I-SPY with my little eye
your best matched treatment

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*University California San Francisco*

*Inspire2Live 2020*
Disclosure

• Co-Founder, part-time employee and stock holder Agendia NV (MammaPrint)

• No off-label use will be discussed
Personalized Healthcare - Precision Oncology

Science based targeted escalation and de-escalation

GOAL

INCREASE EFFICACY

REDUCE MORBIDITY

SCREENING

DIAGNOSIS

TREATMENT

SURVIVORSHIP

PERSONALIZED INTERVENTION

ESCALATE

RISK or RESPONSE

DE-ESCALATE

RISK or RESPONSE
Breast Cancer Therapy – early 2000’s

- 70% cured by surgery and radiotherapy
- 70% of them are offered CTX
- 30% develop distant metastases
- 90% of them are offered CTX

Node negative BC

Overtreatment

High socio-economical burden

Undertreatment

right time. Now.
70 Gene Prognosis Signature - MammaPrint

MammaPrint: ‘07 FDA cleared IVDMIA for prognosis assessment technology – microarray

van’t Veer et al., Nature 415, p. 530-536, 2002
MINDACT Trial: Study Objective

• **Microarray In Node** negative and 1-3 positive node
  **Disease may Avoid ChemoTherapy** (**MINDACT**)

• Designed to provide evidence for the clinical utility of MammaPrint:
  • Use of the **70-gene signature** (**MammaPrint**)
  • In addition to **standard clinical-pathological criteria**

• **Goal:** more accurate selection of patients for adjuvant chemotherapy

• “Precision Medicine/Personalized Medicine”
MINDACT Primary Test and End Point

Primary endpoint:
• Distant Metastasis Free Survival (DMFS) at 5 years

Primary test:
• To assess whether patients with clinical high risk features and a genomic Low Risk profile who did not receive CT would have a 5-year DMFS of ~95%.
  – A non-inferiority boundary of 92% (lower limit confidence interval)

NEJM, 2016
MINDACT Trial Primary Test Analysis: Clin-High / MP Low group- No Chemo (100% compliance)

Primary Test Population, C-high / G-low tumors:
- 58% >2cm
- 93% Grade II or III
- 48% LN+ 1-3
- 98% HR+

Primary Endpoint met!

- 5-Year DMFS for the C-high / G-low (MP Low) group with no CT= 94.7%
  (CI: 92.5 – 96.2%).
- Excludes 92%, positive outcome met.

NEJM, 2016

Piccart M. AACR Podium Presentation, April 18th, 2016
MINDACT Secondary Test

Secondary endpoint:

• Distant Metastasis Free Survival of noCT vs CT

Secondary test:

• To assess significance of survival difference, added clinical benefit of chemotherapy, for patients with clinical high risk features and a genomic Low Risk

NEJM, 2016
Chemo efficacy in Clin-High / MP Low (DMFS)

DMFS: distant relapses deaths all causes

- No statistical difference between CT vs no CT arms
- Excellent survival with no chemotherapy for patients with clinically high risk features (94.4%)

Secondary Endpoint met!

NEJM, 2016
Personalized Healthcare - Precision Oncology

Science based targeted escalation and de-escalation

GOAL
- INCREASE EFFICACY
- REDUCE MORBIDITY

Screening
- Diagnosis
- Treatment

Personalized Intervention

Risk or Response
- Escalate
- De-escalate

I-SPY | The right drug. The right patient. The right time. Now.
Early Breast Cancer
High risk for recurrence – Unmet need

Standard Chemotherapy Only 20% Response
New Targeted Drugs Need Companion Diagnostics
use case: early stage breast cancer - high risk
(stage 2 and 3)
EU: 100K and US: 60K patients/year
Breast cancer molecular subtypes to predict response in a modern treatment landscape
lessons from ~1000 patients across 10 arms of the I-SPY 2 TRIAL

the ‘right bins’

Laura van ‘t Veer, PhD - University California San Francisco

I-SPY Scientists:
Christina Yau, Denise Wolf, Michael Campbell, Chip Petricoin, Julia Wulfkuhle, Mark Magbanua, Lamorna Swigart, Gillian Hirst
and Concept holder scientists

I-SPY 2 Trial PI’s:
Laura Esserman, UCSF
Don Berry, MDAnderson
Trial Sponsor: Quantum Leap
Healthcare Collaborative
Basic Principles of I-SPY Platform Clinical Trial

• Test new drugs where they matter most
  • **Early stage (primary diagnosis)** rather than metastatic disease

• Change the order of therapy: learn about response early in the course of care
  • Neoadjuvant setting (**systemic therapy before surgery**)
  • Primary Endpoint is pathology complete response to therapy (**pCR, evaluated at surgery**)

• Build an efficient engine to evaluate drugs, accelerate knowledge turns
  • Master Protocol, Adaptive Design

• Use imaging and biomarker guidance
  • Focus on the population of patients who are at high risk for EARLY recurrence
  • Insights about who responds to what agents
  • “Graduation” for efficacy = threshold predictive probability of success in next phase III trial

• Collaborative by Design:
  • FDA, IRBs, Pharma, Biotech, Academics, Community Cancer Centers, Advocates
The I-SPY 2 TRIAL Standing Platform for High Risk Early Stage Breast Cancer

- **Phase II, adoptively randomized neoadjuvant trial**
  - Phase II drugs added to standard chemo
  - High-risk disease: MammaPrint high risk, plus all HER2+
- **Shared control arm**
  - Standard neoadjuvant chemotherapy
  - HER2+ also gets standard of care for targeted agents
- **Simultaneous experimental arms**
  - Up to five
- **Primary endpoint**: pathologic complete response (pCR)
  - Defined as **no residual invasive cancer in the breast or lymph nodes**
- **Match therapies (adaptively) with most responsive subtypes**
  - Defined by **HR+/−, HER2+/−, MammaPrint High1/(ultra) High 2 (MP1/2) status**
- **Agents/combinations “graduate” for efficacy** = reaching >85% predictive probability of success in a subsequent phase III trial in the most responsive patient subset (HR/HER2/MPh1-2)

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* HER2 positive participants will also receive Trastuzumab. An investigational agent may be used instead of Trastuzumab.
I-SPY 2 Participating Sites

18 Sites Open and Enrolling, Opening 3 sites Q1 and 3 sites Q2 2020

Status as of January 31st, 2020
Trial Patient Enrollment Overview

Registered (n=2924)
- Actively Being Screened (n=33)
- Did Not Proceed to the Treatment Phase (n=1205)

Randomized (n=1686)

Completed Surgery (n=1508)

Status as of January 31, 2020
I-SPY 2 Investigational Drugs

**Anti-HER family signaling**
- Neratinib
- trastuzumab/pertuzumab
- TDM1/pertuzumab
- trastuzumab/patritumab

**Anti-IGF1R**
- Ganitumab

**AKT inhibition**
- MK2206

**Unfolded protein response inhibition**
- ganetespib (HSP90i)

**PARP inhibition + DNA damage**
- Talazoparib/irinotecan
- veliparib/carboplatin

**Immune checkpoint inhibition**
- Pembrolizumab

**TIE1/2 inhibition**
- AMG386

**Hallmarks of Cancer**
- Sustaining proliferative signaling
- Evading growth suppressors
- Resisting cell death
  - (altered stress response)
- Deregulating cellular energetics
- Genomic instability & mutation
- Inducing angiogenesis
- Activating invasion & metastasis
- Enabling replicative immortality
- Tumor-promoting inflammation
- Avoiding immune destruction

**Drugs Developed Targeting Hallmarks of Cancer**
I-SPY 2 Framework:
Biomarkers Guide Enrichment of Drug Arm with Responding Subtype

**Biomarkers:**
- Imaging
- Pathology
- Molecular Biology
  - 8 subtypes (current) by:
    - Hormone Receptor +/−
    - HER2 +/−
    - MammaPrint high 1/2

**Adaptive Randomization**

**Diagnosis**
MRI to assess tumor volume
Biopsy to assess biological subtype

**Drug Treatment**
Serial MRI (volume change) & Pathology (pCR) at surgery informs adaptive randomization by biological subtype

**Surgery**

**Efficacy endpoint:**
pCR, pathological Complete Response, on surgery specimen
1) Randomization of a drug starts randomly across 8 subtypes
2) Adaptive randomization to 1 of 5 ‘investigational’ arms based on serial MRI response and surgical endpoint seen for each of 8 subtypes (hormone receptor +/-, HER2 +/-, MammaPrint-high 1 or 2), plus 1 in 5 to control
MRI: Rapid Response in TN-BC to Veliparib, Paclitaxel, Carboplatin

MRI imaging volume change at every time point informs adaptive randomization

11/4/2010  
11/22/2010

At Diagnosis

12/13/2010 – post Week 3

After 3 cycles/weeks of treatment

04/18/2011 – pre-surgery

At treatment completion 6 months

Example of 3 week response - (full treatment is ~ 6 months! De-escalate?)
3) **Drug Graduation** based on 10 signatures (single or combinations of 8 subtypes) and 85% success in Phase 3
Timeline of Investigational Drugs and Graduating Signatures
Biomarkers Guide Enrichment of Drug Arm with Responding Subtype-Signature

17 drugs entered the trial:
- 12 completed
- 6 graduated w signatures
- 4 dropped (no increase efficacy)
- 2 halted/stopped (toxicity)
- 1 control arm
- 5 arms ongoing

Overall for graduating signatures for each Drug
the pCR response rates
Doubled or Tripled
pCR at surgery relates to survival regardless of treatment
10 treatment arms, 950 patients, median 3.8 yr follow-up

Event-free Survival

Distant Recurrence-free Survival

3 yr survival (EFS)
pCR is 95%
vs.
nopCR 78%

Across high-risk subtypes, agents

pCR = pathological complete response at surgery

Figure 3: Association between pCR and Survival Outcomes (A) Kaplan Meier curves of EFS by pCR; (B) Kaplan Meier curves of DRFS by pCR.

Yee et al, SABCS 2017; DeMichele et al, ENA 2018; manuscript in revision
## I-SPY 2 is a biomarker rich trial

### STANDARD
- Level 1 evidence
- FDA cleared or approved or IDE filed
- **Used in clinical decision**

HR, HER2, MammaPrint, MR volume

### QUALIFYING
- Level 2 evidence
- Have existing evidence for response prediction
- Based on mechanism of action
- Evaluated in CLIA setting
  - Agilent 44K array (FDA IDE)
  - Phospho-protein array (CLIA)
  - DNA mutation panel (CLIA)
- **Hypothesis testing**
  - Pre-defined biomarkers

### EXPLORATORY
- Biomarker discovery
- **Hypothesis generation**

Example: Veliparib (PARP-inh)/Carboplatin (tested in HER2neg subtypes)

Adaptive Biomarker Subtypes indicated:
- **response** in Triple-Negative (TN) Breast Cancer
- **no response** in Hormone receptor positive Breast cancer (HR+/HER2-)

and the adaptive randomization enriched the VC arm and graduated in TN Breast Cancer

**VC response ~50%!**

Example: Pembroluzimab x4 (anti-PD1) (tested in HER2neg subtypes)
Adaptive randomization indicated:
response and graduated in three biomarker subtypes/signatures

- HER2-
  - **Control:** 17%
  - Pembrolizumab: 44%
  - ~ doubled response
  - all HER2-

- HR-HER2-
  - **Control:** 13%
  - Pembrolizumab: 60%
  - ~ tripled response
  - TN

- HR+HER2-
  - **Control:** 13%
  - Pembrolizumab: 30%
  - ~ doubled response
  - HR+/HER2-

Nanda et al ASCO 2017
Within one subtype response to multiple drugs, though not everyone

### HR-HER2-

- **Ot**: 13% 14% 14%
- **VC**: 17% 14% 14%
- **N**: 15%
- **Total**: 17%

### HR+HER2-

- **Ot**: 22%
- **VC**: 30%
- **N**: 38% 37% 38% 39% 60%
- **Total**: 51%

### HR-HER2+

- **Ot**: 17%
- **N**: 17% 35% 46% 37% 44%
- **Total**: 62%

### HR+HER2+

- **Ot**: 33%
- **N**: 33% 44% 44% 62% 64%
- **Total**: 63%
Response Biomarkers to improve response prediction (Biomarkers level 2)

• Important to get every patient to pCR (increased probability of survival)
• I-SPY 2 randomizes by 8 subtypes (HR +/-, HER2 +/-, MammaPrint High1/High 2; $2^3=8$)

• How can biology further identify responders?

• I-SPY 2 tests ‘Qualifying Biomarkers’, which have existing evidence for response prediction (Biomarkers level 2)
  • Biology of Targeted agent, eg DNA repair deficiency, HER2 signaling, immune signatures, biology subtyping (gene expression, phosphor protein, some DNA mutation)

• Presented here: Individual and Integrated Qualifying Biomarkers
I-SPY 2 is a biomarker rich trial

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  - Pre-defined biomarkers
  - Pre-specified rigorous statistical framework

**EXPLORATORY**
- Biomarker discovery
- Hypothesis generation
Our Pre-specified Qualifying Biomarker Evaluation Methodology is a 3-Step Process

**Step 1:**
Assess relative performance in Exp and control arms

1) Is the biomarker associated with response in experimental arm?
2) Is the biomarker associated with response in the control arm?
3) Is there a treatment x biomarker interaction of $p < 0.05$?

**Step 2:**
Evaluate biomarker in context of graduating signature

Is there a treatment x biomarker interaction of $p < 0.05$ adjusting for subtype?

**Step 3:**
Bayesian modeling of estimated pCR rates

Within each biomarker-defined subset of interest:
1) What is the estimated pCR rates in the experimental and control arms?
2) What is the predictive probability of success in a 300-patient Phase 3 trial?

**PASS – STEP 1**
Biomarker ready for use with validated threshold for drug assignment

**PASS – STEP 2**
Biomarker ready for further second validation

**PASS – STEP 3**
Validates for clinical utility

**Qualifying Biomarker Process:**
Qualifying Biomarkers for all I-SPY 2 Drugs

**Top Qualifying predictive biomarkers**
- Gene expression 44K array
- Phospo-protein array
- (few) DNA NGS panel

**Exploratory by platform**
- IHC multiplex

10 drugs/combinations tested, plus control

5 pathways/hallmarks:
- DNA repair deficiency
- HER2
- Immune
- AKT
- Angiogenesis
Evaluation by receptor subtype: TNBC

- BOTH veliparib/carboplatin (VC) combination therapy AND pembrolizumab (P) graduated in the triple negative (TN) subset
  - Plan to include pembro in Block B; carbo in Block B; more immunotherapy & DRD agents in Block A

- Who should get what and can we prioritize based on biomarkers to improve outcome?

Platinum-based

- 51% estimated pCR rate in VC (vs 26% in control)

Immunotherapy

- 60% estimated pCR rate in P (vs 22% in control)

I-SPY | The right drug. The right patient. The right time. Now.
Immune and DRD biomarkers in TNBC, viewed individually

**DRD+ patients have a high estimated pCR rate to VC (79%)**

**Immune+ patients have a high estimated pCR rate to Pembrolizumab (87%)**
Which drug should be prioritized for whom in TNBC?

- **Immune+/DRD+**
  - Positive for both biomarkers
  - Lower pCR in both (22%, 34%)

- **Immune-/DRD-**
  - Biomarker negative
  - Higher pCR in VC (64%)

- **Immune-/DRD+**
  - Higher pCR in VC (64%)

- **Immune+/DRD-**
  - Higher pCR in Pembro (90%)

- **Immune+/DRD+**
  - High pCR in Pembro (84%) and VC (83%)

- **Immune-/DRD-**
  - Lower pCR in both (22%, 34%)

- **Immune+/-DRD-**
  - Lower pCR in both (22%, 34%)
Immune and DRD biomarkers associate with pCR in HR+HER2- as well, though prevalence differs

- Immune-/DRD- (Neither) 56%
- Immune-/DRD+ (VC) 5%
- Immune+/DRD+ (Pembro or VC) 27%
- Immune+/DRD- (Pembro) 12%

HR+/HER2- Pembro4 graduated with 30% estimated pCR rate in P (vs 13% in control)

HR+/HER2- Veliparib/Carbo did not graduate, but qualifying biomarker DRD+ patients showed increased response

- 39% are Immune+
- 17% are DRD+

HR+HER2- (I-SPY 2 all MammaPrint High Risk)
How do we integrate all this information?

(Our current status based on pre-treatment tumor biomarkers)
From Breast Cancer Receptor Subtypes to Drug Sensitivity Subtypes

~1000 I-SPY 2 patients
Pre-treatment biopsy

Denise Wolf, Christina Yau ao
Integrated Qualifying Biomarkers
~1000 I-SPY 2 patients -> 5 Drug Sensitivity subtypes

Receptor subtypes

Hybrid-5
Drug Sensitivity subtypes

S1 (27.5%)
S3 (40%)
S2 (7.4%)
S4 (18.7%)
S5 (6.1%)

Denise Wolf, Christina Yau ao
Prioritizing Agents per Hybrid Response Subtype

~1000 I-SPY 2 patients
Pre-treatment biopsy

Receptor subtypes

Hybrid-5 Response subtypes

S1 (27.5%)
S2 (7.4%)
S3 (40%)
S4 (18.7%)
S5 (6.1%)

What to do here?

What to do here?

What to do here?

for each Hybrid subtype based in I-SPY 2 responses

some have multiple options, some not yet

hallmarks for other agents?

for the right drug. The right patient. The right time. Now.
From Breast Cancer Receptor Subtypes to Drug Response Subtypes

~1000 I-SPY 2 patients
Pre-treatment biopsy: standard receptor subtypes (left) to best qualifying biology (right), based on 24 per drug qualified biomarkers combined (work in progress)

Denise Wolf, Christina Yau ao

Increase of Response prediction:
1) Standard Chemotherapy
   No subtype selection 20-25%
2) I-SPY 2 Standard chemo with targeted agents on optimal receptor subtype 40%
3) Predicted Response subtypes w preferred targeted agent
   estimated ~60-70% (ongoing)
4) Next: Test in I-SPY 2.2

4 Receptor subtypes
5 Response subtypes
# I-SPY 2 is a biomarker rich trial

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  - Pre-defined biomarkers
  - Pre-specified rigorous statistical framework

## EXPLORATORY

- Biomarker discovery
- **Hypothesis generation**
Circulating Tumor DNA (exploratory biomarker): Personalized 16 tumor mutated specific fragments
Serial liquid biopsies: MK2206 (Akt-inh) plus controls

Signatera platform – Natera Inc

Mark Magbanua et al, SABCS 2018/2019, submitted
Evolution to I-SPY 2.2

Permit de-escalation or escalation of therapy as needed

- Adapt treatment on the individual patient level to maximize pCR and further increase survival probability
- Make use of MRI volume change early
- Guidance by ctDNA *(high need: validation, sequencing)*
- Immune blood marker changes *(high need: sequencing)*
- Integrate all our qualifying biomarker knowledge to guide drugs
  - 24 predictive biomarkers across 10 drugs evaluated on 1000 I-SPY 2 patient
- Introduce ‘window of opportunity’ drug treatment in trial to find signal of response
I-SPY 2 Platform Trial: Learning, Innovating, and Evolving

• The I-SPY 2 trial
  • adaptive randomization of targeted drugs to responding subtypes optimizes complete response and survival for high risk breast cancer

• Patient Centered
  • the best drug for their subtype

• Maximize chance of pCR and cure for each patient
  • pCR results in 95% 3 yr disease-free survival (no-pCR 76-79%)

• Increase chance of pCR and cure for the high risk population
  • Learn, approve drugs and combinations that are effective and less toxic

• A design that patients like, that investigators like, where industry will participate - speeds the chance that patients will survive

• Advances regulatory science
I-SPY 2 Data and Biospecimen Access Process

• Platform Data available for access proposals
• Biospecimen available for proposals

By Data Access and Publication Policy and Concept sheet submission
I-SPY 2™ TRIAL

Acknowledgements

WORKING GROUP CHAIRS

<table>
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<th>Study Pls:</th>
<th>Imaging:</th>
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<th>Safety:</th>
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Swedish: Erin Ellis
UChi: Rita Nanda
UCSD: Anne Wallace
UCSF: A. Jo Chien
UMinn: Doug Yee
UPenn: Amy Clark
USC: Julie Lang
Yale: Tara Sanft
Emory: Jane Meisel
Wake: Alexandra Thomas

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Quantum Leap Healthcare Collaborative

QLHC Board, James Palazzolo, Adam Asare, Kathryn Watson-Feiner, Tracey Heather, Smita Asare, Paul Henderson, Dan Dornbusch, Karyn DiGiorgio, Brendan Raven

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Swigart, Gillian Hirst, Sara Venters, Aye Aye Ma, Elizabeth Bergin, Mark Magbanua & Collaborators

Imaging Lab:
Wen Li, David Newitt, Jessica Gibbs, Melanie Regan, Margarita Watkins

Data Analysis, Data Management & IT:
Ashish Sanil (Berry Consultants), Christina Yau, Amrita Basu, Garry Peterson, Amy Wilson, Sruthi Samineni, Nick O’Grady

PRIOR COLLABORATORS and STAFF

Andres Forero-Torres, Larissa Korde, Rashmi Murthy, Donald Northfelt, Qamar Khan, Kirsten Edmiston, Rebecca Viscusi, Barbara Haley, Amelia Zelnak, Julie Sudduth-Klinger, Nancy Lisser, Meredith Buxton, Melissa Paolini, and Julia Lyanderes

Thank you to the remarkable patients and families, our amazing advocates, all of the investigators, staff, and our DSMB for supporting the trial

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I-SPY 2 Participating Organizations and Funders

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"Here are my genes..."