VANDERBILT UNIVERSITY
MEDICAL CENTER

Study ID: _____
(for staff only)

# CONTACT INFORMATION

**Name**: _____

First Name Middle Initial Last Name

**Address**: _____

Street City State Zip

**Phone Number**: _____

**Alternate Phone Number**: _____

**Social Security Number**: _____

Study ID: _____
(for staff only)

# DEMOGRAPHICS

**Which best describes you**? (Check one)

☐ Melanoma patient ☐ Caregiver

1. **What is your gender?**

☐ Male ☐ Female

2. **What is your race**? (Check all that apply)

☐ American Indian or Alaska Native

☐ Asian

☐ Native Hawaiian or Other Pacific Islander

☐ Black or African American

☐ White

3. **Are you of Hispanic, Latino, or Spanish origin?**

☐ Yes ☐ No

4. **What is your highest level of education?** (Check one)

- ☐ Less than 8th grade
- ☐ Between 9th and 12th grade – no diploma
- ☐ High school graduate, GED or equivalent
- ☐ Some college – no degree
- ☐ Vocational/Technical degree
- ☐ Bachelor's degree (e.g. BA, AB, BS, BBA)
- ☐ Master's degree (e.g. MA, MS, MEng, MBA)
- ☐ Professional school degree (e.g. MD, DDS, JD)
- ☐ Doctoral degree (e.g. PhD, EdD)

5. **What is your employment status?** (Check one)

- ☐ Full-time
- ☐ Part-time
- ☐ Retired
- ☐ Disabled
- ☐ Home-maker
- ☐ Unemployed
- ☐ Other

6. **What is your household income?** (Check one)

- ☐ I would rather not say
- ☐ $10,000 or less
- ☐ $10,001 - $20,000
- ☐ $20,001 - $35,000
- ☐ $35,001 - $55,000
- ☐ $55,001 - $75,000

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## HOSPITAL MATERIALS

For each of the following questions, please check the box ☑ that best reflects your answer.

**1. How confident are you in filling out forms by yourself**?

| ☐ | ☐ | ☐ | ☐ | ☐ |
|---|---|---|---|---|
| 1 | 2 | 3 | 4 | 5 |
| Never | Occasionally | Sometimes | Often | Always |

**2. How often do you have someone help you read hospital materials?**

| ☐ | ☐ | ☐ | ☐ | ☐ |
|---|---|---|---|---|
| 1 | 2 | 3 | 4 | 5 |
| None of the time | A little of the time | Some of the time | Most of the time | All of the time |

**3. How often do you have problems learning about your medical conditions because of difficulty reading hospital materials?**

| ☐ | ☐ | ☐ | ☐ | ☐ |
|---|---|---|---|---|
| 1 | 2 | 3 | 4 | 5 |
| None of the time | A little of the time | Some of the time | Most of the time | All of the time |

Study ID: _____
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# HOW YOU LEARN

For the question below, please check the box ☑ next to the answer that best describes you. Choose more if they apply to you.

**I will recall how to do something a year from now if:**

- ☐ I learn it by reading.
- ☐ I learn it by listening.
- ☐ I learn it by watching.
- ☐ I learn it by doing.

Study ID: _____
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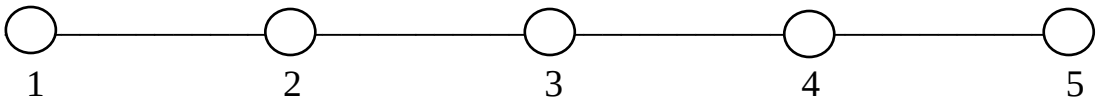
# GENETICS

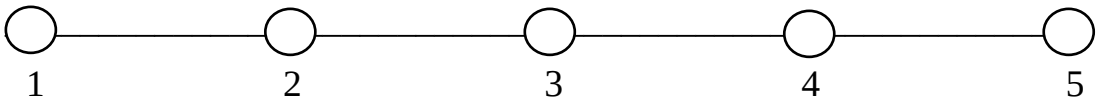
Fill in the circle that best corresponds to your understanding of the following terms.

**1. Gene**




Fully understand its meaning — Somewhat understand its meaning — Never heard of it

**2. DNA**

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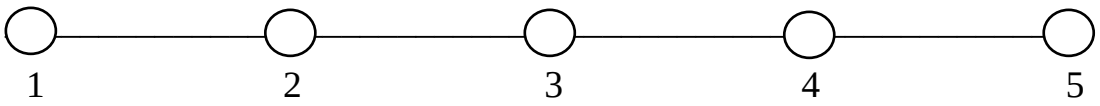
**3. Mutation**

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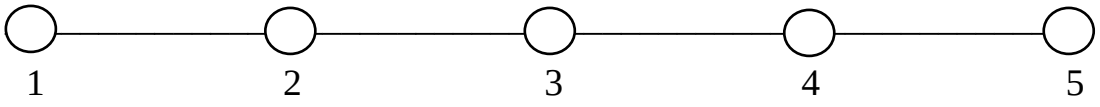
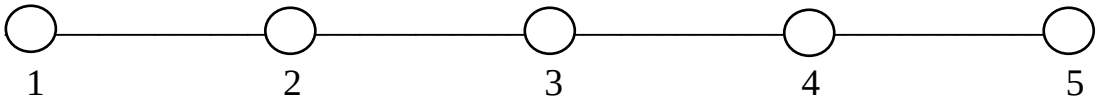
**4. BRAF**




Fully understand its meaning — Somewhat understand its meaning — Never heard of it

**5. Targeted therapy**




Fully understand its meaning — Somewhat understand its meaning — Never heard of it

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# MELANOMA & GENETICS QUESTIONS

Please circle only one answer.

1. **Which of these statements is true about melanoma?**
    a. Melanoma is a type of skin cancer.
    b. Melanoma starts in cells that make melanin.
    c. Melanoma usually starts in areas of skin that are exposed to the sun.
    d. All of the above
    e. Do not know

2. **Besides skin, where else might melanoma appear?**
    a. Heart
    b. Eye
    c. Liver
    d. Lymph nodes
    e. All of the above
    f. Do not know

**3. Melanoma can be treated by:**

a. Surgery

b. Traditional chemotherapy

c. Targeted therapy

d. Immune-based treatment

e. All of the above

f. Do not know

**4. Which of these statements about genes is correct?**

a. Genes come from your parents.

b. You have three copies of each gene.

c. Your genes are exactly the same as everyone else's genes.

d. Genes are not involved in cancer.

e. All of the above

f. Do not know

**5. A mutation can:**

a. Change how a protein works

b. Change the DNA sequence of a gene

c. Cause cancer

d. All of the above

**e.** Do not know

**6. Knowing about mutations in melanoma can help guide treatment options.**

a. True

b. False

c. Do not know

**7. Which of these statements is true about targeted therapies?**

a. They are a type of anti-depressant.

b. They mainly attack cells with a specific mutation.

c. They attack infections.

d. All of the above

e. Do not know

**8. What is BRAF?**

a. BRAF is an amino acid.

b. BRAF is a gene.

c. BRAF is a medication for treating melanoma.

d. BRAF is a protein.

e. Only a and c

f. Only b and d

g. Do not know

**9. How common is the BRAF mutation in melanoma?**

a. All melanomas have BRAF mutations.

b. BRAF mutations are not found in melanoma.

c. Nearly half of all melanomas have BRAF mutations.

d. Do not know

**10. Dabrafenib and vemurafenib are types of:**

a. Pain medications

b. BRAF inhibitors

c. Antibiotics

d. Do not know

# Melanoma and BRAF V600E

## Molecular Profiling of Melanoma

Melanoma is a malignant tumor of melanocytes. The disease is the fifth most common cancer in men and the seventh in women with an estimated 76,100 new cases and 9,710 deaths in 2014 in the U.S. ACS 2014). Melanoma is treated with a combination of surgery, traditional cytotoxic chemotherapy, targeted therapies, and immune-based therapies. Five-year survival rates for patients with metastatic disease, unfortunately, are below 10% (Jemal et al. 2010). Novel therapies and treatment strategies are needed.

Historically, melanoma has been classified according to pathologic and clinical characteristics such as histology (depth, Clark level, ulceration) and anatomic site of origin. Over the past decade, it has become evident that subsets of melanoma can be further defined at the molecular level by recurrent "driver" mutations that occur in multiple oncogenes, including BRAF, GNA11, GNAQ, KIT, MEK1 (MAP2K1), and NRAS (Table 1; Figure 1). Such driver mutations lead to constitutive activation of mutant signaling proteins that induce and sustain tumorigenesis.

Mutations in BRAF, GNA11, GNAQ, KIT, MEK1 (MAP2K1), and NRAS can be found in approximately 70% of all melanomas. In addition, mutations in CTNNB1 have also been described in melanoma. Mutations in more than one of these genes are seldom found concurrently in the same tumor. The distribution of mutations varies by site of origin and also by the absence or presence of chronic sun damage (Table 1; Figure 1). Importantly, targeted small molecule inhibitors are currently available or are being developed for specific molecular subsets of patients with melanoma.

The mutation-specific pages are meant to provide a broad overview of several of the oncogenes known to be important for melanoma pathogenesis. Where possible, the presence of a specific mutation is correlated to clinical parameters as well as response to both conventional chemotherapy and targeted agents. At present, only data for treatment of advanced (stage IV) disease are presented.

**Table 1.** Frequency of Somatic Gene Mutations in Melanoma.

| Mutated Gene | Frequency in Melanoma |
|---|---|
| BRAF | ~50% (COSMIC; Davies et al. 2002; Maldonado et al. 2003) |
| CTNNB1 | ~2-3% (Demunter et al. 2002; Omholt et al. 2001; Pollock and Hayward 2002; Reifenberger et al. 2002; Rimm et al. 1999) |
| GNA11 | 2% (COSMIC) |
| GNAQ | 1% (COSMIC) |
| KIT | ~2-6% (Beadling et al. 2008; Curtin et al. 2006; Handolias et al. 2010; Willmore-Payne et al. 2005) |
| MEK1 | 6% (Nikolaev et al. 2012) |
| NRAS | ~13-25% (Ball et al. 1994; Curtin et al. 2005; van't Veer et al. 1989) |

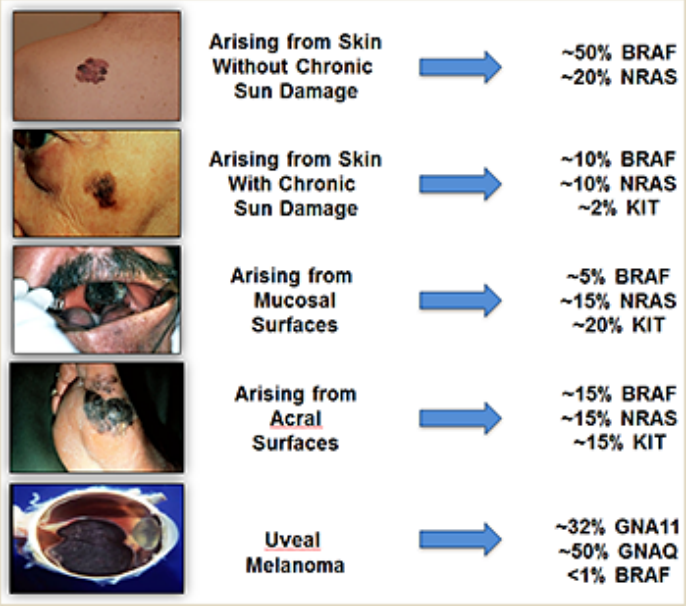



**Figure 1.** Molecular subsets of melanoma. Depicted is the frequency of driver mutations in melanoma related to anatomic location of the primary tumor. Frequencies of MEK1 and CTNNB1 mutations according to anatomic location are not known at present.

# BRAF

BRAF belongs to a family of serine-threonine protein kinases that includes ARAF,

BRAF, and CRAF (RAF1). RAF kinases are central mediators in the MAP kinase signaling cascade and exert their effect predominantly through phosphorylation and activation of MEK. This occurs following the dimerization (hetero- or homo-) of the RAF molecules. As part of the MAP kinase pathway, RAF is involved in many cellular processes, including cell proliferation, differentiation, and transcriptional regulation.

Mutant BRAF has been implicated in the pathogenesis of several cancers, including melanoma, non-small cell lung cancer, colorectal cancer, papillary thyroid cancer, and ovarian cancer (Davies et al. 2002).

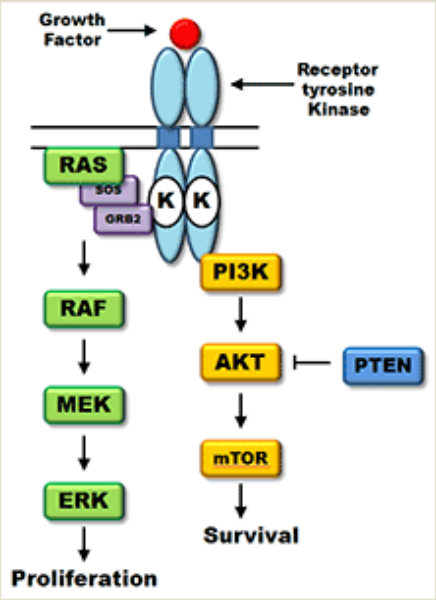



**Figure 1.** Schematic of the MAPK and PI3K pathways. Growth factor binding to receptor tyrosine kinase results in activation of the MAPK signaling pathway (RAS-RAF-MEK-ERK) and the PI3K pathway (PI3K-AKT-mTOR). The letter "K" within the schema denotes the tyrosine kinase domain.

# BRAF Mutations in Melanoma

Somatic mutations in BRAF have been found in ~50% of all malignant melanomas (COSMIC; Davies et al. 2002; Maldonado et al. 2003). BRAF mutations are found in all melanoma subtypes but are the most common in melanomas derived from skin without chronic sun-induced damage (Curtin et al. 2005; Maldonado et al. 2003). In this category of melanoma, BRAF mutations are found in ~59% of samples (Curtin et al. 2005).

The most prevalent BRAF mutations detected in melanoma are missense mutations, which introduce an amino acid substitution at valine 600. Approximately 80-90% of

V600 BRAF mutations are V600E (valine to glutamic acid; COSMIC; Lovly et al. 2012; Rubinstein et al. 2010) while 5-12% are V600K (valine to lysine; COSMIC; Lovly et al. 2012; Rubinstein et al. 2010), and 5% or less are V600R (valine to arginine) or V600D (valine to aspartic acid; COSMIC; Lovly et al. 2012; Rubinstein et al. 2010). The result of these mutations is enhanced BRAF kinase activity and increased phosphorylation of downstream targets, particularly MEK (Wan et al. 2004). In the vast majority of cases, BRAF mutations are non-overlapping with other oncogenic mutations found in melanoma (e.g., NRAS mutations, KIT mutations, etc.).

While BRAF inhibitor therapy is associated with clinical benefit in the majority of patients with BRAF V600E-mutated melanoma, resistance to treatment and tumor progression occurs in nearly all patients, usually in the first year (Chapman et al. 2011; Sosman et al. 2012). A variety of mechanisms have been implicated in primary and acquired resistance to BRAF inhibitors, primarily through reactivation of the MAP kinase pathway and other cell signaling pathways. Secondary BRAF mutations have not been described. Mechanisms of resistance are described below (Table 1); the frequencies of each of these mechanisms of resistance are not yet known. Possible second-line and greater treatment options supported by preclinical rationale are listed, although clinical data are lacking. Additionally, first-line combination therapy with BRAF and MEK inhibitor therapy may delay or prevent some of the mechanisms below (Flaherty et al. 2012).

**Table 1.** Mechanisms of Resistance to BRAF Inhibition.

| Mechanism of resistance | Implications for Targeted Therapeutics |
|---|---|
| BRAF V600 alternate splicing | Unknown at this time[a] |
| BRAF V600 gene amplification | Unknown at this time[b] |
| COT overexpression | Unknown at this time[c] |
| CRAF overexpression | Unknown at this time[d] |
| HGF overexpression | Unknown at this time[e] |
| IGF1R overexpression | Unknown at this time[f] |
| Acquired MEK1 (MAP2K1) mutations | Unknown at this time[g] |
| Acquired NRAS mutations | Unknown at this time[h] |
| PDGFRβ overexpression | Unknown at this time[i] |

[a] See Poulikakos et al. 2011.

[b] See Shi H. et al. 2012.

[c] In preclinical studies, cell lines overexpressing COT (the product of the *MAP3K8* gene) were sensitive to combinations of BRAF and MEK inhibitors but resistant to BRAF or MEK inhibitors alone (Johannesson et al. 2010).

[d] A BRAF inhibitor-resistant cell line demonstrating CRAF (the product of the *RAF1* gene) overexpression was sensitive to the HSP inhibitor geldanamycin in preclinical studies (Montagut et al. 2008).

[e] In preclinical studies, cell lines overexpressing HFG were sensitive to combinations of BRAF and HGF inhibitors and combinations of BRAF and MET inhibitors (Straussman et al. 2012; Wilson et al. 2012).

[f] In preclinical studies, cell lines overexpressing IGF1R were sensitive to combinations of MEK and IGF1R inhibitors and combinations of MEK and PI3K inhibitors (Villanueva et al. 2010).

[g] See Wagle et al. 2011.

[h] NRAS Q61K mutations resulted in increased levels of activated NRAS in two cell lines. These cell lines were sensitive to MEK inhibition with AZD6244 in preclinical studies (Nazarian et al. 2010).

[i] See Nazarian et al. 2010.

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

| **Properties** | |
|---|---|
| Location of mutation | Kinase domain (exon 15) |
| Frequency of BRAF mutations in melanoma | ~50% (COSMIC; Davies et al. 2002; Maldonado et al. 2003) |
| Frequency of V600E mutation among BRAF mutant melanomas | ~80-90% (COSMIC; Lovly et al. 2012; Rubinstein et al. 2010) |

| 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 |

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 (Figure 1). Approximately 80-90% of V600 BRAF mutations are V600E (COSMIC; Lovly et al. 2012; Rubinstein et al. 2010). Mutant BRAF proteins have increased kinase activity and are transforming in vitro (Davies et al. 2002). BRAF mutations are usually found in tumors wild type for NRAS, KIT, and other driver mutations.

[a] BRAF V600E mutations are associated with increased sensitivity to BRAF inhibitors (Chapman et al. 2011; Falchook et al. 2012a; Flaherty et al. 2012a; Flaherty et al. 2010; Hauschild et al. 2012; Sosman et al. 2012).

[b] Patients whose tumors harbored V600E and V600K mutations showed better responses to the MEK inhibitor, trametinib, than to chemotherapy (dacarbazine or paclitaxel; Flaherty et al. 2012b); patients with V600E or V600K-mutant tumors also showed better responses to trametinib than patients with BRAF wild type tumors (Falchook et al. 2012b). No significant differences were observed between patients with BRAF mutant (including V600E, V600K, K601E, and K581S) or wild type tumors, or between patients treated with selumetinib or temozolomide in a phase 2 trial (Kirkwood et al. 2008). However, patients whose tumors harbored BRAF mutations (primarily V600E) showed better response to the MEK inhibitor selumetinib in combination with dacarbazine, docetaxel, or temsirolimus than patients with BRAF wild type tumors in a phase 1 trial (Patel et al. 2012).

| Reference | Study Type / Phase | Line of Treatment | Treatment Agent | Mutation Status | # Patients in Study | Response Rate | PFS | OS |
|---|---|---|---|---|---|---|---|---|
| Flaherty et | phase | 1st line or | vemurafenib | BRAF mutant (intended to | | | >7 months | not |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| al. 2010 | 1 | greater | (BRAF inhibitor) | include only BRAF V600E) | 32 | 81% | (estimated) | reached |
| Chapman et al. 2011 | phase 3 | 1st | vemurafenib (BRAF inhibitor) | BRAF mutant (intended to include only BRAF V600E) | 337 | 48% | 5.3 | 84% at 6 months |
| | | | dacarbazine (crossover to the vemurafenib arm permitted at progression) | BRAF mutant (intended to include only BRAF V600E) | 338 | 5% | 1.6 | 64% at 6 months |
| Sosman et al. 2012 | phase 2 | 2nd line or greater | vemurafenib (BRAF inhibitor) | BRAF V600 mutations | 132 (122 with V600E, 10 with V600K) | 53% | 6.8 | 15.9 |
| | | | | V600K | 10 | 40% | | |
| | | | | V600E | 122 | 54% | | |
| Flaherty et al. 2012a | phase 1 and 2 | 1st line or greater | 150 mg dabrafenib twice daily + 1 mg trametinib per day | V600E or V600K | 54 (45 with V600E, 9 with V600K) | 50% | 9.2 | |
| | | | 150 mg dabrafenib twice daily + 2 mg trametinib per day | V600E or V600K | 54 (47 with V600E, 7 with V600K) | 76% | 9.4 | |
| | | | 150 mg dabrafenib 2x | V600E or V600K | 54 (45 with V600E, 9 | 54% | 5.8 | |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | daily | | with V600K) | | | |
| Falchook et al. 2012a | phase 1 | 1st line or greater | dabrafenib (BRAF inhibitor) | V600, irrespective of brain metastases | 46 | 69% | | |
| | | | | V600, w/o untreated brain metastases | 36 | | 5.5 | |
| | | | | V600E, irrespective of brain metastases | 27 | 78% | 5.5 | |
| | | | | V600K, irrespective of brain metastases | 18 | 39% | 5.6 | |
| | | | | K601E | 1 | 0 | 1.5 | |
| | | | | V600_K601delinsE | 1 | 0 | 1.8 | |
| | | | | K601E | 1 | 0 | 4.2 | |
| Hauschild et al. 2012 | phase 3 | 1st line or greater | dabrafenib (BRAF inhibitor) | V600E | 187 | 50% | 5.1 | |
| | | | dacarbazine (crossover to dabrafenib arm permitted at progression) | V600E | 63 | 6% | 2.7 | |
| Flaherty et al. 2012b | phase 3 | 1st line or greater | trametinib (MEK inhibitor) | V600E or V600K | 214 | | 4.8 | 81% at 6 months |
| | | | chemotherapy (crossover to trametinib arm permitted at | V600E or V600K | 108 | | 1.5 | 67% at 6 months |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | progression) | | | | | |
| Falchook et al. 2012b | phase 1 | 1st line or greater | trametinib (MEK inhibitor) | BRAF mutant and no prior BRAF inhibitors | 30 | 33% | 5.7 | |
| | | | | BRAF mutant, with prior BRAF inhibitors | 6 | 17% (unconfirmed) | | |
| | | | | BRAF wild type | 39 | 10% | | |
| Kirkwood et al. 2008 | Phase 2 | None within five years (see reference for details) | selumetinib (MEK inhibitor) | Any BRAF status | 104 | 5.8% | 78 days | 284 days (TTD) |
| | | | | BRAF mutant | 45 | 11.1% | | 284 days (TTD) |
| | | | temozolomide (crossover to selumetinib arm permitted at progression) | Any BRAF status | 96 | 9.4% | 80 days | 369 days (TTD) |
| | | | | BRAF mutant | 28 | 10.7% | | 369 days (TTD) |
| Patel et al. 2012 | phase 1 | 1st line or greater | selumetinib (MEK inhibitor) in combination with dacarbazine, docetaxel or temsirolimus | BRAF mutant (8 V600E and 1 R603*) | 9 | 56% | 51 weeks (TTP) | |
| | | | | BRAF wild type | 9 | 0% | 12 weeks (TTP) | |

NOTE: PFS = progression-free survival; OS = overall survival; TTD = time to death; TTP = time to progression.

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

Horn, L., Johnson, D. B., Lovly, C. M., Pao, W., & Sosman, J. A. (2014, September 29). BRAF Mutation in Melanoma. Retrieved May 11, 2015, from http://www.mycancergenome.org/content/disease/melanoma/braf/54/

**Appendix C: Consumer-Friendly My Cancer Genome Content**

## Melanoma and the BRAF V600E Mutation

**This material will help you understand:**

- the basics of melanoma
- how the BRAF gene might be involved in melanoma
- if there are any drugs that might work better if you have certain changes in the BRAF gene

**What is melanoma?**

Melanoma is a type of skin cancer. It starts in the cells that make melanin, the substance that gives skin its color.

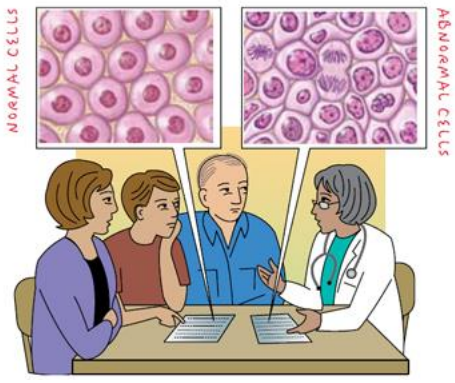
**Where is melanoma found?**

Melanoma usually starts on areas of the skin exposed to the sun. But melanoma can also appear in other parts of your body like the eye, the soles of the feet, under the nails, or inside the mouth.

**What are the most common current treatments for melanoma?**

Doctors may treat melanoma using one or more of these options:

- **Surgery –** removes as much of a cancer tumor as possible.
- **Traditional chemotherapy –** drugs that kill growing cells. All cells grow, but cancer cells grow faster than healthy cells, so more of the cancer cells die. But because these drugs kill healthy cells too, this can cause unwanted side effects.
- **Targeted therapy –** mainly drugs that target proteins involved in cancer. Because these drugs mainly kill cancer cells and not healthy cells, they may have fewer side effects. Two types of targeted therapies for melanoma are:
    - **Immune-based therapy –** works with your body's defense system to fight cancer. Some of these drugs mark cancer cells so they are easier for your immune system to find.
    - **Small molecule therapy** – mainly acts on cells with specific protein changes. Genes contain the instructions for making proteins. Changes in genes, called mutations, may result in changes in proteins. These changes may cause cells to grow out of control which could lead to cancer. So, small molecule therapy uses drugs to target those proteins. Genetic testing can tell if your tumor has protein changes that can be targeted.




**Can mutations found in my cancer cells be passed to my children?**

Mutations that are found only in your cancer cells cannot be passed on to your children

**How well does cancer drug treatment work?**

After a while, your cancer cells may stop responding to the drug(s). This means your cancer may start to grow again. Your doctor will do regular checkups to watch for this. This way, if the cancer starts to come back, your doctor can try another drug or treatment.

**What is BRAF?**

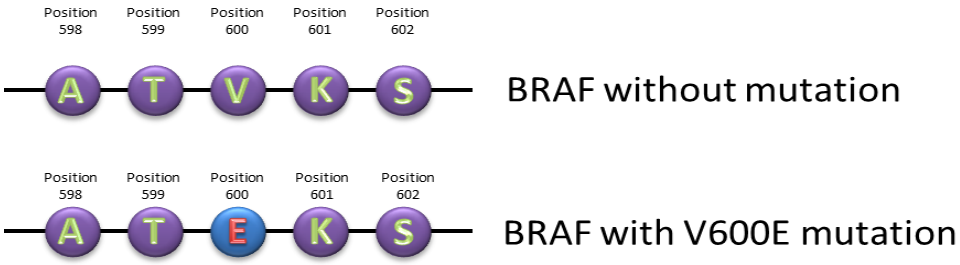
BRAF is the name of both a gene and a protein. The BRAF gene contains the instructions for making the BRAF protein. The main job of the BRAF protein is to help control cell growth. BRAF is part of a chain of proteins, called a signaling pathway. This pathway relays signals from outside of the cell to the cell's nucleus. The nucleus is the control center of the cell. These signals may tell the cell to grow, divide, or die. These signals are turned on and off as needed.

**What is BRAF's role in melanoma?**

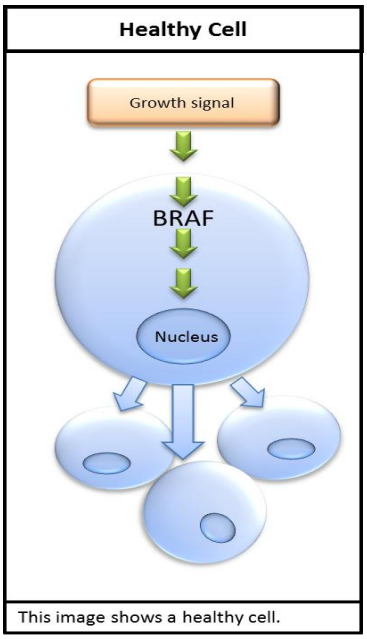
About half of all melanomas have a mutation in the BRAF gene that changes the BRAF protein. BRAF mutations are most common in melanomas found on skin without long-term sun damage. But these mutations can occur in all types of melanoma.

**What is the BRAF V600E mutation?**

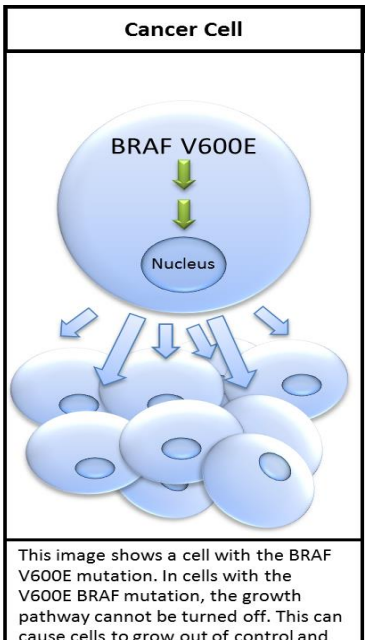
V600E is the most common BRAF mutation in melanoma. Proteins are long chains of amino acids. The BRAF protein has 766 amino acids. The original amino acid at position 600 of BRAF is valine, or V for short. The V600E mutation replaces valine with another amino acid called glutamic acid, or E for short.




In cells with the V600E BRAF mutation, the growth signaling pathway cannot be turned off. This can cause cells to grow out of control and lead to cancer.




This image shows a healthy cell.




This image shows a cell with the BRAF V600E mutation. In cells with the V600E BRAF mutation, the growth pathway cannot be turned off. This can cause cells to grow out of control and lead to cancer.

**Are there targeted therapies for BRAF V600E?**
The BRAF V600E mutation is a target for BRAF inhibitor drugs. The drugs vemurafenib and dabrafenib are BRAF inhibitors. These drugs stop the BRAF protein with the mutation from turning on other proteins needed for cell growth. This stops the growth of these cells and may lead to cell death.

**What if I have a mutation at a different position in BRAF?**
If your BRAF mutation is not at position 600, BRAF inhibitors may not work well for you.

**What if my test results show "no mutation detected" in BRAF?**
"No mutation detected" means that you may still have other mutations in this or other genes that were not tested. However, your genetic test results will still help your doctor determine which one of the available treatments is best for you.

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*This text was translated from MyCancerGenome on August 1, 2014.*

**Appendix D**

**Table 4.** Multivariate linear regression (with question 4 included)

| | Posttest score ($N$ = 88) | | | |
|---|---|---|---|---|
| Variable | *B* | (SE) | t | p (two-tailed) |
| Intercept | 6.93 | (0.83) | 8.31 | <0.0001 |
| Pretest score | 0.36 | (0.076) | 4.82 | <0.0001 |
| Group B | 0.34 | (0.37) | 0.94 | 0.3479 |
| Group C | 0.8 | (0.36) | 2.26 | 0.0267 |
| Age | -0.03 | (0.011) | -3.11 | 0.0025 |
| Health Literacy* | -0.11 | (0.07) | -1.67 | 0.0978 |

*Health literacy is scored on an inverse scale.