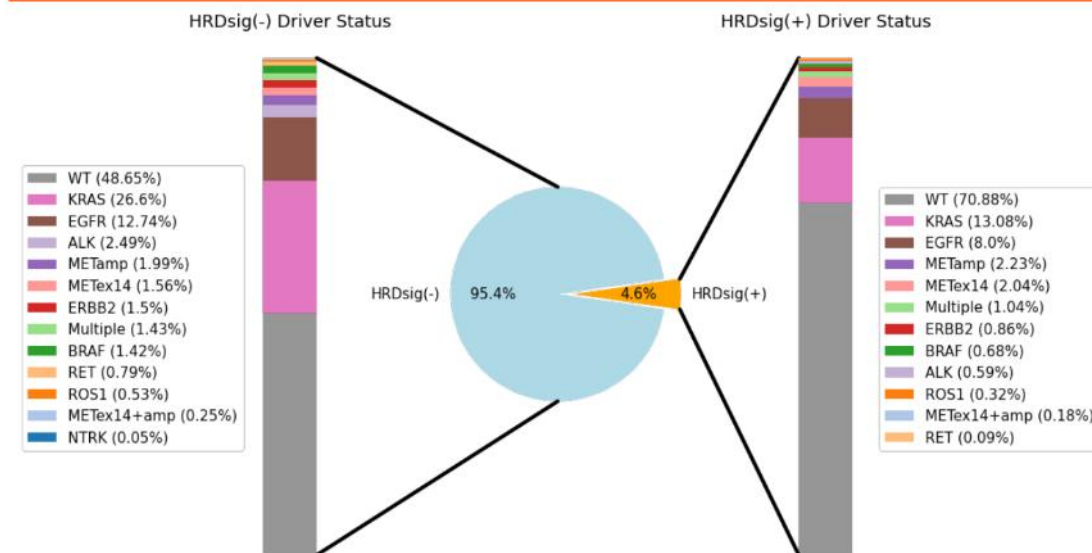


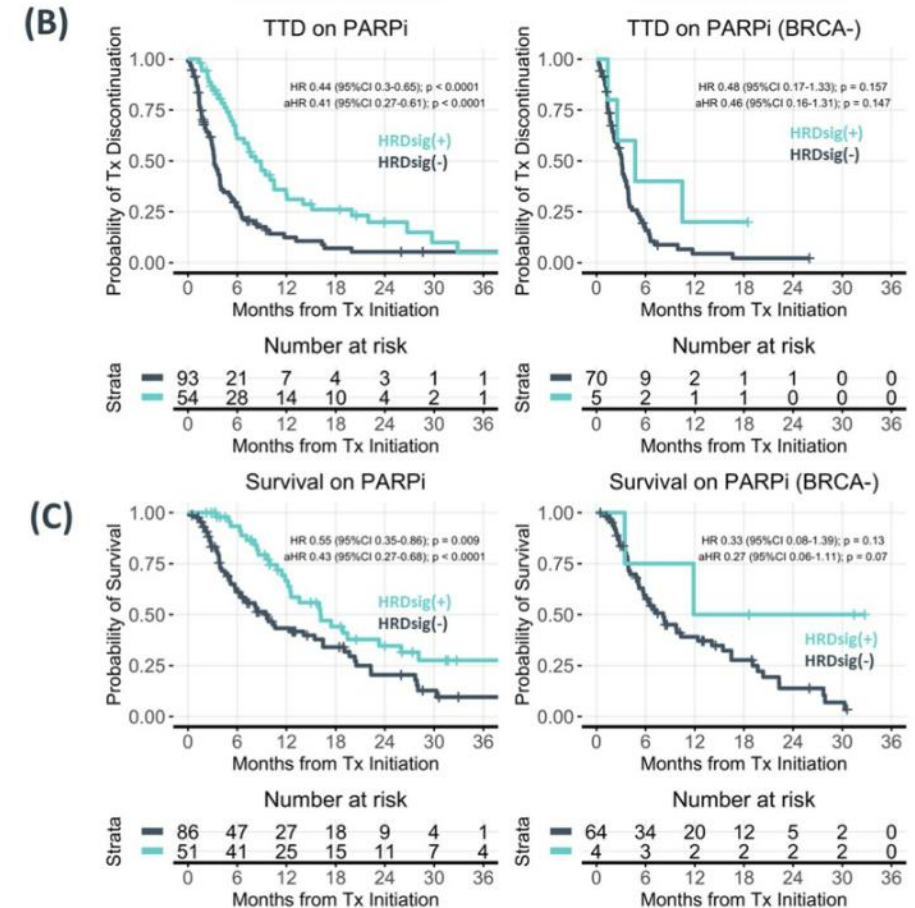
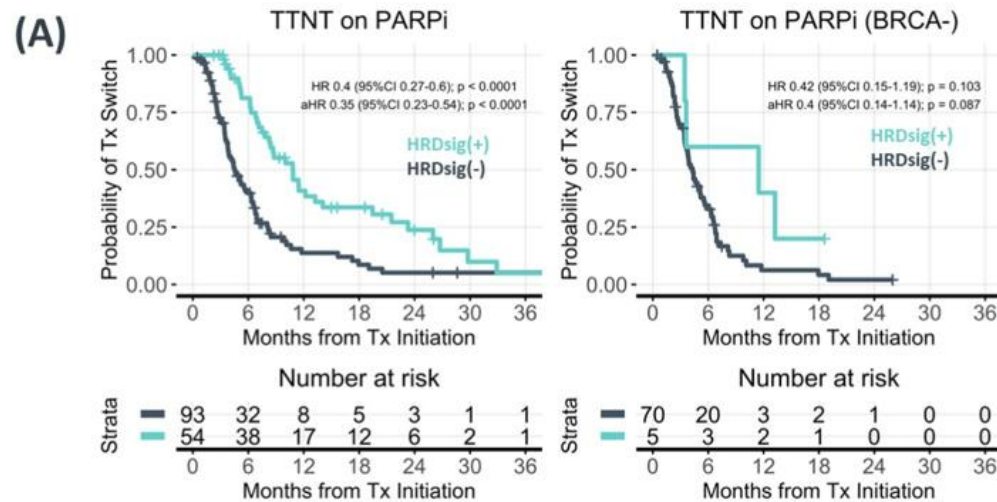
FIGURE 2: Driver alterations were less frequent in HRDsig(+) NSCLC except for MET exon 14 splicing alterations and amplification, both of which were more common in HRDsig(+) NSCLC

FIGURE 6: Patient characteristics from the clinicogenomic subset were like what was observed in the larger genomic database, including increased prevalence of squamous histology and fewer KRAS mutants amongst HRDsig(+) NSCLC. In patients with BRCA altered tumors HRDsig(+) was associated with a nominally longer median rwPFS

Real-world effectiveness of PARP inhibitors (PARPi) in metastatic castration-resistant prostate cancer (mCRPC) by genomic homologous recombination repair (HRR) alterations and homologous recombination deficiency signature (HRDsig)

- This study used the nationwide (US-based) de-identified Flatiron Health-Foundation Medicine Prostate Cancer clinico-genomic database (FH-FMI CGDB), originating from approximately 280 US cancer clinics (~800 sites of care).
- Patients with mCRPC and tumor genomic profiling (tissue or liquid) who underwent single agent PARPi treatment were included.
- 313 patients with mCRPC met inclusion criteria, of which 162 had tissue specimens for genomics evaluations and 151 had liquid profiling.

Figure 4: Novel *HRDsig* is associated with improved PARPi outcomes and identifies a subset of BRCA- patients who may benefit from PARPi. (A) TTNT, (B) TTD, and (C) rwOS stratified by either the entire patient cohort or BRCA- patients only.



CQ.HRDsigについて

注意：HRDsigは、professional serviceとして提供されます。そのため、このバイオマーカーだけではNCCH1901（受け皿試験）の対象とならないことにご注意ください。

HRDsig POSITIVEの場合について

- ・ 卵巣がんの場合、PARP阻害薬を推奨/提案しますか

(1) はい (2) いいえ (3) わからない/棄権

- ・ 卵巣がんの場合で、PARP阻害薬治療歴がある場合PARP阻害薬を推奨/提案しますか

(1) はい (2) いいえ (3) わからない/棄権

- ・ 卵巣がんの場合、Niraparibの受け皿試験を推奨/提案しますか

(1) はい (2) いいえ (3) わからない/棄権

- ・ 前立腺がんの場合、PARP阻害薬を推奨/提案しますか

(1) はい (2) いいえ (3) わからない/棄権

- ・ 前立腺がんの場合、Niraparibの受け皿試験を推奨/提案しますか

(1) はい (2) いいえ (3) わからない/棄権