HER2-Driven Breast Cancer

Panel Discussion:

Moderator: Joyce O’Shaughnessy, MD
Break
Novel Therapies for Triple Negative Breast Cancer

International Congress on
The Future of Breast Cancer
Hotel del Coronado
Coronado, CA

Judy E. Garber, MD MPH
Dana Farber Cancer Institute
5 August 2011 Boston, MA
Disclosure of Information
Speaker: Judy E. Garber, MD MPH

I have the following relationships to disclose:

- Leading PARP inhibitor trials with Astra Zeneca
- Leading PARP inhibitor trial with Abbott Labs, Inc.
- Co-investigator multiple clinical trials through the Dana Farber / Harvard Cancer Center

I will discuss the following off-label uses of the product in approved research protocols in my presentation: None
Triple-negative breast cancer: Range of histology

Basal-like and Triple-Negative Breast Cancers

Low-grade tumors
- Secretory carcinoma
- Adenoid cystic carcinoma

High-grade tumors
- Medullary breast cancer
- Metaplastic breast cancer
- Grade 3 - IDC-NST
Cluster diagrams of breast cancer subtypes highlighting the Basal-like and Claudin-low subtypes

**Basal-like subtype**
1. 10-25% of all tumors
2. ~50-75% of TN tumors
3. Distinct cell type of origin or developmental stage of arrest
4. >50% TP53 mutated
5. Highly proliferative (RB-loss)
6. BRCA1-associated
7. Highly aneuploid

**Claudin-low Subtype**
1. 5-10% of tumors
2. Typically TN
3. Low expression of cell-cell junction proteins
4. Lymphocyte infiltrates
5. Stem cell + EMT features

Perou C M The Oncologist 2011;16:61-70
Natural History and Survival after Breast Ca Diagnosis

Triple-negative cancer

- Brain 30%
- Lung 40%
- Liver 20%
- Bones 10%

Non-triple-negative cancer

- 10% (higher in HER2+ breast cancer)
- 20%
- 30%
- 40%

A Breast-Cancer-Specific Survival According to Immunohistochemical Subtype

- Years after Diagnosis
- Breast-Cancer-Specific Survival
- ER+ or PR+/HER2-
- ER+/HER2+
- ER-/PR-/HER2+/basal marker-
- ER-/PR-/HER2+/basal marker+
- ER-/PR-/HER2+

B Hazard Rate for Distant Recurrence

- Years after Diagnosis
- Hazard Rate
- Triple-negative breast cancers
- Non-triple-negative breast cancers

## Intrinsic Subtypes and AC-T Sensitivity

### Response to Neoadjuvant Anthracycline and Taxane

<table>
<thead>
<tr>
<th>Subtype</th>
<th>Basal-like (n=33)</th>
<th>HER2+/ER- (n=55)</th>
<th>Luminal (n=36)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical Response (CR+PR)</td>
<td>76%</td>
<td>65%</td>
<td>51%</td>
</tr>
<tr>
<td>Path CR</td>
<td>26%</td>
<td>18%</td>
<td>5%</td>
</tr>
</tbody>
</table>

# Overall Survival Rate after Neoadjuvant Chemotherapy in Women with TNBC and Those with Non–TNBC

<table>
<thead>
<tr>
<th>Variable</th>
<th>Triple-Negative Breast Cancer (N = 225)</th>
<th>Non–Triple-Negative Breast Cancer (N = 863)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complete pathological response*</td>
<td>22</td>
<td>11</td>
<td>0.03</td>
</tr>
<tr>
<td>3-Yr overall survival with complete pathological response</td>
<td>94</td>
<td>98</td>
<td>0.24</td>
</tr>
<tr>
<td>3-Yr overall survival after less than complete pathological response</td>
<td>68</td>
<td>88</td>
<td>0.001</td>
</tr>
</tbody>
</table>

* Complete pathological response was determined on the basis of examination of breast tissue removed at the time of definitive surgery. Data are from Liedtke et al.\textsuperscript{49}
Recurrence-Free Survival By ER Status In Dana-Farber Trial For Patients With Residual Disease After Preoperative Chemotherapy

Comparison of Survival Curves

ER positive
N=73

ER Negative
N=37

RR = 0.289 SE = 0.13 p-value = 0.0058

Mayer E et al, ASCO 2009
C9344 disease-free survival for Paclitaxel by ER and HER-2 status

Berry DA et al, JAMA 2006;295:1658-67
Estimated subtype-specific cumulative incidence of relapse over time by treatment group with **CMF v no CMF**

A. TNBC

B. HER2 +

C. Endocrine Receptor +

Colleoni M et al. JCO 2010;28:2966
Triple-Negative Breast Cancers: some potential therapeutic targets

- Cetuximab
- EGFR Tyrosine Kinase
- MAPK inhibitors; NOTCH inhibitors
- Anti-Angiogenesis
- Bevacizumab
- C-KIT tyrosine kinase
- Dasatinib Sunitinib
- MAP Kinase Pathway
- Akt Pathway
- Transcriptional Control
- Cell Cycle
- Cell Death
- DNA Repair pathways
- PARP inhibitors; Trabectedin
- HYPOXIA

After Cleator S et al. Lancet Oncol. 2006:8:235-244
Gene Expression Profiles and IHC of Basal-like Breast Cancers

EGFR-Targeting Trials in Metastatic TNBC

US Oncology

Weekly Irinotecan Carboplatin

Weekly Irinotecan Carboplatin + Cetuximab

Cetuximab

Carboplatin + Cetuximab

Inter-SPORE*

Cetuximab

Carboplatin + Cetuximab

PD

*UNC, UCSF, DFCI, UAB, IU, Mayo, JHU, GT, Baylor, Duke, MDACC, Wash U
# EGFR Inhibition in Basal-like Breast Cancer

## TBCRC 001: Cetuximab + Carboplatin in Pretreated Triple Negative

<table>
<thead>
<tr>
<th>C and P</th>
<th>C</th>
<th>P</th>
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<tbody>
<tr>
<td>N</td>
<td>71</td>
<td></td>
</tr>
<tr>
<td>CR</td>
<td>1%</td>
<td></td>
</tr>
<tr>
<td>PR</td>
<td>15%</td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>23%</td>
<td></td>
</tr>
<tr>
<td>CB</td>
<td>31%</td>
<td></td>
</tr>
<tr>
<td>RR</td>
<td>17%</td>
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</table>

Carey LA, et al. ASCO’08

## US Oncology 225200: Carboplatin + Irinotecan + Cetuximab

<table>
<thead>
<tr>
<th>IP</th>
<th>IP+C</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>69</td>
</tr>
<tr>
<td>N</td>
<td>69</td>
</tr>
</tbody>
</table>

| Overall Response Rate | 31% | 31% |

<table>
<thead>
<tr>
<th>Triple Negative Pts</th>
<th>N</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall Response Rate</td>
<td>30%</td>
<td>49%</td>
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</tbody>
</table>

O’Shaughnessy et al, SABCS ‘07

16 had serial bx with good RNA:

- 4 EGFR not expressed/pathway not activated
- 12 EGFR expressed and “activated:
  - 4 Inhibited (2CB,2PD);
  - 8 NOT inhibited (ALL PD)
BALI-1 Study Design

TNBC
≤1 prior CT for met. disease
Randomized 2:1

Group A
Cisplatin 75 mg/m² IV, d1
3-week cycles x 6
+ cetuximab 400 mg/m² initial dose
then 250 mg/m² weekly

Cetuximab

Group B
Cisplatin 75 mg/m² IV, d1
3-week cycles x 6

Cross over allowed on PD*

*Cetuximab + cisplatin if PD during first 6 weeks;
cetuximab alone if PD after first 6 weeks

CT, chemotherapy; TNBC, triple negative breast cancer; PD, progressive disease
<table>
<thead>
<tr>
<th>Response assessed by investigator</th>
<th>Cetuximab + cisplatin n=115 (100%)</th>
<th>Cisplatin n=58 (100%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complete response</td>
<td>2 (1.7%)</td>
<td>1 (1.7%)</td>
</tr>
<tr>
<td>Partial response</td>
<td>21 (18.3%)</td>
<td>5 (8.6%)</td>
</tr>
<tr>
<td>Stable disease</td>
<td>48 (41.7%)</td>
<td>18 (31.0%)</td>
</tr>
<tr>
<td>Progressive disease</td>
<td>34 (29.6%)</td>
<td>31 (53.4%)</td>
</tr>
<tr>
<td>Not evaluable</td>
<td>10 (8.7%)</td>
<td>3 (5.2%)</td>
</tr>
<tr>
<td><strong>ORR</strong></td>
<td><strong>23 (20.0%)</strong></td>
<td><strong>6 (10.3%)</strong></td>
</tr>
</tbody>
</table>

[95% CI] [13.1% – 28.5%] [3.9% – 21.2%]
BALI-1 Secondary efficacy: PFS

HR [95% CI]: 0.67 [0.47–0.97]  p=0.03

<table>
<thead>
<tr>
<th>Treatment</th>
<th>No. events</th>
<th>Median PFS (months) [95% CI]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cetuximab + cisplatin</td>
<td>92</td>
<td>3.7 [2.8 – 4.3]</td>
</tr>
<tr>
<td>Cisplatin</td>
<td>47</td>
<td>1.5 [1.4 – 2.8]</td>
</tr>
</tbody>
</table>

Patients at risk

<table>
<thead>
<tr>
<th>Treatment</th>
<th>No.</th>
<th>0</th>
<th>3</th>
<th>6</th>
<th>9</th>
<th>12</th>
<th>15</th>
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</thead>
<tbody>
<tr>
<td>Cetuximab + cisplatin</td>
<td>115</td>
<td>53</td>
<td>18</td>
<td>6</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Cisplatin</td>
<td>58</td>
<td>16</td>
<td>5</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>

Probability of PFS vs. Time (months)
BALI-1 Summary and Conclusions

• Addition of cetuximab to cisplatin increased ORR (20.0% vs 10.3%; p=0.11)
  – Primary objective not met since ORR in patients treated with cetuximab plus cisplatin did not exceed 20% (p=0.5)
  – However, chance for response was twice as high for patients receiving cetuximab plus cisplatin (OR=2.126)

• Significant improvement in PFS in patients treated with cetuximab plus cisplatin compared to cisplatin alone (median PFS 3.7 vs 1.5 months, HR=0.67; p=0.03)

• No new safety concerns were identified

• OS results not yet available

• This trial suggests that cetuximab may be a potential addition to the treatment of patients with TNBC
Cis-platinum and Breast Cancer

- Sledge (JCO 1988 6:1811-1814) reported 47% response rate in 20 patients in first line setting
- ~10% response rate after previous chemotherapy for metastatic disease
- Taxanes developed during this time; better therapeutic index
- Recent interest in patients with HER2+ and triple negative breast cancer
Patients with metastatic breast cancer treated with platinum-containing regimens by subtype:

TNBC showed similar response rate (~39%) but worse survival than non-TNBC.
BRCA1-Associated Breast Cancers are Basal-like by Microarray

- 10% of breast cancers
- ER(-) PR(-) HER2(-)
- Poorly differentiated
- High grade, Aneuploid
- EGFR+, cyclin E+
- Express basal keratins
- Little DCIS
- Poor prognosis
- Different stem cell?
Is LOH an indicator of DNA repair defects?

- High genome-wide levels of LOH is a feature shared between sporadic and BRCA1-associated TNBC
- Perhaps defects in DNA repair are responsible for high level of LOH in some TNBC
- Is level of LOH or allelic imbalance in TNBC associated with cisplatin response?

Courtesy of Andrea Richardson
DF/HCC NeoAdjuvant Platinum in Triple Negative Breast Cancer

> 2cm, Stage II/III ER/PR/Her Neg Breast Cancer on Core Biopsy

Cis Platinum 75mg/m² q3wks x 12 weeks*

Standard Adjuvant Therapy per MD

**Tissue:**
- 5q, 8q LOH
- IHC: BRCA1, CK5/6
- Microarrays: cDNA, SNP
- Radiation damage assay

**Blood:**
- BRCA1

**Imaging**
- Mammo
- US
- MRI

* Additional MRI and Core bx after 1 dose of CDDP: radiation damage assay
Neoadjuvant Cisplatin (CDDP) in Triple-Negative Breast Cancer

- N = 28
  - > 2-cm stage II/III triple negative
- Single-agent cisplatin 75 mg/m² q3w x 4 cycles prior to surgery

**Toxicity:**

<table>
<thead>
<tr>
<th>Grade 4</th>
<th>LFT ↑</th>
<th>1 pt</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 3</td>
<td>Neutropenia</td>
<td>2 pts</td>
</tr>
<tr>
<td></td>
<td>Tinnitus</td>
<td>1 pt</td>
</tr>
<tr>
<td></td>
<td>Nausea</td>
<td>1 pt</td>
</tr>
<tr>
<td></td>
<td>Fatigue</td>
<td>1 pt</td>
</tr>
<tr>
<td></td>
<td>Hyperkalemia</td>
<td>1 pt</td>
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<tr>
<td></td>
<td>LFT ↑</td>
<td>1 pt</td>
</tr>
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</table>

**Response:**

<table>
<thead>
<tr>
<th>Pathologic CR</th>
<th>6 (22%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical CR</td>
<td>4 (14%)</td>
</tr>
<tr>
<td>Clinical PR</td>
<td>10 (36%)</td>
</tr>
<tr>
<td>Stable Disease</td>
<td>5 (17%)</td>
</tr>
</tbody>
</table>

- Age associated with pCR (P < .04)
- Both BRCA1+ had path CR

Silver D et al. JCO 2010;28:1145-1153
# Predictors of Response to Neo-Adjuvant CDDP in TNBC

Reference subtype

Cisplatin response

<table>
<thead>
<tr>
<th>Response score</th>
<th>Progress</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5 (pCR)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample No.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>i) BRCA1 mutation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ii) Low BRCA1 mRNA</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>iii) BRCA1 methylation</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>iv) ΔNp63/TAp73 &gt; 2</td>
<td></td>
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</tr>
</tbody>
</table>
What do these results tell us?

- We were right: BRCA1-associated breast cancer is sensitive to cross-linking agents as is a subset of TNBC
- We were partially right: BRCA1-associated breast cancer is sensitive to chemo in general and so is a subset of TNBC
- Platinums should be investigated further
- It’s good to be lucky
- All of the above
- Nothing
### Response to Neoadjuvant Cisplatin in BRCA1+ Breast Cancer Patients

- 10 women with BRCA1 mutations
- 4 cycles CDDP 75mg/m2 q 21d
- Trial is ongoing

#### ASCO Result:

18/25 = 72% pathCR

<table>
<thead>
<tr>
<th>Response</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical Complete Response</td>
<td>9</td>
<td>90</td>
</tr>
<tr>
<td>Clinical Partial Response</td>
<td>1</td>
<td>10</td>
</tr>
<tr>
<td>Complete Pathologic Response</td>
<td>9</td>
<td>90</td>
</tr>
<tr>
<td>Partial Pathologic Response</td>
<td>1</td>
<td>10</td>
</tr>
<tr>
<td>Residual Disease in the Breast</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

#### Number positive nodes

- 0-3: 90%
- 1-3: 10%

TBCRC: Cis- or Carboplatin for metastatic TNBC

Accrual Complete
N=82

TNBC
0-1 prior chemo
FFPE for study
No prior platinum
No active CNS mets

Cisplatin 75/m2
q 21 days

Carboplatin AUC 5
q 21 days

*Not Randomized: Drug Chosen per Site

Sites: BIDMC, DFCI, MGH, MSKCC, UCSF, UNC, Vanderbilt

S. Isakoff, MD PhD, PI
CALGB 40603: Triple Negative Neoadjuvant Trial

N=600 ER/PR/HER2-
Stage II-III B

Paclitaxel
Carboplatin
No Carboplatin

No Paclitaxel
Carboplatin
No Carboplatin

Bevacizumab

Dose-dense AC

Breast imaging
Blood
MUGA
Tumor Biopsy*

Breast imaging
Blood

Breast imaging
Blood
MUGA

W Sikov, PI

RT prn
TNT Trial
KCL / ICR co-sponsors

CA1 genotyping
Rahman ICR

Central ER / PR / HER2
CK5/6 and EGFR
Guy’s KCL Bank

ER-, PR- and HER2-
1st metastatic relapse
(or inoperable and recurrent locally advanced)

RANDOMISE
1:1

Carboplatin
AUC 6 q3w
6* cycles

Upon imaging
confirmed progression**

Docetaxel
100mg/m²
6* cycles

Upon imaging
confirmed progression**

Objective Response
Time to Progression
Objective Response
(2nd line protocol therapy)
Time to Treatment Failure
Toxicity
Overall Survival

Carboplatin
AUC 6 q3w
6* cycles

Docetaxel
100mg/m²
6* cycles

Optional biological
studies

Primary Block
Array CGH/expression
array/TMA

and where feasible
Relapse site
Core Biopsy
Array CGH/expression
array/TMA

Eligible for future phase II trials of novel targeted biological therapeutics
informed by Trans-TNT
When do I use platinums at this moment?

- Therapeutic option in TN and HER2+ Br Ca
- Early agent in TN metastatic disease, particularly if there is CNS involvement
- Rarely as Carbo/Paclitaxel adjuvant therapy for a 2nd or 3rd primary breast cancer in BRCA1/2 mutation carrier
- In combination with PARP inhibitor in therapeutic trials
The IKEA principle: PARP inhibitors

Normal tissue cells

- DNA repair
- Base excision DNA repair
- Homologous recombination (HR) repair

BRCA1/BRCA2 deficient Tumor cells

- DNA repair
- Base excision DNA repair
- PARP inhibitor

Specific tumor cell killing

- DNA repair
- Base excision DNA repair
- PARP inhibitor

Oral Parp-Inhibitor in Metastatic Breast/Ovarian Cancers in BRCA Carriers

Women with germline \( BRCA1/2 \) mutation and metastatic Breast or Ovarian cancers (2 protocols); \( \geq 1 \) prior therapy

Olaparib 400mg or 100mg po BID x 28 day cycles

Phase II multicenter trials

PIs: Breast: Andrew Tutt, MD
Ovarian: MW Audeh, MD

Iceberg Trial
One patient was excluded as only 1 of their 2 target lesions was measured at each assessment.
DF/HCC Trial of Veliparib + TMZ in Breast Cancer

Eligibility
Stage 4 Breast Cancer
Measurable Disease
> 1 Prior Therapy
Archived Tumor

1° Objective: Response Rate
2° Objective: Clinical Benefit Rate
Progression-Free Survival
Safety/Tolerability

S Isakoff, PI, MGH
Waterfall plot of best response

* = BRCA carriers (6 included above, 2 not included due to rapid progression)

Isakoff S et al. ASCO Abst 1019, 2010
Gem/CARBO +/- BSI-201

MBC
Triple Negative
Prior Chemo
N=120

- Gemcitabine 1000 mg/m² d 1,8
- Carbo AUC 2 d 1,8

CYCLES EVERY 21 DAYS

- Gemcitabine 1000 mg/m² d 1,8
- Carbo AUC 2 d 1,8
- BSI-201 5.6 mg/kg d 1,4,8, 11

RESTAGE EVERY 2 CYCLES

O’Shaugnessy J et al, ASCO 2009
Carbo/Gem +/- BSI-201: Overall Survival

BSI-201 + Gem/Carbo (n = 57)
Median OS = 9.2 months

Gem/Carbo (n = 59)
Median OS = 5.7 months

P = 0.0005
HR = 0.348 (95% CI, 0.189-0.649)
Evidence for the Efficacy of Iniparib, a potential PARPi, in BRCA2-associated Pancreatic Cancer

Fogelman DR et al. AntiCancer Res 2011;31:1417-20
Can we define tumors that will respond to PARP inhibitors? A phase II correlative study of olaparib in advanced serous ovarian cancer and triple-negative breast cancer

Gelmon KA et al. Abstract # 3002

TNBC: no responses if BRCA1/2 –
Ovarian: Responses in BRCA1/2+ and WT

ASCO 2010
Olaparib in a Patient with PTEN-Deficient Endometrioid Endometrial Cancer

Registered Trials with PARPi (USA)

- 18 Ovarian / 17 Breast studies with PARPi
  - Hoosier Onc Group: Post-Neoadjuvant Cisplatin 75/m2 x 4 +/- Pfizer Pi (PF-01367338) in women with TNBC and BRCA1/2+
- Iniparib + Irinotecan: CNS mets TNBC
- Veliparib + TMZ in CNS mets in pts ≤ age 21
- Olaparib + Gemcitabine in Pancreatic Cancer
- Olaparib in diverse tumor types, BRCA1/2+

PARPi Abstracts ASCO 2011: 141
<table>
<thead>
<tr>
<th>PARPi</th>
<th>company</th>
<th>phase</th>
<th>route</th>
<th>In vitro synergy</th>
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<tbody>
<tr>
<td>Olaparib</td>
<td>Astra Zeneca</td>
<td>I-II</td>
<td>oral</td>
<td>Platins, tmz, IR</td>
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<tr>
<td>ABT-888</td>
<td>Abbott</td>
<td>I-II</td>
<td>oral</td>
<td>Platins, Tmz, cyclophos, topoisomerase I Inhibs</td>
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<tr>
<td>Veliparib</td>
<td>Abbott</td>
<td>I-II</td>
<td>oral</td>
<td>Platins, Tmz, cyclophos, topoisomerase I Inhibs</td>
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<td>BSI-201?</td>
<td>Sanofi-Aventis</td>
<td>I-III</td>
<td>iv</td>
<td>Gmz/carbo, topotecan, oxaliplatin, IR</td>
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<td>Clovis</td>
<td>I-I</td>
<td>iv</td>
<td>Tmz, topotecan, IR</td>
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<td>MK-4827</td>
<td>Merck</td>
<td>I</td>
<td>oral</td>
<td></td>
</tr>
<tr>
<td>CEP-9722</td>
<td>Cephalon</td>
<td>I</td>
<td>oral</td>
<td>Tmz, Topoisomerase I poisons, platins</td>
</tr>
</tbody>
</table>

In development: “PARP inhibitor-like” Slowed
TRA-8 Ab Death Receptor 5: Mode of Action

Courtesy of A. Forero-Torres
Treatment of Basal-like (2LMP) Orthotopic Tumor Xenografts

Combination treatment most effective

Day

% Original Tumor Size

Control

Nab-Paclitaxel (1/10)

TRA-8 (2/10)

TRA-8 + Nab-Paclitaxel (2/9)

Treatment
TBCRC 019: An Open Label, Randomized, Phase II Trial of Nab-Paclitaxel with or without Tigatuzumab (a Humanized Monoclonal Antibody Targeting Death Receptor 5) in Patients with Metastatic TNBC

UAB and UAB Investigators who are not involved with the clinical trial have a potential conflict of interest with the research agent
129 Trials in TNBC from ClinTrials.gov: Neoadjuvant or Post-Neoadjuvant

- Carboplatin plus Eribulin (V Kaklamani, NWN)
- Sunitinib plus Carbo/Paclitaxel (D Yardley, Sarah Cannon)
- Carbo/Paclitaxel + Gamma Secretase Inhibitor RO4929097 (E Mrozek, Ohio State)
- Hoosier Onc Grp BRE09-146: PF-01367338 (PARPi) After Preop ChemoRx in TNBC or ER/PR +, HER2- With Known BRCA1/2 Mutations: (K Miller, Hoosier Onc Grp)