A Genetic Approach to Melanoma: Targeting of NRAS Disease

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Melanoma driver mutations

- Melanoma is a genetically heterogeneous disease
- Oncogenic driver mutations are present in most tumors
- These mutations have clinical implications

Lovly et al, PLOS One 2012
MyCancerGenome
Melanoma driver mutations

- Clear implications for targeted therapy
  - BRAF V600E
  - KIT exon 11
  - BRAF non-V600
  - BRAF fusions
  - NRAS

Sosman et al NEJM 2012
Carvajal et al, JAMA 2011
Dahlman et al Cancer Discovery 2012
Melanoma TCGA: Initial Results

- Cutaneous melanomas
- Non-glabrous skin
- Mainly regional metastases
- Total accrual goal = 500

NRAS mut (28%)
BRAF mut (52%)
NF1 mut (14%)
MITF amp (4%)
BRAF amp (12%)
MDM2 amp (12%)
NOTCH2 amp (5%)
KIT amp (3%)
KIT mut (3%)
PDGFRα amp (3%)
PDGFRα mut (10%)
CCND1 amp (4%)
TERT amp (5%)
PPP6C mut (7%)
TP53 mut (7%)
PTEN mut (8%)
PTEN del (12%)
CDKN2A mut (13%)
CDKN2A del (40%)

"Wild-Type":

MAPK
Cell Cycle

MAPK dependent tumors can be divided into two classes:

**BRAF^mut**

- Active monomer (mutant RAF, RAS, NF1, RAF translocations, atypical RAF mutants)
- Driven by RAF dimers
- Sensitive to MEK and/or ERK inhibitors (low therapeutic index)

**BRAF^wt**

- Active monomer (V600 BRAF mutants in tumors with low RAS.GTP)
- Driven by RAF monomers
- Sensitive to RAF inhibitors (high therapeutic index)
NRAS mutant melanoma

- NRAS mutant melanoma comprises 15-20% of all melanomas
- Has been shown to confer poor prognosis
- No targeted agents (yet) available
- MEK inhibitors (binimetinib) and combinations being developed

Jakob et al, Cancer 2012
MEK162 Clinical Activity in NRAS-Mutated Melanoma

CR = complete response; PR = partial response; SD = stable disease.
CDK4 as a Target in Melanoma

Clinical Significance of Genomic Alterations of the CDK4-pathway and Sensitivity to the CDK4 Inhibitor PD 0332991 In Melanoma.

1Grant A. McArthur, 1Richard J. Young, 1Karen E. Sheppard, 1Victoria Mar, 1Kelly Waldeck, 1Stephen B. Fox, 1Fergal C. Kelleher, 1Wendy Liu, 1Alexander Dobrovic, 1Richard Pearson, 1John Kelly, 1James G. Christensen, 1Sophia Randolph.

2Peter MacCallum Cancer Centre, Melbourne, Australia; 3Victorian Melanoma Service, Alfred Hospital, Melbourne, Australia; 4Department of Cancer Research, Pfizer Global Research and Development, La Jolla, CA; Pfizer Oncology, San Diego, CA.

Figure 3
Copy number variations according to mutation status.

<table>
<thead>
<tr>
<th>Copy Number Variations</th>
<th>Wildtype</th>
<th>BRAF mutation</th>
<th>NRAS mutation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>%</td>
<td>n</td>
</tr>
<tr>
<td>CyclinD1 gain</td>
<td>13</td>
<td>19</td>
<td>4</td>
</tr>
<tr>
<td>CDK4 gain</td>
<td>14</td>
<td>38</td>
<td>10</td>
</tr>
<tr>
<td>CDKN2A loss</td>
<td>33</td>
<td>53</td>
<td>18</td>
</tr>
</tbody>
</table>

Figure 4
Frequency of individual and multiple copy number variations in invasive primary melanoma.
68 cases were scored for all three genes: 25% (17/68) showed no gene alterations, 47% (32/68) showed a single gene alteration, 24% (16/68) showed a double gene alteration, and 4% (3/68) showed alterations in all three genes.

Figure 6
CDKN2A status and Sensitivity to PD-991
CDKN2A loss (low CDKN2A mRNA expression or mutation or loss of CDKN2A gene) was significantly (Fisher's exact test p < 0.006) associated with sensitivity to PD-991.

MEKi + CDK4i for NRAS-Mutant Melanoma

- GEMM: Compared NRAS extinction (doxy removal) vs MEKi
  - MEKi deficient in blocking proliferation, key node = CDK4
  - MEKi + CDK4i tumor regression in GEMM and human NRAS-mutant line (SB2)

A Phase 1b/2 Study of LEE011 in Combination With Binimetinib (MEK162) in Patients With Advanced NRAS-Mutant Melanoma: Early Encouraging Clinical Activity

Jeffrey A. Sosman,1 Muaiad Kittaneh,2 Martijn P. Lolkema,3 Michael Postow,4 Gary Schwartz,5 Catherine Franklin,6 Lyh Ping Lam,6 Alessandro Matano,7 Suraj Bhansali,8 Sudha Parasuraman,6 Kevin Kim9

1 Vanderbilt University Medical Center, Nashville, TN, USA; 2 Eisenberg Center for Translational Therapeutics, Karmanos Cancer Institute, Detroit, MI, USA; 3 University Medical Center Utrecht, Utrecht, Netherlands; 4 Memorial Sloan-Kettering Cancer Center, New York, NY, USA; 5 Columbia University Medical Center, New York, NY, USA; 6 Novartis Institutes for BioMedical Research, Cambridge, MA, USA; 7 Novartis Pharma AG, Basel, Switzerland; 8 Novartis Pharmaceuticals Corporation, East Hanover, NJ, USA; 9 The University of Texas MD Anderson Cancer Center, Houston TX, USA
Binimetinib & LEE011

• Binimetinib is an oral selective MEK1/2 inhibitor\(^1\)
  – Binimetinib has shown preliminary antitumor activity in multiple cancer types in phase 1 studies, including in patients with melanoma or other solid tumors\(^2-4\)
  – Acceptable safety for binimetinib has been demonstrated for doses up to 60 mg bid (RP2D, 45 mg bid)\(^2-4\)
  – A phase 2 trial showed clinical activity in NRAS-mutant melanoma;\(^5\) phase 3 trial in progress (NEMO)\(^6\)

• LEE011 is an oral selective inhibitor of CDK4/6\(^7\)
  – LEE011 has shown antitumor activity in multiple tumor xenograft models as a single agent and in combination with other targeted agents
  – In a phase 1 study, LEE011 demonstrated acceptable safety at doses up to 900 mg qd and preliminary clinical activity in patients with advanced solid tumors, including breast cancer and melanoma, or lymphomas\(^7\)

Presented by: Jeffrey A. Sosman, MD
**Study Design and Objectives**

**Phase 1b/Dose Escalation**
- Metastatic or Locally-Advanced NRAS-Mutant Melanoma $N \geq 15$
- LEE011 + Binimetinib
- MTD and/or RP2D

**Phase 2/Dose Expansion**
- Metastatic or Locally-Advanced NRAS-Mutant Melanoma ($N \approx 40$)

**Phase 1b Endpoints**
- **Primary objective:** determine the MTD and/or RP2D using a Bayesian logistic regression model with overdose control principle
- **Secondary objectives:** characterize safety and tolerability, PK, and clinical efficacy

**Study Status**
- As of March 21, 2014, 22 patients were treated in the phase 1b/dose-escalation part of the study
- Defining of a RP2D is ongoing

### Phase 1b Doses and Schedule\(^a\)

<table>
<thead>
<tr>
<th>Cohort</th>
<th>Binimetinib mg (\text{bid})</th>
<th>LEE011 mg (\text{qd})</th>
<th>Patients, (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>45</td>
<td>200</td>
<td>9</td>
</tr>
<tr>
<td>2</td>
<td>45</td>
<td>250</td>
<td>3</td>
</tr>
<tr>
<td>3</td>
<td>30</td>
<td>300</td>
<td>4</td>
</tr>
<tr>
<td>4</td>
<td>45</td>
<td>300</td>
<td>6</td>
</tr>
</tbody>
</table>

\(^a\)LEE011 for 21 consecutive days followed by a 7-day planned break and binimetinib on a continuous dosing schedule.

bid, twice daily; MTD, maximum tolerated dose; qd, once daily; RP2D, recommended phase 2 dose.

Presented by: Jeffrey A. Sosman, MD
## Results: Patient Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Patients With NRAS-Mutant Melanoma (n = 21)</th>
<th>Patients With KRAS-Mutant Pancreatic Cancer (n = 1)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age, median (range), y</strong></td>
<td>58 (21-78)</td>
<td></td>
</tr>
<tr>
<td><strong>Sex, n (%)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>13 (59)</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>9 (41)</td>
<td></td>
</tr>
<tr>
<td><strong>ECOG performance score, n (%)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>9 (41)</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>11 (50)</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>2 (9)</td>
<td></td>
</tr>
<tr>
<td><strong>Elevated LDH level at study entry, n (%)</strong></td>
<td>12 (55)</td>
<td></td>
</tr>
<tr>
<td><strong>Disease stage at study entry</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M1a</td>
<td>3 (14)</td>
<td></td>
</tr>
<tr>
<td>M1b</td>
<td>0 (0)</td>
<td></td>
</tr>
<tr>
<td>M1c</td>
<td>17 (81)</td>
<td></td>
</tr>
<tr>
<td>IIIIC</td>
<td>1 (5)</td>
<td></td>
</tr>
<tr>
<td>NA</td>
<td>1(NA)</td>
<td></td>
</tr>
<tr>
<td><strong>Patients with CNS metastases, n (%)</strong></td>
<td>2 (9)</td>
<td></td>
</tr>
<tr>
<td><strong>Prior systemic anticancer therapies, n (%)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-1</td>
<td>13 (59)</td>
<td></td>
</tr>
<tr>
<td>2-3</td>
<td>7 (32)</td>
<td></td>
</tr>
<tr>
<td>≥ 4</td>
<td>2 (9)</td>
<td></td>
</tr>
<tr>
<td><strong>Patients with prior immunotherapy, n (%)</strong></td>
<td>11 (50)</td>
<td></td>
</tr>
</tbody>
</table>

*CNS, central nervous system; ECOG, Eastern Cooperative Oncology Group; LDH, lactate dehydrogenase; NA, not applicable.*

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*a Calculated for patients with melanoma only; NA includes patient with KRAS-mutant pancreatic cancer.*

Presented by: Jeffrey A. Sosman, MD
## Results: Safety

### Treatment-Related Adverse Events (≥ 20% of All Patients)

<table>
<thead>
<tr>
<th>Adverse Event, n (%)</th>
<th>LEE011 200 mg + Binimetinib 45 mg (n = 9)</th>
<th>LEE011 250 mg + Binimetinib 45 mg (n = 3)</th>
<th>LEE011 300 mg + Binimetinib 30 mg (n = 4)</th>
<th>LEE011 300 mg + Binimetinib 45 mg (n = 6)</th>
<th>All (N = 22)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All Grades</td>
<td>Grade 3/4</td>
<td>All Grades</td>
<td>Grade 3/4</td>
<td>All Grades</td>
</tr>
<tr>
<td>CPK elevation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>4 (44)</td>
<td>1 (11)</td>
<td>2 (67)</td>
<td>0</td>
<td>3 (75)</td>
</tr>
<tr>
<td>Acneiform dermatitis</td>
<td>6 (67)</td>
<td>0</td>
<td>1 (33)</td>
<td>1 (33)</td>
<td>1 (25)</td>
</tr>
<tr>
<td>Nausea</td>
<td>4 (44)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2 (50)</td>
</tr>
<tr>
<td>Rash</td>
<td>2 (22)</td>
<td>0</td>
<td>2 (67)</td>
<td>0</td>
<td>1 (25)</td>
</tr>
<tr>
<td>Edema</td>
<td>3 (33)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1 (25)</td>
</tr>
<tr>
<td>Creatinine elevation</td>
<td>1 (11)</td>
<td>0</td>
<td>1 (33)</td>
<td>0</td>
<td>2 (50)</td>
</tr>
<tr>
<td>Leukopenia</td>
<td>3 (33)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1 (25)</td>
</tr>
<tr>
<td>Neutropenia</td>
<td>2 (22)</td>
<td>2 (22)</td>
<td>1 (33)</td>
<td>0</td>
<td>2 (50)</td>
</tr>
<tr>
<td>ALT elevation</td>
<td>2 (22)</td>
<td>1 (11)</td>
<td>1 (33)</td>
<td>0</td>
<td>1 (25)</td>
</tr>
<tr>
<td>AST elevation</td>
<td>2 (22)</td>
<td>0</td>
<td>1 (33)</td>
<td>0</td>
<td>1 (25)</td>
</tr>
<tr>
<td>Anemia</td>
<td>2 (22)</td>
<td>0</td>
<td>1 (33)</td>
<td>0</td>
<td>1 (25)</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>3 (33)</td>
<td>0</td>
<td>1 (33)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Fatigue</td>
<td>3 (33)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2 (50)</td>
</tr>
</tbody>
</table>

- No clinically significant QTcF prolongation observed

ALT, alanine aminotransferase; AST, aspartate aminotransferase; CPK, creatine phosphokinase; QTcF, Friderica-corrected QT interval.

Presented by: Jeffrey A. Sosman, MD
Dose Limiting Toxicities

• LEE011 200 mg + binimetinib 45 mg (n = 1):
  – Grade 3 acute renal injury

• LEE011 300 mg + binimetinib 45 mg (n = 2):
  – Grade 4 atrial fibrillation and grade 3 edema
    • Cardiomyopathy and death: Patient, entering study with multiple comorbid conditions, experienced several treatment-related toxicities and died suddenly due to cardio-pulmonary arrest after 2+ months of treatment. Cause of death unclear
  – Grade 4 asymptomatic CPK elevation

• LEE011 300 mg + binimetinib 30 mg (n = 1):
  – Intracranial bleed and death: Patient with early signs of clinical response, normal coagulation parameters, and no known risk factors for bleeding, died of intracranial hemorrhage (ICH) on cycle 1, day 11. Cause of ICH suspected to be due to undetected intracranial metastases

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*a Creatinine > 3-fold higher than baseline or > 4.0 mg/dL; hospitalization indicated.
CPK, creatine phosphokinase; DLT, dose limiting toxicity.

Presented by: Jeffrey A. Sosman, MD
Several patients had early tumor shrinkage with major symptomatic improvement.

*a* Includes 1 patient with KRAS-mutant pancreatic cancer.

RECIST, Response Evaluation Criteria In Solid Tumors.

Presented by: Jeffrey A. Sosman, MD
Results: Duration of Treatment Exposure

Patients remaining in the study (n = 12, 55%) have had exposure to the study drugs ranging 2 to 8 months.

- Arrowhead indicates patient ongoing as of April 14, 2014.
- K denotes patient with KRAS-mutant pancreatic cancer.

Presented by: Jeffrey A. Sosman, MD
Results: Patient Case Studies

Patients with NRAS Q61R–Mutant Melanoma Treated with LEE011 + Binimetinib

Pretreatment

31-yo male, previously untreated, with bleeding gastric mass

Week 8

65-yo female, previously treated with ipilimumab 10mg/kg adjuvant with PD, then MK-3475 with PD

PD, progressive disease; yo, year old.

Presented by: Jeffrey A. Sosman, MD
10 of 15 patient tumors (67%) showed concurrent mutation/loss of NRAS and CDKN2A

- 4 samples also showed CDKN2B loss
- 2 samples had a mutation in PIK3CA

Presented by: Jeffrey A. Sosman, MD
Conclusions

• Combined LEE011 and binimetinib demonstrated promising preliminary antitumor activity in patients with advanced NRAS-mutant melanoma

• Common treatment-related AEs included elevated CPK and creatinine; skin, hematologic, and GI events; edema; and fatigue
  – The combination was associated with frequent AEs, necessitating dosing interruptions and reductions

• Pharmacokinetics of the combination was generally comparable to that of the single agents, suggesting lack of drug-drug interaction

• The MTD for the current dosing schedule was determined to be LEE011 200 mg qd (3-weeks-on, 1-week-off schedule) and binimetinib 45 mg bid (continuous schedule)

• Exploration of intermittent dosing schedules to establish a more tolerable RP2D and further analysis of effect of additional genetic alterations (FM) on clinical outcome are underway

AE, adverse event; bid, twice daily; CPK, creatine phosphokinase; FM, Foundation Medicine; GI, gastrointestinal; MTD, maximum tolerated dose; qd, once daily; RP2D, recommended phase 2 dose.

Presented by: Jeffrey A. Sosman, MD
Phase Ib/II Trametinib + Palbociclib

Part 1
Dose Finding

- 3+3 dose escalation
- Dose Level 1A
- Dose Level 2A
- Dose Level 2B
- Dose Level 3A
- Dose Level 3B
- PK, safety
- RCR

Part 2
PD

- BRAF^{WT}/NRAS^{MUT}
  Cutaneous Melanoma
- BRAF^{WT}/NRAS^{WT}
  Cutaneous Melanoma

Part 3
Phase II

- BRAF^{WT}/NRAS^{TBD}
  Cutaneous Melanoma
- Randomize

- n = TBD
  Combination
- n = TBD
  trametinib

IF RR ≥10%
- continue enrollment
- n = TBD
  Combination
- n = TBD
  trametinib
Tumor genetics and immunotherapy

- Does genetics play a role in response to immune therapy?
- BRAF V600E mutation influences VEGF and IL-10 production
- BRAF inhibition also induces tumor infiltrating lymphocytes and may also affect PD-L1
- PTEN loss induces PD-L1 expression in glioblastoma

- If so...
  - Improved patient selection
  - Clues about other biomarkers
  - Clinical testing implications

Tompers Frederick et al, CCR 2013
NRAS mutation and immune based therapy

- NRAS mutant melanoma may have improved prognosis in patients treated with IL-2
- Not clear whether this effect is seen with checkpoint inhibitors
- Unclear mechanism

<table>
<thead>
<tr>
<th>Variable</th>
<th>NRAS n = 15</th>
<th>BRAF and WT n = 88</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>CR/PR</td>
<td>7 (47%)</td>
<td>27 (19%)</td>
<td>0.04*</td>
</tr>
<tr>
<td>OS (y)</td>
<td>5.3</td>
<td>2.4</td>
<td>0.30†</td>
</tr>
<tr>
<td>PFS (d)</td>
<td>214</td>
<td>70</td>
<td>0.13†</td>
</tr>
</tbody>
</table>

*Fisher exact test.
†Log-rank test.
CR indicates complete responder; OS, overall survival; PFS, progression-free survival; PR, partial responder; SD, stable disease.

Joseph et al, J. Immunotherapy 2012
## Response Rates

### Best Response to Any Line of Immune based therapy

<table>
<thead>
<tr>
<th></th>
<th>NRAS mutant (n=60)</th>
<th>WT (n=116)</th>
<th>BRAF mutant (n=53)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>CR/PR</td>
<td>20 (33%)</td>
<td>21 (18%)</td>
<td>12 (23%)</td>
<td>$p = 0.03$</td>
</tr>
<tr>
<td>SD/ PD</td>
<td>40 (67%)</td>
<td>95 (82%)</td>
<td>41 (77%)</td>
<td></td>
</tr>
<tr>
<td>CR/PR/SD</td>
<td>30 (50%)</td>
<td>34 (29%)</td>
<td>16 (30%)</td>
<td>$p &lt; 0.01$</td>
</tr>
<tr>
<td>PD</td>
<td>30 (50%)</td>
<td>82 (71%)</td>
<td>37 (70%)</td>
<td></td>
</tr>
</tbody>
</table>

Johnson et al, ASCO 2013
# Response Rates

## Responses by Immune Therapy Type

<table>
<thead>
<tr>
<th></th>
<th>NRAS mutant</th>
<th>BRAF mutant</th>
<th>WT</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anti PD-1/PD-L1</strong></td>
<td>N = 11</td>
<td>N = 14</td>
<td>N = 23</td>
</tr>
<tr>
<td>Objective Response</td>
<td>8 (73%)</td>
<td>3 (21%)</td>
<td>7 (30%)</td>
</tr>
<tr>
<td>Clinical Benefit</td>
<td>8 (73%)</td>
<td>3 (21%)</td>
<td>9 (41%)</td>
</tr>
<tr>
<td><strong>Ipilimumab</strong></td>
<td>N = 43</td>
<td>N = 31</td>
<td>N = 95</td>
</tr>
<tr>
<td>Objective Response</td>
<td>8 (19%)</td>
<td>4 (13%)</td>
<td>10 (11%)</td>
</tr>
<tr>
<td>Clinical Benefit</td>
<td>18 (42%)</td>
<td>5 (16%)</td>
<td>19 (20%)</td>
</tr>
<tr>
<td><strong>IL-2</strong></td>
<td>N = 15</td>
<td>N = 29</td>
<td>N = 19</td>
</tr>
<tr>
<td>Objective Response</td>
<td>5 (33%)</td>
<td>6 (21%)</td>
<td>5 (26%)</td>
</tr>
<tr>
<td>Clinical Benefit</td>
<td>5 (33%)</td>
<td>11 (34%)</td>
<td>7 (37%)</td>
</tr>
</tbody>
</table>

Johnson D. et al, ASCO 2013
Fig 3: a) Dose levels and b) study schema of pembrolizumab, binimetinib, and LEE011

**Dose levels**

<table>
<thead>
<tr>
<th></th>
<th>Binimetinib 45mg bid</th>
<th>LEE011 300mg daily</th>
<th>Pembrolizumab 200mg IV q3W (all patients)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Binimetinib 30mg bid</td>
<td>LEE011 250mg daily</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Binimetinib 30mg bid</td>
<td>LEE011 200mg daily</td>
<td></td>
</tr>
</tbody>
</table>

**Study Schema**

- **Week 0**: Biopsy, Peripheral blood
- **Week 1**: Pembrolizumab
- **Week 2**: Pembrolizumab + LEE011
- **Week 3**: Pembrolizumab + LEE011
- **Week 4**: Pembrolizumab
- **Week 5**: Pembrolizumab + LEE011
- **Week 6**: Pembrolizumab + LEE011
- **Week 7**: Pembrolizumab
- **Week 8**: Pembrolizumab + LEE011
- **Week 9**: Pembrolizumab + LEE011

**Criteria for Dose Level Changes**

- If 0-1 DLTs: Early Expansion Cohort Enroll 10 patients
- If 2+ DLTs: Decrease binimetinib 1 dose level, continue LEE011, N = 3
- Decrease LEE011 by 1 dose level, continue binimetinib, N = 3

**Treatment Regimen**

- Treatment for 2 years, progression, or unacceptable toxicity
- Disease assessment q12 weeks
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Su2C MRA-AACR Melanoma Dream Team