



THE CLARITY FOUNDATION

Transforming treatment and improving survival for ovarian cancer patients



March 25, 2012



Overview

- **Introduction to Clarity Foundation**
- **Clarity profiling panel**
- **Interpretation of results utilizing the Diane Barton Database**
- **Case studies**
 - **Patient in second recurrence that went on to receive chemotherapy**
 - **Patient in first recurrence that went onto a clinical trial of a molecular-targeted agent combined with chemotherapy**
 - **Recurrent vs primary specimens**
- **Utilizing the TCGA groupings for selection of clinical trials**
 - **Case study of BRCAness**
- **Q&A**

The Clarity Foundation launched as a non-profit organization in 2008 to:

- **Bring molecular profiling to the forefront of ovarian cancer diagnosis and treatment**
- **Assist doctors in prioritizing therapy for recurrent ovarian cancer informed by their patient's tumor molecular profile**
- **Expedite the clinical development of novel targeted agents for ovarian cancer**
- **Increase the probability of success by utilizing molecular profiling to select patients for clinical trials**

Leading advisors and scientific findings presented

Scientific Advisory Board	
Beth Karlan, MD, Chair	Cedars Sinai & UCLA Medical Center
Doug Levine, MD	Memorial Sloan Kettering Cancer Center
Johnathan Lancaster, MD	Moffitt Cancer Center
Julie Cherrington, PhD	Pathway Therapeutics
Ursula Matulonis, MD	Dana Farber Cancer Center & Harvard Medical School
Deb Zajchowski, PhD	Clarity Foundation Scientific Director

Mol Cancer Ther; 11(2) February 2012:
Treatment-related protein biomarker expression differs between primary and recurrent ovarian carcinomas DA Zajchowski, BY Karlan and LK Shawver

ASCO 2011: Expression Profiles in Matched Primary and Recurrent Ovarian Carcinomas
 DA Zajchowski, BY Karlan and LK Shawver,

AACR 2011: Molecular Profiling in Recurrent Ovarian Cancer Patients DA Zajchowski, C Bentley, J Gross, BY Karlan and LK Shawver

AACR 2010: Selecting Patients for Ovarian Cancer Clinical Trials by Profiling Tumors against a Broad Panel of Molecular Markers
 DA Zajchowski, J Gross, BY Karlan, K Bloom, D Loesch, A Alarcon and LK. Shawver

Accomplishments in less than four years

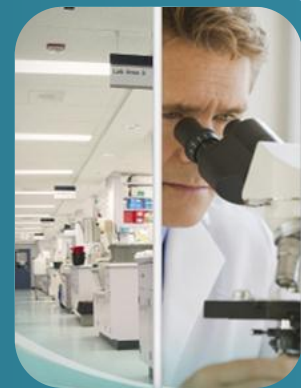
- **Developed diagnostic protocols with latest technologies and input from expert advisors**
- **Created the Diane Barton Database, a platform for:**
 - **Compiling test results from multiple labs**
 - **Tracking patient outcomes**
 - **Establishing assay cut-points to prioritize treatment options**
 - **Utilizing markers for clinical trial enrollment**
 - **Comparing tumor profiling results from patient to patient**
- **Formed web-based informational tools and patient support process**
- **Provided access to molecular profiling for ~200 women with ovarian cancer**

How we work

Oncologists
and Patients

Clarity Profiling Services:

- Physician and patient education
- Coordination with CLIA labs to test
- Secure database for patient data
- Data integration/ analysis
- Results reporting



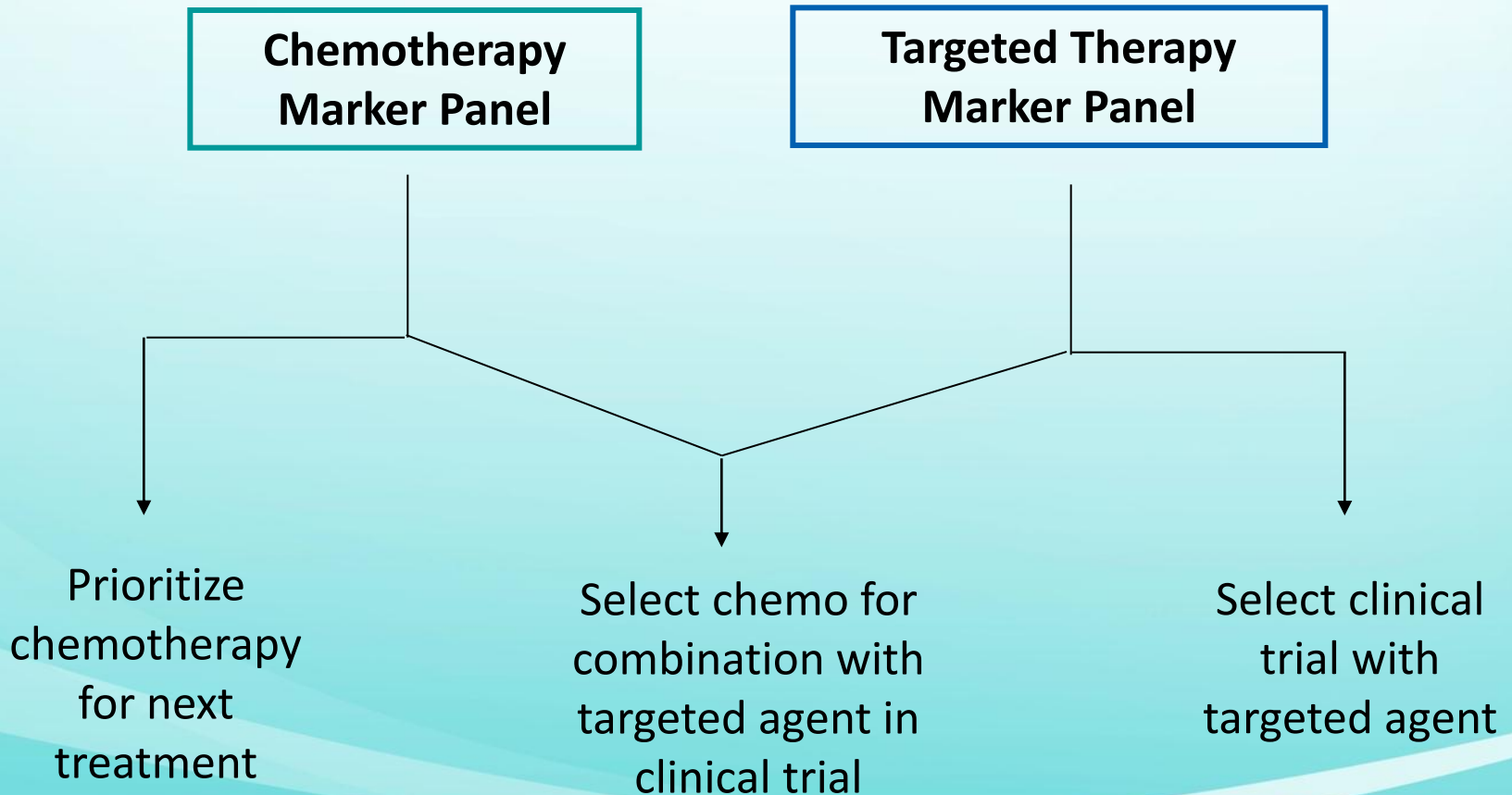
Medical team utilizes
molecular profiles to prioritize
therapeutic options

NCCN Guidelines for Epithelial Ovarian Cancer/Fallopian Tube Cancer/Peritoneal Cancer 2.2.2011

ACCEPTABLE RECURRENCE THERAPIES (1 of 2)¹

Agents	Cytotoxic Therapy	Hormonal Therapy	Targeted Therapy	Radiation Therapy													
Preferred Agents	<p><u>Combination if platinum sensitive</u> Carboplatin/paclitaxel (category 1)^{2,3} Carboplatin/weekly paclitaxel^{2,4} Carboplatin/docetaxel^{2,5,6} Carboplatin/gemcitabine^{2,7} Carboplatin/liposomal doxorubicin^{2,8} Cisplatin/gemcitabine^{2,9}</p> <p><u>Single-agent if platinum sensitive</u> Carboplatin⁷ Cisplatin⁷</p> <p><u>Single-agent non-platinum based if platinum resistant</u> Docetaxel¹⁰ Etoposide, oral¹¹ Gemcitabine^{12,13} Liposomal doxorubicin^{12,13} Paclitaxel, weekly¹⁴ Topotecan¹⁵</p>		Bevacizumab														
Other Potentially Active Agents	<p><u>Single Agents</u>¹⁶</p> <table border="0"> <tr> <td>Altretamine</td> <td>Paclitaxel</td> </tr> <tr> <td>Capecitabine</td> <td>Paclitaxel, albumin bound (nab-paclitaxel)</td> </tr> <tr> <td>Cyclophosphamide</td> <td>Pemetrexed</td> </tr> <tr> <td>Ifosfamide</td> <td>Vinorelbine</td> </tr> <tr> <td>Irinotecan</td> <td></td> </tr> <tr> <td>Melphalan</td> <td></td> </tr> <tr> <td>Oxaliplatin</td> <td></td> </tr> </table>	Altretamine	Paclitaxel	Capecitabine	Paclitaxel, albumin bound (nab-paclitaxel)	Cyclophosphamide	Pemetrexed	Ifosfamide	Vinorelbine	Irinotecan		Melphalan		Oxaliplatin		Anastrozole Letrozole Leuprolide acetate Megestrol acetate Tamoxifen	Palliative localized radiation therapy
Altretamine	Paclitaxel																
Capecitabine	Paclitaxel, albumin bound (nab-paclitaxel)																
Cyclophosphamide	Pemetrexed																
Ifosfamide	Vinorelbine																
Irinotecan																	
Melphalan																	
Oxaliplatin																	

Use tumor molecular profiles to prioritize choice of chemotherapy and/or clinical trial



Current panel of tests

Growth Factors/ Receptors

EGFR*	Her2*	IGF1R	c-Met	VEGF	PDGFR
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Cytoplasmic Signal Transducers and Apoptosis Regulators

K-ras**	B-raf**	PIK3CA**	PTEN	Bcl-2	Survivin	Cox-2
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Nuclear Signaling Proteins

Hormone Receptors/Transcription Factors				Cell Cycle			
ER	AR	PR	HIF1A	Ki67	p21	p16	Rb

Chemotherapy Sensitivity Markers

DNA Synthesis/Transcription			ECM
TLE3	Topo1	Top2A	SPARC

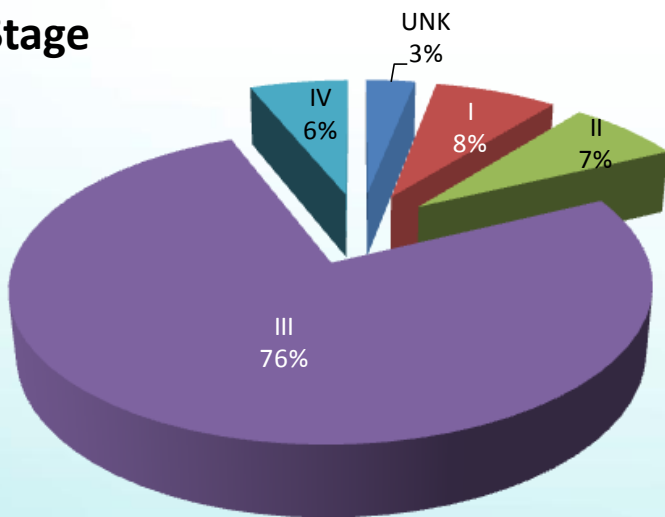
Chemotherapy Resistance Markers

Drug Transporters			DNA Repair/ Modification		DNA Synthesis/Cell Division		
BCRP	MRP1	MDR1/PGP	ERCC1	MGMT	RRM1	TS	TUBB3

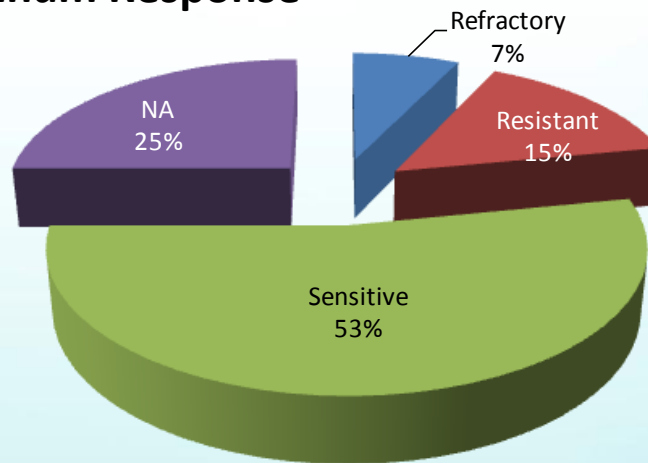
* DNA amplification ** DNA mutational analysis

Data stored and analyzed in Diane Barton Database

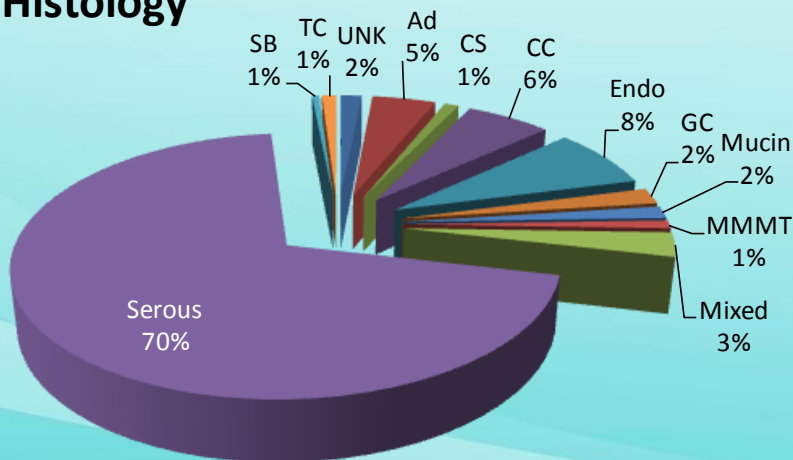
Stage



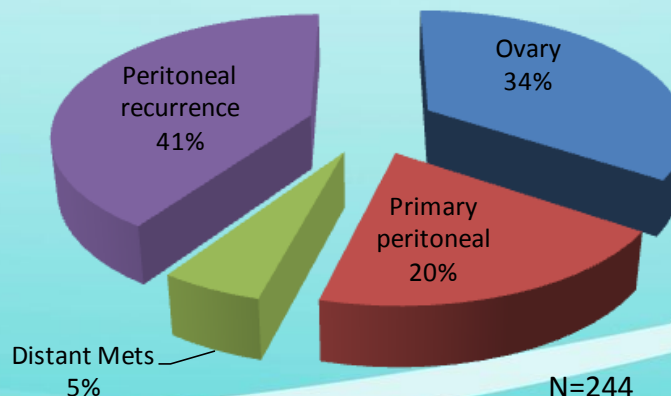
Platinum Response



Histology



Specimen Source



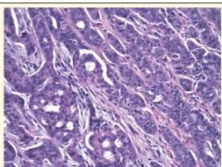
N=244

Example laboratory read-out for IHC

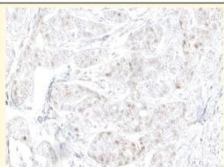
IHC Report

Omentum with tumor Tissue S08-47586-A2

10% Neutral Buffered Formalin (6-48 hrs)



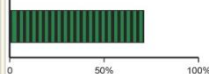
H&E



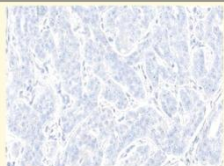
Review: Manual
Nuclei Stained: 70%
Intensity: 2
of fields: N/A

Clone: 1D5
AssayType: IVD
Reference Range
Negative < 1%
Positive >= 1%

POSITIVE



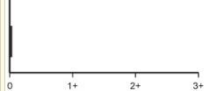
Estrogen Receptor



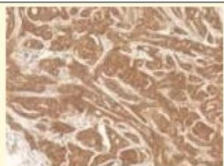
Review: Image
Score: 0
Fractional Score: 0.0
Max Score: 0.0

Kit: HercepTest
AssayType: FDA
Reference Range
Negative 0 - 1+
Equivocal 2+
Positive 3+

NEGATIVE



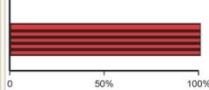
Her-2/neu



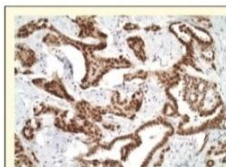
Review: Manual
Tumor Stained: 100%
Intensity: 3
of fields: N/A

Clone: 87
AssayType: HOMEBREW
Reference Range
Negative < 1%
Positive >= 1%

POSITIVE



eIF-4E

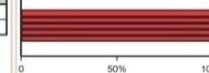


Androgen Receptor

Review: Image
Nuclei Stained: 100%
Intensity: 3
of fields: 3

Clone: AR441
AssayType: IVD
Reference Range
Negative <= 10%
Positive > 10%

POSITIVE

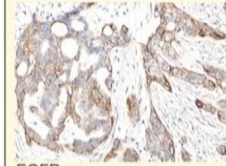
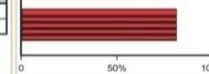


COX-2

Review: Manual
Tumor Stained: 80%
Intensity: 1-2
of fields: N/A

Clone: COX229
AssayType: IVD
Reference Range
Negative < 10%
Positive >= 10%

POSITIVE

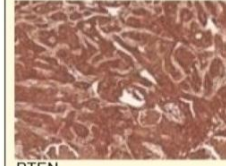
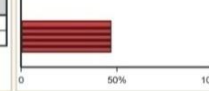


EGFR

Review: Image
Tumor Stained: 46%
Intensity: 2
of fields: 2

Clone: 31G7
AssayType: IVD
Reference Range
Negative < 1%
Positive >= 1%

POSITIVE

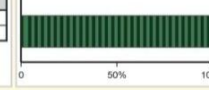


PTEN

Review: Manual
Tumor Stained: 100%
Intensity: 3
of fields: N/1

Clone: 6H2.1
AssayType: IVD
Reference Range
Loss of Expression < 5%
No Loss of Expression >= 5%

NO LOSS OF EXPRESSION



Data collected as histoscores

Patient Name
Date of Birth
Gender
Medical Record #
Master Accession
Case No



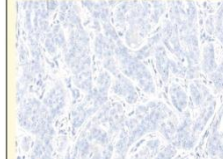
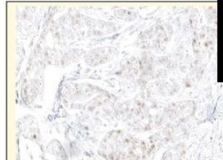
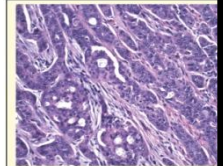
EGFR

Review:	Image
Tumor Stained:	46%
Intensity:	2
# of fields:	2

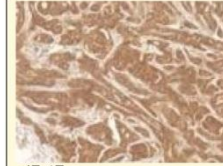
Histoscore =
% tumor stained x
Intensity = 92

Patient Name: The Clarity Foundation | Cedars-Sinai
MC: 241272
Referring Physician: Dr. Beth Karlan
Reporting Physician: Dr. Beth Karlan
Collection Time: 12/10/2008
Receipt Time: 12/16/2008 9:05:00 PM

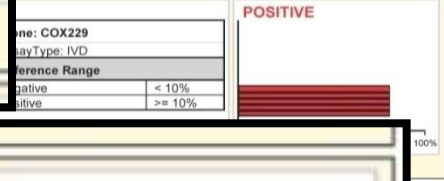
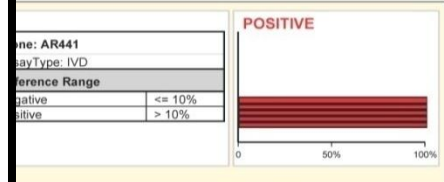
Omentum with tumor Tissue



Review: Image
Score: 0
Fractional Score: 0.0
Max Score: 0.0



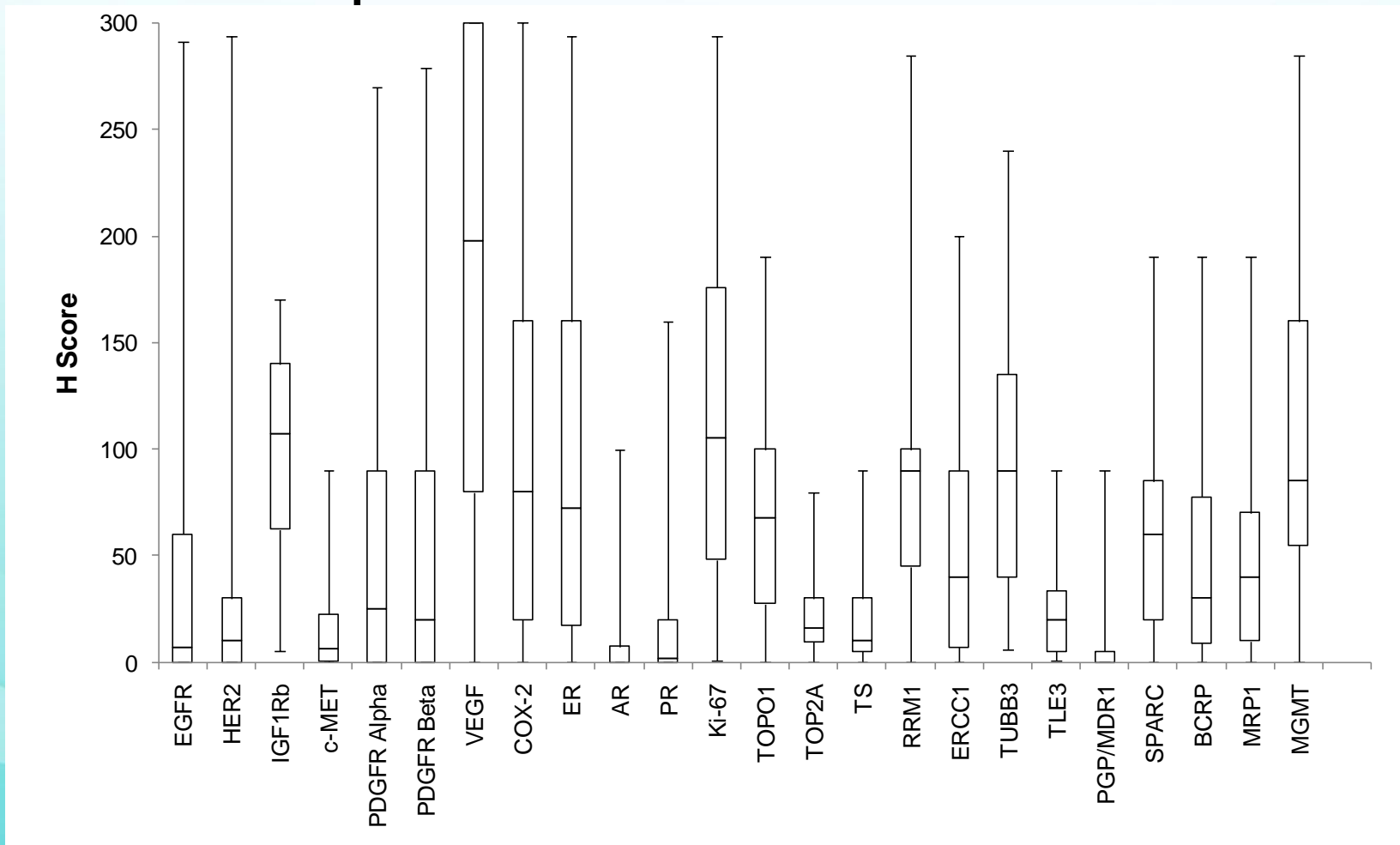
Review: Manual
Tumor Stained: 100%
Intensity: 3
of fields: N/A



Clone: 31G7	
AssayType: IVD	
Reference Range	
Negative	< 1%
Positive	>= 1%

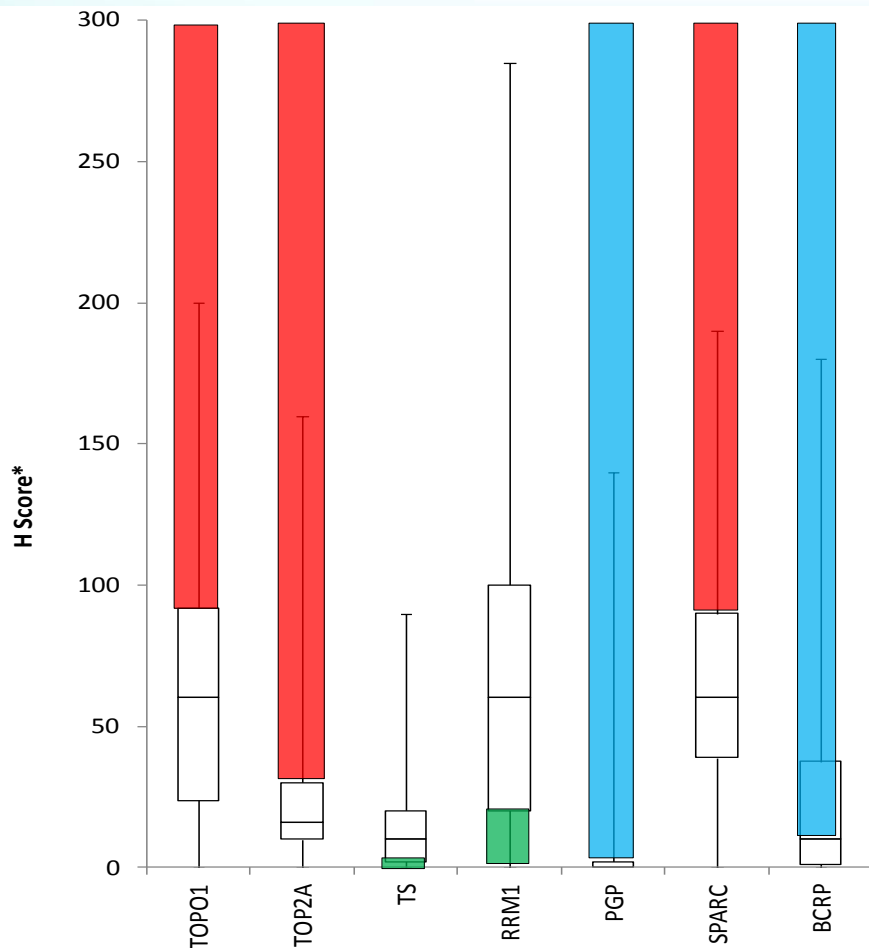
POSITIVE

Marker expression in all patients provides basis for interpretation of individual results



Box, inter-quartile range; line, median; whiskers, maximum and minimum values

Chemotherapy selection using published evidence and expression cut-offs derived from current database



High Topo I → Irinotecan, topotecan

High Topo II → Doxorubicin, etoposide

Low RRM1 → Gemzar

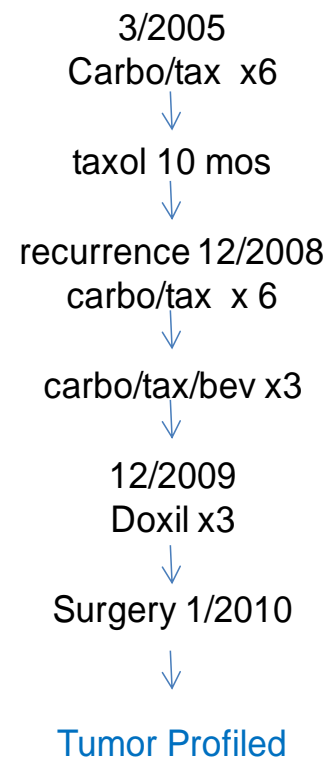
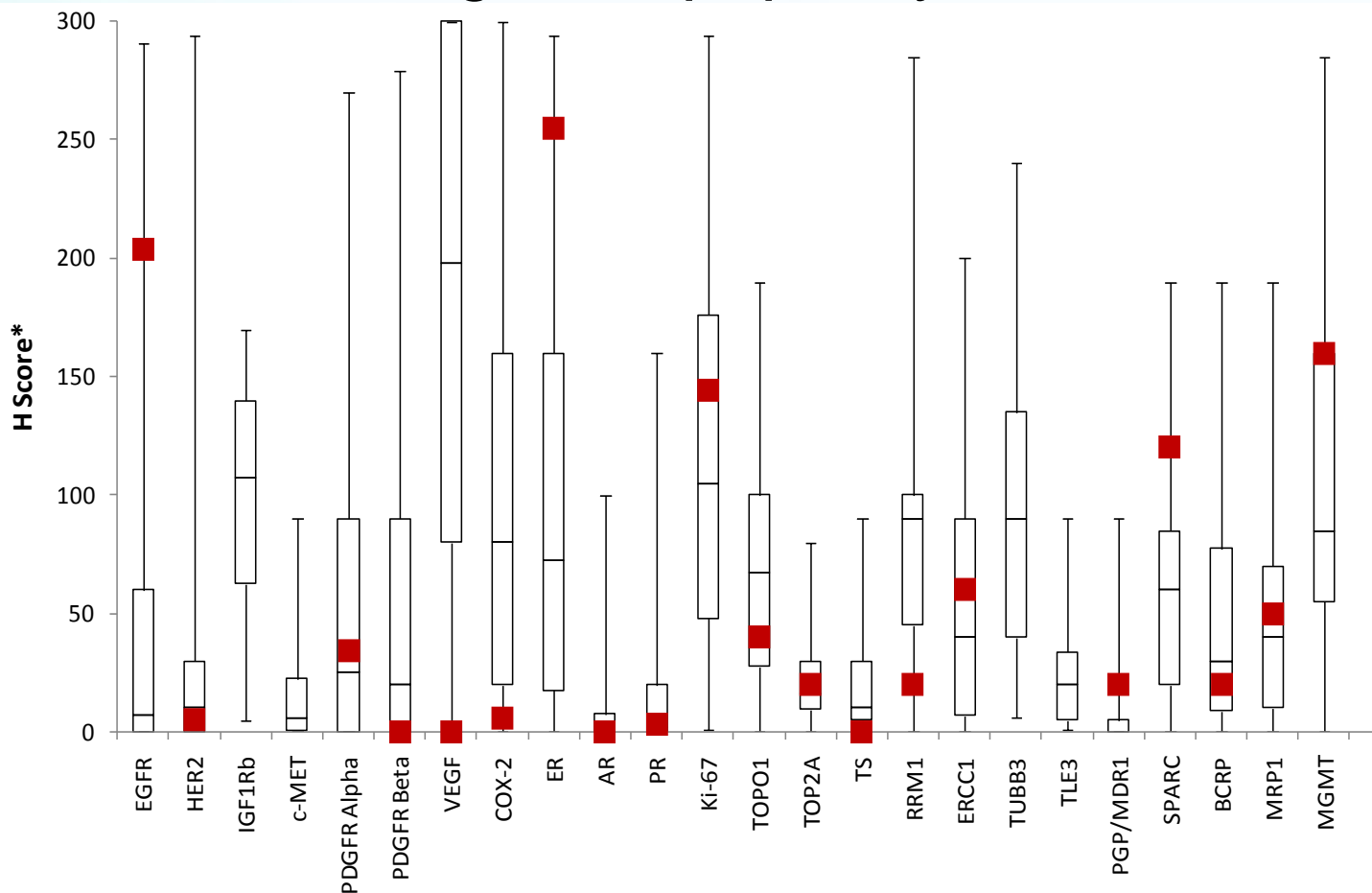
Low TS → Fluoropyrimidines

High SPARC → nab-Paclitaxel

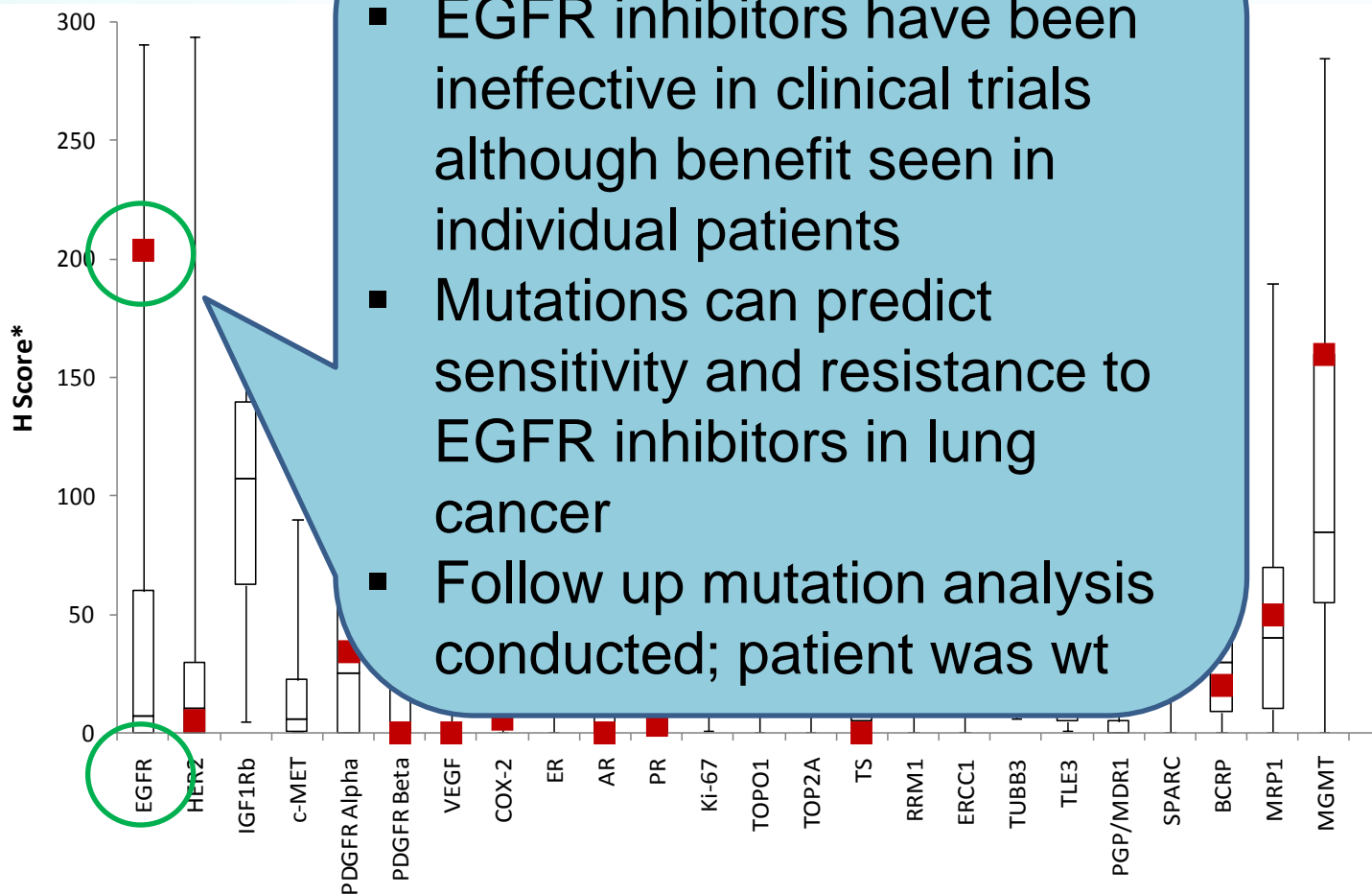
High PGP → No Taxane, no doxil

High BCRP → No Topotecan

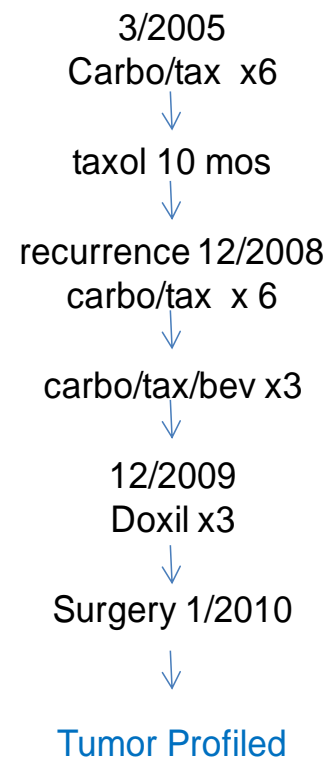
Case Study: profile for patient diagnosed in 3/2005 with stage IIB papillary serous carcinoma



Case Study: profile for patient diagnosed in 3/2005 with High EGFR (98th percentile) carcinoma

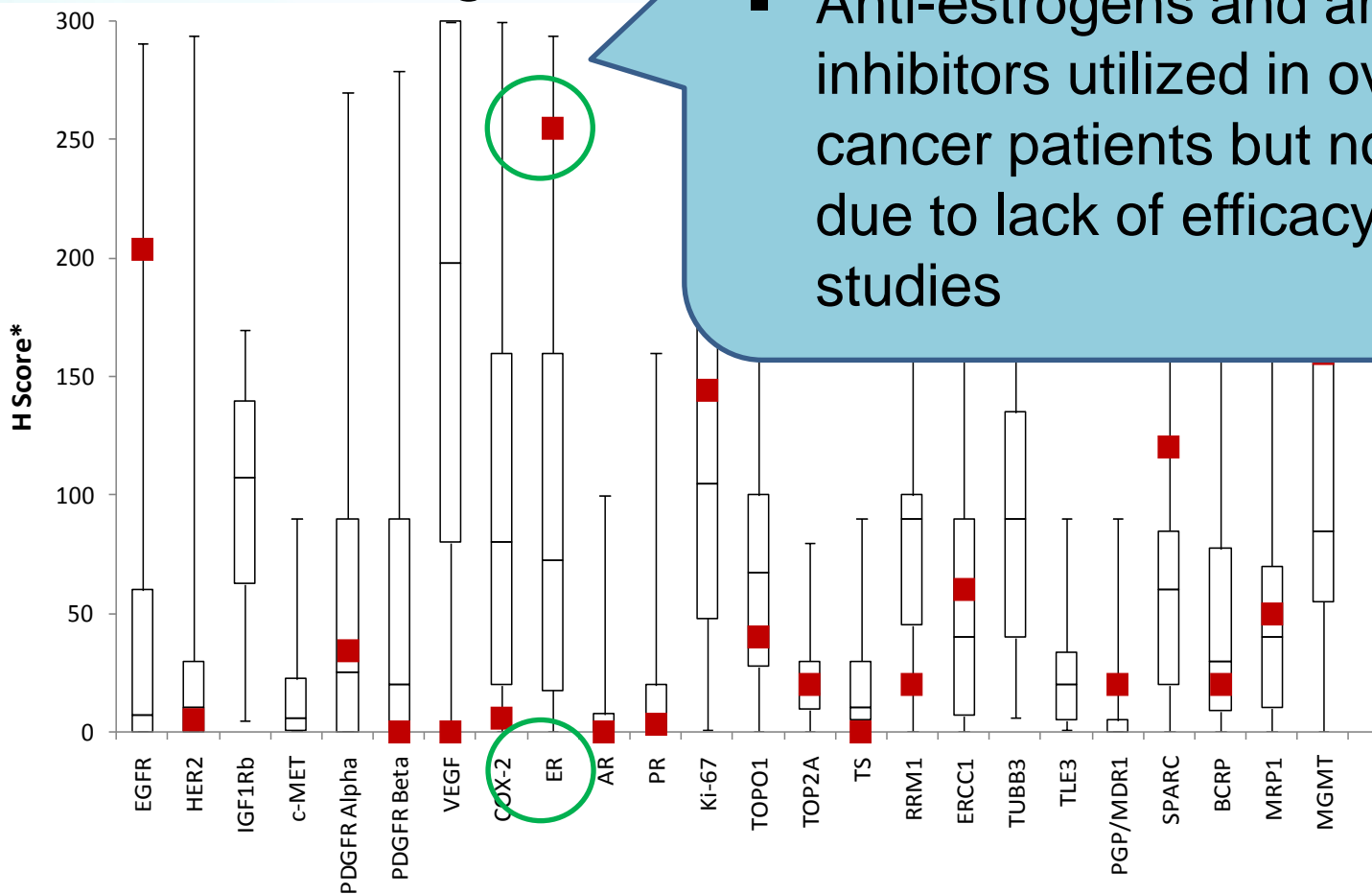


- EGFR inhibitors have been ineffective in clinical trials although benefit seen in individual patients
- Mutations can predict sensitivity and resistance to EGFR inhibitors in lung cancer
- Follow up mutation analysis conducted; patient was wt



Case Study: profile for patient diagnosed in 3/2005 with stage IIIB High ER

- Anti-estrogens and aromatase inhibitors utilized in ovarian cancer patients but not approved due to lack of efficacy in clinical studies

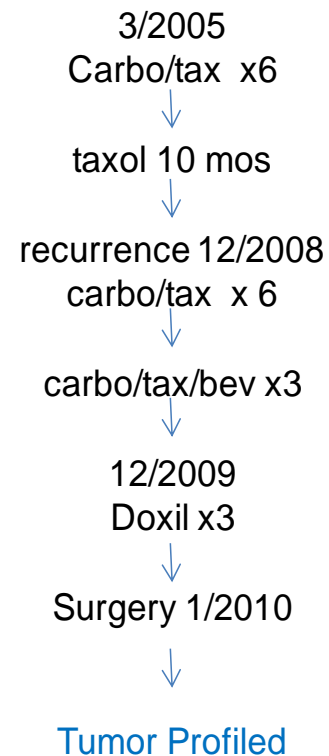
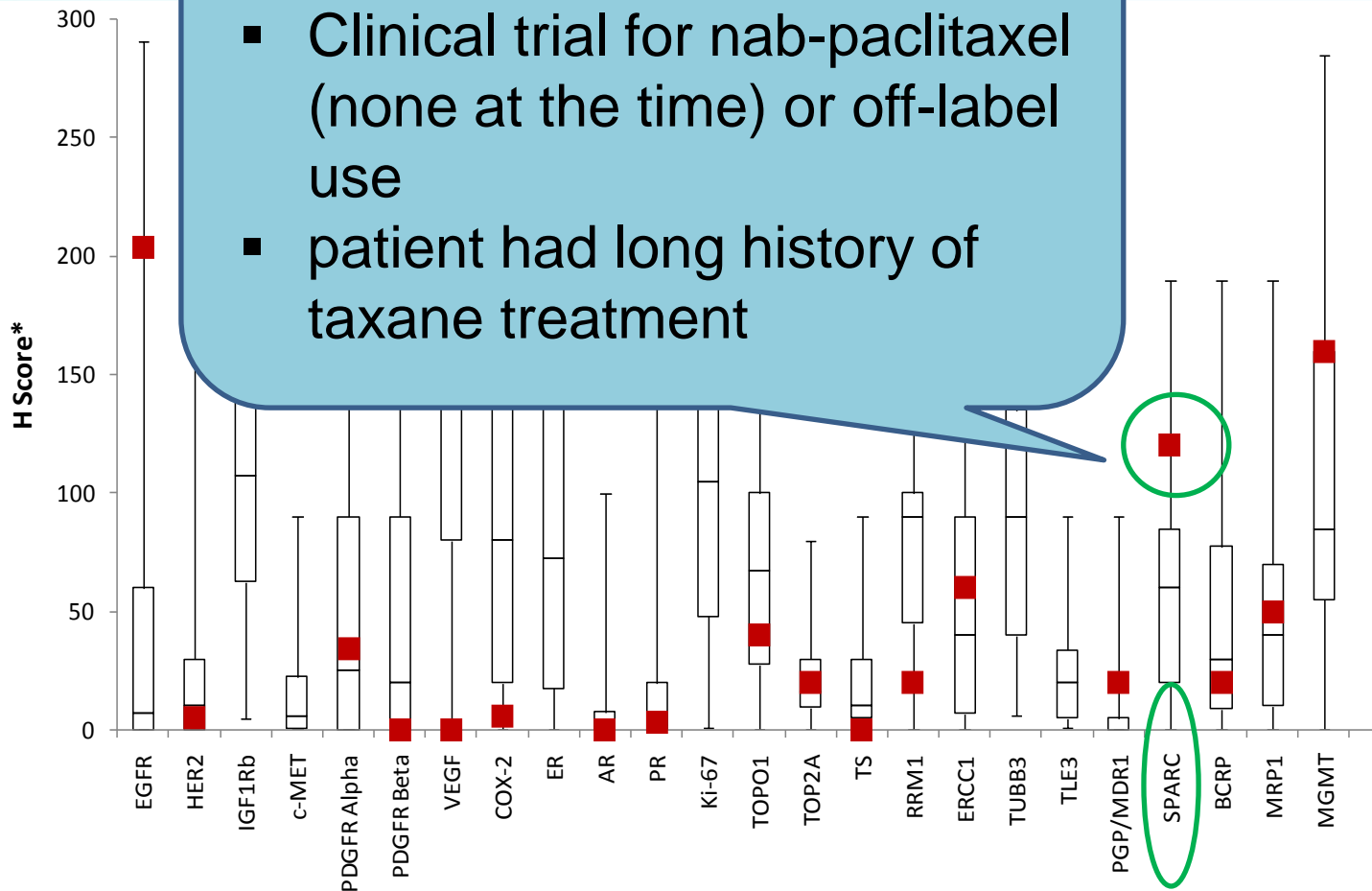


carbo/tax/bev x3
 ↓
 12/2009
 Doxil x3
 ↓
 Surgery 1/2010
 ↓
 Tumor Profiled

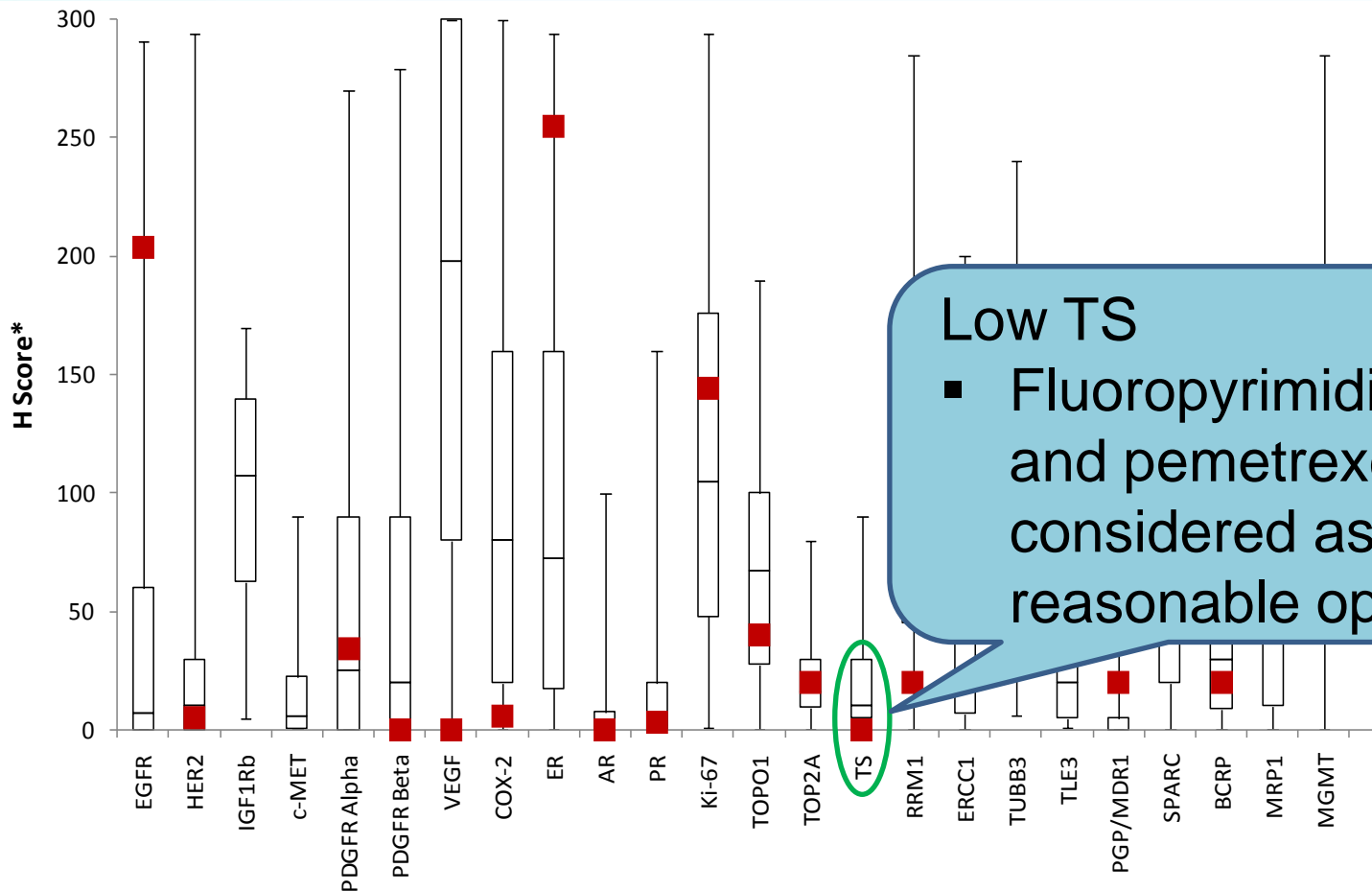
Case Study: profile for patient diagnosed in 3/2005 breast carcinoma

High SPARC

- Clinical trial for nab-paclitaxel (none at the time) or off-label use
- patient had long history of taxane treatment



Case Study: profile for patient diagnosed in 3/2005 with stage IIB papillary serous carcinoma



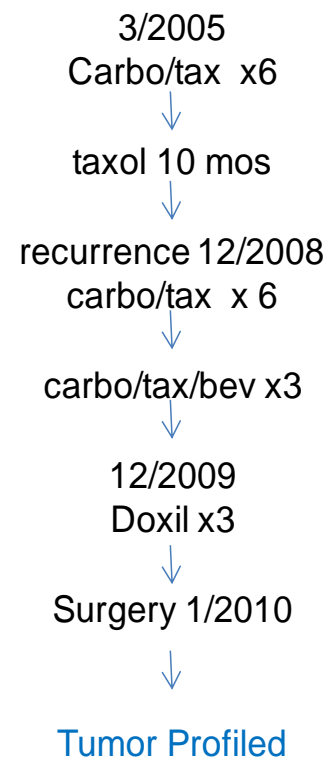
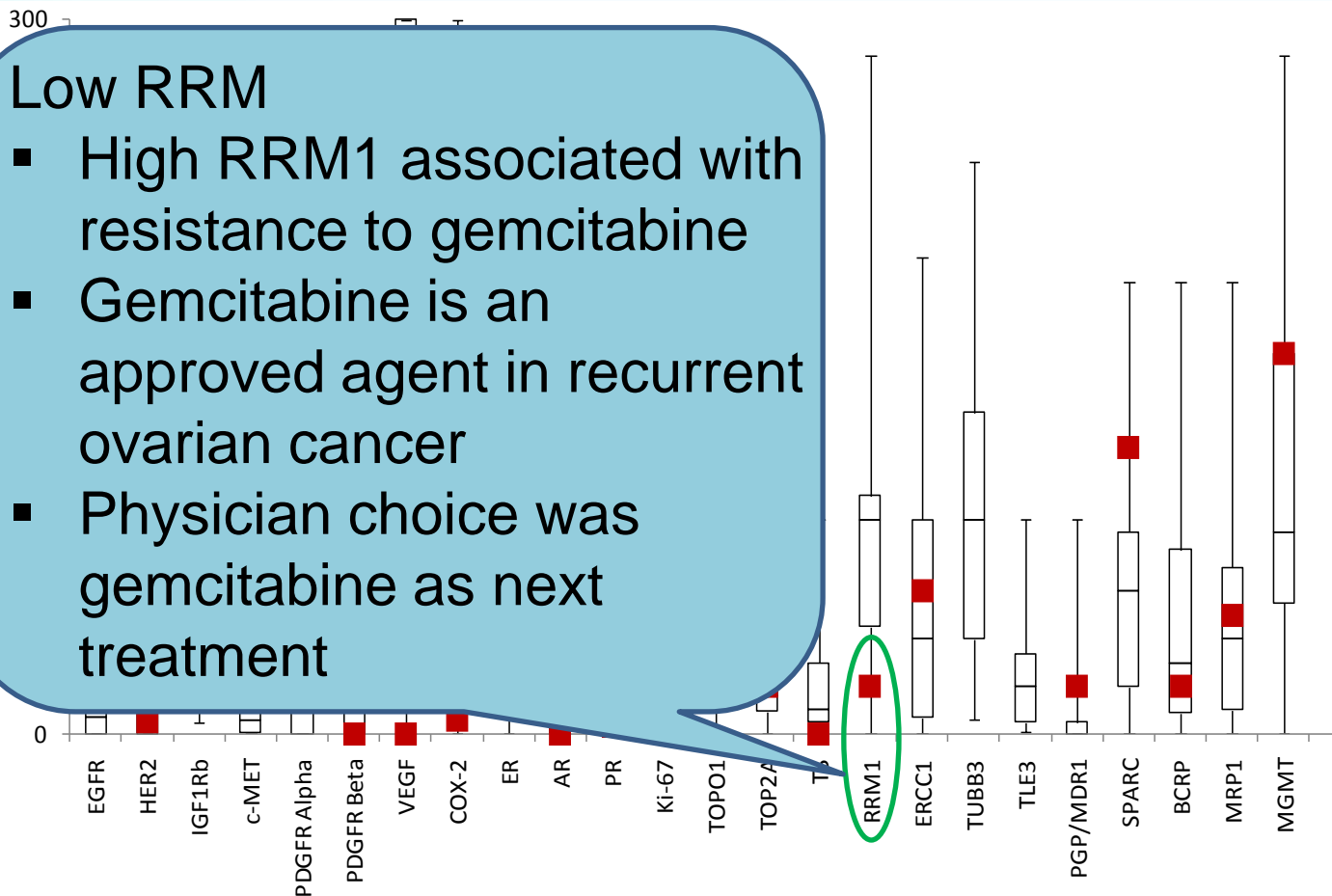
3/2005
 Carbo/tax x6
 ↓
 taxol 10 mos
 ↓
 12/2008
 Carboplatin/tax x6
 ↓
 tax/bev x3
 ↓
 2/2009
 Carboplatin/docetaxel x3
 ↓
 Surgery 1/2010
 ↓
Tumor Profiled

Low TS
 ■ Fluoropyrimidines and pemetrexed considered as a reasonable option

Case Study: profile for patient diagnosed in 3/2005 with stage IIIB papillary serous carcinoma

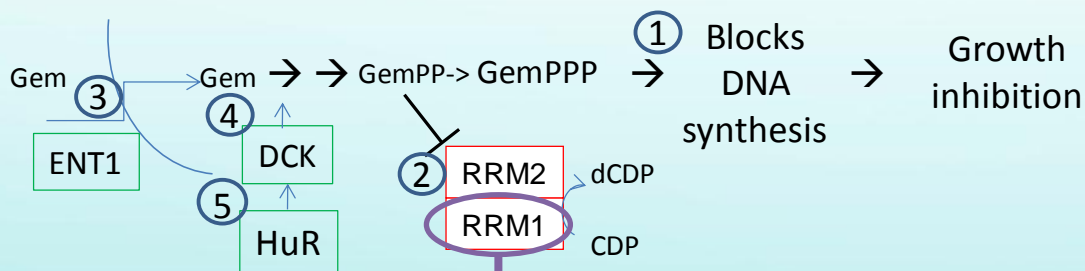
Low RRM

- High RRM1 associated with resistance to gemcitabine
- Gemcitabine is an approved agent in recurrent ovarian cancer
- Physician choice was gemcitabine as next treatment



Chemotherapy as targeted agents - clinical research evidence

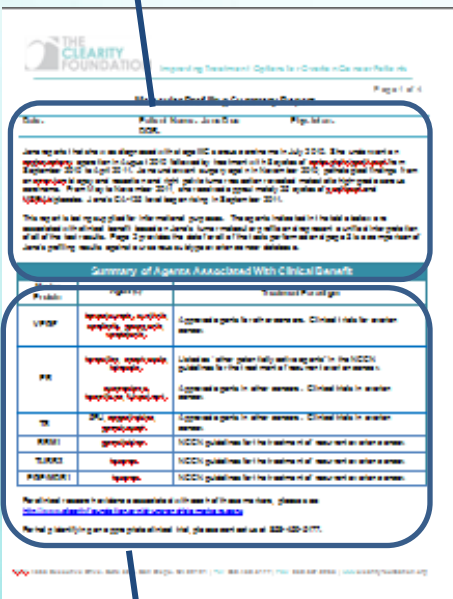
Drug	Mechanism of Action
Gemcitabine	Inhibits cell division by blocking DNA synthesis



Gemcitabine Resistance Markers				
Marker	Name	Biological Role	Evidence	References
RRM1	ribonucleotide reductase, regulatory subunit M1	Enzyme synthesizes deoxyribonucleosides from ribonucleoside precursors	High protein levels associated with poor response and outcome in pancreatic, biliary, and NSCLC patients after gemcitabine-based therapy	Akita, Zheng et al. 2009; Reynolds, Obasaju et al. 2009; Nakamura, Kohya et al. 2010

Summary of relevant patient medical history

Clarity molecular profiling summary report



Summary of Agents Associated With Clinical Benefits

Agent	Target	Approved Indication
LY2957449	PI3K	Approved for the treatment of recurrent and/or metastatic endometrial cancer.
PI3K	PI3K	Approved for the treatment of recurrent and/or metastatic endometrial cancer.
TRK	TRK	Approved for the treatment of recurrent and/or metastatic endometrial cancer.
PARP	PARP	Approved for the treatment of recurrent and/or metastatic endometrial cancer.
PARP	PARP	Approved for the treatment of recurrent and/or metastatic endometrial cancer.
PARP	PARP	Approved for the treatment of recurrent and/or metastatic endometrial cancer.

Summary of agents associated with clinical benefit extracted from pg 2

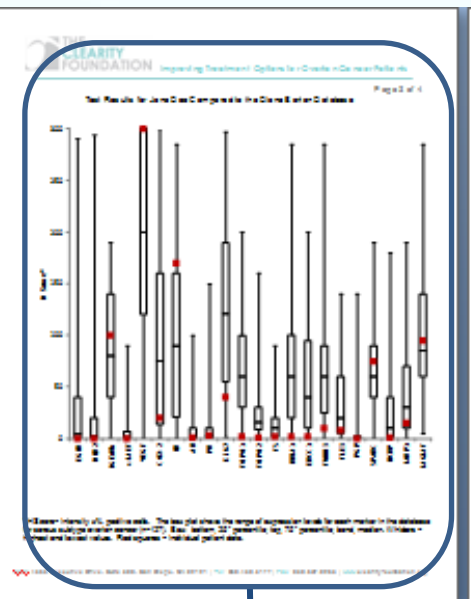


Molecular Profiling Data Summary

High Path in Tumor (HPI) (HPI Score)

Gene	Pathway	Interpretation
BRCA1	BRCA1 Pathway	Highly expressed in tumor
BRCA2	BRCA2 Pathway	Highly expressed in tumor
PTEN	PTEN Pathway	Highly expressed in tumor
PIK3CA	PIK3CA Pathway	Highly expressed in tumor
ERBB2	ERBB2 Pathway	Highly expressed in tumor
ERBB3	ERBB3 Pathway	Highly expressed in tumor
ERBB4	ERBB4 Pathway	Highly expressed in tumor
EGFR	EGFR Pathway	Highly expressed in tumor
HER3	HER3 Pathway	Highly expressed in tumor
HER4	HER4 Pathway	Highly expressed in tumor
IGF1R	IGF1R Pathway	Highly expressed in tumor
IGFBP3	IGFBP3 Pathway	Highly expressed in tumor
IGFBP5	IGFBP5 Pathway	Highly expressed in tumor
IGFBP6	IGFBP6 Pathway	Highly expressed in tumor
IGFBP7	IGFBP7 Pathway	Highly expressed in tumor
IGFBP8	IGFBP8 Pathway	Highly expressed in tumor
IGFBP9	IGFBP9 Pathway	Highly expressed in tumor
IGFBP10	IGFBP10 Pathway	Highly expressed in tumor
IGFBP11	IGFBP11 Pathway	Highly expressed in tumor
IGFBP12	IGFBP12 Pathway	Highly expressed in tumor
IGFBP13	IGFBP13 Pathway	Highly expressed in tumor
IGFBP14	IGFBP14 Pathway	Highly expressed in tumor
IGFBP15	IGFBP15 Pathway	Highly expressed in tumor
IGFBP16	IGFBP16 Pathway	Highly expressed in tumor
IGFBP17	IGFBP17 Pathway	Highly expressed in tumor
IGFBP18	IGFBP18 Pathway	Highly expressed in tumor
IGFBP19	IGFBP19 Pathway	Highly expressed in tumor
IGFBP20	IGFBP20 Pathway	Highly expressed in tumor
IGFBP21	IGFBP21 Pathway	Highly expressed in tumor
IGFBP22	IGFBP22 Pathway	Highly expressed in tumor
IGFBP23	IGFBP23 Pathway	Highly expressed in tumor
IGFBP24	IGFBP24 Pathway	Highly expressed in tumor
IGFBP25	IGFBP25 Pathway	Highly expressed in tumor
IGFBP26	IGFBP26 Pathway	Highly expressed in tumor
IGFBP27	IGFBP27 Pathway	Highly expressed in tumor
IGFBP28	IGFBP28 Pathway	Highly expressed in tumor
IGFBP29	IGFBP29 Pathway	Highly expressed in tumor
IGFBP30	IGFBP30 Pathway	Highly expressed in tumor
IGFBP31	IGFBP31 Pathway	Highly expressed in tumor
IGFBP32	IGFBP32 Pathway	Highly expressed in tumor
IGFBP33	IGFBP33 Pathway	Highly expressed in tumor
IGFBP34	IGFBP34 Pathway	Highly expressed in tumor
IGFBP35	IGFBP35 Pathway	Highly expressed in tumor
IGFBP36	IGFBP36 Pathway	Highly expressed in tumor
IGFBP37	IGFBP37 Pathway	Highly expressed in tumor
IGFBP38	IGFBP38 Pathway	Highly expressed in tumor
IGFBP39	IGFBP39 Pathway	Highly expressed in tumor
IGFBP40	IGFBP40 Pathway	Highly expressed in tumor
IGFBP41	IGFBP41 Pathway	Highly expressed in tumor
IGFBP42	IGFBP42 Pathway	Highly expressed in tumor
IGFBP43	IGFBP43 Pathway	Highly expressed in tumor
IGFBP44	IGFBP44 Pathway	Highly expressed in tumor
IGFBP45	IGFBP45 Pathway	Highly expressed in tumor
IGFBP46	IGFBP46 Pathway	Highly expressed in tumor
IGFBP47	IGFBP47 Pathway	Highly expressed in tumor
IGFBP48	IGFBP48 Pathway	Highly expressed in tumor
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IGFBP51	IGFBP51 Pathway	Highly expressed in tumor
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IGFBP59	IGFBP59 Pathway	Highly expressed in tumor
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IGFBP61	IGFBP61 Pathway	Highly expressed in tumor
IGFBP62	IGFBP62 Pathway	Highly expressed in tumor
IGFBP63	IGFBP63 Pathway	Highly expressed in tumor
IGFBP64	IGFBP64 Pathway	Highly expressed in tumor
IGFBP65	IGFBP65 Pathway	Highly expressed in tumor
IGFBP66	IGFBP66 Pathway	Highly expressed in tumor
IGFBP67	IGFBP67 Pathway	Highly expressed in tumor
IGFBP68	IGFBP68 Pathway	Highly expressed in tumor
IGFBP69	IGFBP69 Pathway	Highly expressed in tumor
IGFBP70	IGFBP70 Pathway	Highly expressed in tumor
IGFBP71	IGFBP71 Pathway	Highly expressed in tumor
IGFBP72	IGFBP72 Pathway	Highly expressed in tumor
IGFBP73	IGFBP73 Pathway	Highly expressed in tumor
IGFBP74	IGFBP74 Pathway	Highly expressed in tumor
IGFBP75	IGFBP75 Pathway	Highly expressed in tumor
IGFBP76	IGFBP76 Pathway	Highly expressed in tumor
IGFBP77	IGFBP77 Pathway	Highly expressed in tumor
IGFBP78	IGFBP78 Pathway	Highly expressed in tumor
IGFBP79	IGFBP79 Pathway	Highly expressed in tumor
IGFBP80	IGFBP80 Pathway	Highly expressed in tumor
IGFBP81	IGFBP81 Pathway	Highly expressed in tumor
IGFBP82	IGFBP82 Pathway	Highly expressed in tumor
IGFBP83	IGFBP83 Pathway	Highly expressed in tumor
IGFBP84	IGFBP84 Pathway	Highly expressed in tumor
IGFBP85	IGFBP85 Pathway	Highly expressed in tumor
IGFBP86	IGFBP86 Pathway	Highly expressed in tumor
IGFBP87	IGFBP87 Pathway	Highly expressed in tumor
IGFBP88	IGFBP88 Pathway	Highly expressed in tumor
IGFBP89	IGFBP89 Pathway	Highly expressed in tumor
IGFBP90	IGFBP90 Pathway	Highly expressed in tumor
IGFBP91	IGFBP91 Pathway	Highly expressed in tumor
IGFBP92	IGFBP92 Pathway	Highly expressed in tumor
IGFBP93	IGFBP93 Pathway	Highly expressed in tumor
IGFBP94	IGFBP94 Pathway	Highly expressed in tumor
IGFBP95	IGFBP95 Pathway	Highly expressed in tumor
IGFBP96	IGFBP96 Pathway	Highly expressed in tumor
IGFBP97	IGFBP97 Pathway	Highly expressed in tumor
IGFBP98	IGFBP98 Pathway	Highly expressed in tumor
IGFBP99	IGFBP99 Pathway	Highly expressed in tumor
IGFBP100	IGFBP100 Pathway	Highly expressed in tumor

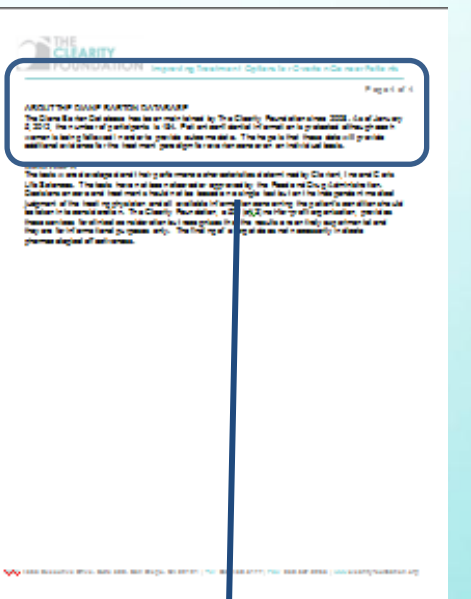
Compilation of data from all labs with interpretation (percentile rank, potential drugs)



Test Results for Gene Data Compared to the Diagnostics Database

Box plot showing test results for various genes compared to the Diagnostics Database. The y-axis represents the test result value, and the x-axis lists the genes. The plot shows the distribution of test results for each gene, with a red dot indicating the patient's result.

Individual profile compared to ovarian cancer population

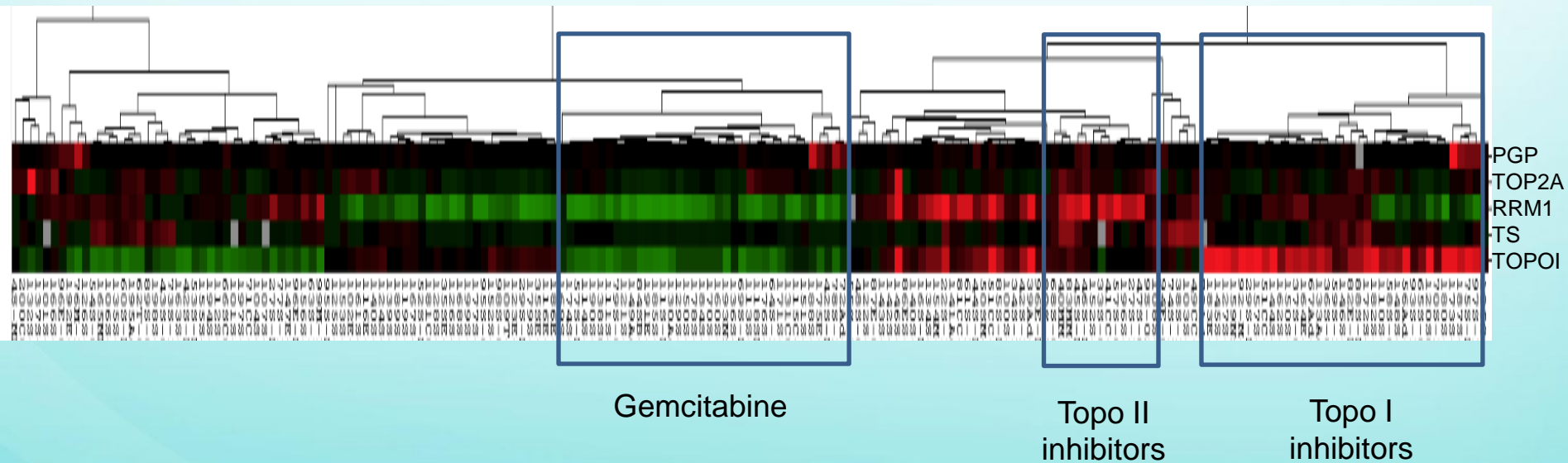


ABOUT THE DIANE BARTON DATABASE

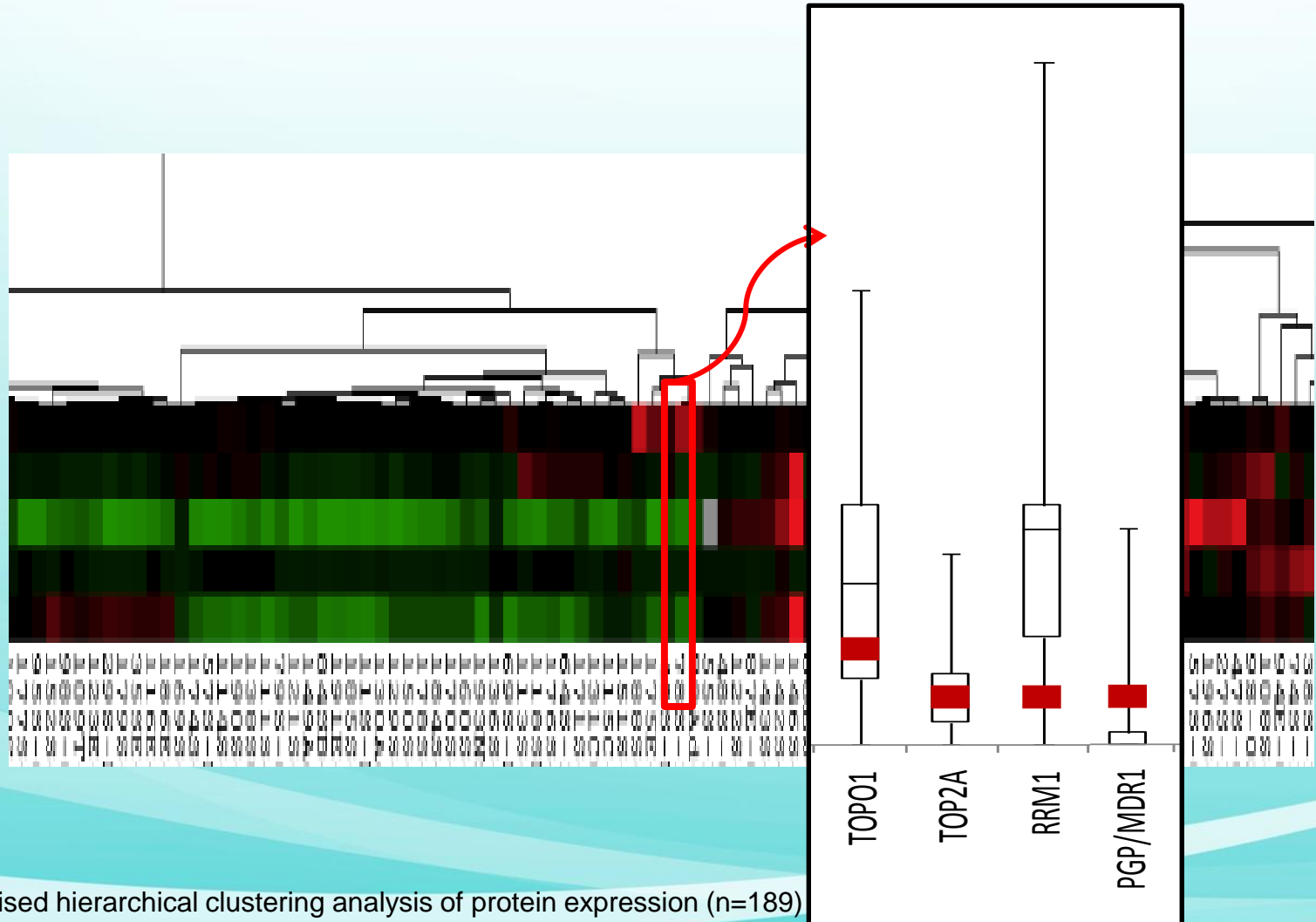
The Diane Barton Database was developed by The Clarity Foundation in 2012. It is a comprehensive database of ovarian cancer patients who have been treated with platinum-based chemotherapy. The database includes information on patient demographics, clinical history, and treatment outcomes. The database is used to compare patient test results to the results of other patients in the database to identify potential therapeutic targets.

Number of patients whose data are included in Diane Barton Database

Often, only one of the commonly used agents to treat recurrent ovarian cancer is prioritized by the profile



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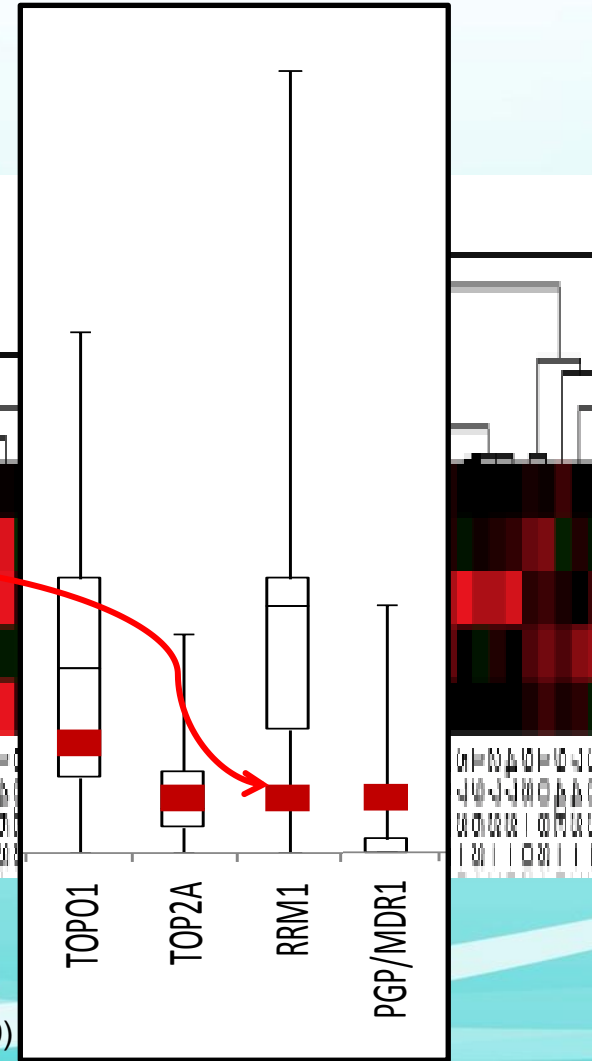
Often, only one of the commonly used agents to treat recurrent ovarian cancer is prioritized by the profile

High Topo I → Irinotecan, Topotecan

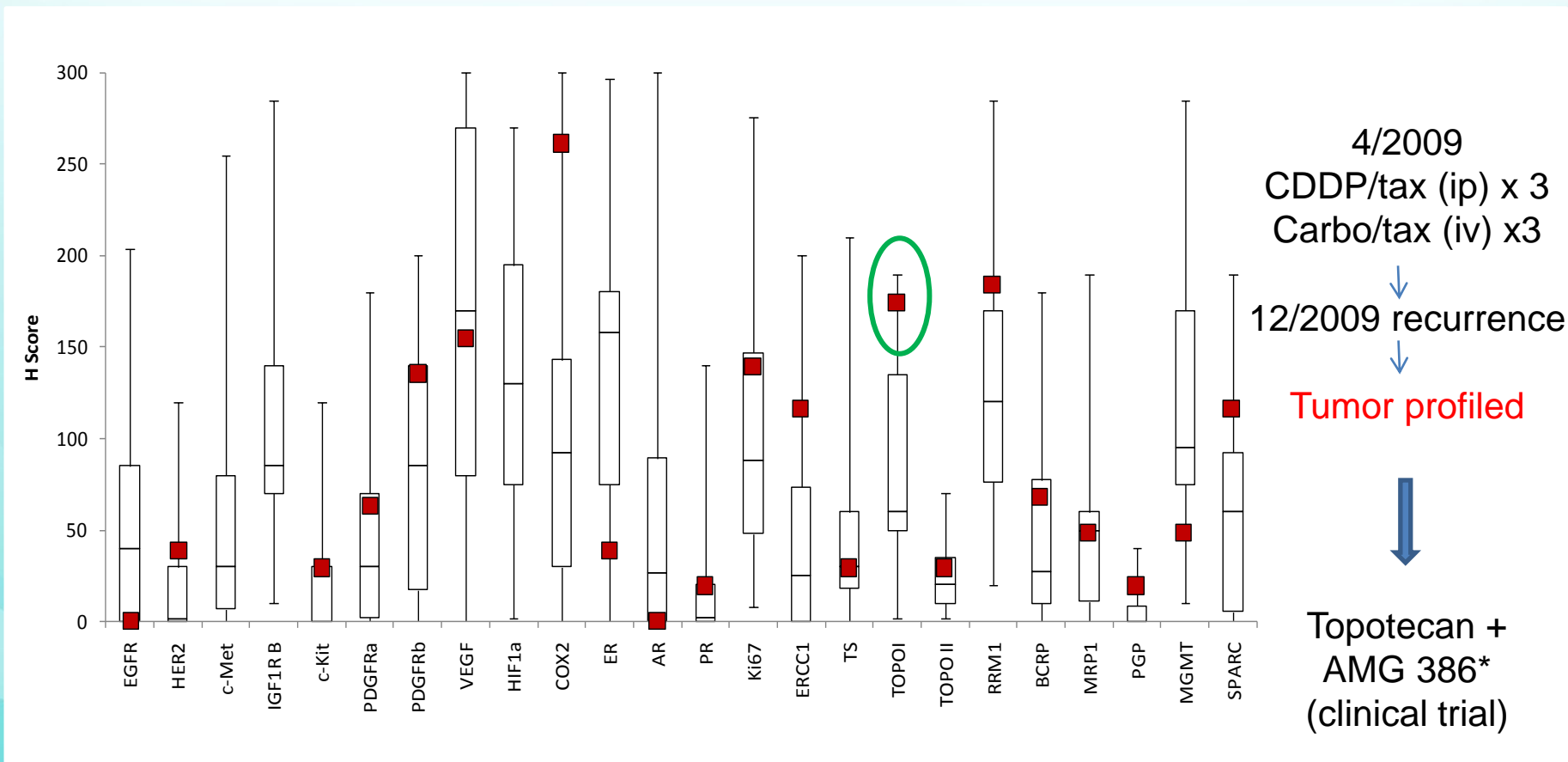
High Topo II → doxil, etoposide

High PGP → No Taxane, no doxil

Low RRM1 → Gemzar



Case Study: profile for patient diagnosed in 2009 with stage IIIC clear cell carcinoma



Biopsy of recurrent disease may be needed to obtain relevant profiling information

Mol Cancer Ther; 11(2) February 2012

Molecular Medicine in Practice

Molecular
Cancer
Therapeutics

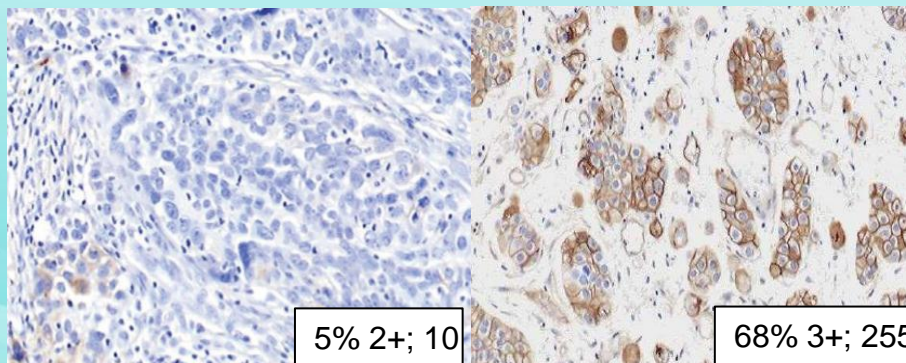
Treatment-Related Protein Biomarker Expression Differs between Primary and Recurrent Ovarian Carcinomas

Deborah A. Zajchowski¹, Beth Y. Karlan², and Laura K. Shawver¹

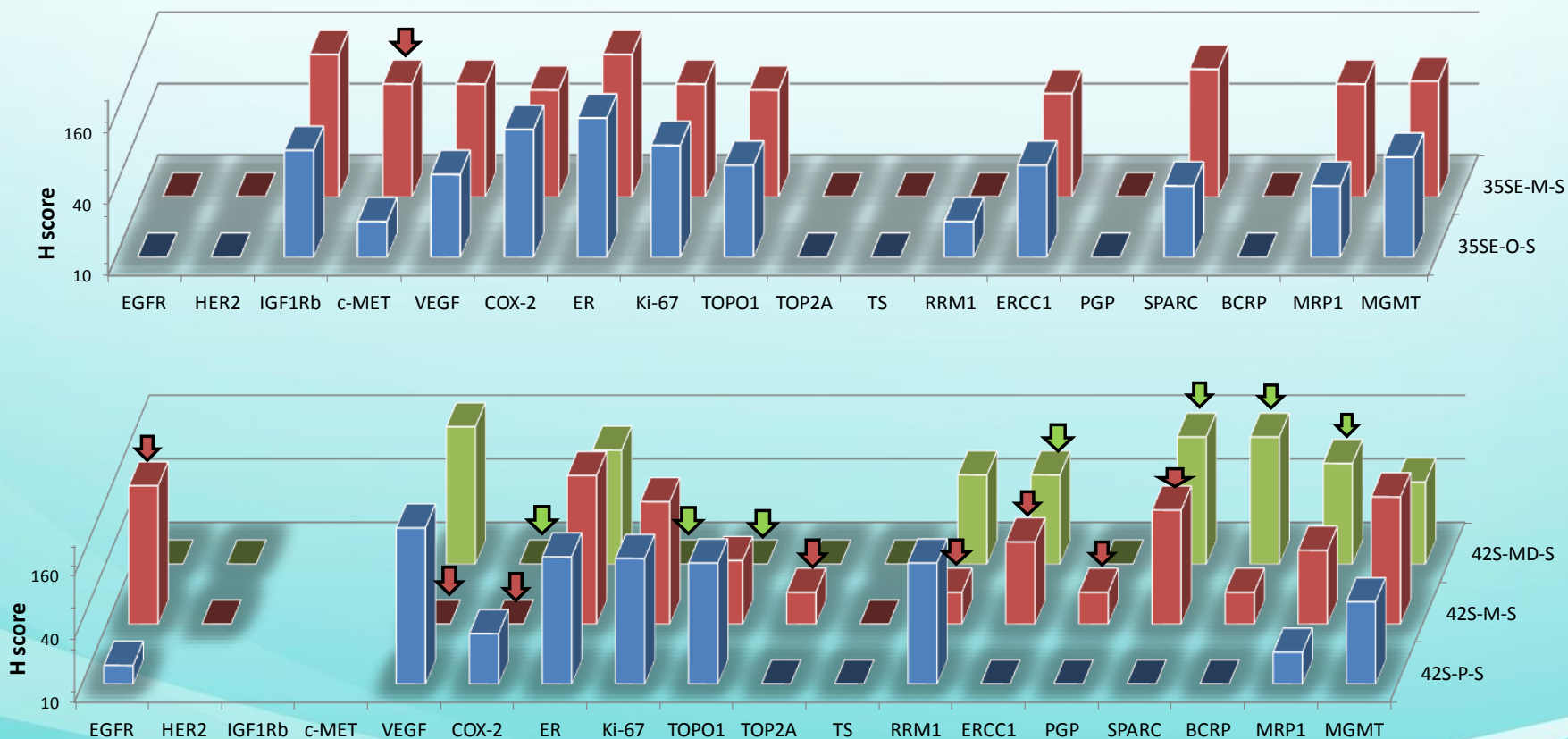
Primary

Recurrence

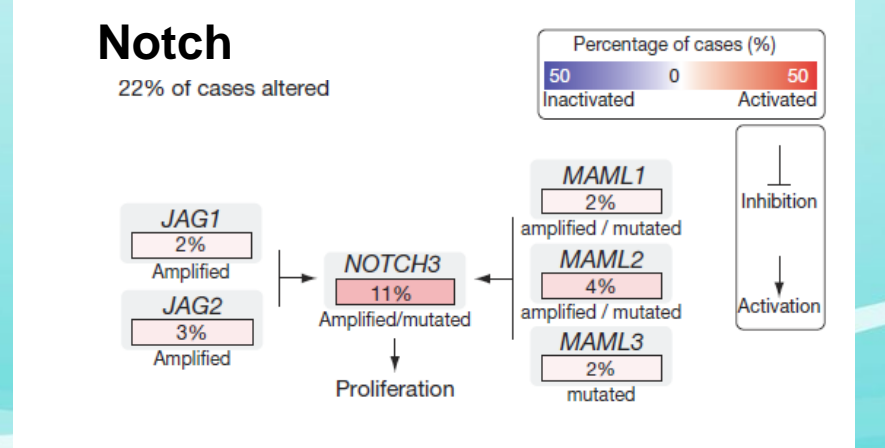
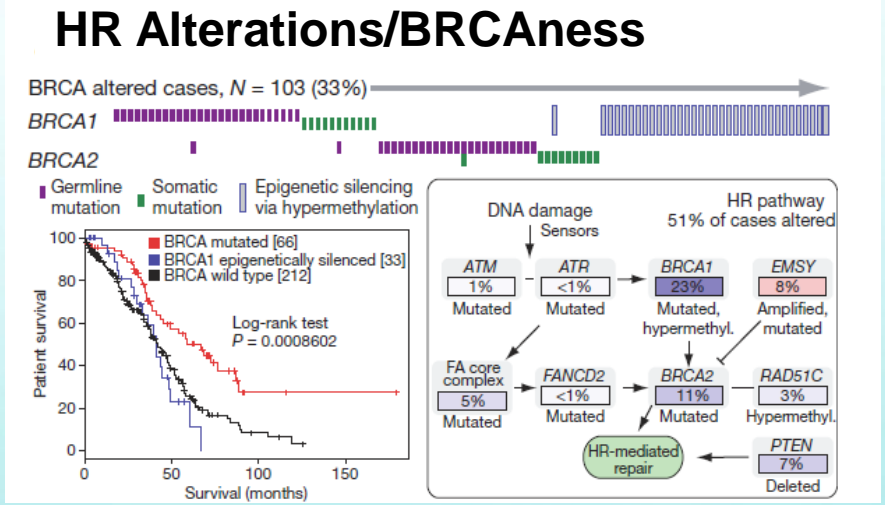
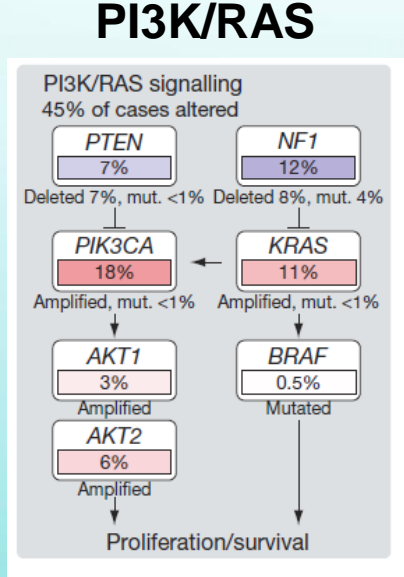
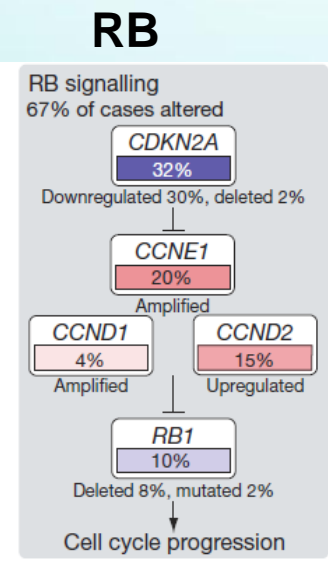
EGFR



Marker expression differences in patient-matched primary and recurrent samples



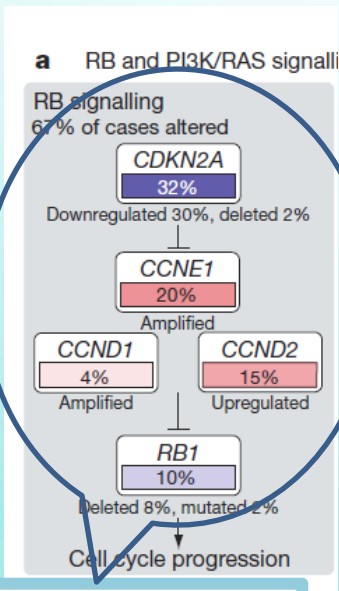
Low frequency of specific genetic aberrations → extensive interrogation necessary to characterize tumors



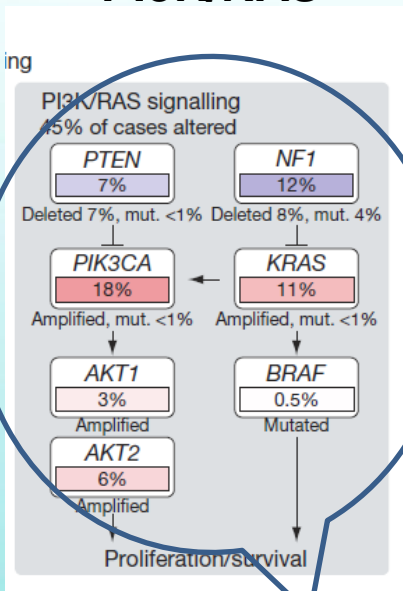
PARP inhibitors

Genomic markers can be used to assign patients to clinical trial agents

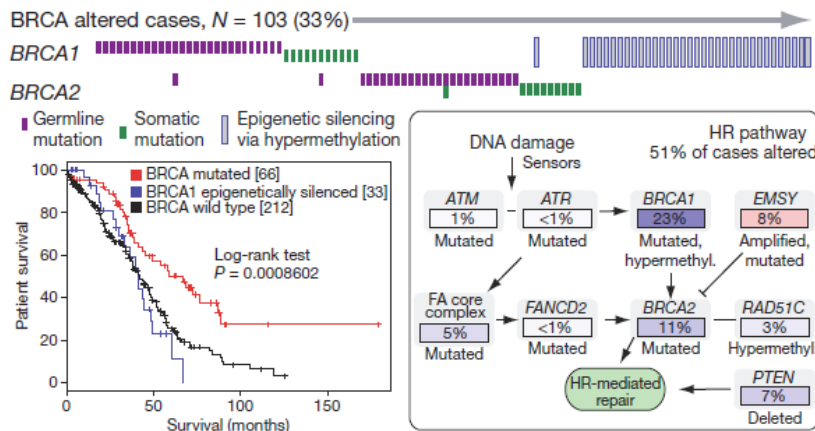
RB



PI3K/RAS



HR Alterations/BRCAness

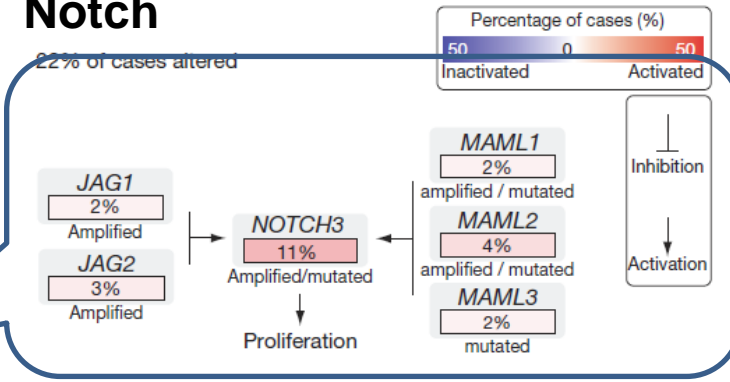


CDK inhibitors
AURK inhibitors

PI3K/AKT/mTOR inhibitors
MEK inhibitors

Notch inhibitors

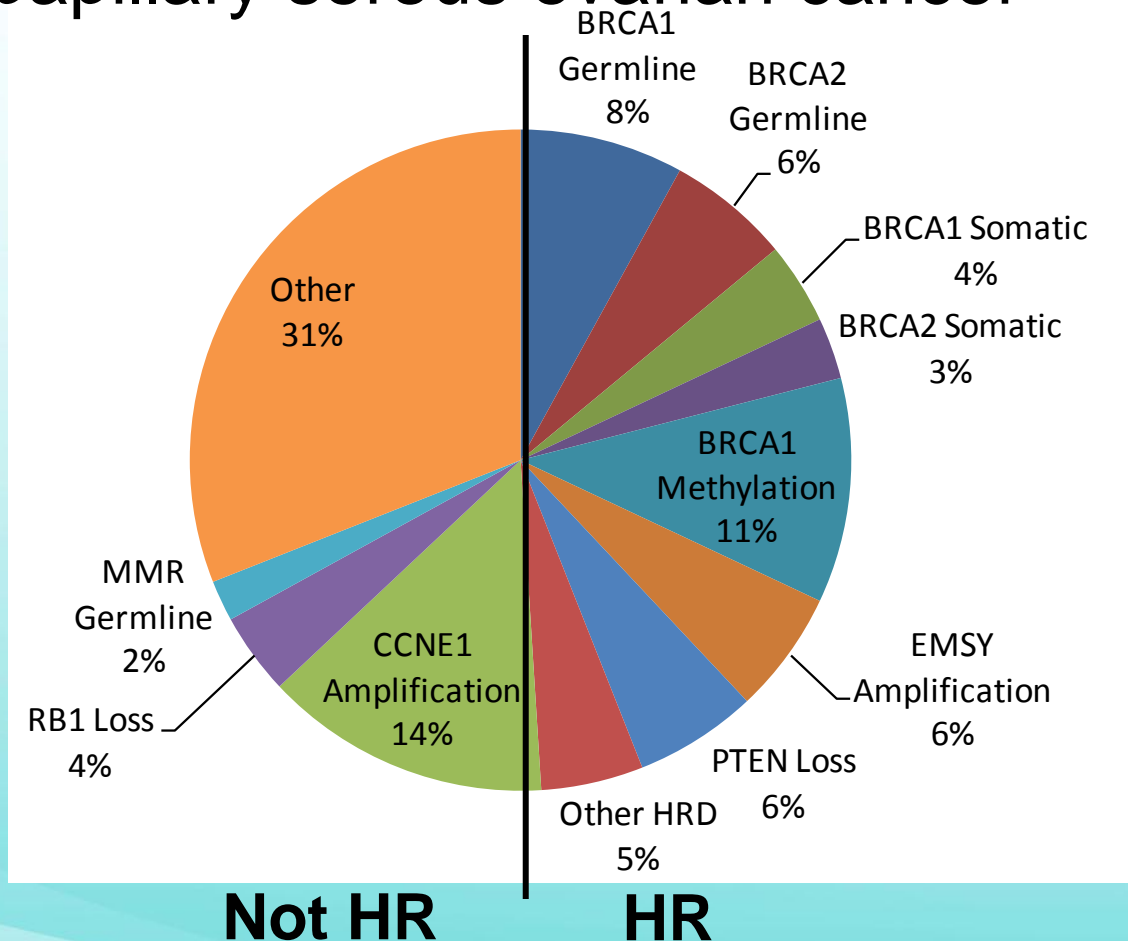
Notch



BRCAness Case Study

- **Stage IIIC diagnosed June 2007**
- **BRCA1 and 2 tested at Myriad – no mutations detected**
- **Carbo/docetaxel x 6 followed by 11 cycles of maintenance docetaxel the last 6 with the addition of bevacizumab**
- **Completed treatment in Nov 2008 and recurred in April 2009 (measurable disease by CT and increased CA125)**
- **No response to tamoxifen. 2nd remission achieved with Carbo/doxil**
- **Entered double-blind PARP inhibitor clinical trial Dec 2009 testing olaparib as maintenance to prevent recurrence**
 - **AZ announced 12.20.11 that the drug will not progress to Phase 3 but drug is provided for women who continue to benefit and**
 - **patient remains in remission and continues on study agent**
- **Sequencing of coding regions of ~200 genes implicated in cancer performed on tumor sample December 2011**
- **Somatic BRCA2 mutation detected**

HR deficiencies may be identified in up to 50% of papillary serous ovarian cancer



Summary

- Ovarian cancer is heterogeneous and a broad profiling panel is needed to capture data relevant to each individual
- Commonly utilized agents for treatment of recurrent ovarian cancer can be prioritized using molecular markers
- Obtaining biopsies at recurrence is optimal
- Incorporation of molecular markers can help prioritize clinical trials for patients
 - Single agents
 - And combinations





THE
CLARITY
FOUNDATION

www.clarityfoundation.org

