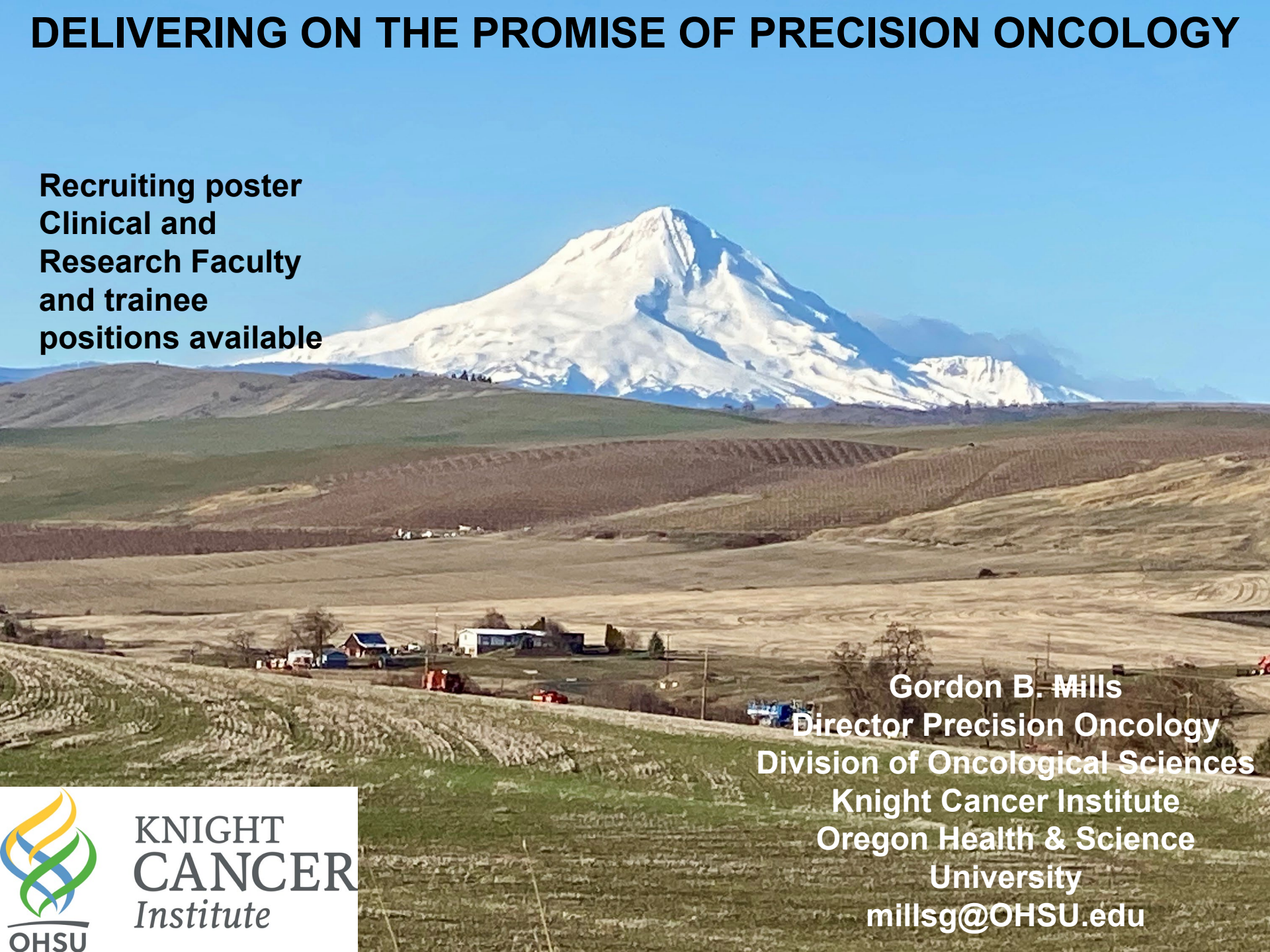
DELIVERING ON THE PROMISE OF PRECISION ONCOLOGY



Recruiting poster
Clinical and
Research Faculty
and trainee
positions available

Gordon B. Mills
Director Precision Oncology
Division of Oncological Sciences
Knight Cancer Institute
Oregon Health & Science
University
millsg@OHSU.edu

OHSU KNIGHT CANCER *Institute*

GOALS

- A failure of confidence from the public and government
- What is the state of progress in improving outcomes in cancer
- What is Precision Oncology/Personalized Medicine
- Challenges to Precision Oncology
- Precision Oncology and SMMART Trials at OHSU

POTENTIAL CONFLICT OF INTEREST DISCLOSURES

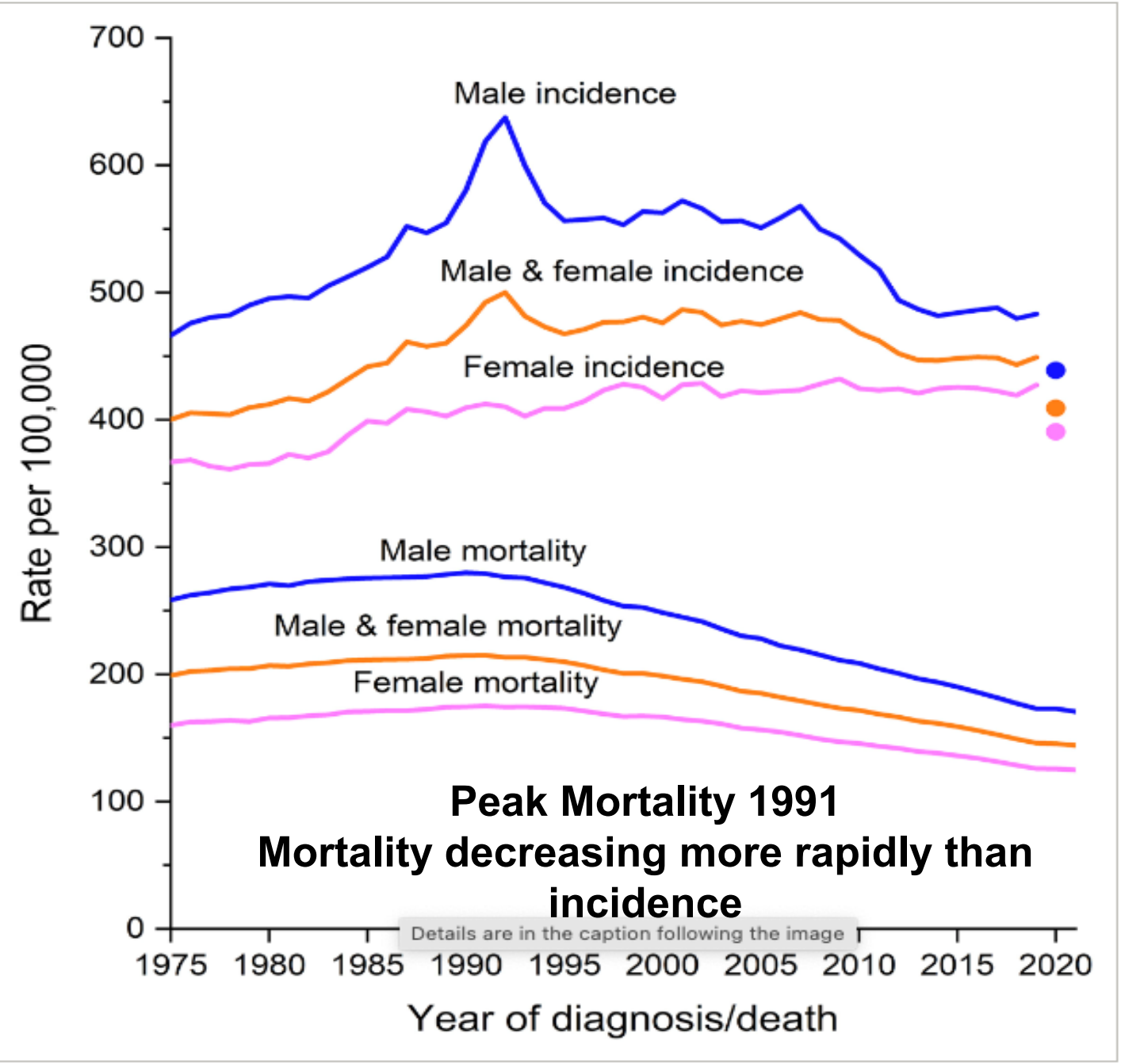
- **Financial Relationships Current**
  - **SAB/Consultant:**
    - **Amphista, Astex, AstraZeneca, BlueDot, Chrysallis Biotechnology, Ellipses Pharma, ImmunoMET, Infinity, Ionis, Lilly, Medacorp, Nanostring, Nuvectis, PDX Pharmaceuticals, Roche, Signalchem Lifesciences, Tarveda, Turbine, Zentalis Pharmaceuticals**
  - **Stock/Options/Financial:**
    - **Bluedot, Catena Pharmaceuticals, ImmunoMet, Nuvectis, SignalChem, Tarveda, Turbine**
  - **Licensed Technology**
    - **HRD assay to Myriad Genetics, DSP patents with Nanostring**
  - **Sponsored research**
    - **AstraZeneca, Nanostring Center of Excellence, Ionis (Provision of tool compounds)**
  - **Clinical trials support (funding or in kind)**
    - **AstraZeneca, Genentech, GSK, Lilly**
  - **I will discuss off label use and/or investigational use of drugs**

WINNING THE WAR AGAINST CANCER

1953	5 year survival	30%
1971	National cancer act	
	5 year survival	50%
	Cancer survivors	3 million
1990s	Mortality rate declines (per 100,000 individuals)	
2001	Cancer survivors	9.8 million
2003	Absolute death rate declines	
2010	Breast cancer death rate declines	2% per year
2013	Cancer survivors	14.5 million
2015	5 year survival (ACS)	66%
2016	21st Century cures act	
2019	Cancer death rate has declined by 32% from peak in 1991	
	2% per year from 2014-2019	
2019	Breast cancer peaked 1989 42% decrease from peak	
2022	Moonshot 2.0	

40% of men and 38%women will develop cancer in their lifetime

WE ARE MAKING REAL PROGRESS

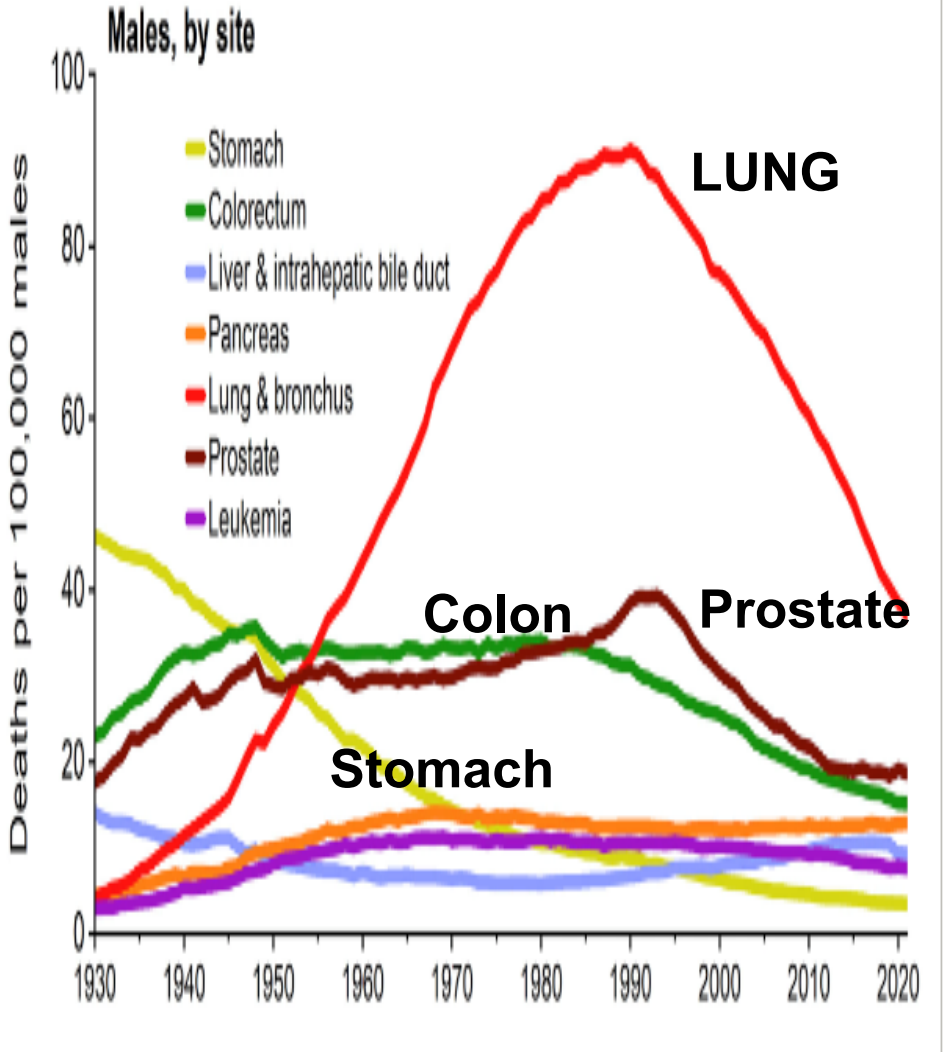


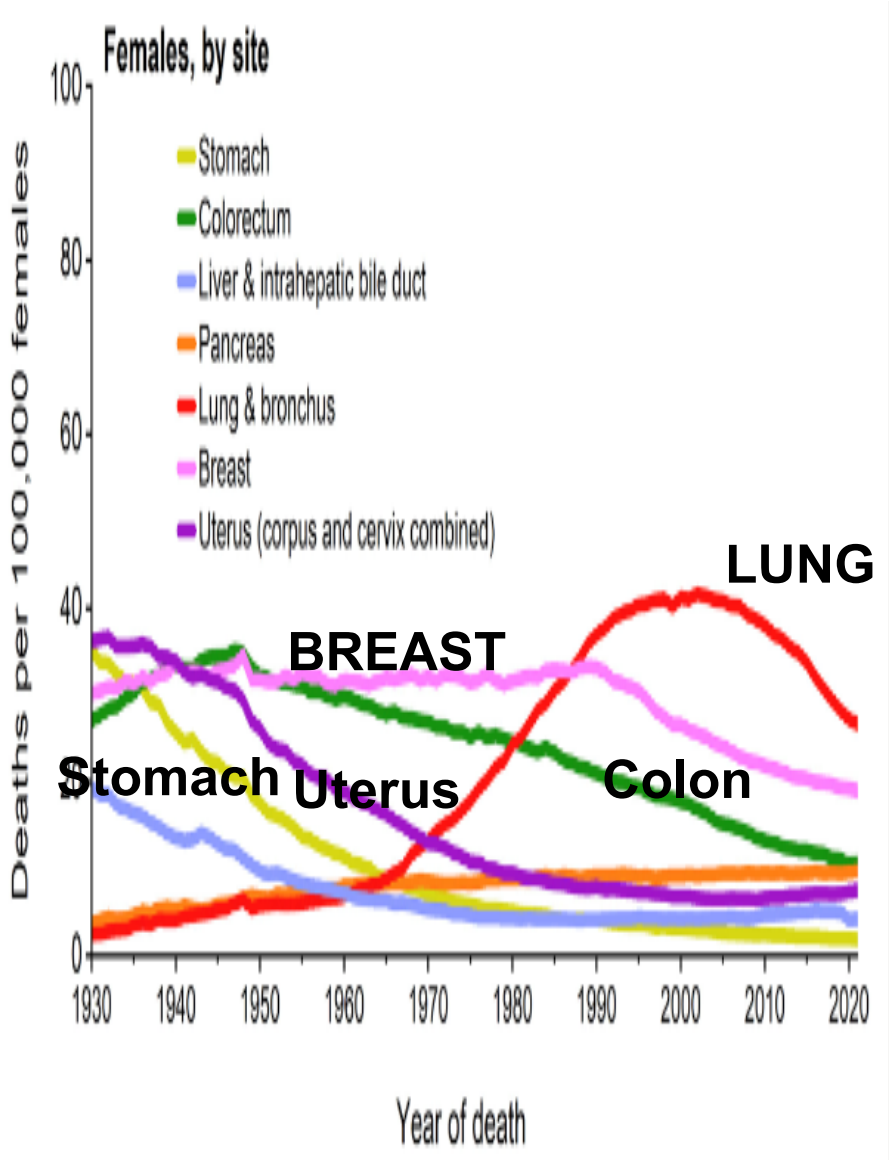
Why

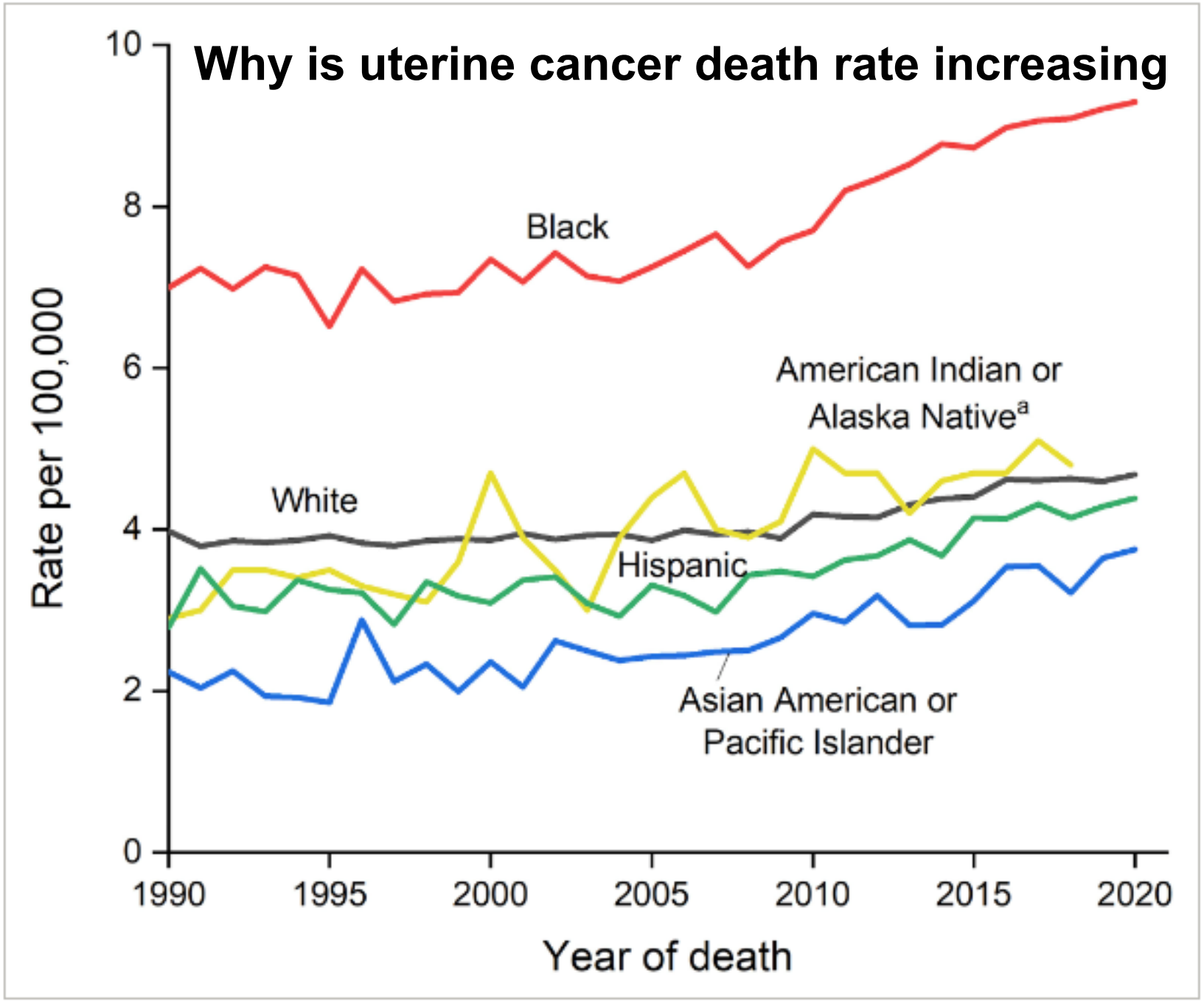
1. **Lifestyle**
   - **Smoking**
   - **Obesity**
2. **Early Detection**
   - **Cervix**
   - **Breast**
3. **Improved Therapy**

WE ARE MAKING REAL PROGRESS

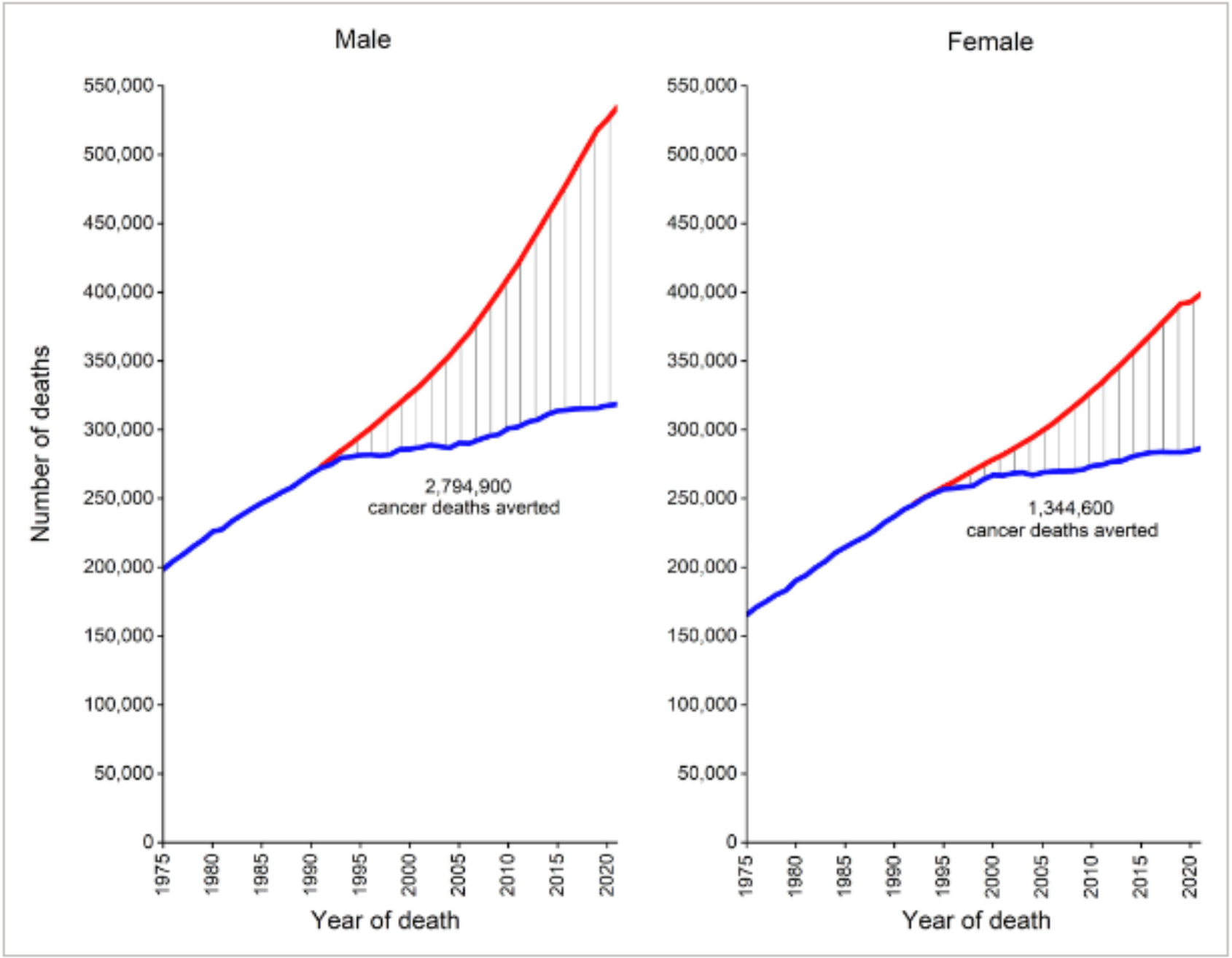
Lung, breast, colon Why stomach and prostate?






Why is uterine cancer death rate increasing
Rate per 100,000
10
8
6
4
2
0
1990
1995
2000
2005
2010
2015
2020
Year of death
Black
American Indian or
Alaska Native[a]
White
Hispanic
Asian American or
Pacific Islander

Cancer deaths averted



WHY AREN'T WE MAKING MORE PROGRESS

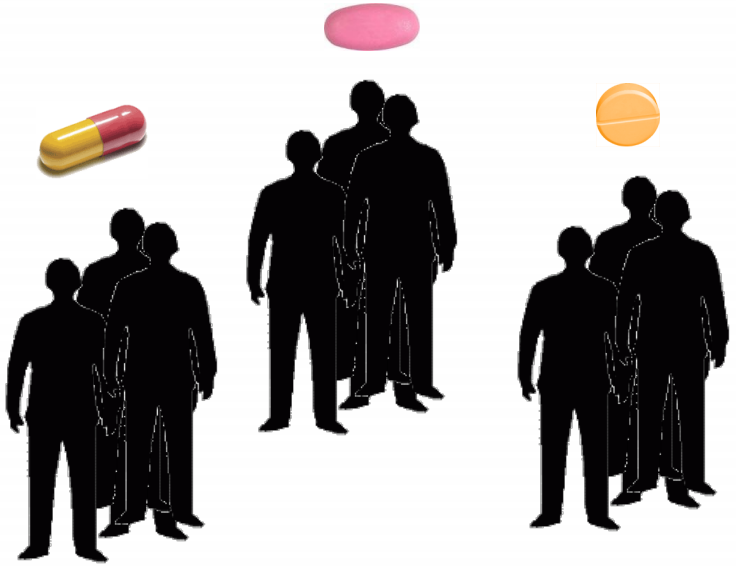
- There are many types of cancer
  - Every person's cancer is unique
- Cancers are frequently detected late
  - Early detection is hard
    - While cancer over a lifetime is common, incidence is low
    - Most effective approaches are 2 stage
    - Multi-disease methylation assays promising
- Cancers have characteristics that are derived from normal cells
  - Therapeutic index determines utility
- Tumors become resistant to therapy
- Silos
- FUNDING

What is Personalized Medicine?

Current Practice

Trial and error

Personalized Medicine

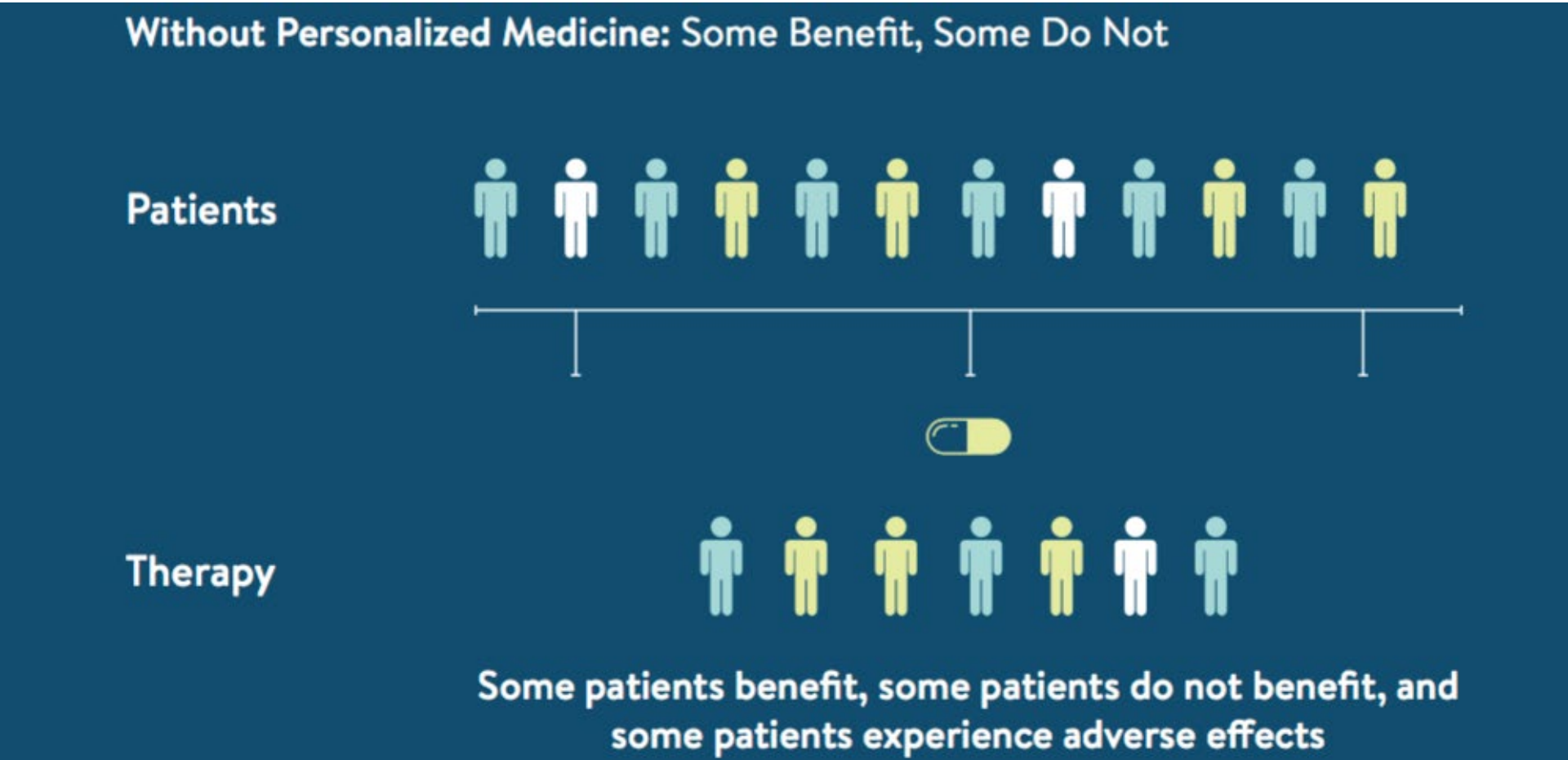


The right treatment for the right person at the right time

LET THE PATIENT TEACH US WHAT IS IMPORTANT

PMC Personalized Medicine Coalition

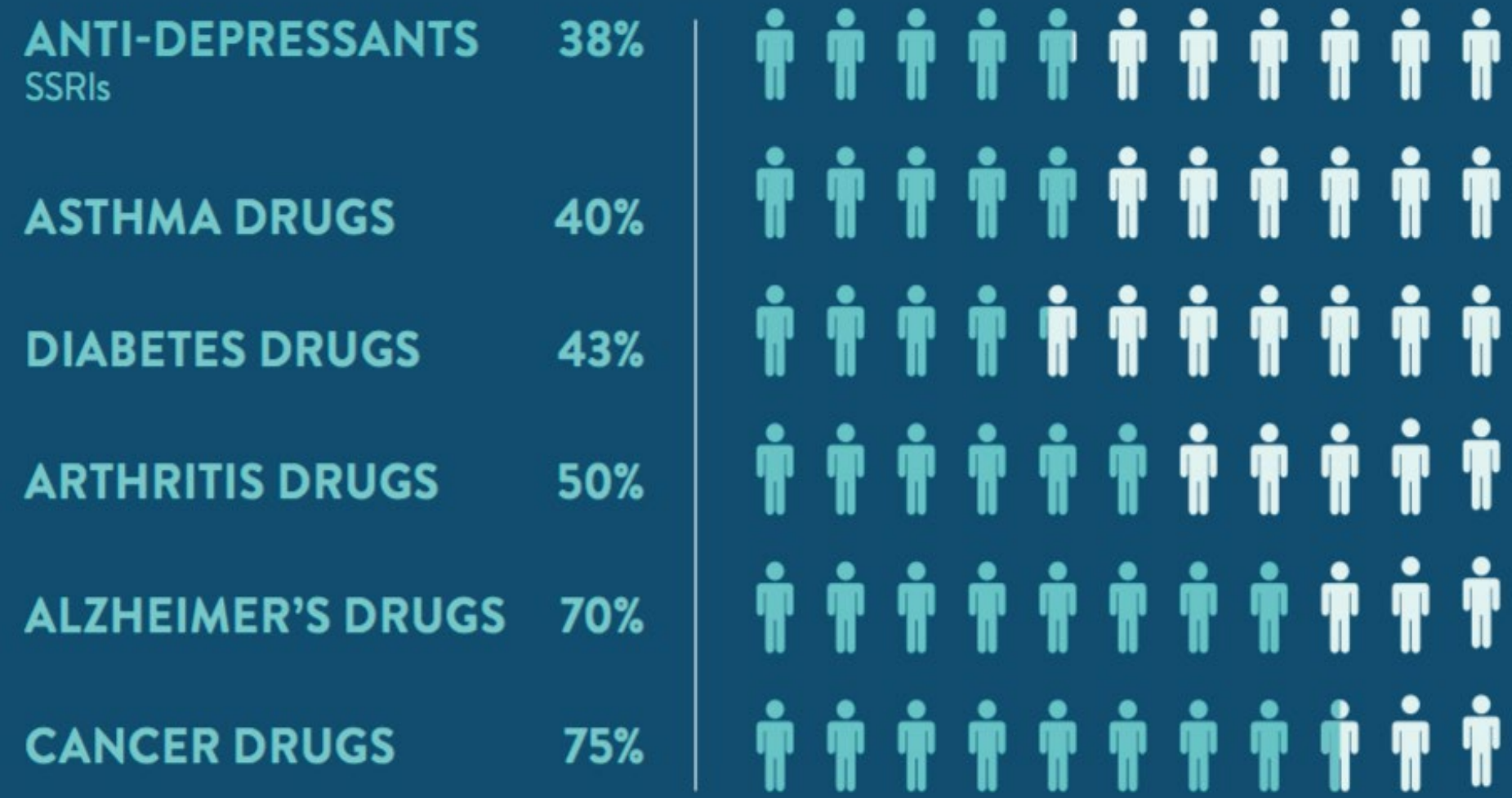
Current approach: treat patients based on pathology



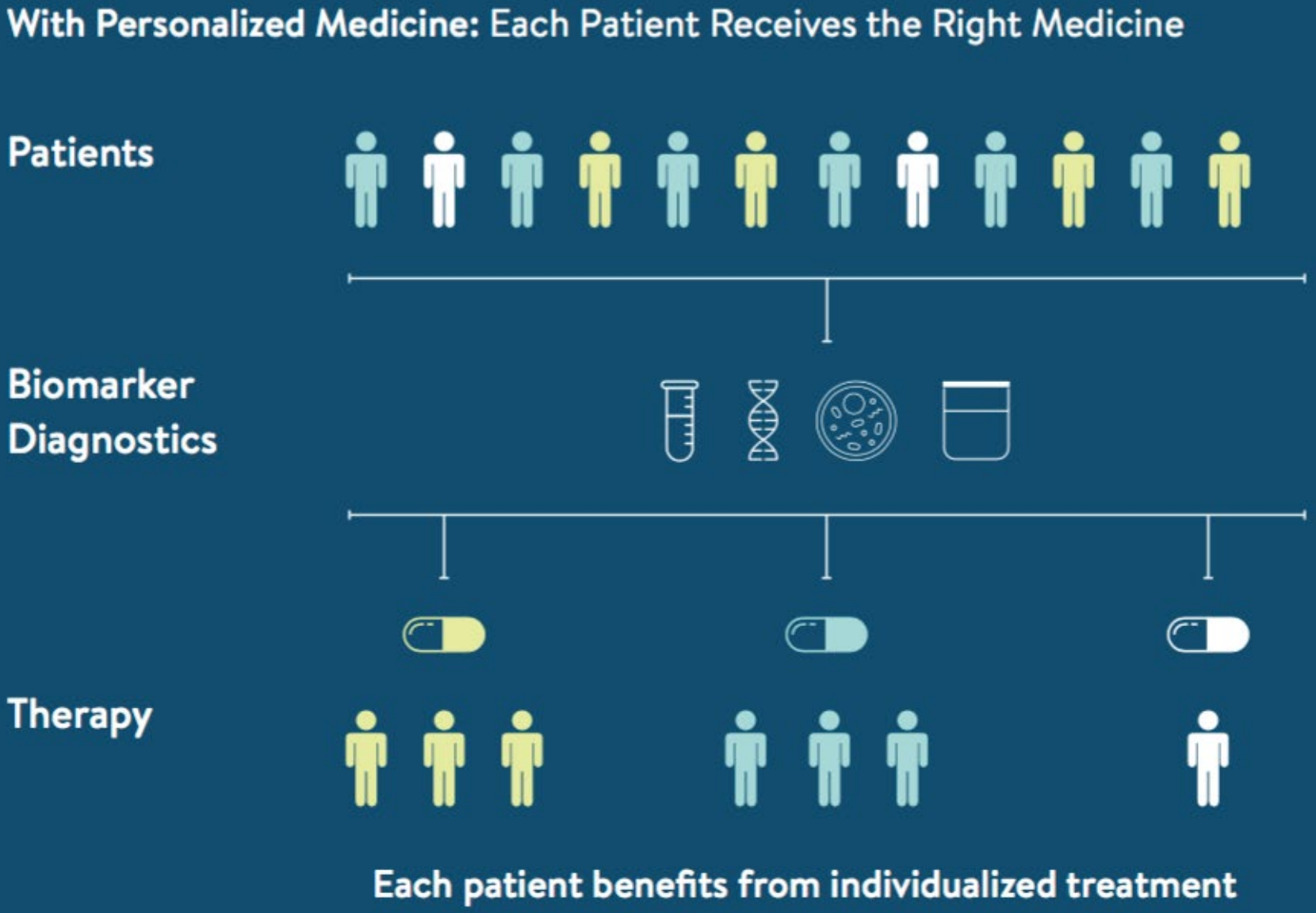
Remarkable progress: but not where we want to be

Current approach: Limited benefit for most

Percentage of the patient population for which a particular drug in a class is ineffective, on average.

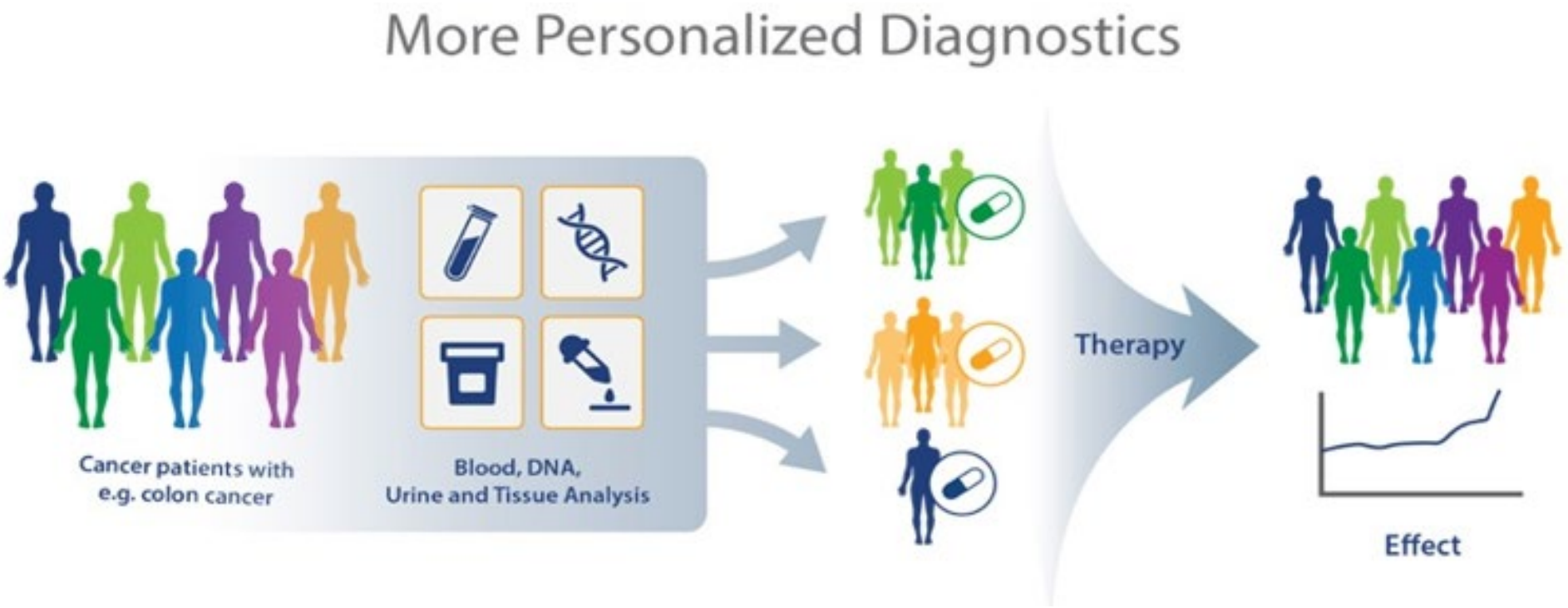


Basic Precept: manage each patient based on biomarkers



Still not where we want to be: limited information and montherapy

Personalized medicine



Barbeau, J. "PDX and Personalized Medicine" https://blog.crownbio.com/pdx-personalized-medicine

Multiple Editorials Question Benefits of Personalized Medicine

The NEW ENGLAND JOURNAL of MEDICINE

SOUNDING BOARD

Limits to Personalized Cancer Medicine

Ian F. Tannock, M.D., Ph.D., and John A. Hickman, D.Sc.

Harvard Business Review

POLICY

Will Personalized Medicine Mean Higher Costs for Consumers?

by Michael Geruso, Anupam B. Jena, and Timothy J. Layton

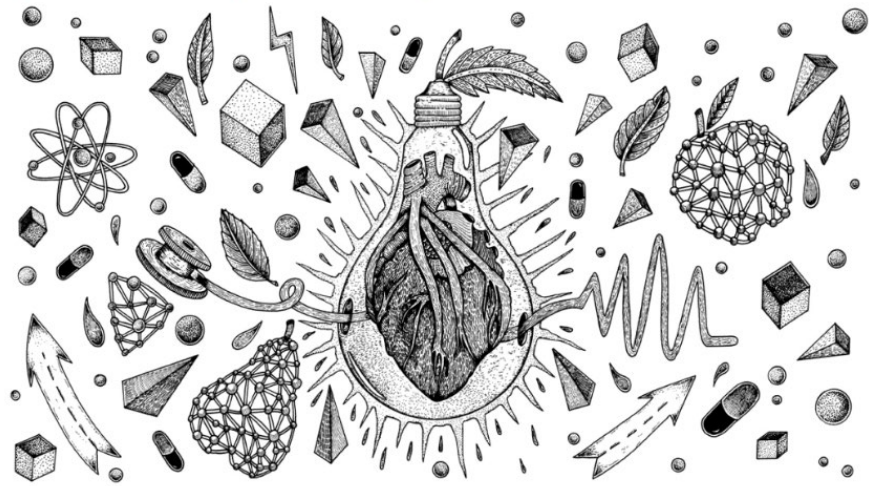
MARCH 01, 2018

THE NEW YORKER

ELEMENTS

THE PROBLEM WITH PRECISION MEDICINE

By Cynthia Graber February 5, 2015



LAURA SCHNEIDER FOR HBR

The excitement surrounding personalized, genetics-based medicine has so far outpaced the science.

Photograph by Dilip Vishwanat/The New York Times/Redux

Will Precision Oncology Benefit All Patients

Precision Oncology

We are at the end of the beginning: monotherapy Testing

Many questions remain

Stratified Medicine

Homogenous patient groups

Ductal Breast Cancer

8 subclasses

A set of orphan diseases

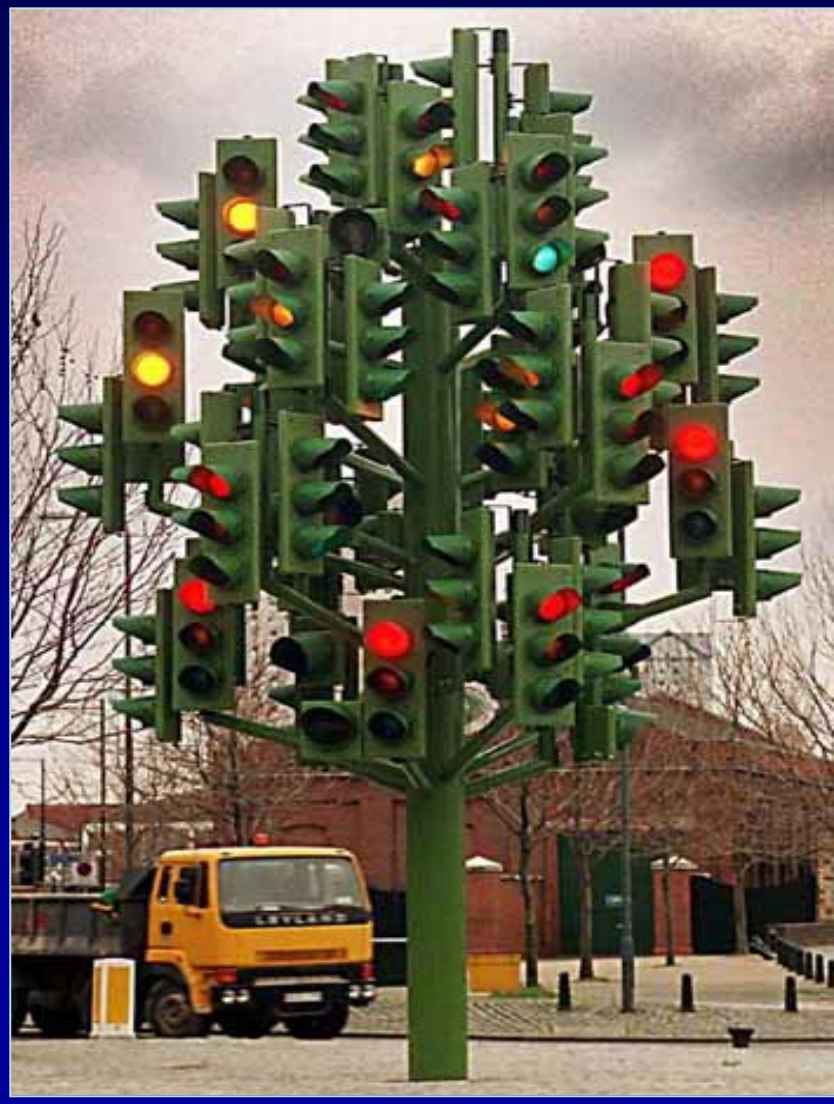
What proportion of patients will benefit

Will it benefit patients when all other therapies have failed

Will there be a cost benefit for patients

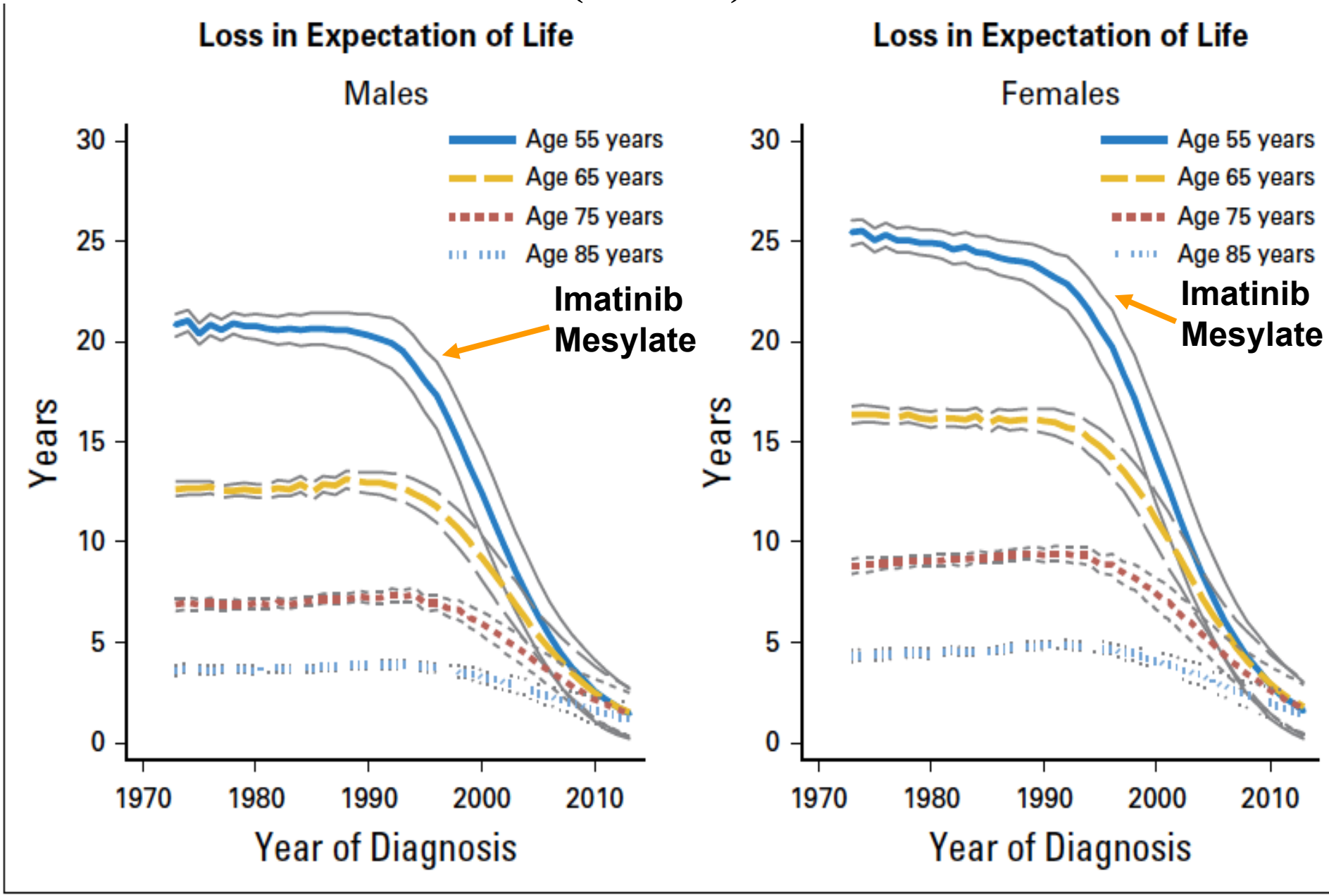
How many patients need to benefit for precision oncology to be a success

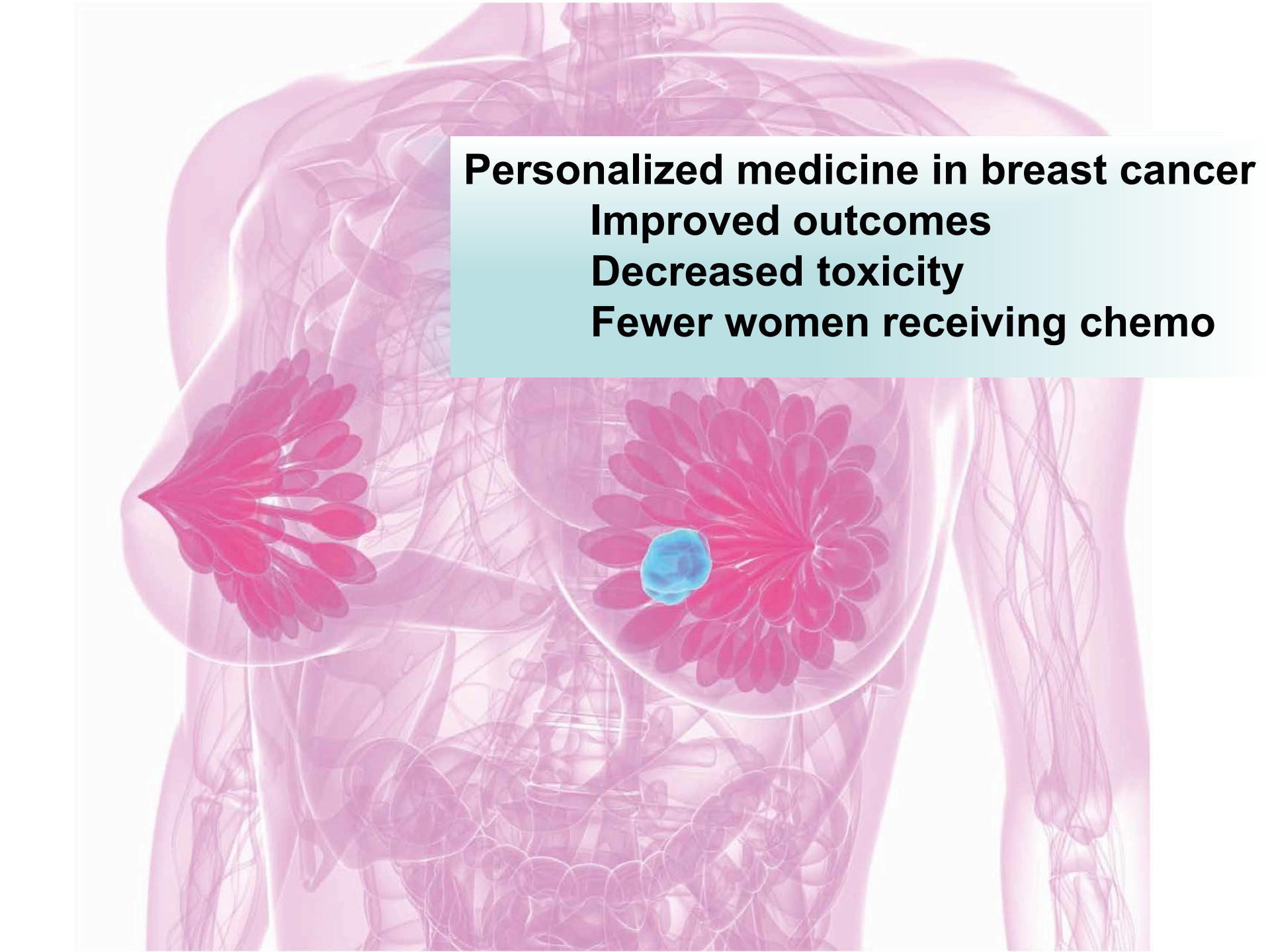
CHALLENGES TO PERSONALIZED TARGETED THERAPY



Patient life span is not shortened by having CML

(Sweden)

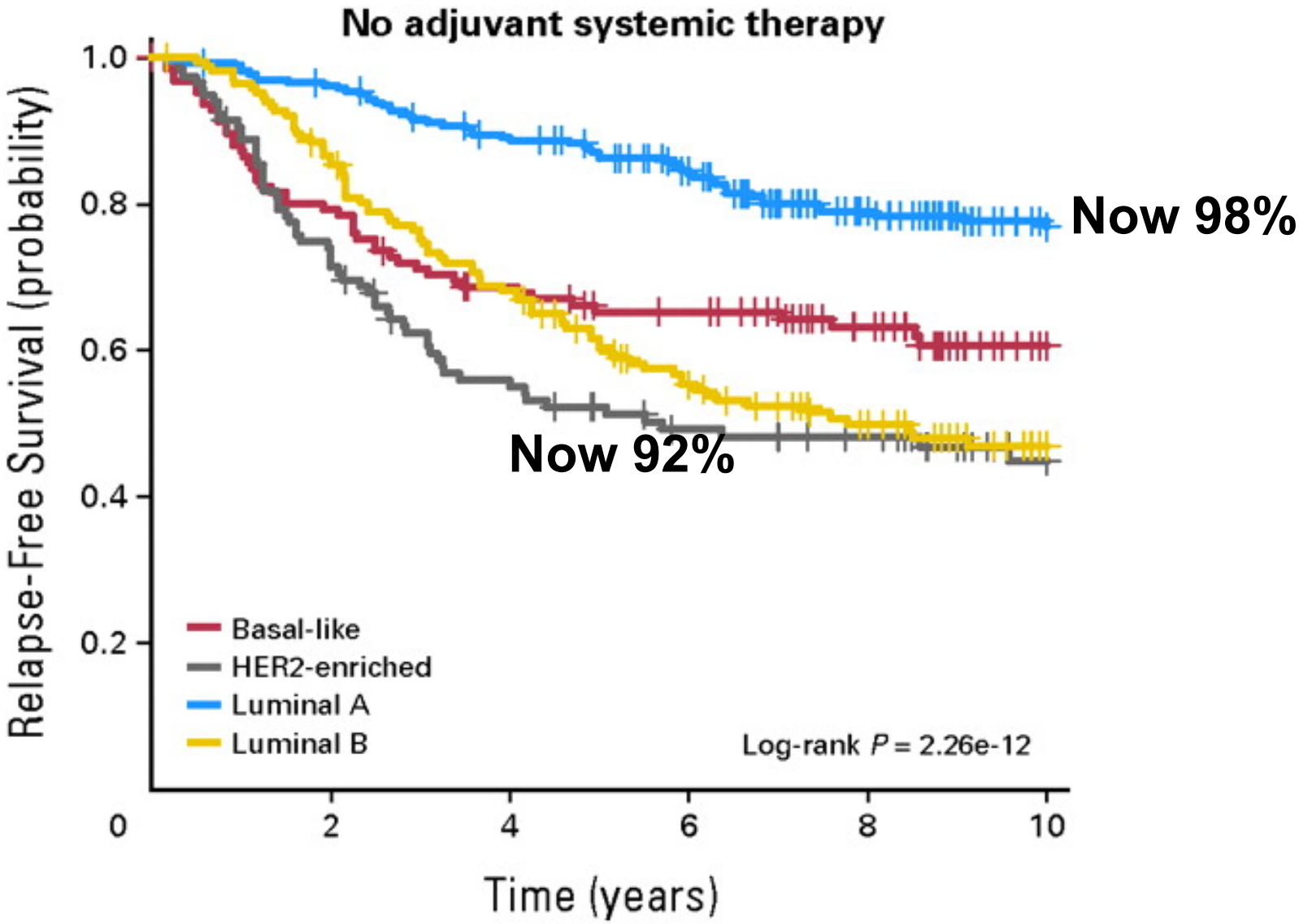


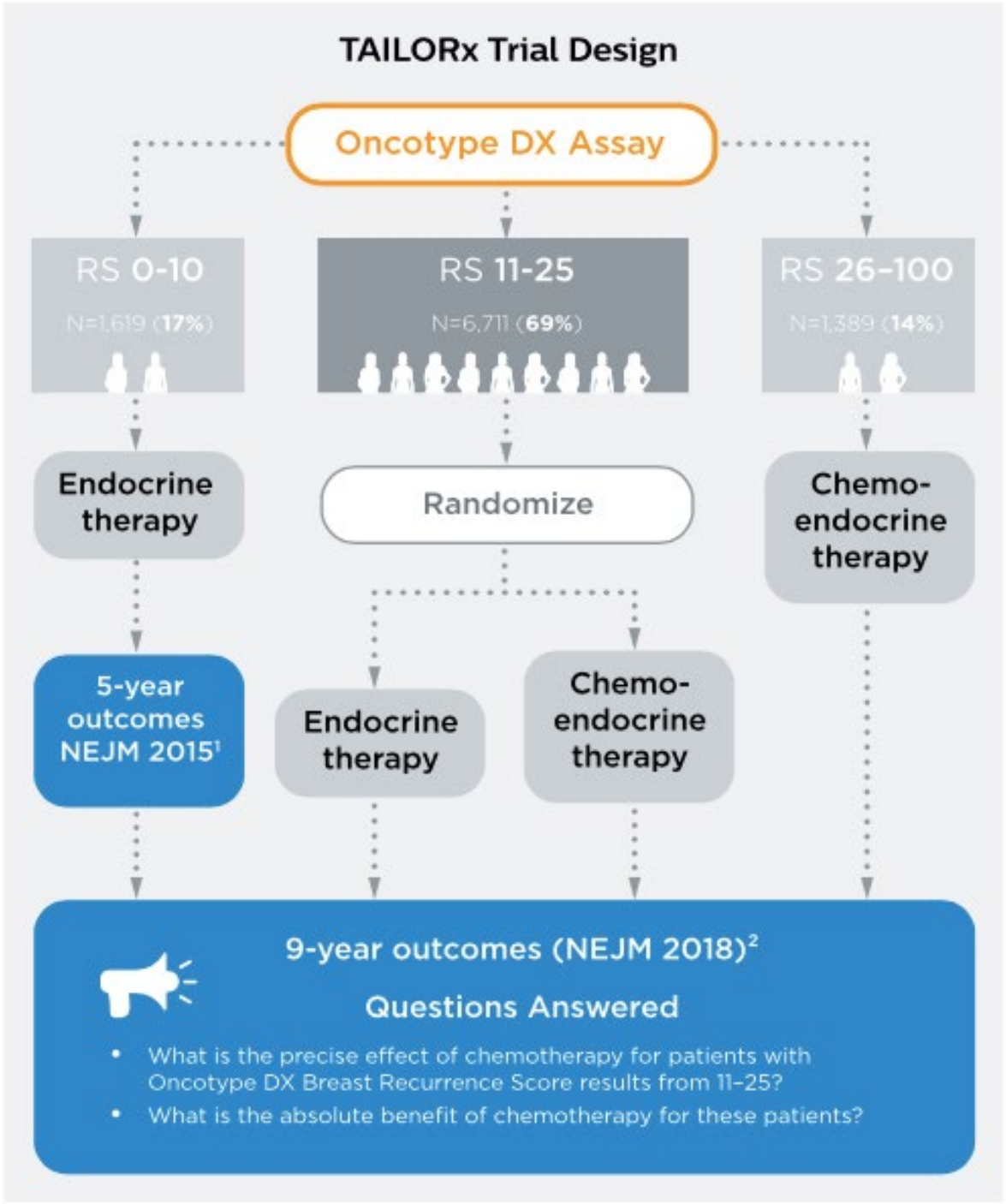


Personalized medicine in breast cancer

- **Improved outcomes**
- **Decreased toxicity**
- **Fewer women receiving chemo**

Change in outcomes for breast cancer
Targeted therapy has changed the natural history



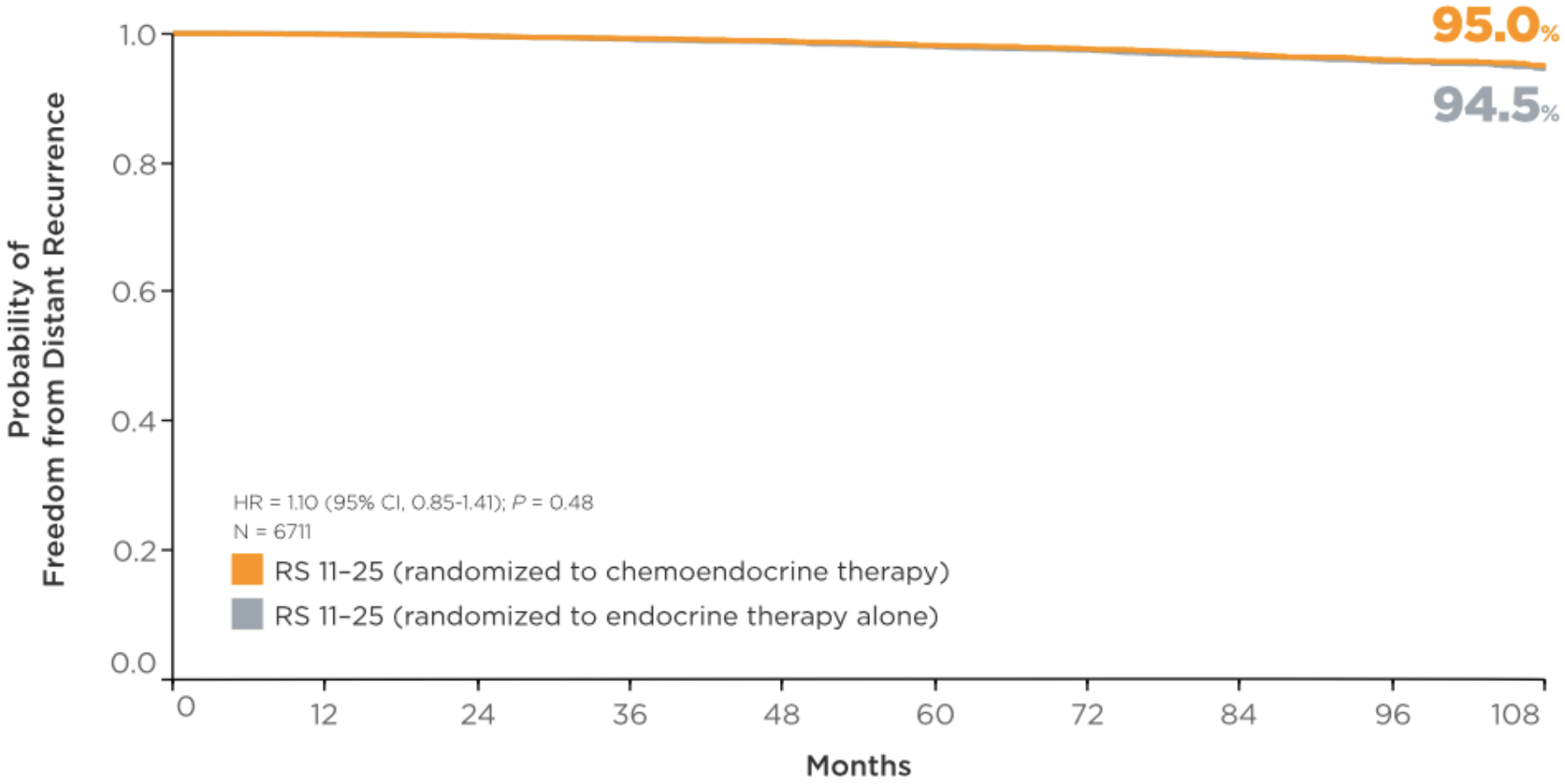


TAILORx

Personalized medicine to decrease toxicity

Overtreatment in breast cancer

Outcomes indistinguishable for low and intermediate risk Distant recurrence



"[The top challenges facing personalized medicine are] reimbursement, reimbursement, and reimbursement."

— **Alexis Borisy**
Partner, Third Rock Ventures

Oncotype Dx Saves 2,256 per patient tested and costs 1,944 per year of life saved

KRAS testing for EGFR inhibitors would save 604 million per year

Intermountain Health:

- **Charges per PFS week lower at 4,665 vs 5000**
- **Increased PFS 21.4 wks vs 11wks**
- **Total costs (Mainly drug costs) higher $91,790 vs 40,782**

Medical Decision-Support

Personalized Cancer Therapy Website https://pct.mdanderson.org

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

Personalized Cancer Therapy

Knowledge Base for Precision Oncology

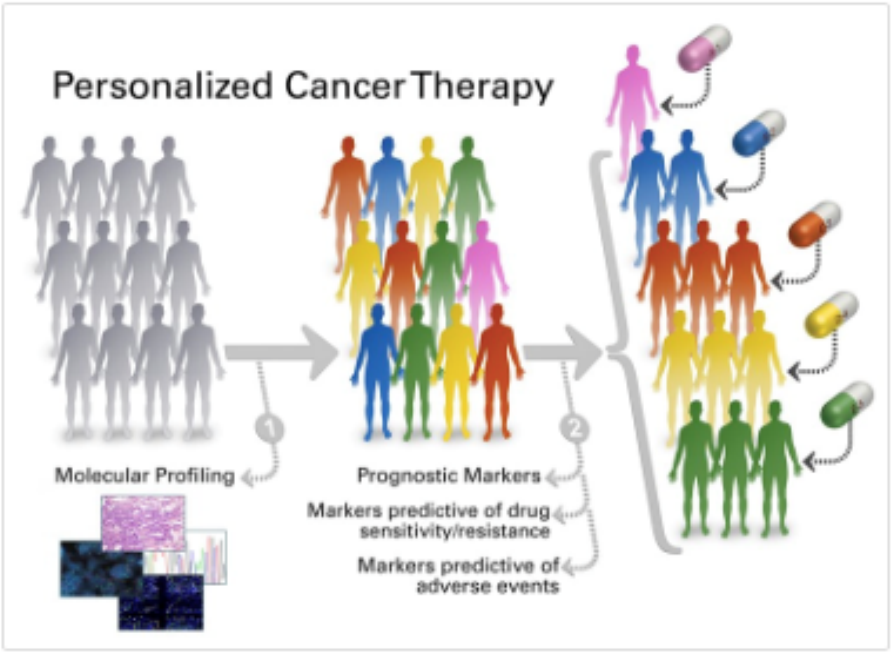
Who We Are | What We Do | Vision and Mission | Knowledge Base Generation | Contact Us

Search for gene information

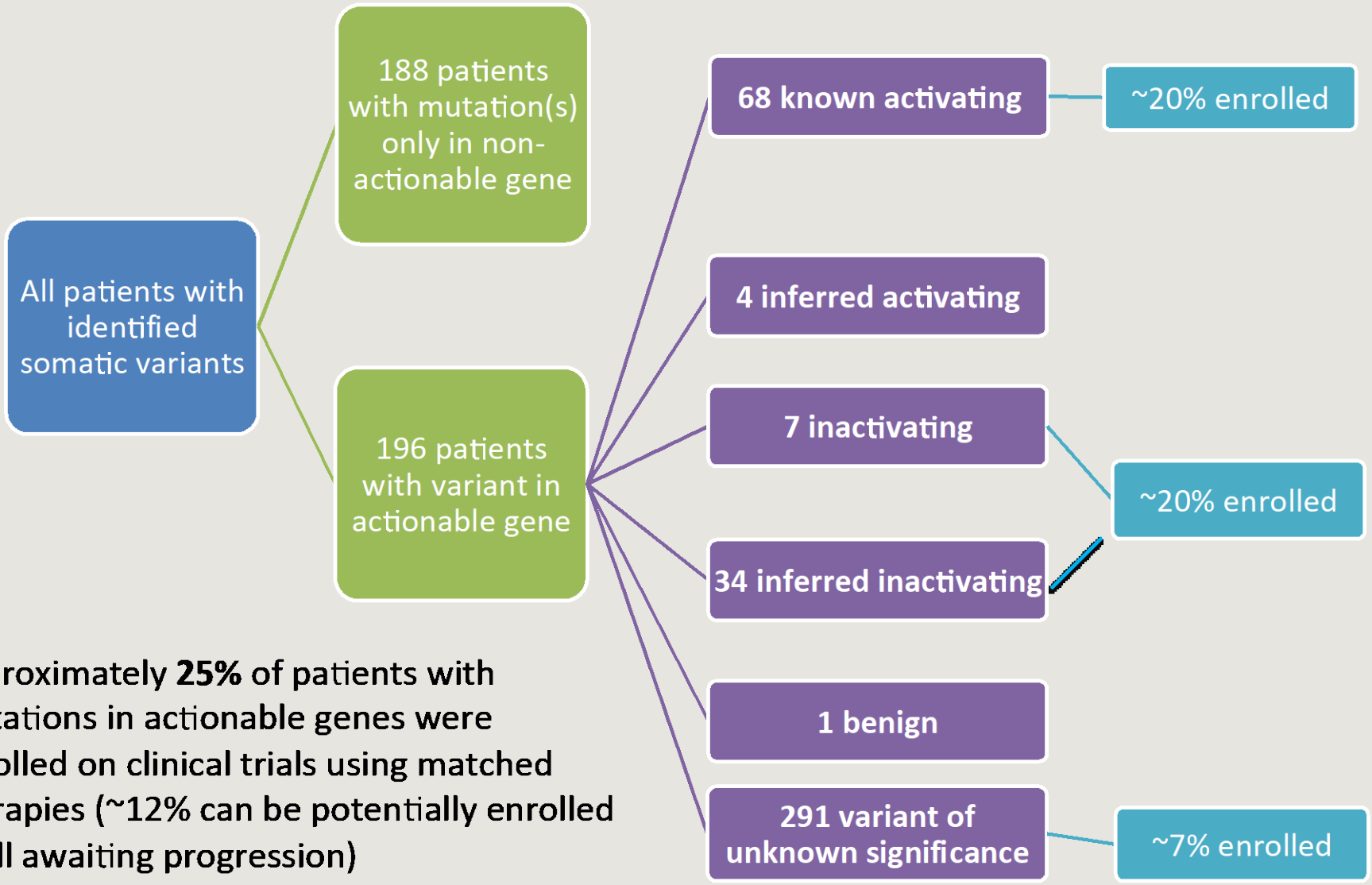
Select gene

Personalized cancer therapy is a treatment strategy centered on the ability to predict which patients are more likely to respond to specific cancer therapies. This approach is founded upon the idea that tumor biomarkers are associated with patient prognosis and tumor response to therapy. In addition, patient genetic factors can be associated with drug metabolism, drug response and drug toxicity. Personalized tumor molecular profiles, tumor disease site and other patient characteristics are then potentially used for determining optimum individualized therapy options.

Tumor biomarkers can be DNA, RNA, protein and metabolomic profiles that predict therapy response. However, the most recent approach is the sequencing of tumor DNA, which can reveal genomic alterations that have implications for cancer treatment. This Personalized Cancer Therapy website was specifically developed as a tool for physicians and patients to assess potential therapy options based on specific tumor biomarkers.



Decision Support in Real Time Improves 'Matching' to 'Right' Drug



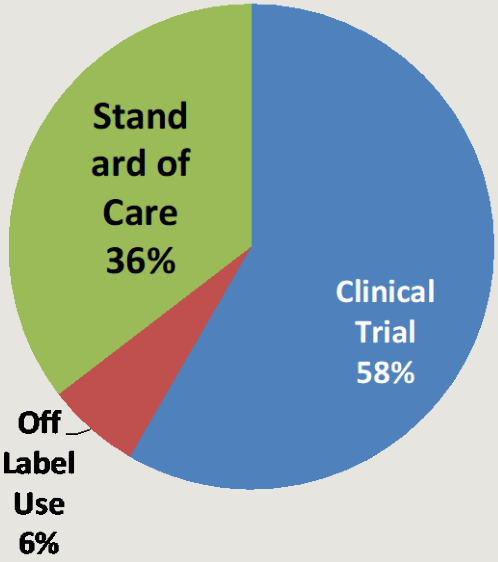
Approximately **25%** of patients with mutations in actionable genes were enrolled on clinical trials using matched therapies (~12% can be potentially enrolled - still awaiting progression)

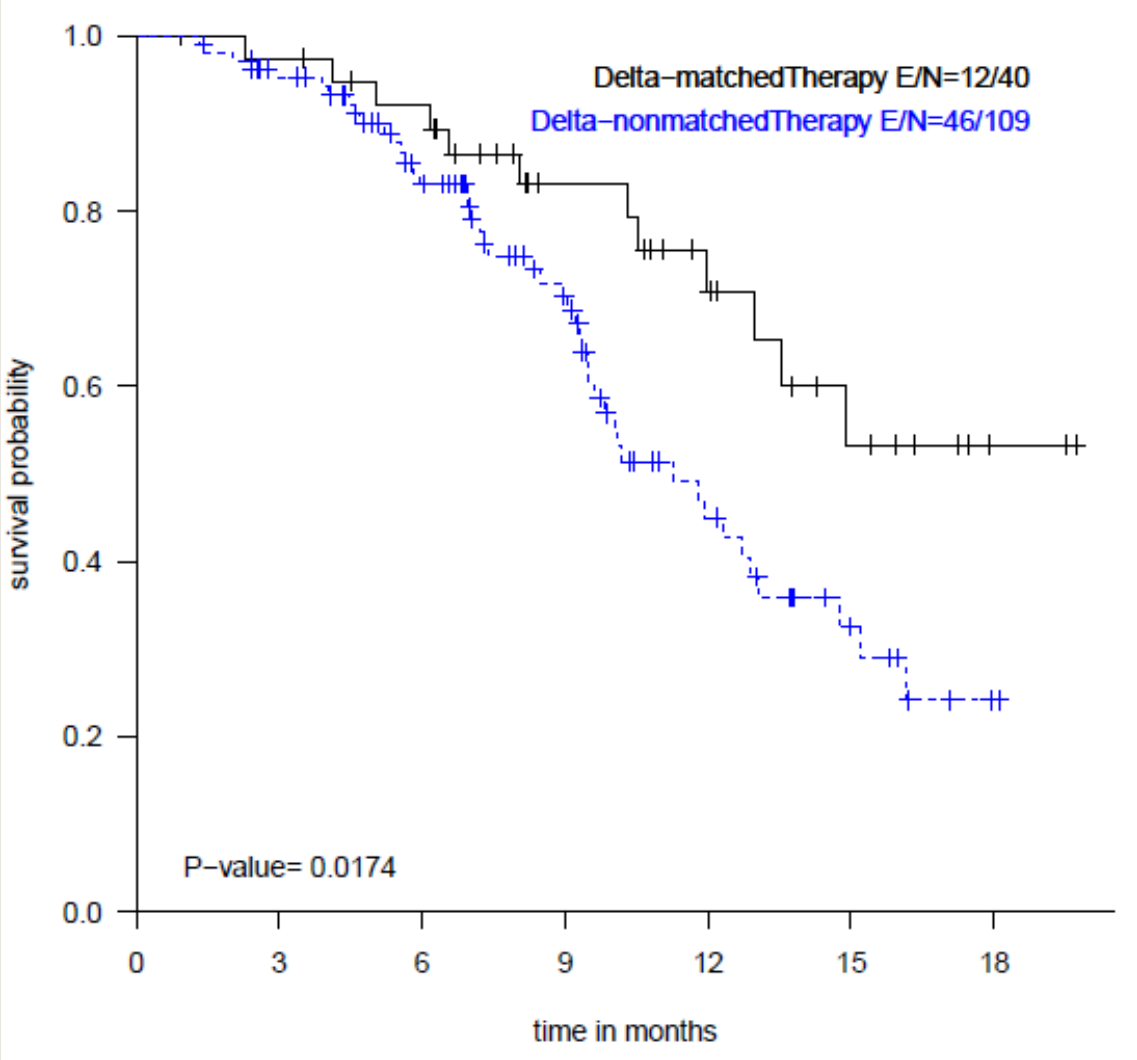
‘Genomic testing is associated with improved outcomes even in phase I trials when drugs are available

All patients were enrolled on ongoing phase I trials

Markedly increased repertoire of drugs

Does not count patients entered on standard of care





Unpublished data from S. Kopetz, J. Lee, R. Broaddus & K. Shaw.

END OF THE BEGINNING
Tumor intrinsic, monotherapy, silos

Targeted Therapy

Even for patients with the biomarker only subpopulations of patients benefit from monotherapy: Usually short term

Emergence of resistance is almost universal

Immunotherapy

When benefit occurs, tends to be durable

Remarkable effects in some diseases:
Leukemia, melanoma, lung, bladder

In most diseases benefit is modest

Few effective biomarkers

Need to apply precision oncology precepts

Patient specific combination therapy

Needed to fulfill promise

Limited ability to predict combinations

Deep longitudinal spatial analysis incorporating structure, DNA, RNA, protein, and phenotype

SMMART®

Serial Measurements of Molecular and Architectural Responses to Therapy: Beyond genetics (60% without actional genetic events)

Patients are treated based on a pre-therapy biopsy: Many years prior

Malignant cells and tumor ecosystem adapt rapidly to therapy

Hypothesis: Optimum patient outcome will accrue from treating patients based on how the tumor and tumor ecosystem adapts to therapy

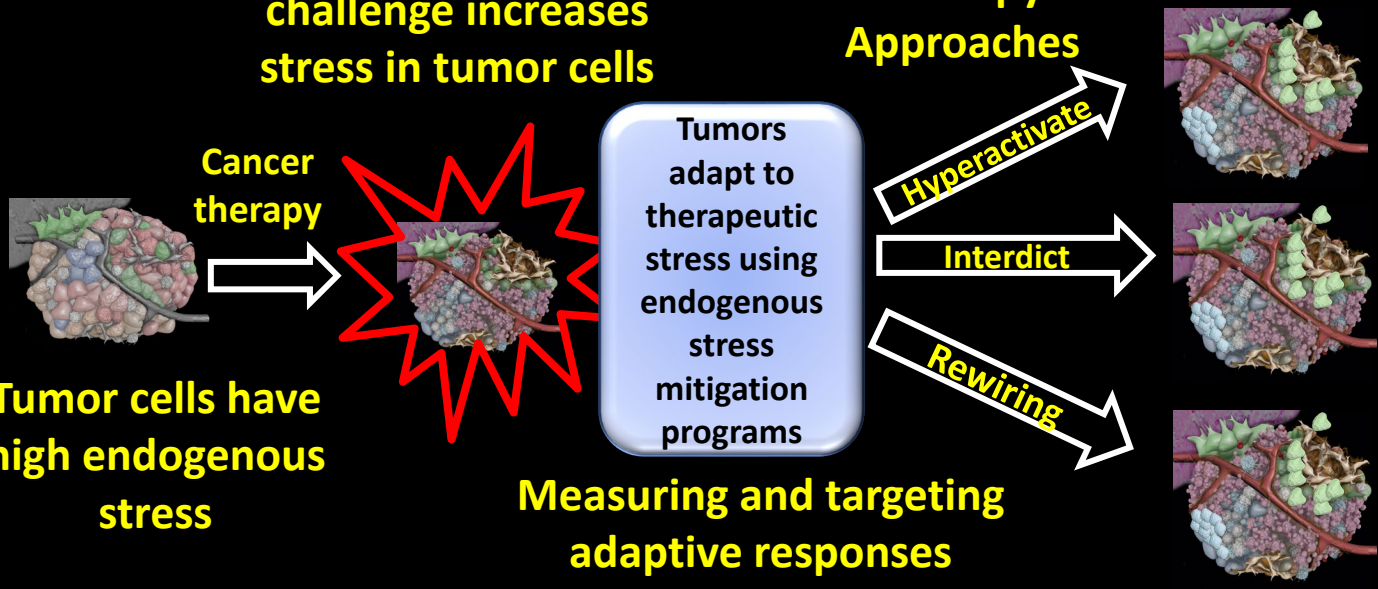
Corollary: Requires acting on spatial analysis of malignant cells and the tumor ecosystem including immune cells and communities as they adapt to therapy

Requires comprehensive analysis of DNA, RNA, protein with single cell spatial resolution in real time (DNA/RNASeq, cycIF, mIHC, EM, scRNA Seq, DSP, CosMx)

Tumor Cells Engage Endogenous Stress Mitigation Programs to Survive Therapeutic Stress: Adaptive responses represent therapeutic opportunities

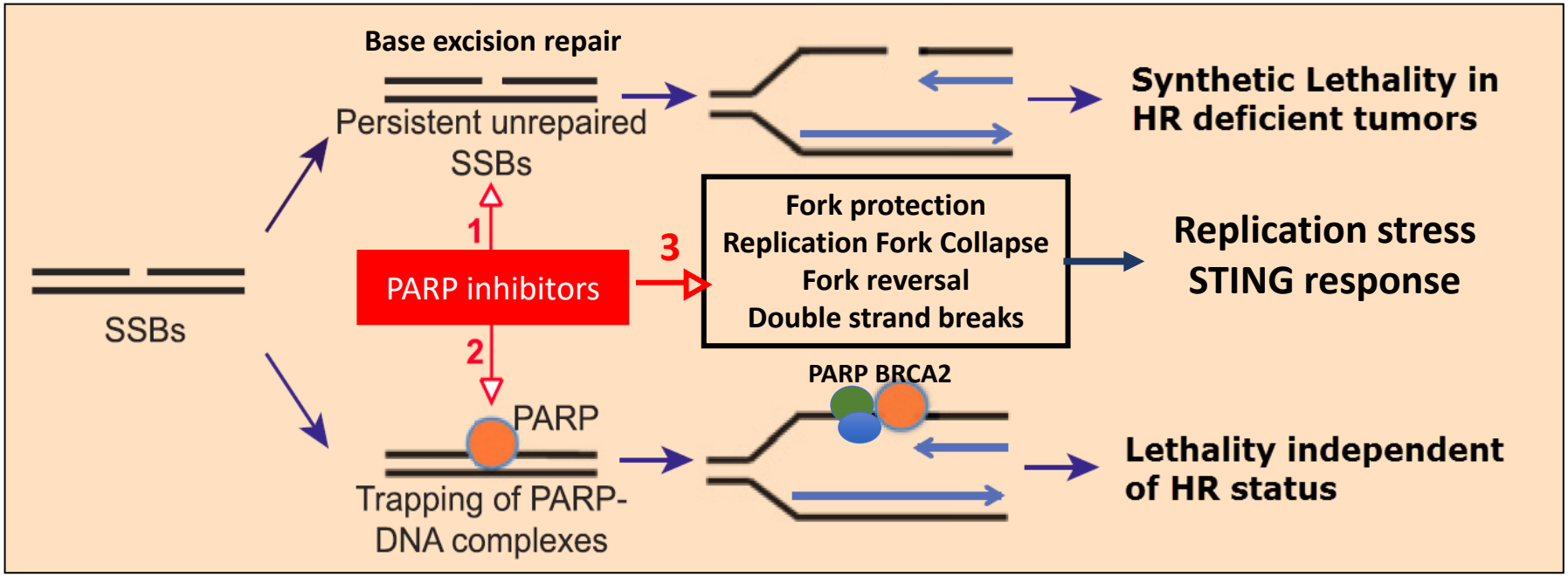
Labrie M., Brugge J., Mills GB., Zervantonakis I 2022 Therapeutic opportunities created by adaptive responses to target therapies in cancer Nat Rev Cancer. doi: 10.1038/s41568-022-00454-5

Adapted from Joan Brugge



Adaptive responses are mediated by state and state change: not genetics

Capitalization on DNA damaging activity of PARP inhibitors outside of HRD



Base excision repair
Replication fork protection
Trapping PARP on DNA

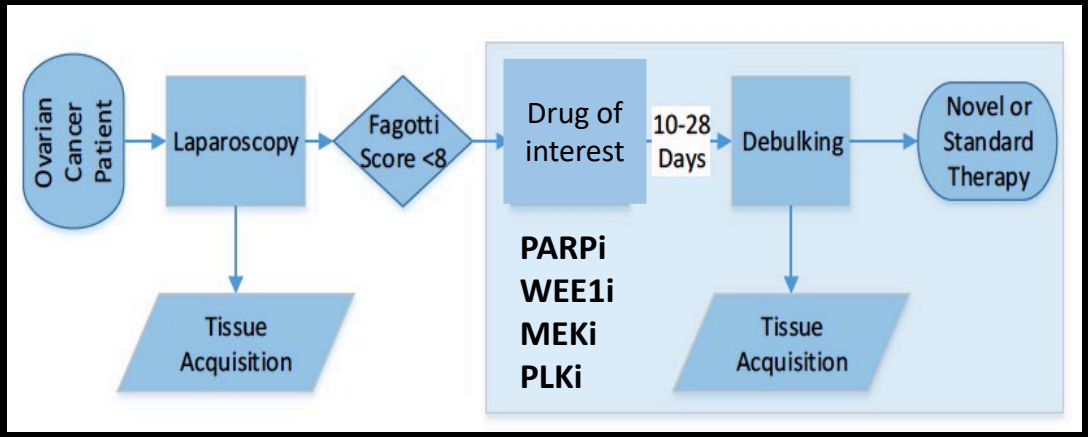
Single strand breaks
Replication stress
Difficult to repair double strand breaks

KNIGHT CANCER Institute
OHSU

Prerequisites for targeting adaptive responses

- **Adaptive responses are conserved across different lesions in an individual patient:** Limited intrapatient heterogeneity of change in signal
- **Adaptive responses are different across individuals:** Interpatient heterogeneity require personalized therapy
- **There are a limited number of adaptive responses**
- **Adaptive responses predict rational combinations** Must measure response
- Do adaptive response predict response to subsequent therapy better than a pre-therapy biopsy

Window of opportunity trial in ovarian and metastatic pancreatic cancer



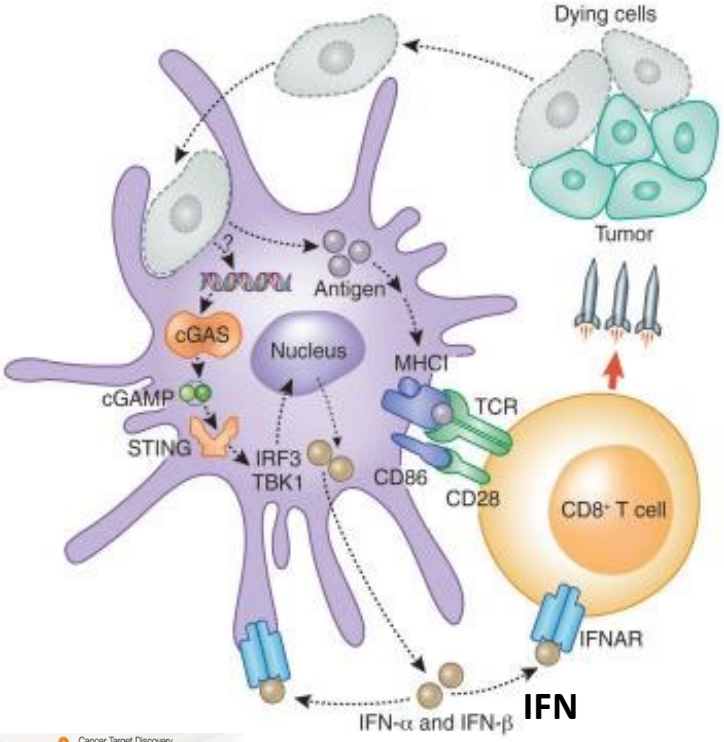
Adaptive Responses to PARP inhibitor Converge on a limited number of targetable pathways

Ongoing and completed biopsy driven trials

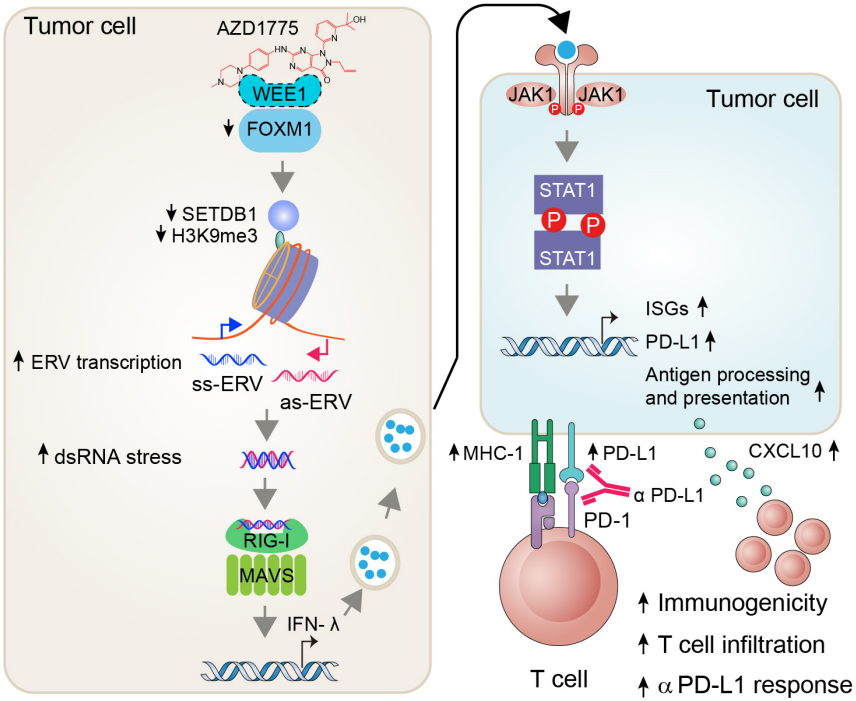
PARP and PDL1 inhibitors (AMTEC)
PARP and PI3K/AKT/mTOR inhibitors (Octopus)
PARP and MEK inhibitors (Solar)
PARP and WEE1 inhibitors: Combined and sequential (Effort/STAR)
PARP and ATR inhibitors (AMTEC)
PARP and CDK4/6 inhibitors (PANNTHR)
Reversing PARP resistance WEE1 combination and monotherapy (Effort/STAR)

PARPi induced dsDNA STING and dsRNA RIG1/MAVS induced interferon production in tumor cells and the microenvironment:PDL1?

dsDNA Fragments



ERV reactivation and dsRNA fragments



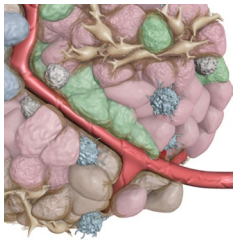
CTD² Cancer Target Discovery and Development

Chaoyang Sun

OHSU KNIGHT CANCER Institute

AMTEC: Adaptive Multi-Drug Treatment of Evolving Cancers
BRCA1/2 wild type

Resting Tumor Ecosystem



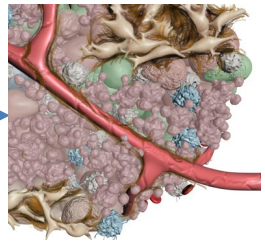
Limited systems information

Real-Time Deep Spatially Resolved Characterization of DNA, RNA, Protein from Tumor and ctDNA

Adaptation to Therapeutic stress

Olaparib
28 Days

Perturbed Tumor Ecosystem



Assess Adaptation to PARPi

Real-Time Deep Spatially Resolved Characterization of DNA, RNA, Protein from Tumor and ctDNA

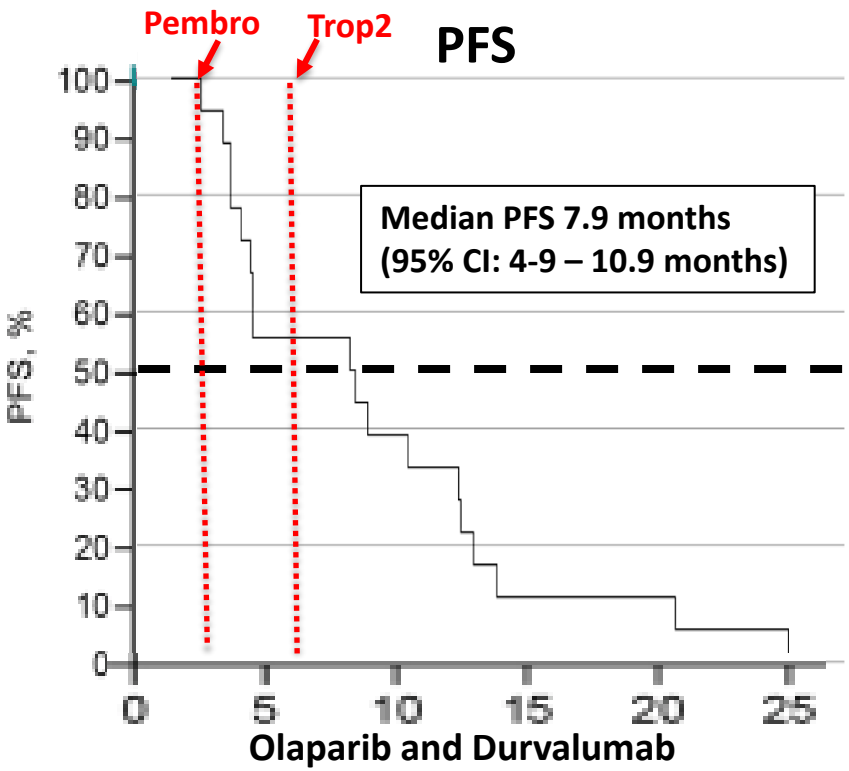
Olaparib
PDL1 Durvalumab
Combination

Bayesian analysis:
Phase 1: 15 patients:
Phase 2: 13 patients
Phase 1 patients used to develop or refine markers to be evaluated in phase 2

Alexandra Zimmer, Evie Hobbs, Zahi Mitri

AMTEC and Pilot

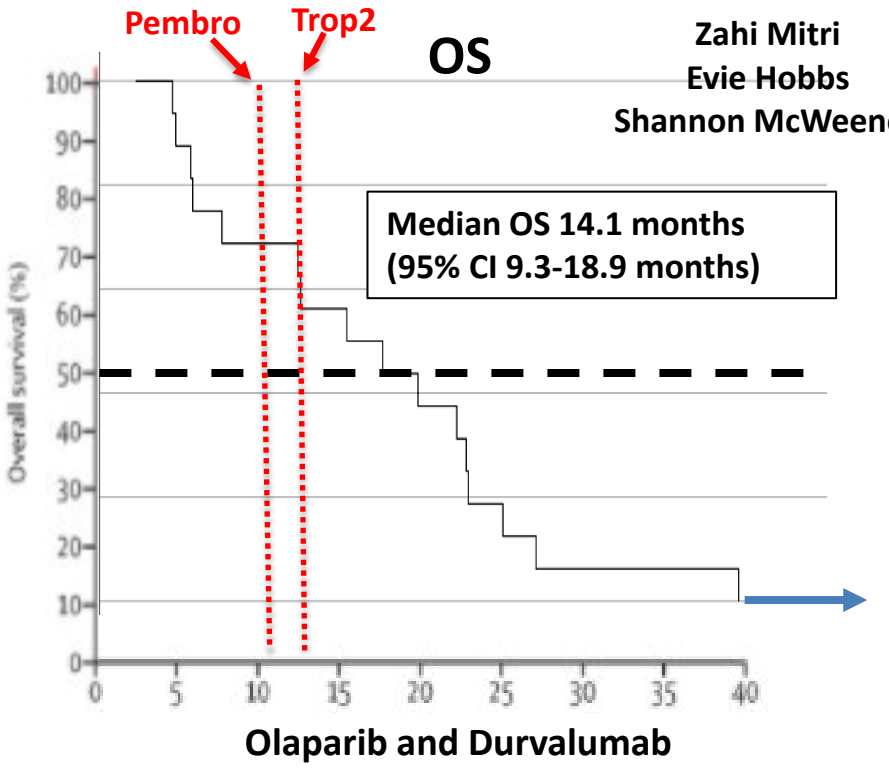
Olaparib 28 days then olaparib and durvalumab 18 patients



Olaparib and Durvalumab

Ascent TROP2 5.6 months

Keynote 119 Pembrolizumab 3.3 months vs physicians choice chemo 1.7 months



Olaparib and Durvalumab

Ascent TROP2 12.1 months

Keynote 119 Pembrolizumab vs physicians choice chemo 10.7 months

Zahi Mitri
Evie Hobbs
Shannon McWeeney

SMMART Analytics Linked to a Strong Comp Bio Program
RUO with transition to CLIA First multiplex protein assay in CLIA

CLIA (in close collaboration with Dr. Chris Corless and the Knight Diagnostic Laboratory)	
IHC	Proliferation (Ki67), cell lineage (ER, PR, HER2), PDL1, CD4/8 lymphocytes
GeneTrails	Deep analysis of 256 candidates for mutation, copy number and TMB
RNASeq	Analysis of RNA levels, fusion genes, pathway analysis, GSVA (requires high tumor content)
Digital Spatial Profiling (DSP)* IMCO	Proteins in tumor and stroma; 100+ proteins (signaling pathways, lineage, rep stress, DNA damage, immune)
Research Use Only (in close collaboration with OHSU/Knight Cancer Institute research laboratories)	
cycIF IMCO	Single-cell, spatially-resolved analysis of tumor and immune contexture Tumor and immune: 2 slides 60 markers
mIHC IMCO	Single-cell, spatially-resolved analysis of immune contexture (Discovery panel = 23 antibodies). Deep analysis of specific immune compartments available
scRNAseq	Single-cell analysis of disaggregated tumor cells for tumor, stroma and immune component
RPPA	Analysis of 486 proteins in tissue lysates
Spatial Molecular Imaging (CosMx)	Single-cell, subcellular resolution with RNA (2000) and Protein (200)
Liquid Biopsy**	Circulating cytokines: Proteins miRNA, and mRNA in exosomes
Electron Microscopy**	Large field EM for architecture (2D/3D)

Liquid Biopsies**	
ctDNA	Response to therapy (Natera)
O-link IMCO	>100 cytokines
CyTOF*	Peripheral immune content

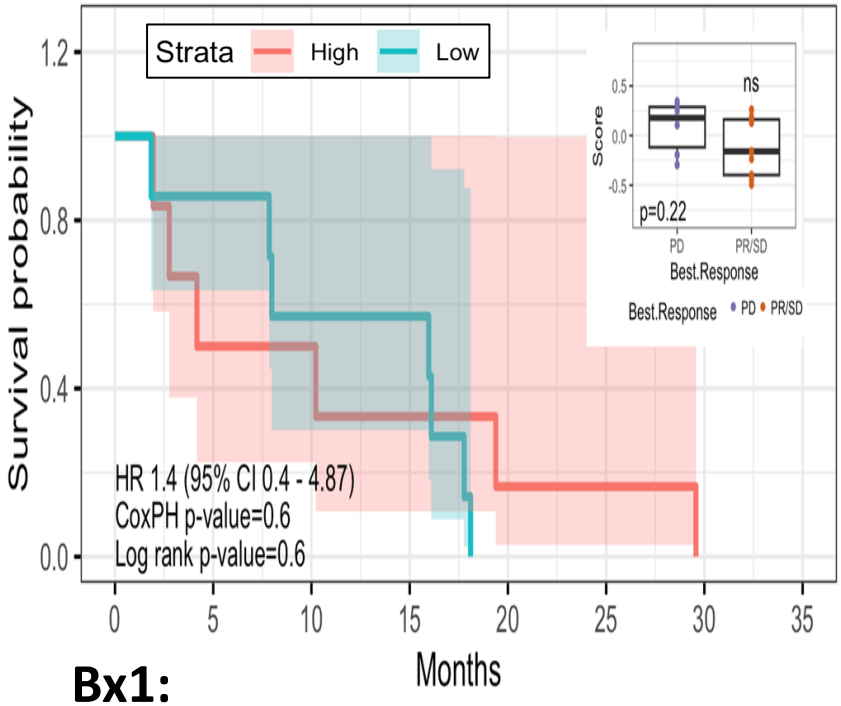
- **Linked to a strong computational biology platform**
- ***Joanna Pucilowska, PhD/IMCO**
- **Chris Corless Knight Diagnostic Laboratory Pathology**
- **All assays are available (**denotes special request)**

OHSU KNIGHT CANCER Institute

Tumor microenvironment predictors

Angiogenesis RNA Signature: May explain activity of PARPi and anti VEGFR

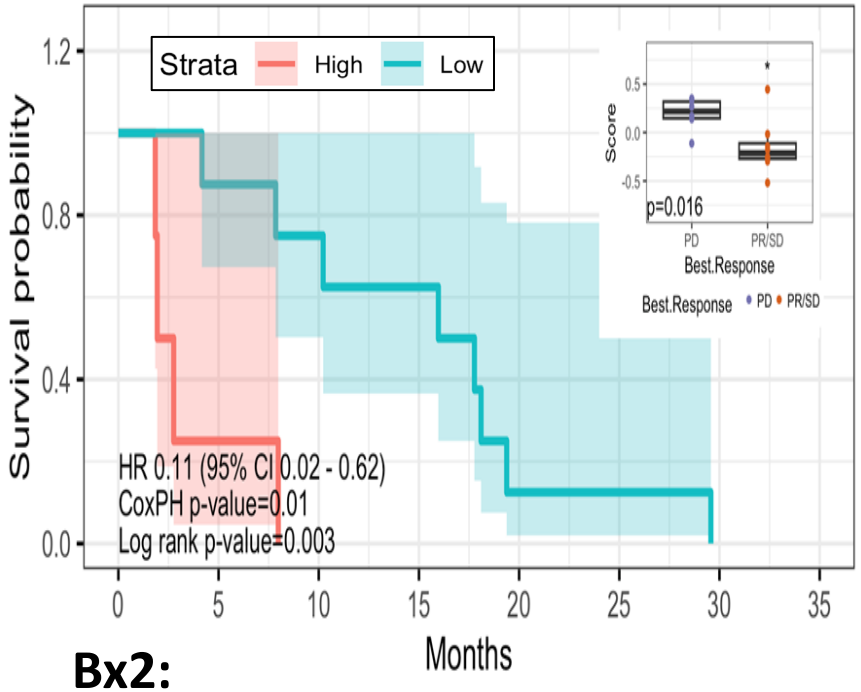
Allison Creason
Jayne Stommel



Bx1:

Overall Survival Probability **P = 0.6**

Best Response t-test **P = 0.22**



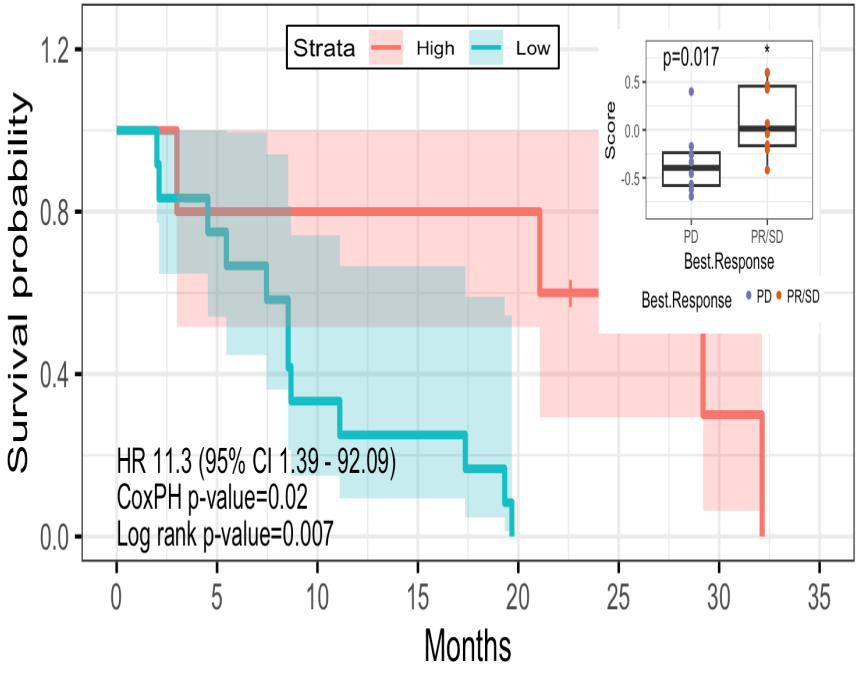
Bx2:

Overall Survival Probability **P = 0.003**

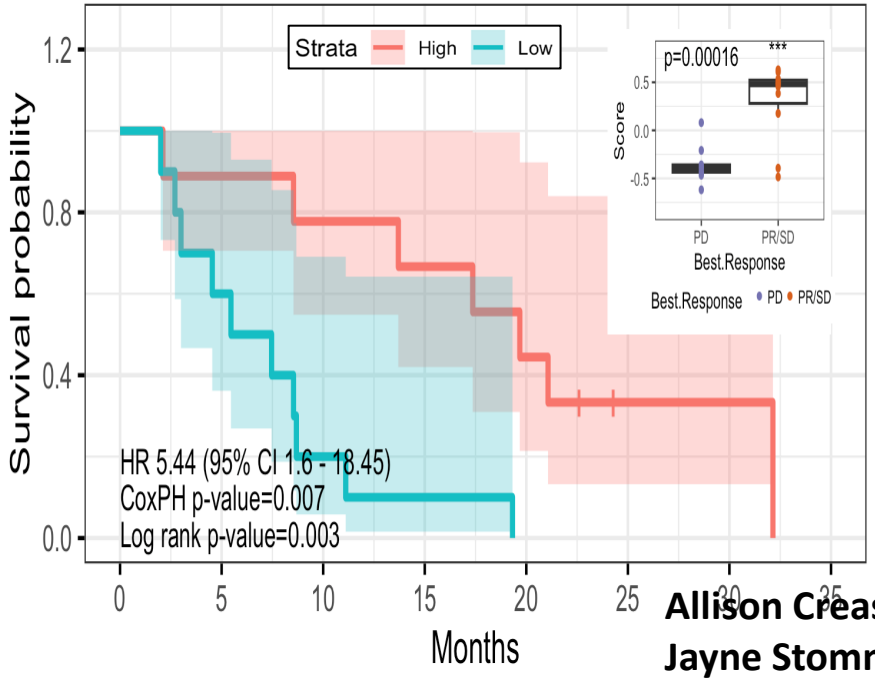
Best Response t-test **P = 0.016**

Tumor microenvironment predictors: RNA
Extended sample set Pilot: phase 1 and phase 2

Interferon Gamma Response
OS Bx1: P=0.007
Best Response P=.0017



Interferon gamma response
OS Bx2: P=0.003
Best Response P=0.00016

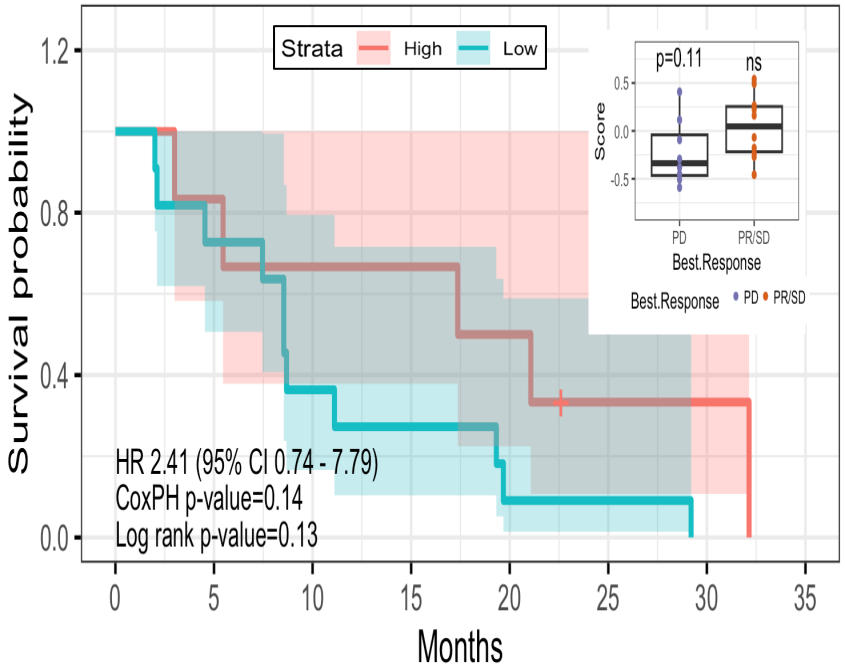


Allison Creason
Jayne Stommel

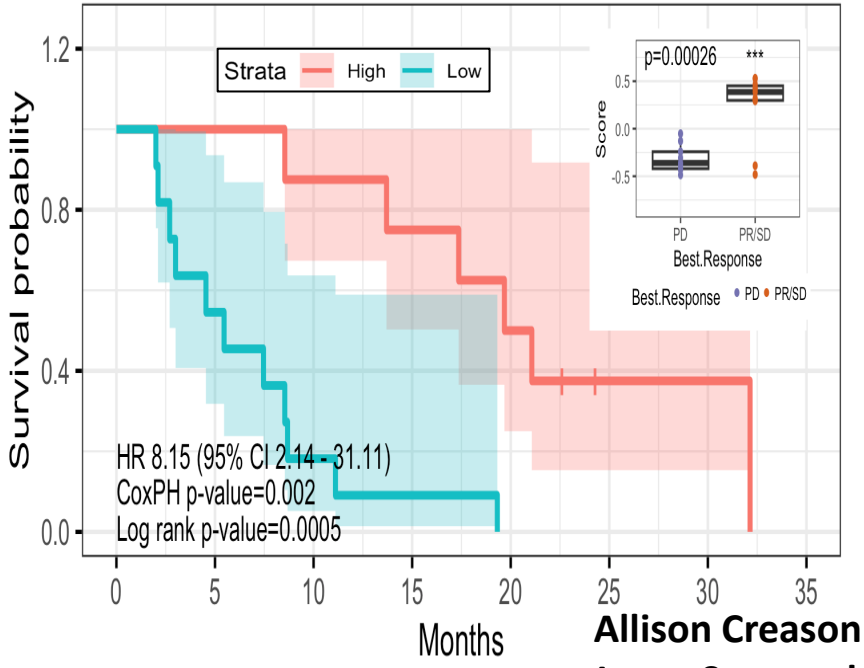
Tumor microenvironment predictors: RNA Hallmarks

Extended sample set Pilot: phase 1 and phase 2

JAK Stat3 Signaling
OS Bx1: P=0.13
Best Response P=11

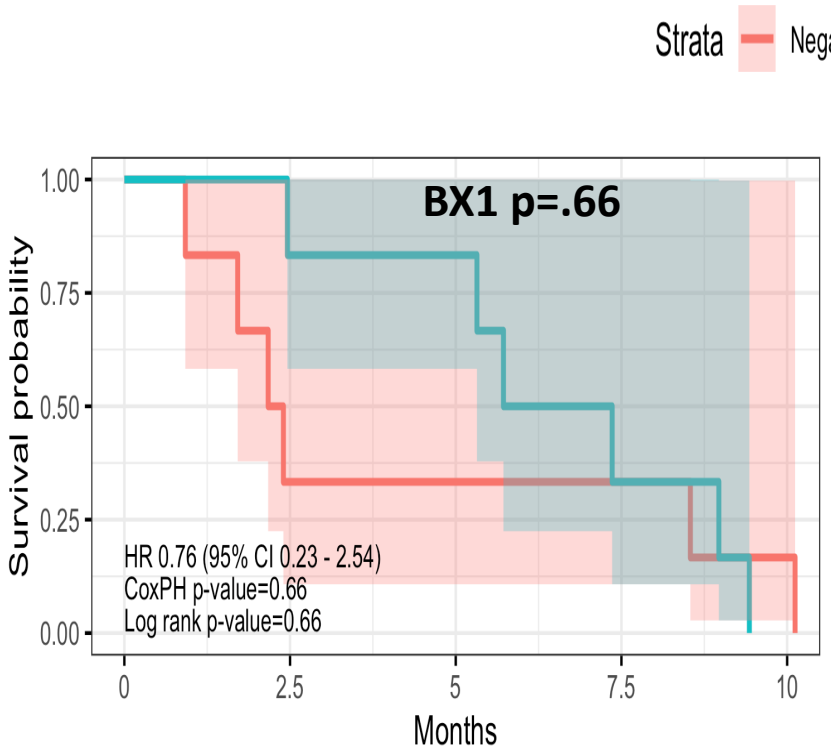


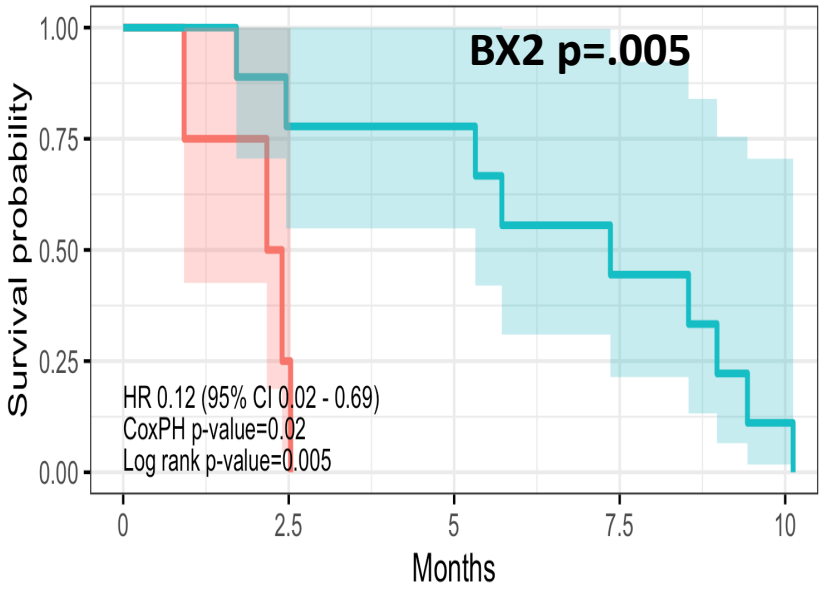
JAK Stat3 signaling
OS Bx2: P=0.0005
Best Response P=0.0026



Allison Creason
Jayne Stommel

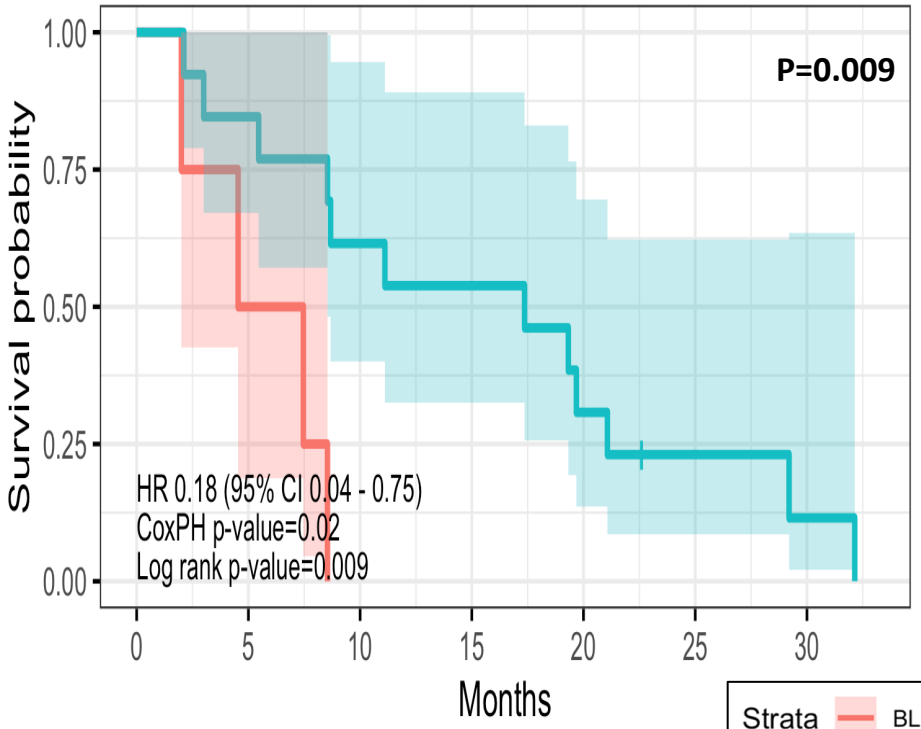
Tumor microenvironment predictors:
PFS Consensus Immune RNA/DSP/mIHC Phase 1 only

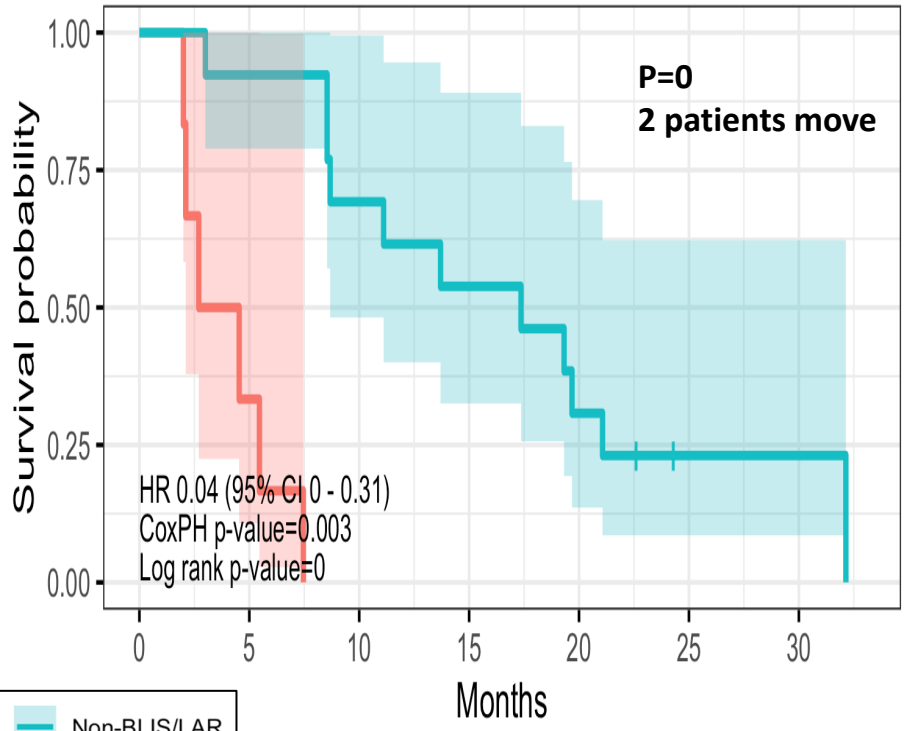




Allison Creason
Jayne Stommel

AMTEC OS: Extended set Basal-Like Immune Activated (BLIA), Basal-Like Immune Suppressed (BLIS), Luminal Androgen Receptor (LAR), Mesenchymal (Mes)

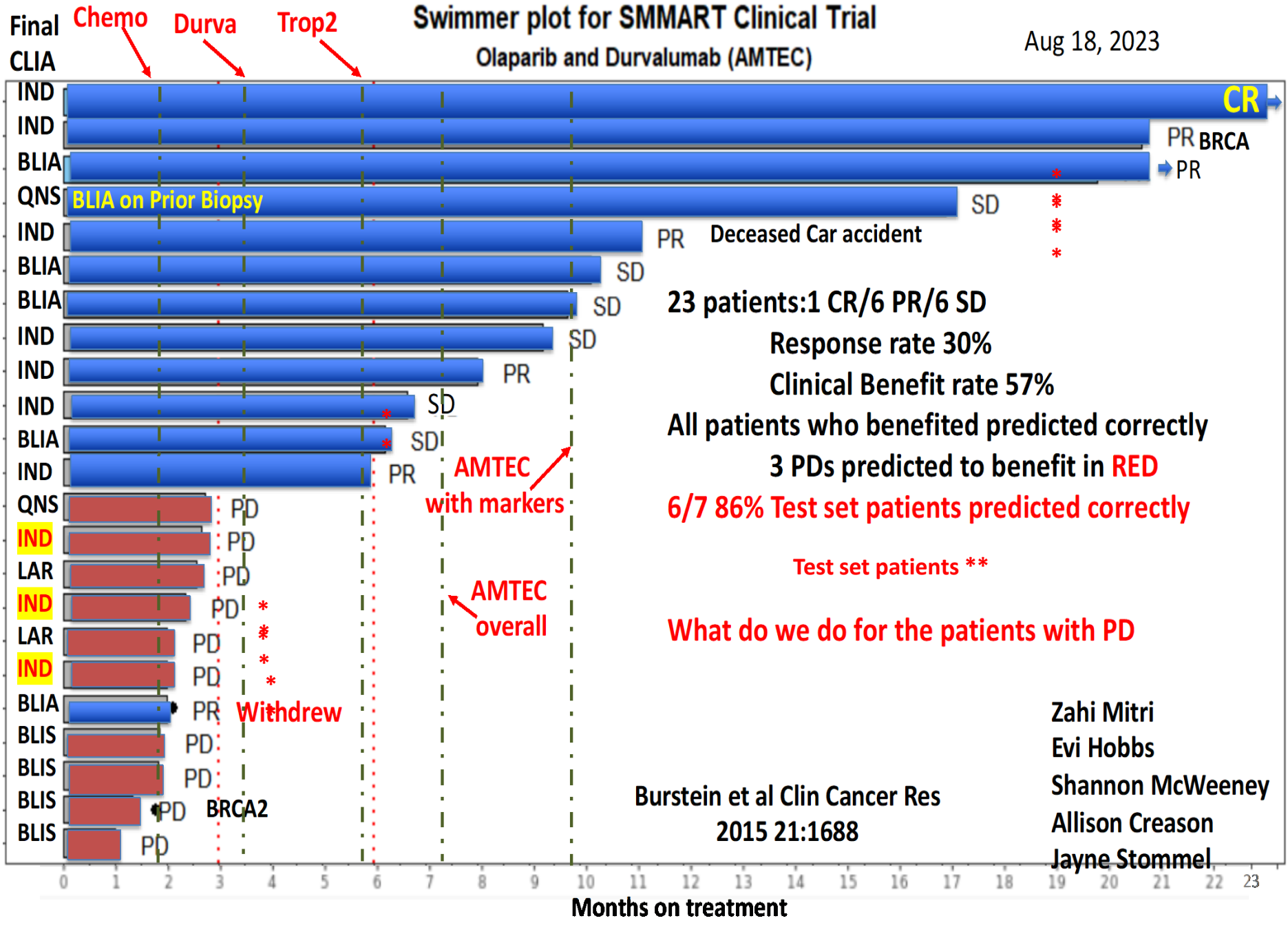


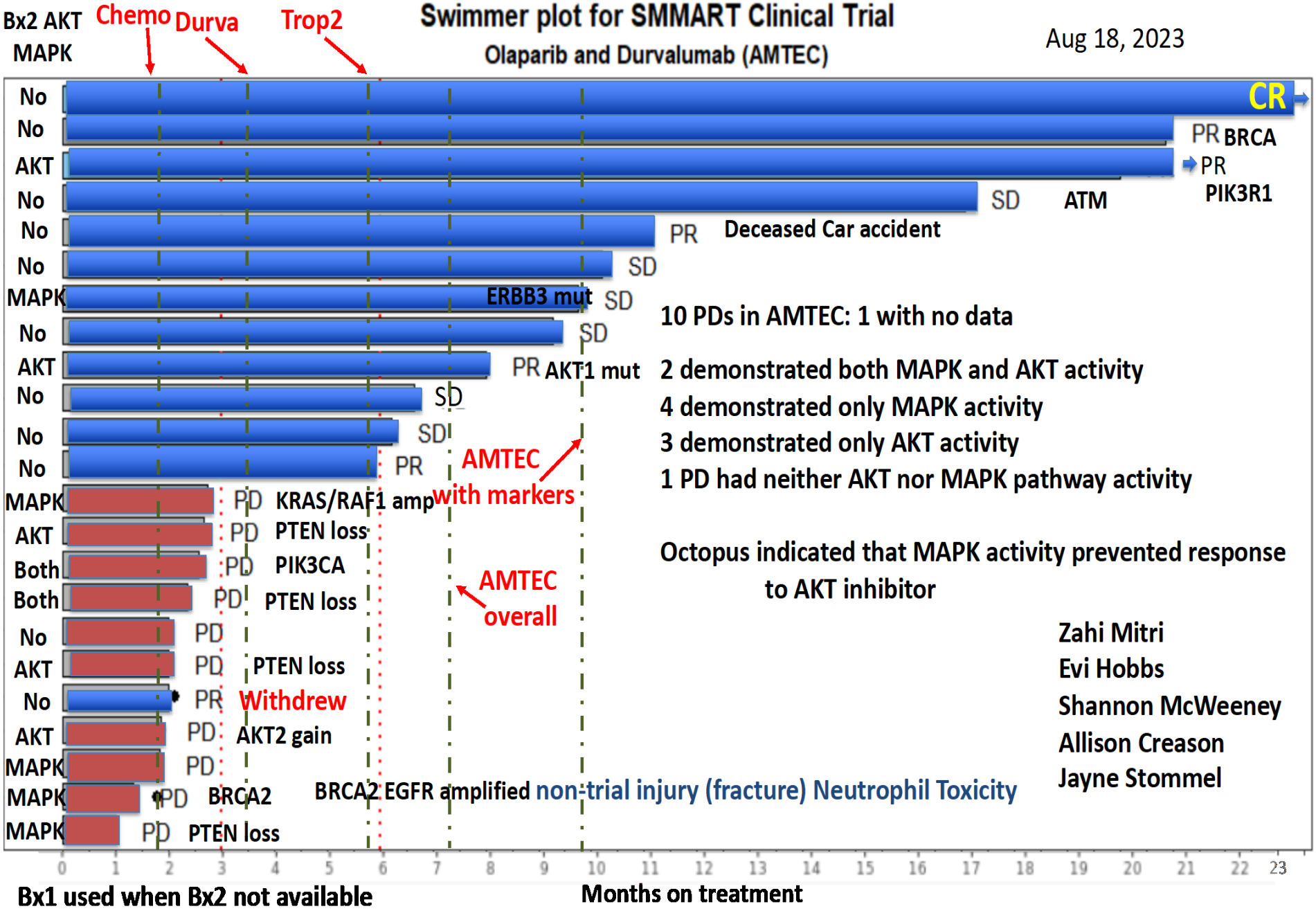


Burstein et al Clin Cancer Res
2015 21:1688

AMTEC 17.5 months
Ascent Trop 2 12.1 months
Keynote 119 Pembro 10.7

Zahi Mitri, Evie Hobbs, Alexandra Zimmer
Allison Creason, Aurora Blucher
Alfonso Poire, Marilyne Labrie
Jeremy Goecks, Shannon McWeeney

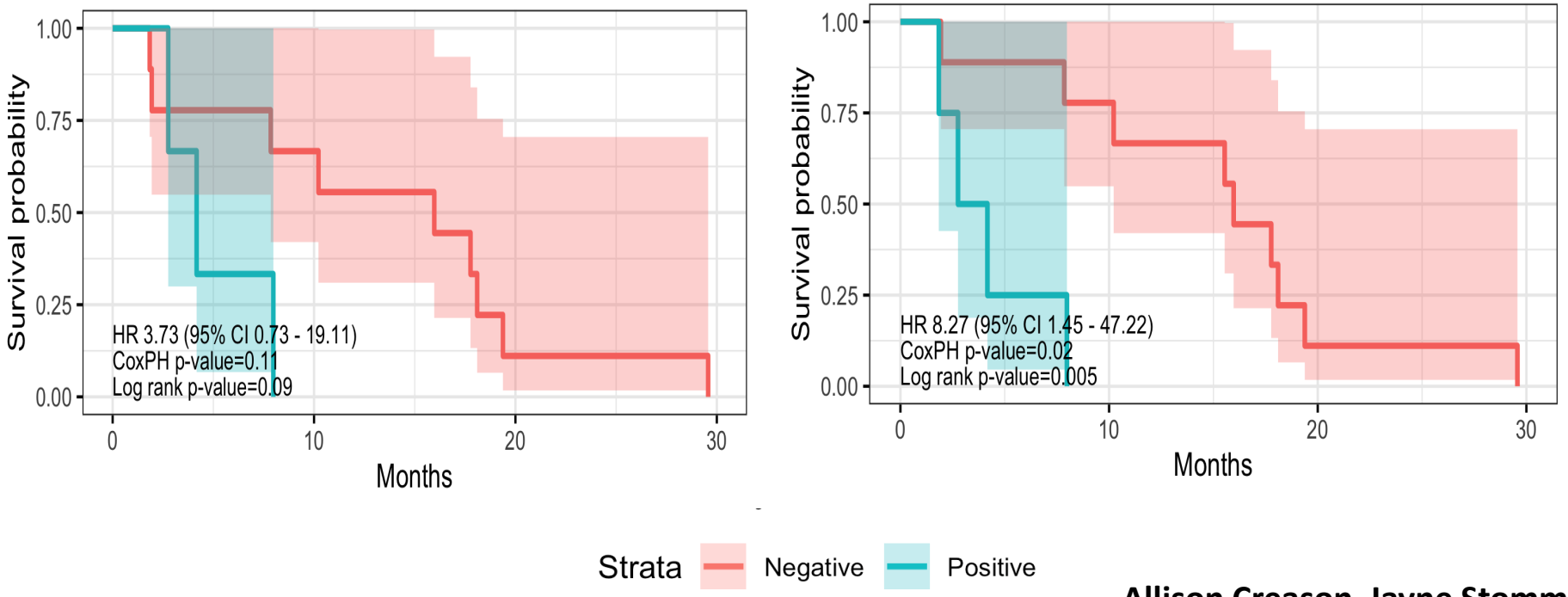

Swimmer plot for SMMART Clinical Trial
Olaparib and Durvalumab (AMTEC)
Aug 18, 2023
Final CLIA
Chemo
Durva
Trop2
IND
IND
BLIA
QNS
IND
BLIA
BLIA
IND
IND
IND
BLIA
IND
QNS
IND
LAR
IND
LAR
IND
BLIA
BLIS
BLIS
BLIS
BLIS
CR
PR BRCA
PR
BLIA on Prior Biopsy
SD
PR
Deceased Car accident
SD
SD
SD
PR
SD
SD
PR
PD
PD
PD
PD
PD
PD
PR
Withdrew
PD
PD
PD
BRCA2
23 patients:1 CR/6 PR/6 SD
Response rate 30%
Clinical Benefit rate 57%
All patients who benefited predicted correctly
3 PDs predicted to benefit in RED
6/7 86% Test set patients predicted correctly
Test set patients **
What do we do for the patients with PD
AMTEC with markers
AMTEC overall
Burstein et al Clin Cancer Res 2015 21:1688
Zahi Mitri
Evi Hobbs
Shannon McWeeney
Allison Creason
Jayne Stommel
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23
Months on treatment


Swimmer plot for SMMART Clinical Trial
Olaparib and Durvalumab (AMTEC)
Aug 18, 2023
Bx2 AKT MAPK
Chemo
Durva
Trop2
No
No
AKT
No
No
No
MAPK
No
AKT
No
No
No
MAPK
AKT
Both
Both
No
AKT
No
AKT
MAPK
MAPK
MAPK
CR
PR BRCA
PR PIK3R1
SD ATM
PR Deceased Car accident
SD
ERBB3 mut SD
SD
PR AKT1 mut
SD
SD
PR
PD KRAS/RAF1 amp
PD PTEN loss
PD PIK3CA
PD PTEN loss
PD
PD PTEN loss
PR Withdrew
PD AKT2 gain
PD
PD BRCA2
PD PTEN loss
AMTEC with markers
AMTEC overall
10 PDs in AMTEC: 1 with no data
2 demonstrated both MAPK and AKT activity
4 demonstrated only MAPK activity
3 demonstrated only AKT activity
1 PD had neither AKT nor MAPK pathway activity
Octopus indicated that MAPK activity prevented response to AKT inhibitor
Zahi Mitri
Evi Hobbs
Shannon McWeeney
Allison Creason
Jayne Stommel
BRCA2 EGFR amplified non-trial injury (fracture) Neutrophil Toxicity
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23
Bx1 used when Bx2 not available
Months on treatment

PI3K and MAPK activity predicts outcomes in AMTEC Pilot, phase 1 and phase 2

AKT or MAPK Pathway Activation DSP

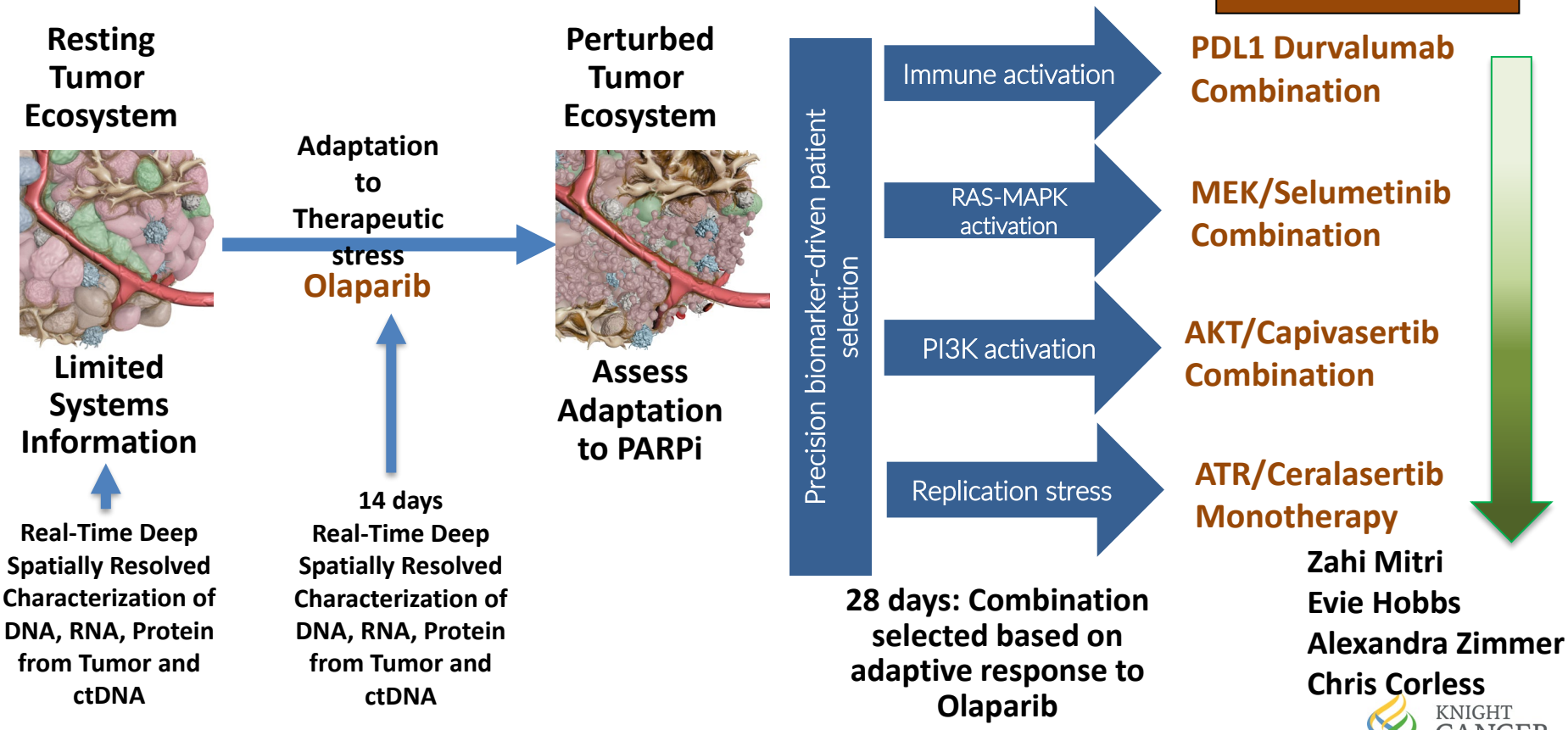
OS Bx2: P=0.14



Allison Creason Jayne Stommel

AMTEC: Adaptive Multi-Drug Treatment of Evolving Cancers

First trial that treats patients based on how tumors adapt to therapy



Biggest challenge: Measuring adaptive responses in real time

Zahi Mitri
Evie Hobbs
Alexandra Zimmer
Chris Corless

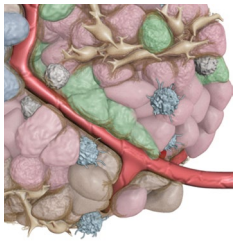
KNIGHT CANCER Institute
OHSU

AMTEC: Adaptive Multi-Drug Treatment of Evolving Cancers

First trial that treats patients based on how tumors adapt to therapy

TARGET THE ADAPTIVE RESPONSE

Resting Tumor Ecosystem



Limited Systems Information

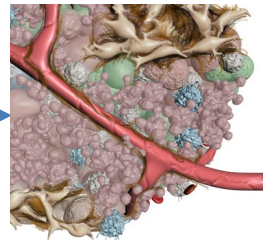
Real-Time Deep Spatially Resolved Characterization of DNA, RNA, Protein from Tumor and ctDNA

Adaptation to Therapeutic stress

Olaparib

14 days Real-Time Deep Spatially Resolved Characterization of DNA, RNA, Protein from Tumor and ctDNA

Perturbed Tumor Ecosystem



Assess Adaptation to PARPi

Precision biomarker-driven patient selection

- Immune activation → **PDL1 Durvalumab Combination**
- RAS-MAPK activation → **MEK/Selumetinib Combination**
- PI3K activation → **AKT/Capivasertib Combination**
- Replication stress → **ATR/Ceralasertib Monotherapy**
- Retained Angiogenesis → **VEGFR/Cediranib Combination**

28 days: Combination selected based on adaptive response to Olaparib

Biggest challenge: Measuring adaptive responses in real time

Zahi Mitri
Evie Hobbs
Alexandra Zimmer
Chris Corless

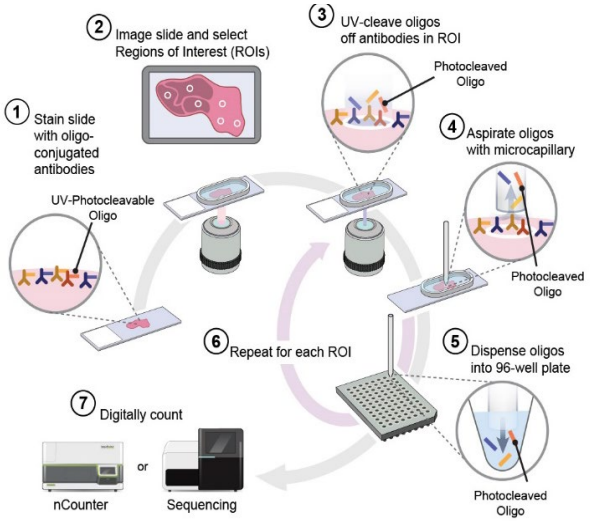
KNIGHT CANCER Institute

Digital Spatial Profiling
Spatially oriented ROI

LETTERS
https://doi.org/10.1038/s41587-020-0472-9
nature biotechnology

Multiplex digital spatial profiling of proteins and RNA in fixed tissue

Christopher R. Merritt[1], Giang T. Ong[1], Sarah E. Church[1], Kristi Barker[1], Patrick Danaher[1], Gary Geiss[1], Margaret Hoang[1], Jaemyeong Jung[1], Yan Liang[1], Jill McKay-Fleisch[1], Karen Nguyen[1], Zach Norgaard[1], Kristina Sorg[1], Isaac Sprague[1], Charles Warren[1], Sarah Warren[1], Philippa J. Webster[1], Zoey Zhou[1], Daniel R. Zollinger[1], Dwayne L. Dunaway[1], Gordon B. Mills[2] and Joseph M. Beechem[1]



Immune Cell Profiling Core	Pan tumor module	Cell Death Module	Oncogenic Signaling - PI3K/AKT	Oncogenic Signaling - RAS.MAPK	DNA damage signaling panel
PD-1	MART1	BAD	PTEN	MET	Cyclin E1
CD68	NY-ESO-1	BCL6	AKT-pS473	p-cMET (Y1234/1235)	ATM_pS1981
HLA-DR	S100B	BCLXL	GSK3ALPHABETA_ pS21/S9	EGFR	ATR_S428
Beta-2-microglobulin	EpCAM	BIM	GSK3_pS9	p-EGFR (Y1068)	c-Myc
CD11c	p21	p53	PRAS40_pT246	Her2	ARID1A
CD20	GAPDH	GZMA	TUBERIN_pT1462	**p-HER2 (Y877)	Cyclin B1
CD3	ER-alpha	CD95/Fas	INPP4B	FGFR1	Cyclin D1
CD4	PR	PARP	PLCG1	Phospho-MEK1 (S217/S221)	AR
CD45	Histone H3	Cleaved caspase 9	Pan-AKT	Phospho-p90 RSK (T359/S363)	p-HER2 (Tyr1248)
CD56	Total Rb	*NF1B	p-4EBP1 (Thr37/46)	p-ERK1/2 (T202/Y204)	p-gammaH2AX (Ser139)
CD8	p-RB (S807)	Mcl-1	S6	p-cMYC	p-Chk2
CTLA4	Ki-67	cleaved PARP	p-S6		p-Histone H3
GZMB	Tumor targets	Bcl-2	IRF1		total ATM
PD-L1	CDK4	Controls			SLFN11
PanCk	CDK6	Rb IgG			
SMA	RRM2	Ms IgG1			
Fibronectin	Trop2	Ms IgG2a			

CLIA Nanostring DSP:90 antibodies on one FFPE slide.
Tumor intrinsic
Immune and tumor microenvironment
ADCs

Your Samples IMCO Your Data Knight Cancer Institute
Chris Corless Joanna Pucilowska
<pucilows@ohsu.edu>

March 2023

OHSU KNIGHT CANCER Institute

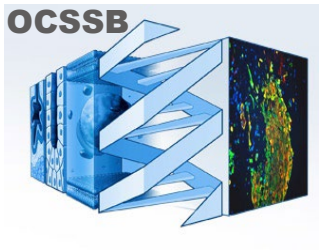
TEAM THAT MAKES IT HAPPEN

Others not pictured:
Allison Creason, Shannon McWeeney, Clayton Kills First, Nat Tilden, Dayana Rojas-Rodriguez, Jordan Teicher, Andrew Silvernail

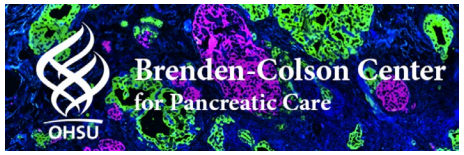
- **ALL OF OUR PATIENTS**
- **Knight Leadership:** Brian Druker
- **SMMART Leadership:** Gordon Mills, Chris Corless, Shannon McWeeney, Jeff Tyner
- **SMMART Operations:** Christina Zheng, Kiara Siex, Jayne Stommel, Allison Solanki
- **Clinicians & Clinical Teams:** Lara Davis, Alexandra Zimmer, Charles Lopez, Adel Kardosh, Tanja Pejovic, Ronan Swords, Shivaani Kummar, Brian Druker, Alexandra Sokolova, Elizabeth Munro, Christopher Ryan, Ted Braun, Curtis Lachowiez and more
- **Biology/Pathology/Radiology/Surgery:** Laura Heiser, Lisa Coussens, Chris Corless, Chris Suciu, George Thomas, Alex Guimaraes, Brett Sheppard, Monika Davare, and many more
- **SMMART Clinical Team:** Kiara Siex, Nat Tilden, Jules Amaya
- **SMMART Translational Operations:** Jayne Stommel, Ben Kong, Jamie Keck, Brett Johnson, Heidi Feiler
- **SMMART Data Team:** Lauren Murray, Ana Olson, Dayana Rojas-Rodriguez, Jordan Teicher
- **Knight Data Team:** Christina Zheng, Matt Viehdorfer, Andrew Silvernail
- **Knight Computational Biology Team:** Allison Creason, Emek Demir and team
- **Research Lab Staff:** Allison Creason, Koei Chin, Jessica Riesterer, Boyoung Jeong, Tugba Ozmen, Furkan Ozmen, Alfonso Poire, David Kilburn, Sam Sivagnanam, many more
- **Knight Diagnostics Lab/BioLibrary/Histopathology Core Teams**
- **Immune Monitoring & Cancer Omics (IMCO):** Joanna Pucilowska and team
- **Brendon Colson Center for Pancreatic Health:** Rosie Sears, Jonathan Brody and teams
- **CEDAR:** Sadik Esener and team
- **Biostatistics Shared Resource**
- **Knight Finance and Knight CRM Finance**
- **Knight Disease Teams and Knight CRM Group**
- **Knight Clinical Research and Quality Teams**
- **OHSU Tech Transfer, CTO, OPAM and CRSO**
- **Patient Advocates**
- **Prior Leads:** Joe Gray, Ray Bergan, Tom Beer, Annette Kolodzie, Marlana Klinger, Rochelle Williams-Belizaire, Souraya Mitri, Marilyne Labrie, Nathan McMahon, Patrick Leyshock, Chaoyang Sun, and many more
- **AND MANY MORE!!**

OHSU KNIGHT CANCER Institute

SMMART® Foundational Partners



Cancer Early Detection Advanced Research Center



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Beat AML

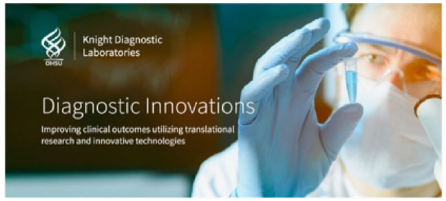
STAND UP TO CANCER

NIH Microenvironmental Perturbagen LINCS

NCI Center for Cancer Systems Biology

CANCER SYSTEMS BIOLOGY CONSORTIUM

NCI CANCER MOONSHOT
Human Tumor Atlas Network
A program of the National Cancer Institute of the National Institutes of Health



susan g. komen.

Prospect Creek Foundation

ALL PATIENTS

Patient Advocates

Research Management/ Quality & Administration

OHSU/Knight Leadership

Pathology/Surgery

Cell Biology

Cancer Center

SMMART Leadership

Bioinformatics/Technical

Bioinformatics Lab/

Unnamed but Appreciated

Genetics

SMMART Operations

KNIGHT CANCER Institute OHSU