Genes & Cancer Predisposition

• 5-10% of cancers attributable to mutations in specific genes inherited from biological parents
  – Breast and ovarian cancers – BRCA1, BRCA2
  – Li-Fraumeni Syndrome – P53 gene

• Most cancer causes by somatic mutations of genes and other biomarkers
Example from lab to clinic – imatinib
The Promise of Targeted Therapy

• Begins in 1960 with discovery of “Philadelphia chromosome”

Normal Blood Smear

Chronic Myeloid Leukemia
The Philadelphia Chromosome Gene Translocation

Normal

9q+

Ph (or 22q-)

FUSION CANCER GENE

CML

9q+

FUSION CANCER GENE

bcr - abl

bcr

22

abl
Imatinib is a drug that blocks BCR-ABL

Goldman JM, Melo JV. NEJM. 344:1084-1086
Efficacy results

Important efficacy endpoints were cytogenetic response and complete hematologic response:

Primary Endpoint: Ph chromosome going away

- **Major cytogenetic response (MCyR)** was documented in 49% (265/532) and a **complete cytogenetic response (CCyR)** in 30% (160/532). Median time to MCyR was 3 months. Median response duration cannot be determined yet because of short follow-up.

Secondary Endpoint: Reduction in number of cancer cells in the blood

- **Complete hematologic response (CHR)** was documented in 88% (467/532). Median time to CHR was 22 days. Because of short follow-up, the median duration of CHR cannot be determined, but it must be a minimum of 6 months; more than half of entered patients (63%) were in CHR at the 6 month evaluation.
Media Reaction: “The magic bullet”
Imatinib: 10 years Later: GIST
2011 ASCO Annual Meeting Plenary

SSGXVIII: Recurrence-free survival (ITT)

- 36 Months
- 12 Months

Hazard ratio 0.46
(95% CI, 0.32-0.65)

$P < .0001$

No. at risk (n=397)

<table>
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<tr>
<th>Years</th>
<th>0</th>
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<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
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<th>7</th>
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<tbody>
<tr>
<td>36 Months of imatinib</td>
<td>198</td>
<td>184</td>
<td>173</td>
<td>133</td>
<td>82</td>
<td>39</td>
<td>8</td>
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<tr>
<td>12 Months of imatinib</td>
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<td>177</td>
<td>137</td>
<td>88</td>
<td>49</td>
<td>27</td>
<td>10</td>
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</tr>
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</table>

Median follow-up time 54 months
Major Challenges in Completing Cancer Molecular Map

1. Sequencing exomes vs. whole genomes
2. Differentiating between driver & passenger mutations in genetically unstable tumors
3. Integrating data across platforms
4. Finding solutions to problems associated with multiple testing

Tran B “Cancer Genomics: Technology, Discovery, and Translation” JCO 30:6, February 20, 2012
Genomics – From Research to Clinic

- Multiple targets, continue to mutate, and mutations alone don’t dictate behavior
- Pace of genomic discovery is rapid, but development of agents is not
- Hype for transforming cancer care significant, but can we deliver?
- Quality diagnostics at the community level?
- How do we help general oncologists keep up?
Project to Recommend Resources
Membership Need

- Group of ASCO leaders in training currently investigating genomics resources for non-academic oncologists

<table>
<thead>
<tr>
<th>Interviewee</th>
<th>N = 31</th>
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<tr>
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<tr>
<td>Academic Med Onc</td>
<td>1</td>
</tr>
<tr>
<td>ASCO Clinical Practice Committee Leadership</td>
<td>4</td>
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<tr>
<td>Academic Surg Onc</td>
<td>2</td>
</tr>
<tr>
<td>Academic Pathology</td>
<td>2</td>
</tr>
</tbody>
</table>
Current Resources

• New evidence emerging at overwhelming pace
• Difficult to distinguish what is clinically actionable
• No standard resources currently available
• Use path reports & drug/device reps, but “doesn’t seem right”
What is Needed

• Simple, searchable, clinically relevant, updated, user-friendly, patient-friendly
  – “not laundry list”

• Guideline format

• Organized by FDA approved
Type of Resource Desired

• Website (majority)
  – “I’d find it, bookmark it, and use it”
  – Mobile Device App

• Interactive spreadsheet

• Decision support tools embedded in EMR
Education & Training
ACGME Program Requirements for Medical Oncology
Approved: February 5, 2011; Effective: July 1, 2012

• Must demonstrate knowledge of genetics and developmental biology, including:
  – molecular genetics;
  – the nature of oncogenes and their products; and,
  – cytogenetics

• Must demonstrate knowledge of physiology and pathophysiology, including
  – principles of oncogenesis; and, tumor immunology
  – immune markers, immunophenotyping, flow
  – cytometry, cytochemical studies, and
  – cytogenetic and DNA analysis of neoplastic disorders; …and
  – gene therapy

ASCO-Self Evaluation Program®

• Print, e-Book, and mobile products
  – Online Question Bank
  – Downloadable app
  – ASCO Flashcards

• Chapters on Molecular Biology and Biologic Therapy
2013 ASCO Annual Meeting

• 1.5 day Seminar: “The Genetics and Genomics for the Practicing Clinician”
  o Topics: tumor and germline DNA, available diagnostic tests (clinical utility and timing)

• Several 75-minute Educational Sessions, e.g.
  o Squamous Non-Small Cell Lung Cancer: Understanding the Genomics, Treatment and Outcomes
  o Bridging Science and Clinical Practice: How to Use Molecular Markers When Caring for a Patient with Brain Cancer
  o Barriers in Expanding Access to Genomic Information
ASCO University

• eLearning center that hosts online, digital and app-based educational products

• CME-accredited courses
  – Cancer Genetics Review
  – Molecular Oncology Review
  – Genetic Testing in Oncology
ASCO Practice Guideline Development Process

• Systematic Review as the Basis for ASCO Guidelines

• Formal Consensus Development Process Used with Insufficient Evidence

• Focus on Evidence That Informs the Clinical Utility of Genomic Assay Results
Recent Guideline Collaborations

- ASCO-College of American Pathologists (CAP) HER-2 Testing Guideline
- ASCO-CAP ER/PR Testing Guideline
- CAP-Association for Molecular Pathology-ASCO Colorectal Cancer Markers Guideline
Policy on Release of Joint Guidelines

- Concurrent Posting, Publication of Joint Guidelines on Respective Website or Journal
- Careful Coordination with Sister Societies Around Press Releases, Communication Strategy
ASCO Plans for Rapid Learning System

- Embed genomic information
- Provide clinical decision support
- Assemble and analyze data from multiple practices
- Evaluate trends and associations to form hypotheses
Next Steps for ASCO

- Recommendations to ASCO Board May 2013 from Leadership Development Program
- Incorporate genomics into RLS plans
- Discussions with other societies, e.g., pathologists
- Involvement in follow-up from today’s meeting
Traditional vs. Molecular View of Cancer

Melanoma

- Arising from Skin Without Chronic Sun Damage
- Arising from Skin With Chronic Sun Damage
- Arising from Mucosal Surfaces
- Arising from Acral Surfaces

Vanderbilt Molecular Profiling
784 Samples
7-1-10 To 1-8-13

- BRAF 40.3%
- NRAS 17.4%
- No Mutation Detected 33.7%
- KIT 2.8%
- GNAQ 3.4%
- GNA11 1.5%
- CTNNB1 0.9%
Impact at Vanderbilt-Ingram

Melanoma patients with advanced disease and mutation detected

- 61% went onto clinical trial
- 64% received gene directed therapy

2,115 Specimens tested
July 2010 – Dec 2012

Lung Cancer
Melanoma
Breast Cancer
Colorectal Cancer
Other
“Three new surveys… reveal a disconnect in the understanding of and communication about genetic mutation testing among healthcare professionals and cancer patients.”
Old Method for Reporting Mutation Results in the Electronic Medical Record

Old Method:
• Report Template
• Scanned into Electronic Health Record as image file (not computable)

Challenges:
• How to report > 40 mutations in 8 genes?
• Whose role to curate knowledge regarding clinical significance?
• Lack clinical trial information

The epidermal growth factor receptor (EGFR) gene, mapped to 7p12, encodes a transmembrane glycoprotein that is a member of the protein kinase superfamily. EGFR protein is expressed on the cell surface and as a receptor, binds to epidermal growth factor (EGF). The protein-ligand interaction induces receptor dimerization and tyrosine autophosphorylation resulting in cell proliferation. Somatic mutations in the tyrosine kinase-binding domain of the EGFR gene are associated with non-small cell lung carcinoma, primarily moderately to well-differentiated adenocarcinoma. EGFR mutations have been observed in approximately 10% of lung adenocarcinomas in patients from the United States and are significantly associated with Asian ethnicity, female gender and never-smokers.

ERBB2 is a member of the EGFR family of receptor tyrosine kinases and plays important roles in the pathogenesis of several human cancers. Somatic mutations in the form of in-frame duplications and/or deletions in a small stretch of exon 20 have been reported in non-small cell lung carcinomas. Of interest, exon 20 insertion mutations in ERBB2 or EGFR are significantly more prevalent in the same subpopulations in which other EGFR mutations occur.

Progressive and/or metastatic non-small cell lung carcinomas can be treated with inhibitors of the EGFR receptor. Somatic mutations in the tyrosine kinase domain of the EGFR gene present in lung adenocarcinomas can affect a patient’s response to EGFR inhibitors. 90% of EGFR mutations in this population include short in-frame deletions in exon 19 and a T > G point mutation in exon 21 at codon 858 (L858R). The presence of either mutation correlates with sensitivity to EGFR inhibitors. Conversely, insertion mutations in exon 20 of either ERBB2 or EGFR gene appear to be less responsive to therapy.

DNA extracted from this patient’s tumor was amplified for EGFR exons 19 and 20 and ERBB2 exon 20 using multiplex fluorescent PCR to detect small deletions or insertions. Detection of mutation L858R was performed using fluorescent PCR coupled with restriction endonuclease digestion with Sau96I. All amplicons were analyzed using capillary electrophoresis. An in frame deletion in exon 19 of the EGFR gene was detected.

In summary, the results of this study demonstrate that this patient does have an exon 19 deletion of the EGFR gene. The presence of this mutation indicates that this tumor is likely to be responsive to EGFR inhibitors. It is important to note that this assay is specific for these four mutations and does not rule out the presence of other EGFR or ERBB2 mutations that may be present but not detected by this assay which may affect treatment response.
<table>
<thead>
<tr>
<th>MR#</th>
<th>Patient Name</th>
<th>Actions</th>
<th>Tumor Gene Mutations</th>
</tr>
</thead>
<tbody>
<tr>
<td>03</td>
<td>81 A, B M.</td>
<td>Actions</td>
<td></td>
</tr>
<tr>
<td>03</td>
<td>56 A, P</td>
<td>Actions</td>
<td></td>
</tr>
<tr>
<td>03</td>
<td>35 B, J A</td>
<td>Actions</td>
<td></td>
</tr>
<tr>
<td>01</td>
<td>80 B, S A</td>
<td>Actions</td>
<td></td>
</tr>
<tr>
<td>02</td>
<td>29 E, J E</td>
<td>Actions</td>
<td></td>
</tr>
<tr>
<td>02</td>
<td>27 F, R M</td>
<td>Actions</td>
<td></td>
</tr>
<tr>
<td>02</td>
<td>77 G, T</td>
<td>Actions</td>
<td></td>
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<tr>
<td>02</td>
<td>73 H, A</td>
<td>Actions</td>
<td></td>
</tr>
<tr>
<td>03</td>
<td>64 S, C</td>
<td>Actions</td>
<td></td>
</tr>
<tr>
<td>02</td>
<td>79 S, A S</td>
<td>Actions</td>
<td></td>
</tr>
<tr>
<td>02</td>
<td>40 W, J E I</td>
<td>Actions</td>
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<tr>
<td>03</td>
<td>74 W, C L</td>
<td>Actions</td>
<td></td>
</tr>
</tbody>
</table>

- **BRAF c.1798_1799GT>AG (V600R)** Not Detected
- **BRAF c.1798_1799GT>AA (V600K)** Not Detected
- **BRAF c.1799T>A (V600E)** Detected
- **BRAF c.1799_1800TG>AA (V600E)** Not Detected
- **BRAF c.1798G>A (V600M)** Not Detected
- **BRAF c.1799T>G (V600G)** Not Detected
- **BRAF c.1799_1800TG>AT (V600D)** Not Detected
BRAF c.1799T>A (V600E) Mutation in Melanoma

Properties

<table>
<thead>
<tr>
<th>Location of mutation</th>
<th>Kinase domain (exon 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency of BRAF mutations in melanoma</td>
<td>44% \textit{(COSMIC)}</td>
</tr>
<tr>
<td>Frequency of V600E mutation among BRAF mutant melanomas</td>
<td>\textasciitilde 85–90%</td>
</tr>
</tbody>
</table>

Implications for Targeted Therapeutics

<table>
<thead>
<tr>
<th>Response to BRAF inhibitors</th>
<th>Confers increased sensitivity\textasciitilde</th>
</tr>
</thead>
<tbody>
<tr>
<td>Response to MEK inhibitors</td>
<td>Unknown at this time\textsuperscript{b}</td>
</tr>
<tr>
<td>Response to KIT inhibitors</td>
<td>Unknown at this time</td>
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</table>

BRAF V600E mutation

<table>
<thead>
<tr>
<th>Treatment Agent</th>
<th>Drug Class</th>
<th>Line of Treatment</th>
<th># pts in study</th>
<th>Response Rate</th>
<th>PFS (months)</th>
<th>OS (months)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vemurafenib (PLX4032)</td>
<td>Mutated BRAF TKI\textsuperscript{c}</td>
<td>1st to &gt;3rd</td>
<td>32\textsuperscript{c}</td>
<td>81%</td>
<td>&gt;7 (estimated)</td>
<td>Not reached</td>
<td>Flaherty et al. 2010</td>
</tr>
<tr>
<td></td>
<td>Mutated BRAF TKI\textsuperscript{c}</td>
<td>1st</td>
<td>337</td>
<td>48%</td>
<td>5.3</td>
<td>84% at 6 mos</td>
<td>Chapman et al. 2011</td>
</tr>
<tr>
<td>Dacarbazine</td>
<td>Cytotoxic chemotherapy</td>
<td>1st</td>
<td>338</td>
<td>5%</td>
<td>1.6</td>
<td>64% at 6 mos</td>
<td>Chapman et al. 2011</td>
</tr>
</tbody>
</table>

\textsuperscript{c} This trial intended to include only patients with BRAF V600E mutations.
BRAF V600E (c.1799T>A) mutation in Melanoma

**BRAF V600E mutation**

<table>
<thead>
<tr>
<th>Treatment Agent</th>
<th>Drug Class</th>
<th>Line of Treatment</th>
<th># pts in study</th>
<th>Response Rate</th>
<th>PFS (months)</th>
<th>OS (months)</th>
<th>Level of evidence</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>vemurafenib</td>
<td>Mutated BRAF TKI</td>
<td>1st to 3rd</td>
<td>32</td>
<td>81%</td>
<td>&gt; 7 months (estimated)</td>
<td>Not reached</td>
<td>II-1</td>
<td>(Flaherty, 2010)</td>
</tr>
<tr>
<td>vemurafenib</td>
<td>Mutated BRAF TKI</td>
<td>1st</td>
<td>337</td>
<td>48%</td>
<td>5.3</td>
<td>84% at 6 mos</td>
<td>I</td>
<td>(Chapman, 2011)</td>
</tr>
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<td>dacarbazine</td>
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<td>I</td>
<td>(Chapman, 2011)</td>
</tr>
</tbody>
</table>

The V600E mutation results in an amino acid substitution at position 600 in BRAF, from a Valine (V) to a glutamic acid (E). This mutation occurs within the activation segment of the kinase domain (Fig. 2). Approximately 70-90% of V600 BRAF mutations are V600E (Rubinstein, 2010). Mutant BRAF proteins have increased kinase activity and are transforming in vitro (Davies, 2002). BRAF mutations are usually found in tumors wildtype for NRAS, KIT, and other driver mutations.

In the initial phase I trial, patients with metastatic melanoma whose tumor harbored a BRAF V600E mutation displayed an 81% response rate to vemurafenib (PLX4032), an orally available inhibitor of mutated BRAF. The estimated progression-free survival was > 7 months and overall survival had not been reached at the time of study publication (Flaherty, 2010). In the follow-up randomized phase III trial comparing vemurafenib to dacarbazine in previously untreated, metastatic melanoma with the BRAF V600E mutation, vemurafenib improved rates of overall survival and progression-free survival (Chapman, 2011).

Pre-clinical data has correlated the presence of activating mutations in BRAF with sensitivity to non-ATP competitive MEK inhibitors, AZD6244 and CI-1040 (Davies, 2007; Sliwkowski, 2006). In a Phase II clinical trial of AZD6244 versus temozolomide, 5 of 42 melanoma patients with BRAF V600E mutation had confirmed partial responses (12% objective response rate) (Ommen, 2009).
BRAF Mutation Directed Melanoma Clinical Trials

Great effort was made to include all clinical trials relevant for this mutation. However, the completeness of this information cannot be guaranteed.

At Vanderbilt (4)

<table>
<thead>
<tr>
<th>Protocol No.</th>
<th>Title</th>
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</thead>
<tbody>
<tr>
<td>VICCPHI1075</td>
<td>A Phase Ib, Open Label, Dose-Escalation, Study Evaluating the Safety, Tolerability and Pharmacokinetics of RO5185426 in Combination with GDC-0973 when Administered in Patients with BRAFV600E-Positive Metastatic Melanoma Who Have Progressed After Treatment with RO5185426</td>
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<tr>
<td>06/01/2011</td>
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<tr>
<td>VICCMEL1091</td>
<td>BRF113929: A Phase II Open-Label, Two-Cohort, Multicentre Study of GSK2118436 as a Single Agent in Treatment Naive and Previously Treated Subjects with BRAF Mutation-Positive Metastatic Melanoma to the Brain</td>
</tr>
<tr>
<td>Pending</td>
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<tr>
<td>VICCPHI1076</td>
<td>A Phase I, Randomized, Open-Label, Multi-Center, Two Period Crossover Study to Investigate the Effect of Food on the Pharmacokinetics of a Single Oral Dose of RO5185426, Followed by Administration of 960mg RO5195426 Twice Daily to BRAF-V600E Positive Metastatic Melanoma Patients</td>
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<tr>
<td>Pending</td>
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</tr>
<tr>
<td>VICCMEL1083</td>
<td>An Open-Label, Dose-Escalation, Phase I/II Study to Investigate the Safety, Pharmacokinetics, Pharmacodynamics, and Clinical Activity of the BRAF Inhibitor GSK2118436 in Combination with the MEK Inhibitor GSK1120212 in Subjects with BRAF Mutant Metastatic Melanoma</td>
</tr>
</tbody>
</table>

Melanoma Clinical Trials at Vanderbilt (7)

Tennessee (4)

United States (13)

Internationally (12)
Clinical Trial VICCPHI1075

Title
A Phase Ib, Open Label, Dose-Escalation, Study Evaluating the Safety, Tolerability and Pharmacokinetics of RO5185426 in Combination with GDC-0973 when Administered in Patients with BRAFV600E-Positive Metastatic Melanoma Who Have Progressed After Treatment with RO5185426

Principal Investigator(s)
Igor Puzanov

Description
The purpose of this study is to test the combination of the investigational drugs RO5185426 (BRAF inhibitor) and GDC-0973/XL518 (MEK inhibitor) in order to find a safe and tolerated dose when taking these drugs together.

Eligibility

Details

Learn more
- Call toll-free number: 1-800-811-8480
- Use our Online self-referral form
- Print this page for your doctor
<table>
<thead>
<tr>
<th>Protocol No.</th>
<th>Title</th>
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</thead>
<tbody>
<tr>
<td>NCT01271803</td>
<td>A Study of RO5185426 And GDC-0973 in Patients With BRAF-Mutation Positive Metastatic Melanoma</td>
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<tr>
<td>NCT01350401</td>
<td>Phase I/II Study to Assess the Safety and Activity of Enhanced TCR Transduced Autologous T Cells in Metastatic Melanoma</td>
</tr>
<tr>
<td>NCT01390818</td>
<td>Trial of MEK Inhibitor and PI3K/mTOR Inhibitor in Subjects With Locally Advanced or Metastatic Solid Tumors</td>
</tr>
<tr>
<td>NCT01136967</td>
<td>An Open-Label, 2-Cohort, Multicenter, Study of E7080 in Previously Treated Subjects With Unresectable Stage III or Stage IV Melanoma</td>
</tr>
<tr>
<td>NCT00866177</td>
<td>Phase II Study of MEK Inhibitor AZD6244 in Patients With BRAF-Mutated or NRAS-Mutated, Unresectable Stage III or IV Melanoma</td>
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<tr>
<td>NCT00948467</td>
<td>Study of TAK-733 in Adult Patients With Advanced Nonhematologic Malignancies</td>
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<tr>
<td>NCT01248936</td>
<td>A Study of RO5185426 in Patients With Metastatic Melanoma</td>
</tr>
<tr>
<td>NCT01266967</td>
<td>A Study of GSK2118436 in BRAF Mutant Metastatic Melanoma to the Brain</td>
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<tr>
<td>NCT01072175</td>
<td>Investigate Safety, Pharmacokinetics and Pharmacodynamics of GSK2118436 &amp; GSK1120212</td>
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</tbody>
</table>

**United States (13)**

**Internationally (12)**
Trial of MEK Inhibitor and PI3K/mTOR Inhibitor in Subjects With Locally Advanced or Metastatic Solid Tumors

This study is currently recruiting participants.
Verified on July 2011 by EMD Serono
First Received on April 18, 2011. Last Updated on July 8, 2011

Sponsor: EMD Serono
Collaborator: Sanofi-Aventis
Information provided by: EMD Serono
ClinicalTrials.gov Identifier: NCT01390818

Purpose

This research trial is testing a combination of two experimental drugs, MSC1936356B (Mitogen-activated protein extracellular signal-regulated kinase (Mek) Inhibitor) and SAR245409 (Phosphatidylinositol 3-kinase (PI3K)/Mammalian Target of Rapamycin (mTOR) inhibitor), in the treatment of locally advanced or metastatic solid tumours. The primary purpose of the study is to determine the maximum tolerated dose of the drug combination.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Intervention</th>
<th>Phase</th>
</tr>
</thead>
<tbody>
<tr>
<td>Locally Advanced Solid Tumor</td>
<td>Drug: MSC1936356B and SAR245409</td>
<td>Phase I</td>
</tr>
<tr>
<td>Metastatic Solid Tumor</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Study Type: Interventional
Study Design: Endpoint Classification: Safety/Efficacy Study
Intervention Model: Single Group Assignment
Masking: Open Label
Primary Purpose: Treatment

Official Title:
An Open-Label, Phase Ib Dose Escalation Trial of Oral Combination Therapy With MSC1936356B and SAR245409 in Subjects With Locally Advanced or Metastatic Solid Tumors

Resource links provided by NLM:
MedlinePlus related topics: Cancer
Drug Information available for: Sirolimus, Everolimus, CCI 779
U.S. FDA Resources

Further study details as provided by EMD Serono:
Find a Cancer Mutation

Disease (required): Select Disease
Gene (optional):
Variant (optional):

GO

Find Clinical Trials

Lists trials by Disease or Gene for all national and international trials registered within PDQ and clinicaltrials.gov.

Disease (optional): Enter a Disease
Gene (optional): Enter a Gene

GO

Molecular Medicine

› Articles of Interest
› List of Targeted Therapies

Clinical trial search
36,167 Cancer Trials (PDQ)
135 Cancer Diagnoses
437 Cancer Genes (COSMIC)

• 17 Cancers
• 25 Genes
• 289 Disease-Gene-Variant Relationships
Find a Cancer Mutation

Disease (required): Select Disease

Gene (optional): 

Variant (optional): 

Find Clinical Trials

Lists trials by Disease, gene, and/or variant. Ask your doctor about clinical trials registered with Vanderbilt-Ingram Cancer Center.

Disease (optional): Enter a Disease

Gene (optional): Enter a Gene

GO

Database of treatment outcomes of patients with rare mutations

Internet’s only complete list of targeted therapeutics

Molecular Medicine

› Articles of Interest
› List of Targeted Therapies
› Overview on Targeted Therapies for Cancer
› How Gene Alterations are Detected

Feedback

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More...
Worldwide Collaboration

- 49 Contributors
- 18 Institutions
- 8 Countries
Visits from 134 countries and territories
Heatmap of worldwide MCG visits: darker green = more visits