Sandra M. Swain, MD,
ASCO President & Medstar Washington Hsp Ctr

William Pao, MD, PhD
Co-Chair Genomics Seminar & Vanderbilt-Ingram Cancer Ctr

NIH Genomic Medicine 4

January 28, 2013

Genes & Cancer Predisposition

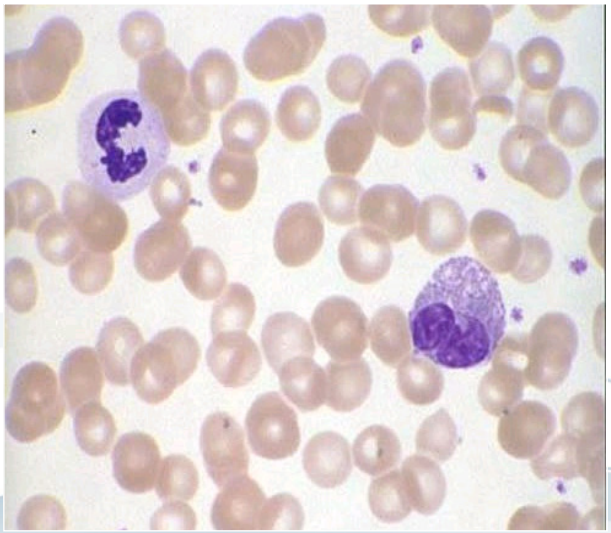
- 5-10% of cancers attributable to mutations in specific genes inherited from biological parents
  - Breast and ovarian cancers – BRCA1, BRCA2
  - Li-Fraumeni Syndrome – P53 gene
- Most cancer causes by somatic mutations of genes and other biomarkers

Example from lab to clinic – imatinib

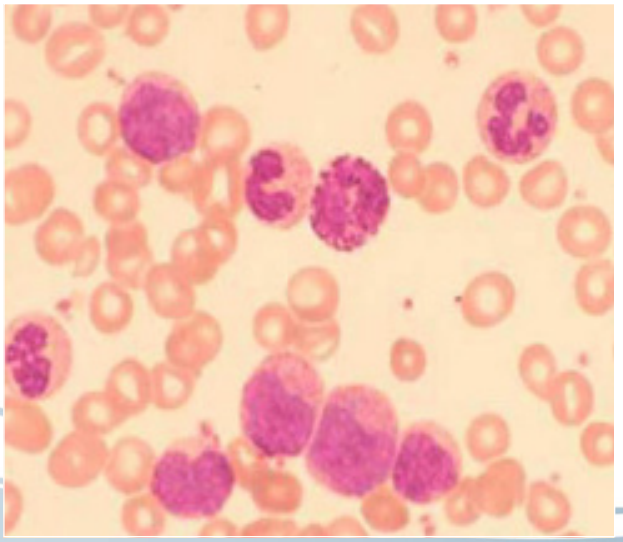
The Promise of Targeted Therapy

- **Begins in 1960 with discovery of "Philadelphia chromosome"**

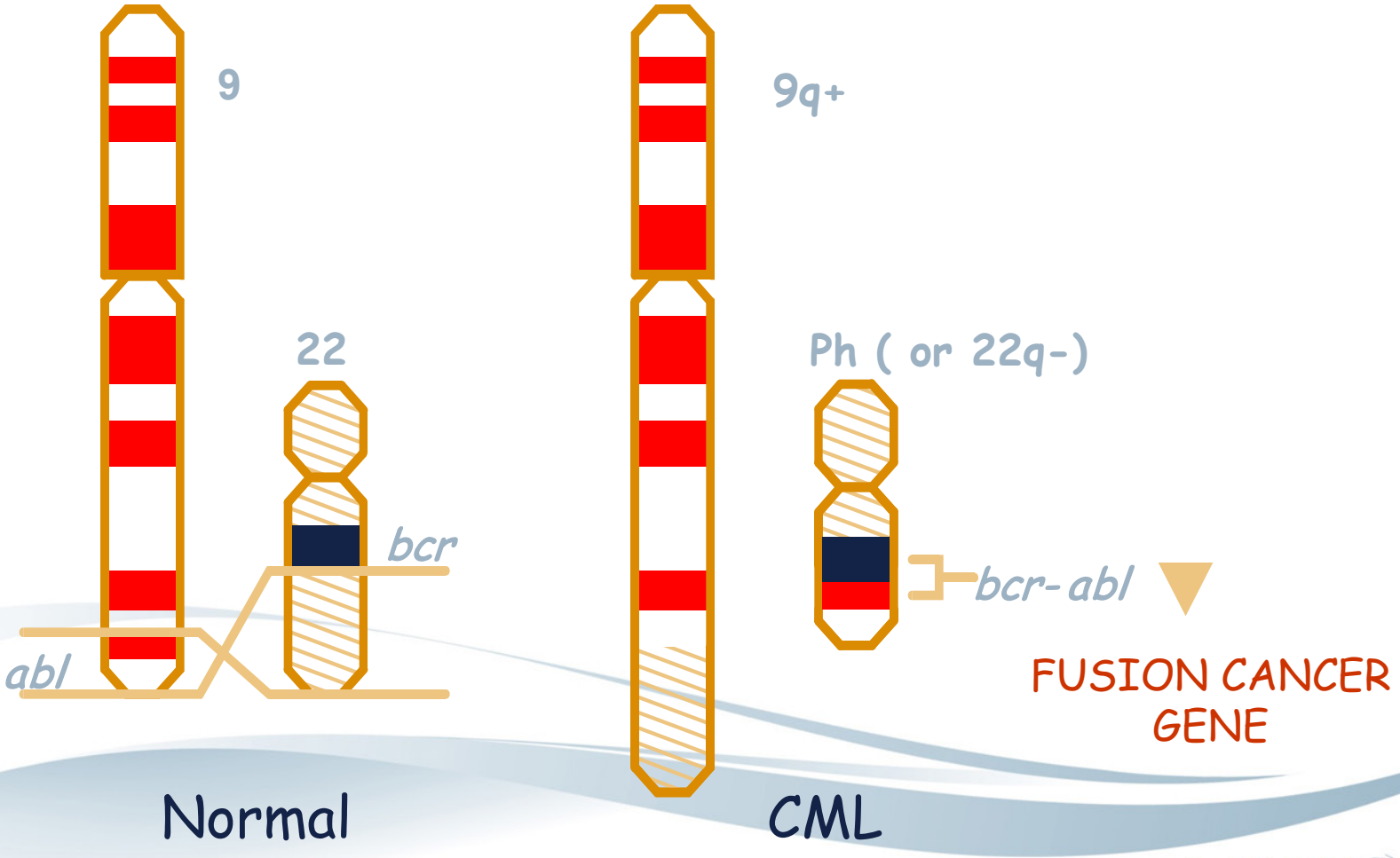
Normal Blood Smear



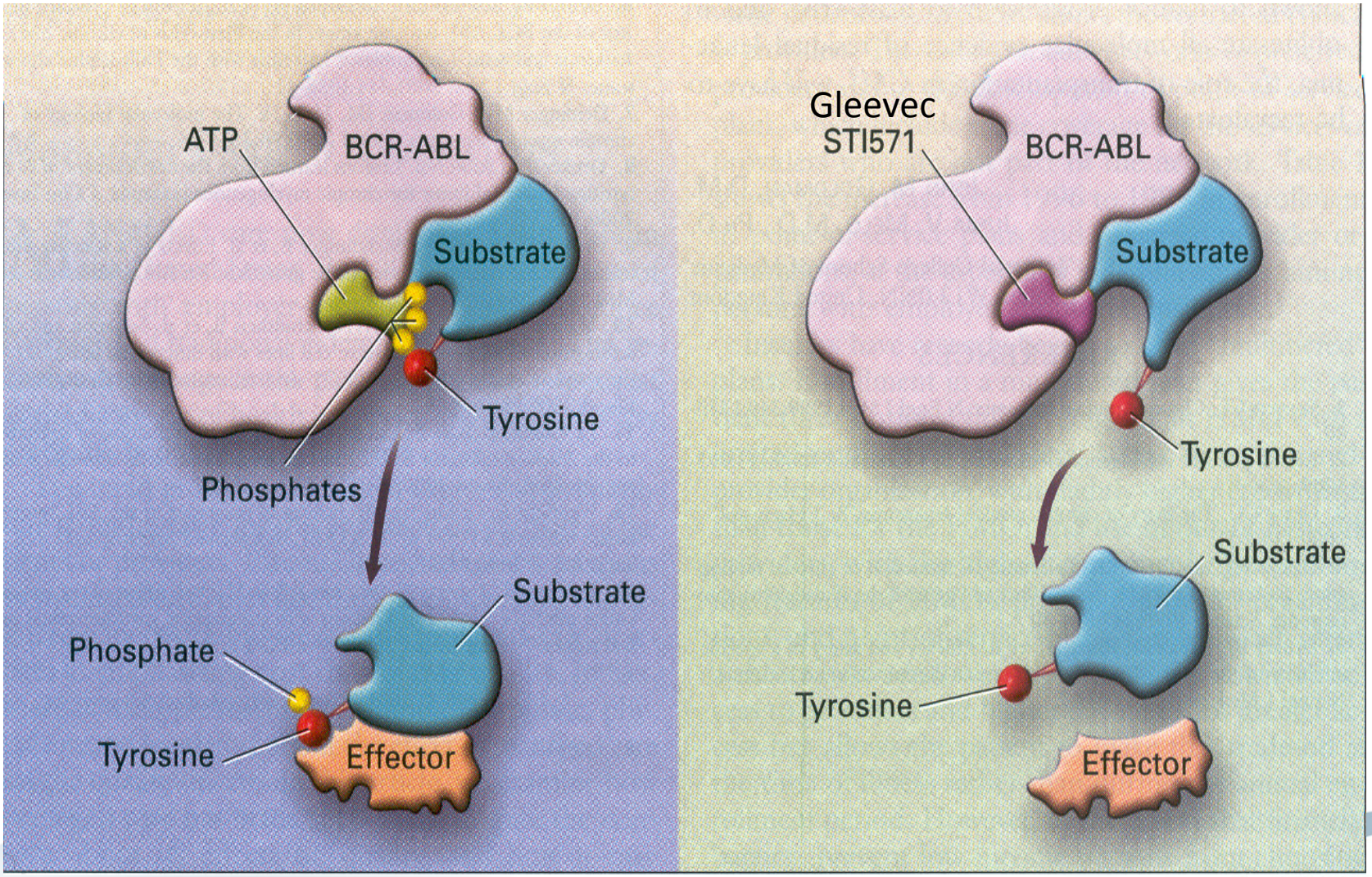
Chronic Myeloid Leukemia



The Philadelphia Chromosome Gene Translocation



Imatinib is a drug that blocks BCR-ABL



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

FDA Accelerated Approval
May 2001

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.**

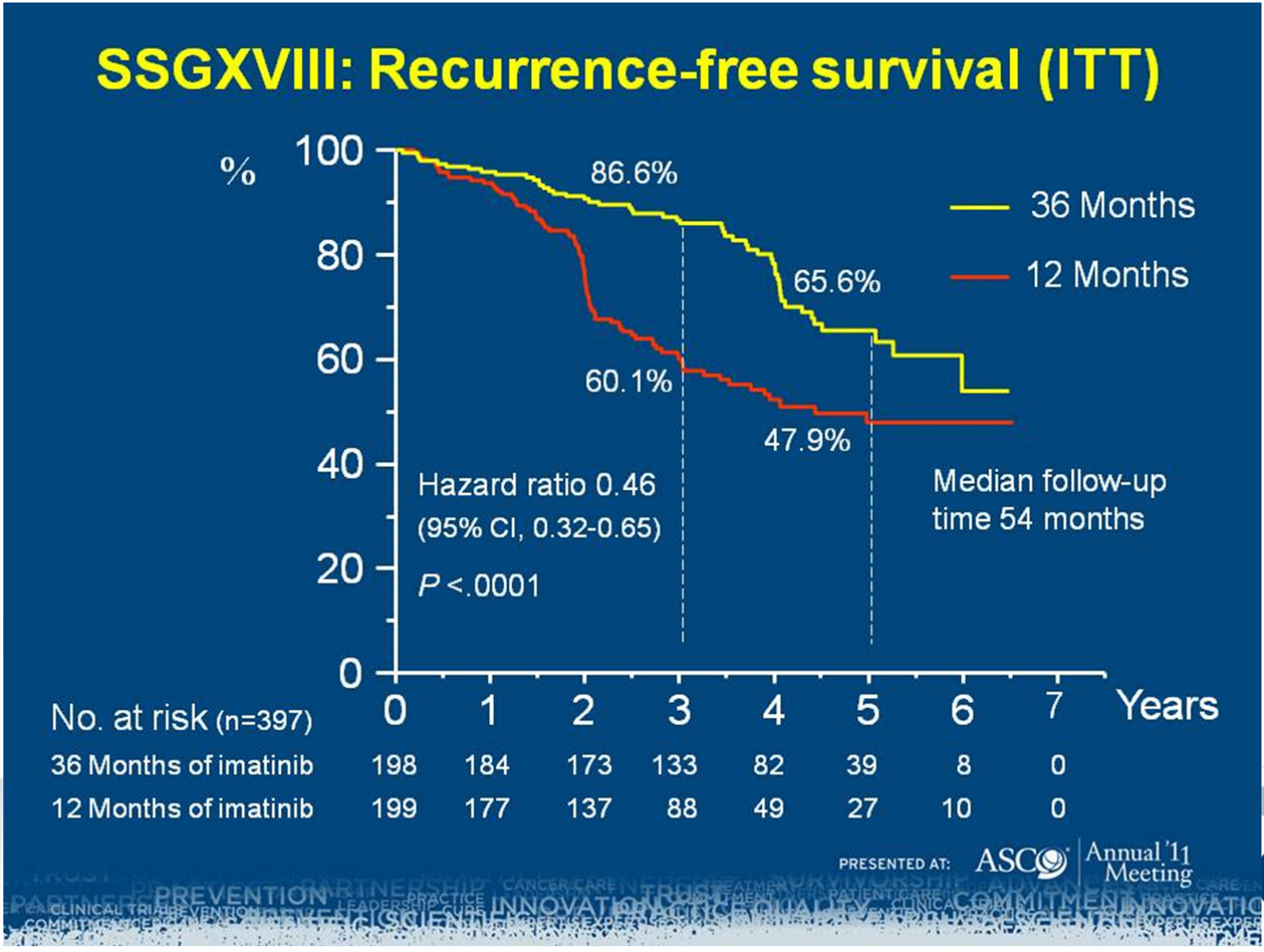
Secondary Endpoint: Reduction in number of cancer cells in the blood

- **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



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

Interviewee	N = 31
Community Med Onc	22
Academic Med Onc	1
ASCO Clinical Practice Committee Leadership	4
Academic Surg Onc	2
Academic Pathology	2

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

Accessed 12_17_12: http://www.acgme.org/acgmeweb/Portals/0/PFAssets/2013-PR-FAQ-PIF/147_medical_oncology_int_med_07132013.pdf

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"*
  - Topics: tumor and germline DNA, available diagnostic tests (clinical utility and timing)
- Several 75-minute Educational Sessions, e.g.
  - Squamous Non-Small Cell Lung Cancer: Understanding the Genomics, Treatment and Outcomes
  - Bridging Science and Clinical Practice: How to Use Molecular Markers When Caring for a Patient with Brain Cancer
  - 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

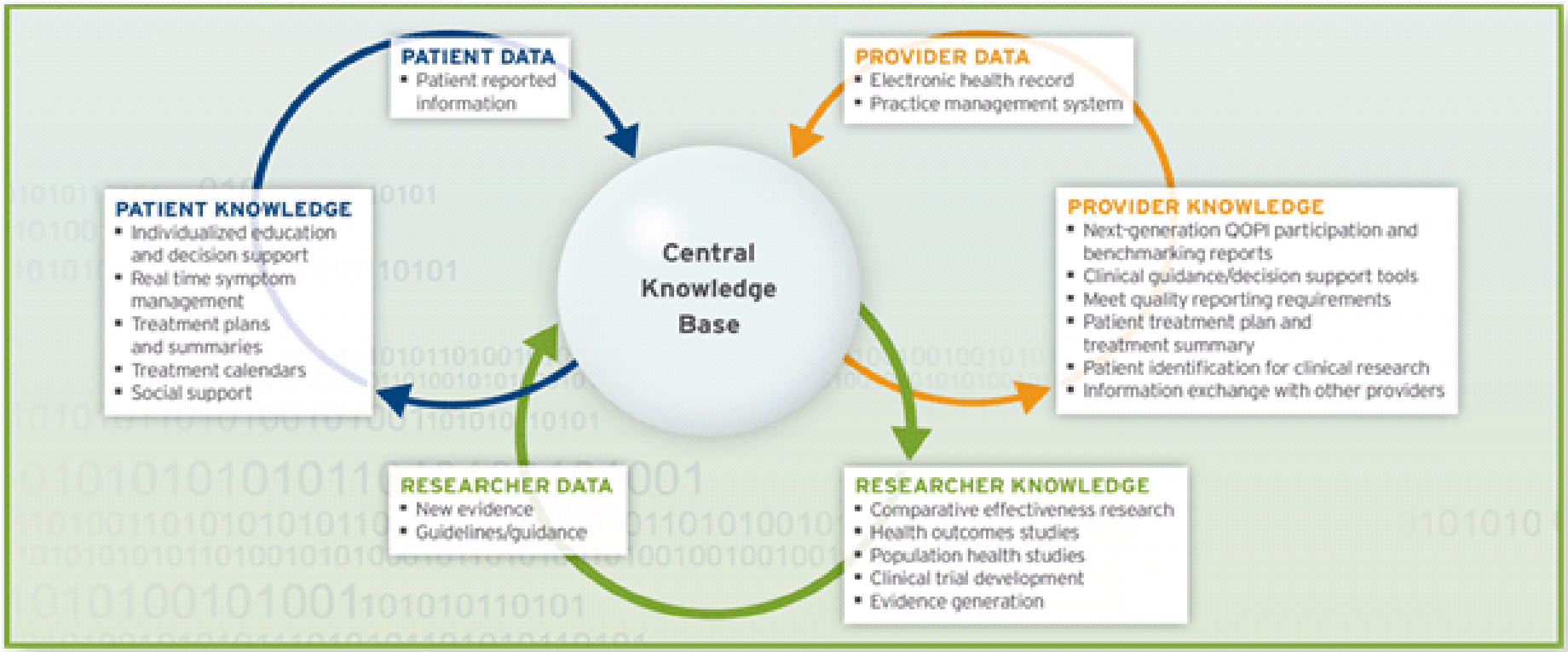
- ASCO-College of American Pathologists (CAP) HER-2 Testing Guideline
- ASCO-CAP ER/PR Testing Guideline
- CAP-Association for Molecular Pathology-ASCO Colorectal Cancer Markers Guideline

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

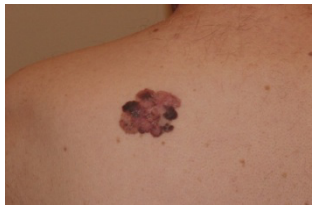


Next Steps for ASCO

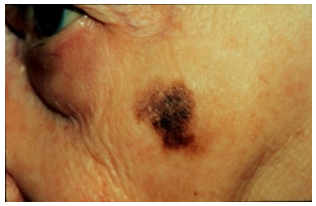
- Recommendations to ASCO Board May 2013 from Leadership Development Program
- Incorporate genomics into RLS plans
- Discussions with other societies, e.g., pathologists
- Involvement in follow-up from today's meeting

Traditional vs. Molecular View of Cancer

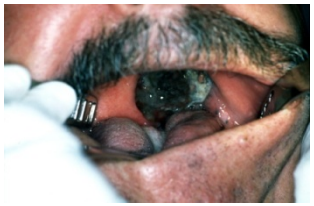
Melanoma



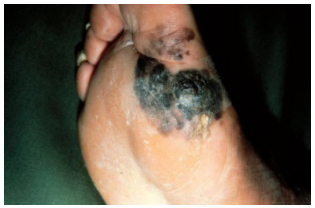
Arising from Skin Without Chronic Sun Damage



Arising from Skin With Chronic Sun Damage

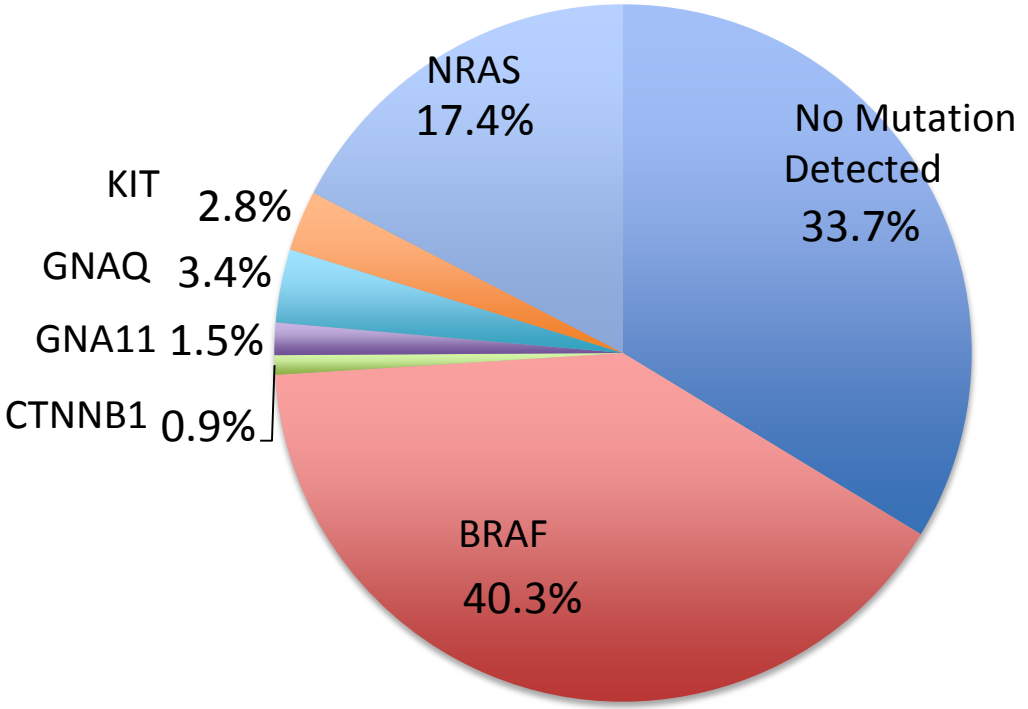


Arising from Mucosal Surfaces



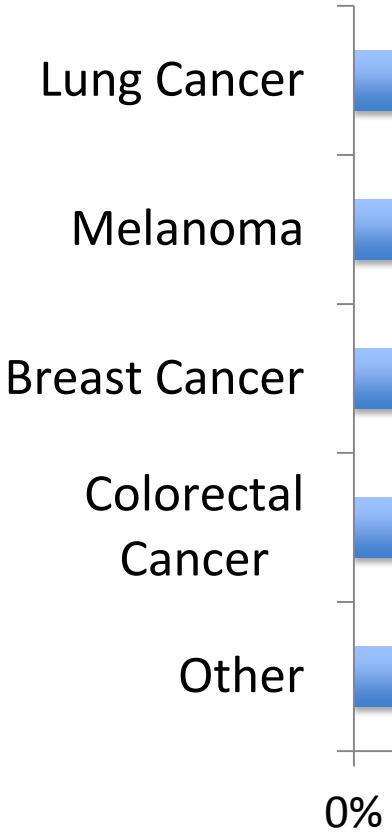
Arising from Acral Surfaces

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



Vanderbilt-Ingram Cancer Center Personalized Cancer Medicine Initiative

2,115 Specimens tested
July 2010 – Dec 2012



Impact at Vanderbilt-Ingram

Melanoma patients with **advanced disease** and **mutation detected**

61% went onto **clinical trial**

64% received **gene directed therapy**

Need for MCG: Knowledge Gap

Surveys Reveal Wide Gaps in Knowledge of Genetic Mutation Testing Exist Between Oncologists, Nurses and Cancer Patients

RIDGEFIELD, Conn., Nov. 16, 2011 /PRNewswire/ -- Despite guidelines calling for genetic mutation testing in certain patients with lung cancer, three new surveys fielded by Harris Interactive reveal a disconnect in the understanding of and communication about genetic mutation testing among healthcare professionals and cancer patients. Results of the surveys were announced today by Boehringer Ingelheim Pharmaceuticals, Inc., which sponsored the surveys in partnership with the Association of Community Cancer Centers (ACCC), ONS:Edge and the National Lung Cancer Partnership (NLCP).

Surveys of 95 comm … in October 2011 to measure per … h.

Cancer researchers … rmally and form cancers, includ … informed treatment decisions, … ce has not been widely adopted …

The surveys found th … ients, only 17 percent of lung cancer patients surveyed were aware of genetic mutation testing. Nearly half of oncology nurses (44 percent) did not discuss genetic mutation testing with patients, primarily because they felt that they lacked the knowledge to discuss it (56 percent) or didn't have the proper resources to share with their patients (33 percent). These findings highlight the need for a greater understanding of genetic mutation testing.

> "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

Name: **Sex:** **Laboratory Number:** **VUH#:**

Referral Source:

Reason for Request: DNA Analysis for *EGFR* Mutations

Type of Specimen: _____ (Block #_____)

Date Received:

Date of Report:

Interpretation: ***EGFR* Mutation Detected: Exon 19 deletion**

EGFR Mutations Tested Include:
Exon 19 deletion, Exon 21 (L858R), Exon 20 insertion

ERBB2 Mutation Tested:
Exon 20 insertion

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 EGF 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 insertions 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 *Sau*96I. 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 will likely be responsive to EGFR inhibitors. It is importatnt 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 and which may affect treatment response.

New Method for Reporting Mutation Results in the EMR

MR#	Patient Name	Actions	Tumor Gene Mutations H-SMP	BRAF	CTNNB1	GNA11	GNAQ	KIT	NRAS
03 81	A, B M.	Actions							
03 56	A, P	Actions							
03 35	B, J A	Actions							
01 80	B, S A	Actions							
02 29	E, J E	Actions							
02 27	F, R M	Actions							
02 77	G, T	Actions							
02 73	H, A	Actions							
03 64	S, C	Actions	A						
02 79	S, A S	Actions	R						
02 40	W, J E I	Actions							
03 74	W, C L	Actions							

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	
Location of mutation	Kinase domain (exon 15)
Frequency of BRAF mutations in melanoma	44% (COSMIC)
Frequency of V600E mutation among BRAF mutant melanomas	~85–90%
Implications for Targeted Therapeutics	
Response to BRAF inhibitors	Confers increased sensitivity[a]
Response to MEK inhibitors	Unknown at this time[b]
Response to KIT inhibitors	Unknown at this time

BRAF V600E mutation

Treatment Agent	Drug Class	Line of Treatment	# pts in study	Response Rate	PFS (months)	OS (months)	Reference
Vemurafenib (PLX4032)	Mutated BRAF TKI[c]	1st to >3rd	32[c]	81%	>7 (estimated)	Not reached	Flaherty et al. 2010
	Mutated BRAF TKI[c]	1st	337	48%	5.3	84% at 6 mos	Chapman et al. 2011
Dacarbazine	Cytotoxic chemotherapy	1st	338	5%	1.6	64% at 6 mos	Chapman et al. 2011

[c] This trial intended to include only patients with BRAF V600E mutations.

- Melanoma Overview
- BRAF
  - BRAF c.1798_1799GT>AG (V600R)
  - BRAF c.1798_1799GT>AA (V600K)
  - BRAF c.1799T>A (V600E)
  - BRAF c.1799_1800TG>AA (V600E)
  - BRAF c.1798G>A (V600M)
  - BRAF c.1799T>G (V600G)
  - BRAF c.1799_1800TG>AT (V600D)
- CTNNB1
- GNA11
- GNAQ
- KIT
- NRAS

- Other Cancers

- Molecular Pathology
- Take Our Survey
- Glossary
- News
- Our Team
- Acknowledgments

BRAF V600E (c.1799T>A) mutation in Melanoma

What is BRAF? | BRAF in Melanoma | BRAF V600E mutation | Clinical Trials

BRAF c.1799T>A (V600E) mutation in Melanoma

BRAF V600E mutation	
Properties	
Location of mutation	Kinase domain (exon 15)
Frequency of BRAF V600E	~85-90% of BRAF mutant melanoma
Implications for Targeted Therapeutics	
Response to BRAF inhibitors	Confers increased sensitivity*
Response to MEK inhibitors	Uncertain at this time#
Response to KIT inhibitors	Uncertain at this time

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.

Figure 2: Schematic of BRAF V600E mutation. Functional domains of BRAF are depicted. CR1: conserved regions 1. CR2: conserved region 2.

*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; Solit, 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) (Dummer, 2008).

BRAF V600E mutation								
Treatment Agent	Drug Class	Line of Treatment	# pts in study	Response Rate	PFS (months)	OS (months)	Level of evidence	Reference
vemurafenib (PLX4032)	Mutated BRAF TKI^	1st to >3rd	32^	81%	> 7 months (estimated)	Not reached	II-1	(Flaherty, 2010)
vemurafenib (PLX4032)	Mutated BRAF TKI^	1st	337	48%	5.3	84% at 6 mos	I	(Chapman, 2011)
dacarbazine	Cytotoxic chemotherapy	1st	338	5%	1.6	64% at 6 mos	I	(Chapman, 2011)

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)

Protocol No.	Title
VICCPHI1075 06/01/2011	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
VICCMEL1091 Pending	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
VICCPHI1076 Pending	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
VICCMEL1083 Pending	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

Melanoma Clinical Trials at Vanderbilt (7)

Tennessee (4)

United States (13)

Internationally (12)

What is BRAF? | BRAF in Melanoma | BRAF V600E mutation | Clinical Trials

BRAF Mutation

Great effort was made to in
guaranteed.

▼ At Vanderbi

Protocol No.	Title
VICCPHI1075 06/01/2011	A Ph Phar Patie Trea
VICCMEL1091 Pending	Br Ager Meta
VICCPHI1076 Pending	A Ph the Adm Mela
VICCMEL1083 Pending	An C Phar with

▼ Melanoma (

▼ 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

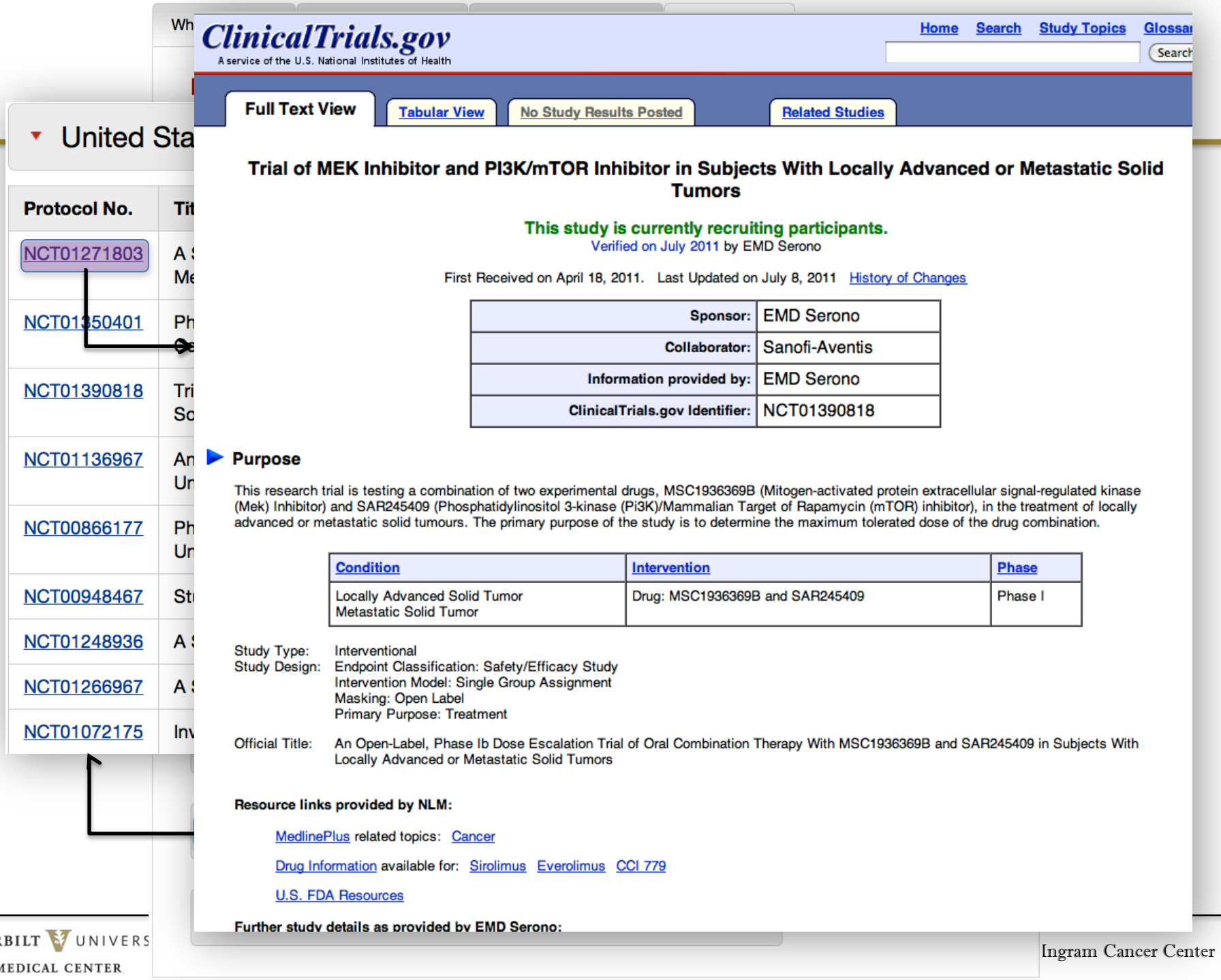
BRAF Mutation Directed Melanoma Clinical Trials

United States (13)

Protocol No.	Title
NCT01271803	A Study of RO5185426 And GDC-0973 in Patients With BRAF-Mutation Positive Metastatic Melanoma
NCT01350401	Phase I/II Study to Assess the Safety and Activity of Enhanced TCR Transduced Autologous T Cells in Metastatic Melanoma
NCT01390818	Trial of MEK Inhibitor and PI3K/mTOR Inhibitor in Subjects With Locally Advanced or Metastatic Solid Tumors
NCT01136967	An Open-Label, 2-Cohort, Multicenter, Study of E7080 in Previously Treated Subjects With Unresectable Stage III or Stage IV Melanoma
NCT00866177	Phase II Study of MEK Inhibitor AZD6244 in Patients With BRAF-Mutated or NRAS-Mutated, Unresectable Stage III or IV Melanoma
NCT00948467	Study of TAK-733 in Adult Patients With Advanced Nonhematologic Malignancies
NCT01248936	A Study of RO5185426 in Patients With Metastatic Melanoma
NCT01266967	A Study of GSK2118436 in BRAF Mutant Metastatic Melanoma to the Brain
NCT01072175	Investigate Safety, Pharmacokinetics and Pharmacodynamics of GSK2118436 & GSK1120212

United States (13)

Internationally (12)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Protocol No.
NCT01271803
NCT01350401
NCT01390818
NCT01136967
NCT00866177
NCT00948467
NCT01248936
NCT01266967
NCT01072175
ClinicalTrials.gov
A service of the U.S. National Institutes of Health
Home
Search
Study Topics
Full Text View
Tabular View
No Study Results Posted
Related Studies
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 History of Changes
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, MSC1936369B (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.
Condition
Intervention
Phase
Locally Advanced Solid Tumor
Metastatic Solid Tumor
Drug: MSC1936369B and SAR245409
Phase I
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 MSC1936369B 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:

My Cancer Genome

Genetically Informed Cancer Medicine

Home | DIRECT | About Us

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

- **17** Cancers
- **25** Genes
- **289** Disease-Gene-Variant Relationships

Molecular Medicine

- Articles of Interest
- List of Targeted Therapies

Clinical trial search

36,167 Cancer Trials (PDQ)

135 Cancer Diagnoses

437 Cancer Genes (COSMIC)

My Cancer Genome

Genetically Informed Cancer Medicine

Home | DIRECT | About Us

Find a Cancer Mutation

Disease (required): Select Disease

Gene (optional):

Variant (optional):

Find Clinical Tria

Lists trials by Dis
trials registered v

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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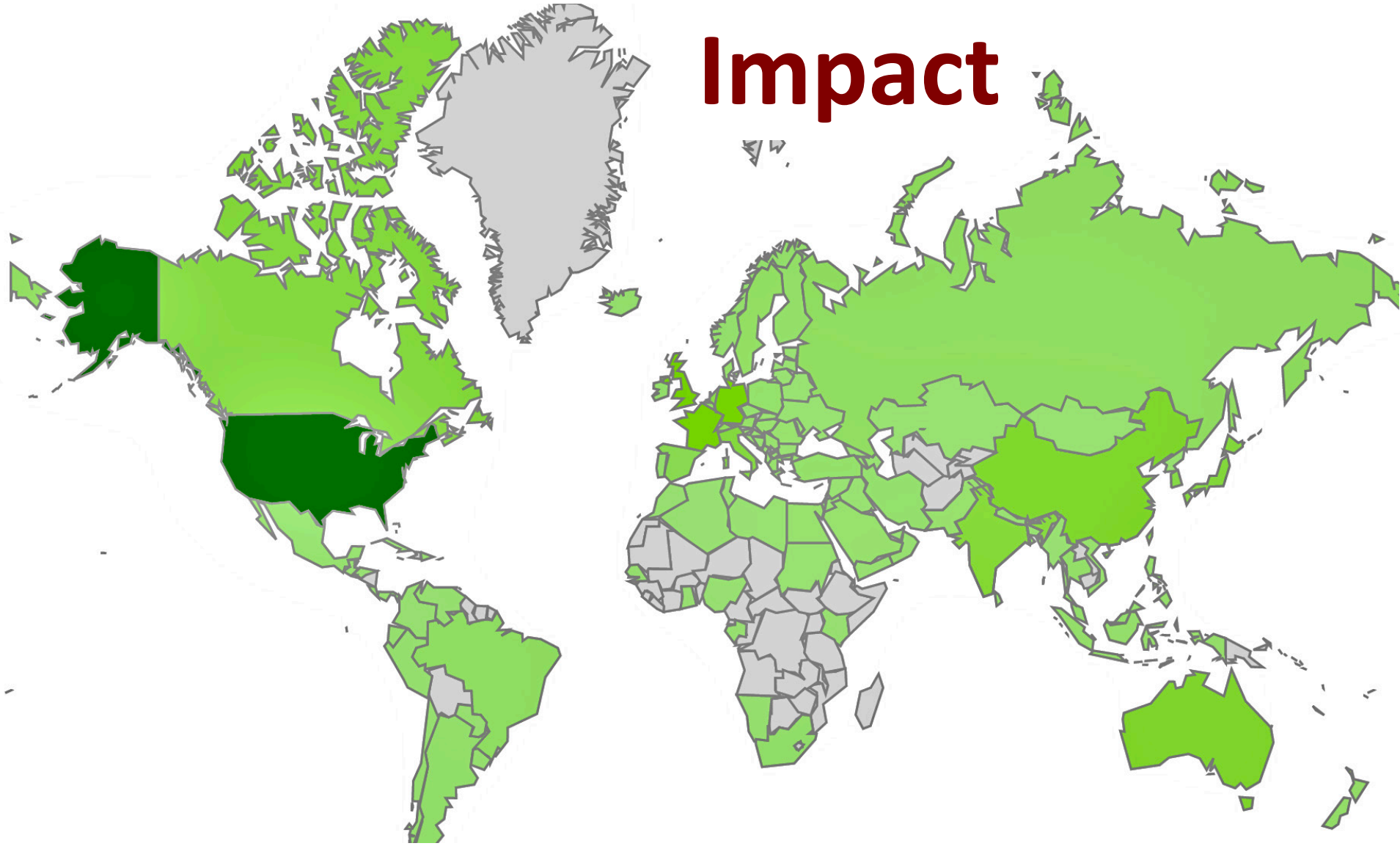
More...

Worldwide Collaboration

MyCancerGenome.org

>2000 site visits per week

Impact



Visits from 134 countries and territories

Heatmap of worldwide MCG visits: darker green = more visits

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HHS/Office for the National Coordinator for HIT (ONC) public data and cancer challenge to create health IT applications that use public data and existing technology to help patients and health care professionals prevent, detect, diagnose and treat cancer (Health 2.0)

GE Healthymagination