Empowering STAT DNA Testing for Molecular Oncology Applications Using A Fully Automated Platform

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Outline

- Human cancer
- How the diagnosis is made
- Precision/personalized medicine in oncology
- The role of molecular testing
- To NGS or not to NGS
- The role of STAT DNA testing

A MESSAGE OF HOPE

CANCER is a curable disease.

CANCER is neither contagious nor hereditary. Yearly 90,000 people (1 in 10 over 40 years old) die of this disease in this country. Many of these victims could have been cured had they gone to a reputable doctor immediately. "Immediately" means as soon as symptoms are noticed.

Shown for the
American Society for the Control of Cancer—A Benevolent Organization.
370 Seventh Avenue, New York City.

Human Cancer - Cancer Statistics, 2018

Estimated New Cases

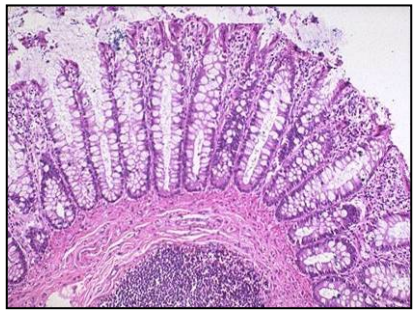
Males			Females		
Prostate	164,690	19%	Breast	266,120	30%
Lung & bronchus	121,680	14%	Lung & bronchus	112,350	13%
Colon & rectum	75,610	9%	Colon & rectum	64,640	7%
Urinary bladder	62,380	7%	Uterine corpus	63,230	7%
Melanoma of the skin	55,150	6%	Thyroid	40,900	5%
Kidney & renal pelvis	42,680	5%	Melanoma of the skin	36,120	4%
Non-Hodgkin lymphoma	41,730	5%	Non-Hodgkin lymphoma	32,950	4%
Oral cavity & pharynx	37,160	4%	Pancreas	26,240	3%
Leukemia	35,030	4%	Leukemia	25,270	3%
Liver & intrahepatic bile duct	30,610	4%	Kidney & renal pelvis	22,660	3%
All Sites	**856,370**	**100%**	**All Sites**	**878,980**	**100%**

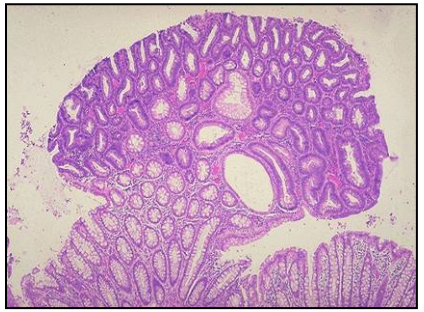
Estimated Deaths

Males			Females		
Lung & bronchus	83,550	26%	Lung & bronchus	70,500	25%
Prostate	29,430	9%	Breast	40,920	14%
Colon & rectum	27,390	8%	Colon & rectum	23,240	8%
Pancreas	23,020	7%	Pancreas	21,310	7%
Liver & intrahepatic bile duct	20,540	6%	Ovary	14,070	5%
Leukemia	14,270	4%	Uterine corpus	11,350	4%
Esophagus	12,850	4%	Leukemia	10,100	4%
Urinary bladder	12,520	4%	Liver & intrahepatic bile duct	9,660	3%
Non-Hodgkin lymphoma	11,510	4%	Non-Hodgkin lymphoma	8,400	3%
Kidney & renal pelvis	10,010	3%	Brain & other nervous system	7,340	3%
All Sites	**323,630**	**100%**	**All Sites**	**286,010**	**100%**

CA: A Cancer Journal for Clinicians
Volume 68, Issue 1, pages 7-30, 4 JAN 2018 DOI: 10.3322/caac.21442
http://onlinelibrary.wiley.com/doi/10.3322/caac.21442/full#caac21442-fig-0001

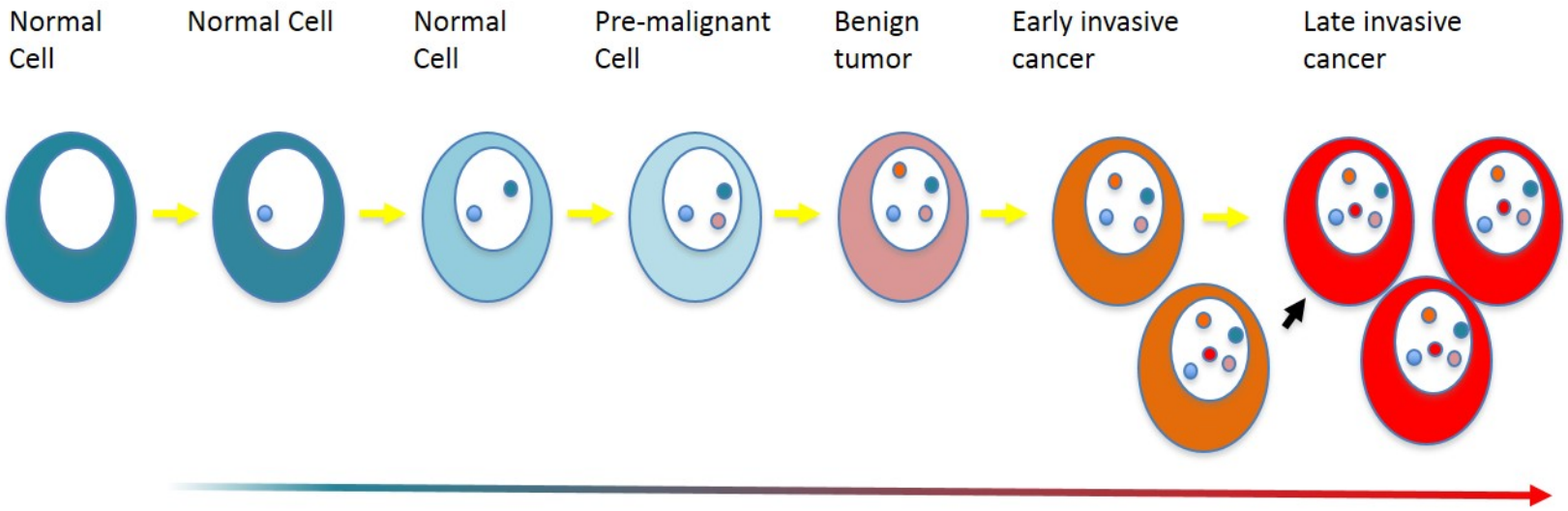
Human Cancer





1. Unregulated (clonal) cell growth
2. Impaired cellular differentiation
3. Invasiveness
4. Metastatic potential

Human Cancer as a Genetic Disease

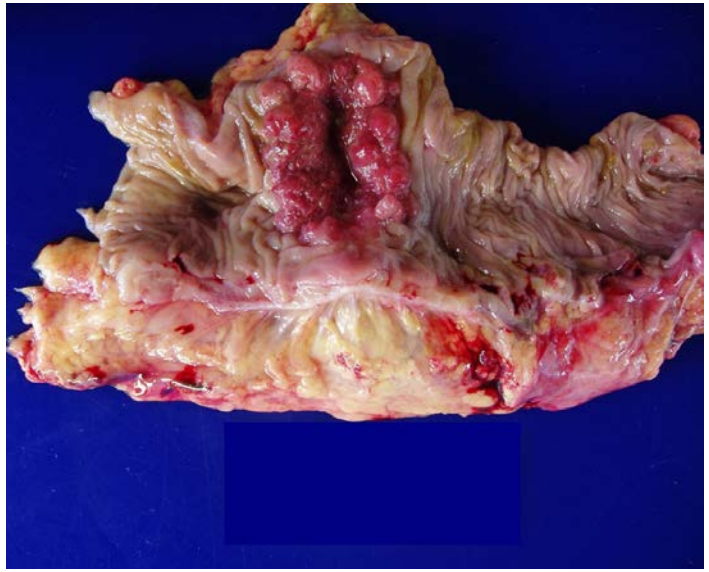


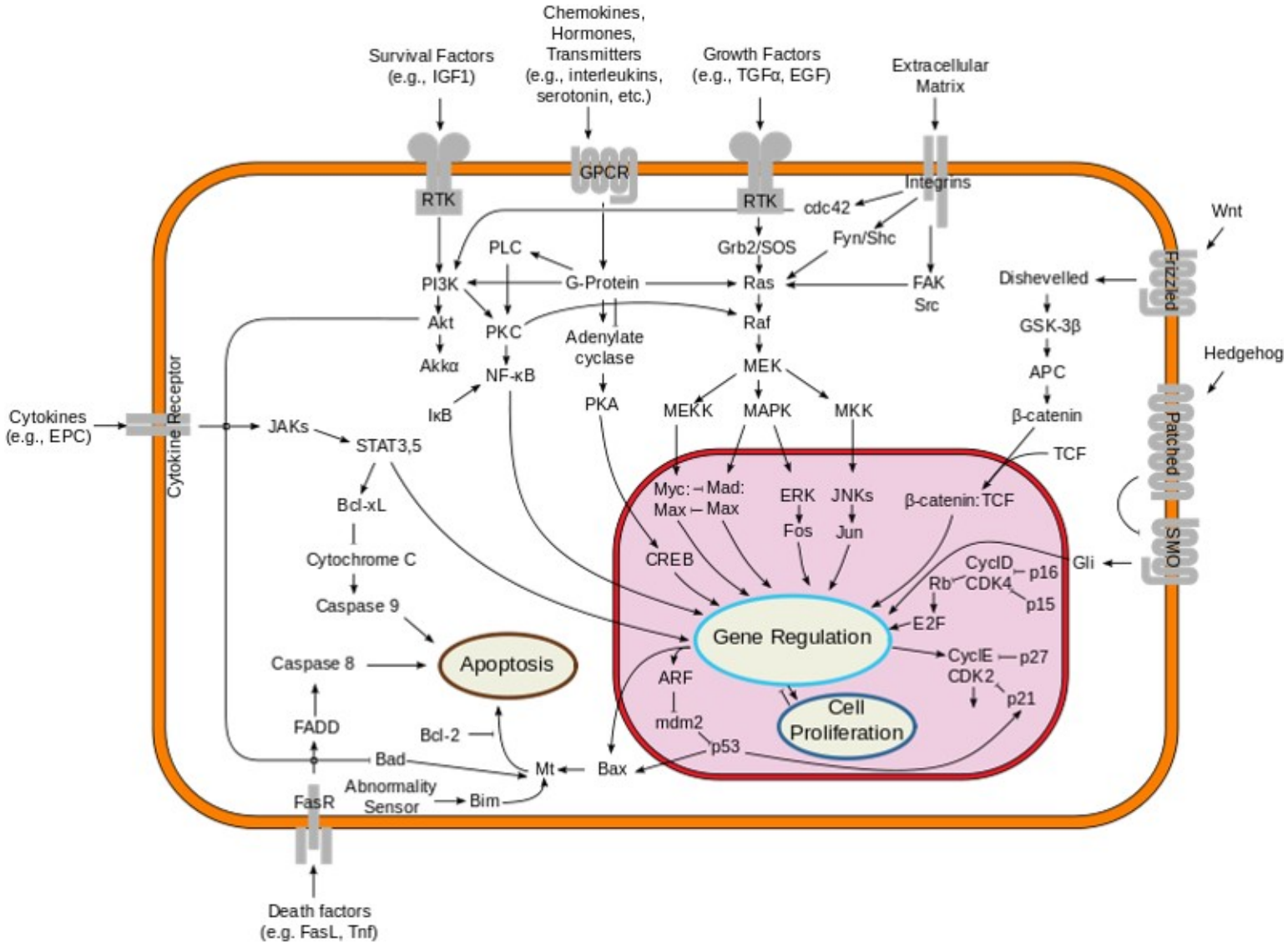
Cancer results from the disruption of important genes and gene products.

Human Cancer as a Genetic Disease

- ~30,000 genes in human genome
- Only a small fraction of these genes have the potential to cause cancer when mutated
- Oncogenes
- Tumor Suppressor Genes

Human Cancer as a Genetic Disease





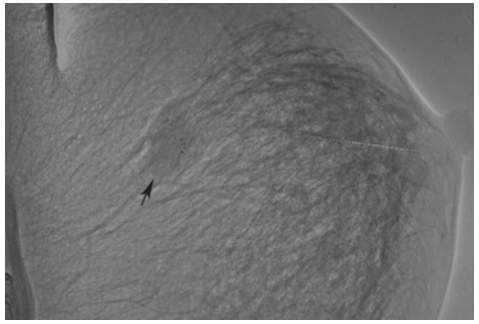
Human Cancer – The Diagnosis

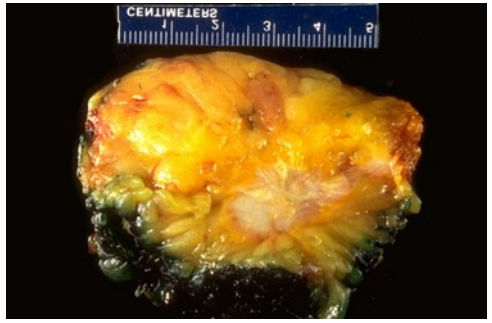
H&E for Morphology

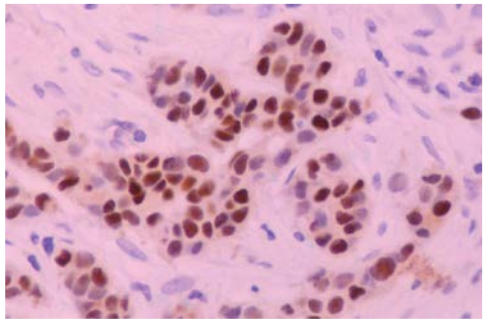
Further Diagnostic Work-up
- Imaging (CT, MRI, PET)
- Immuno (IHC), molecular,

1.4M New Cancers (US)

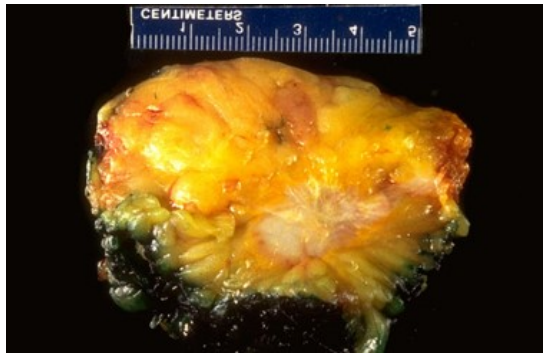
Therapeutic management

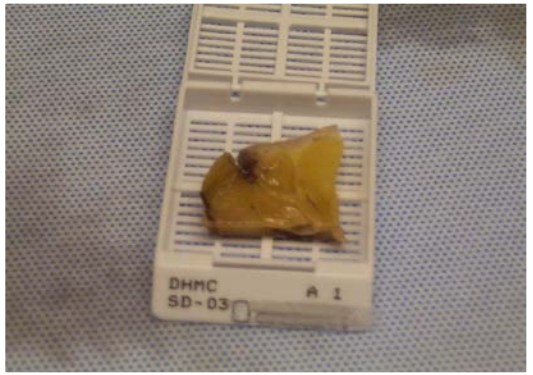


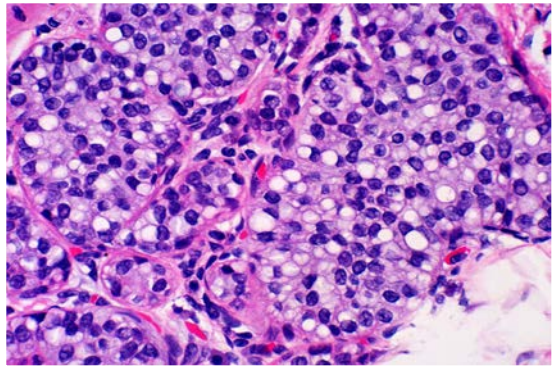




Human Cancer – The Diagnosis

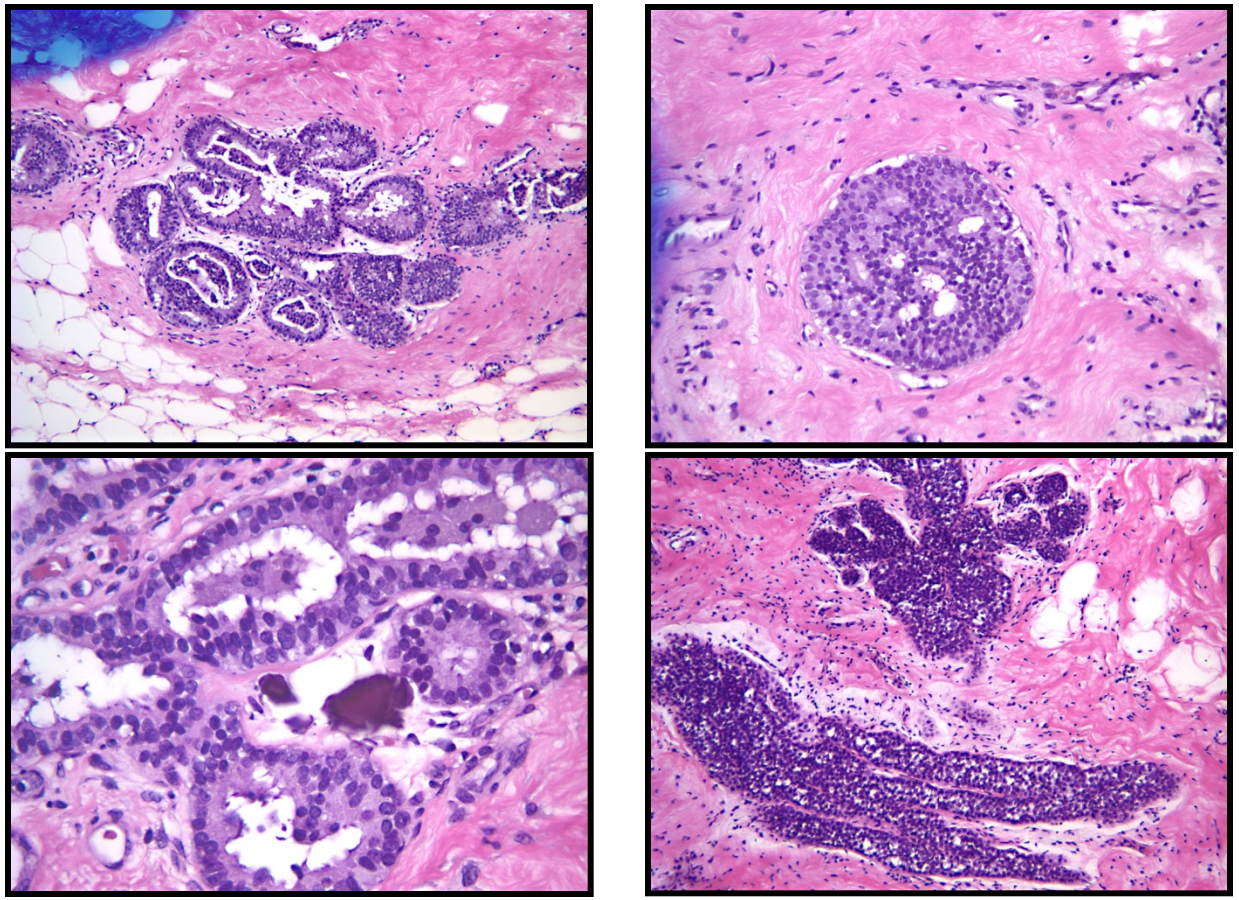






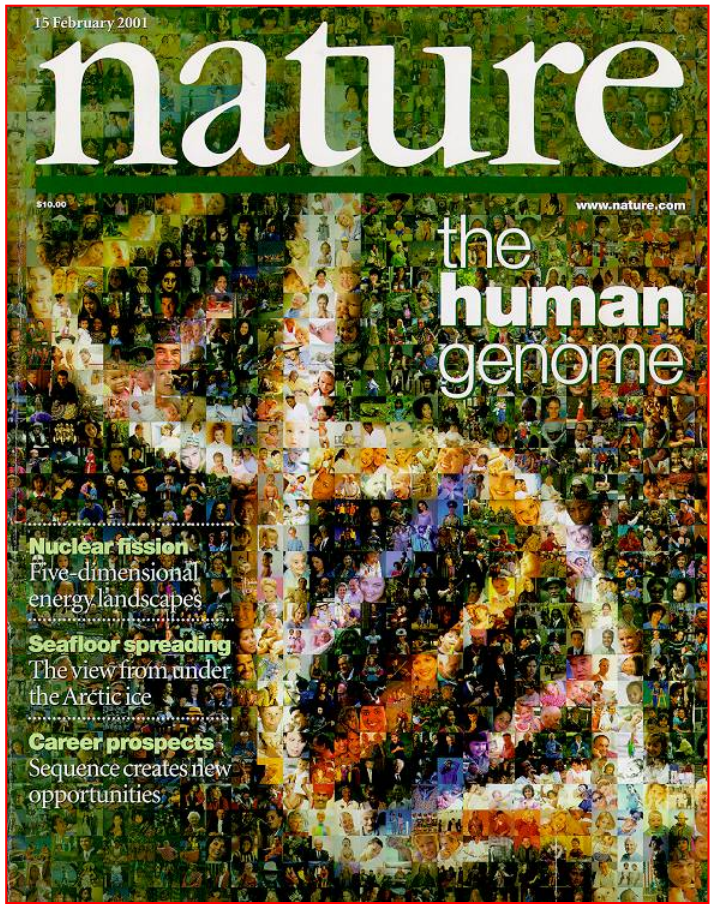
Human Cancer – The Diagnosis

Nothing Else Looks Like This!



Promises of the Human Genome

- Diagnostic
- Prognostic
- Predictive
- Therapeutic



Human Cancer - Precision Medicine

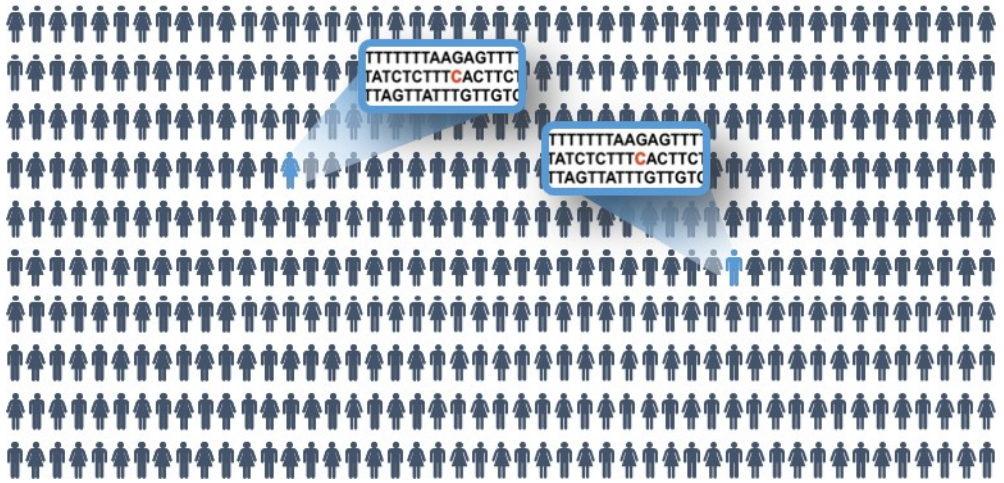
- >2 million ADRs occur annually in US
- ~100,000 deaths (4th leading cause of death)
- >$76 billion - cost of drug-related morbidity & mortality
- 4% of new drugs are withdrawn due to ADRs
  - 1995-2005: 34 drugs withdrawn mainly due to hepatotoxic or cardiotoxic effects
- Therapeutics effective in 25-60 % of patients
- Genetics accounts for ~24% of drug disposition and effects.
  - Due to polymorphisms in drug metabolizing enzymes, transporters, and targets (receptors)

Human Cancer - Precision Medicine

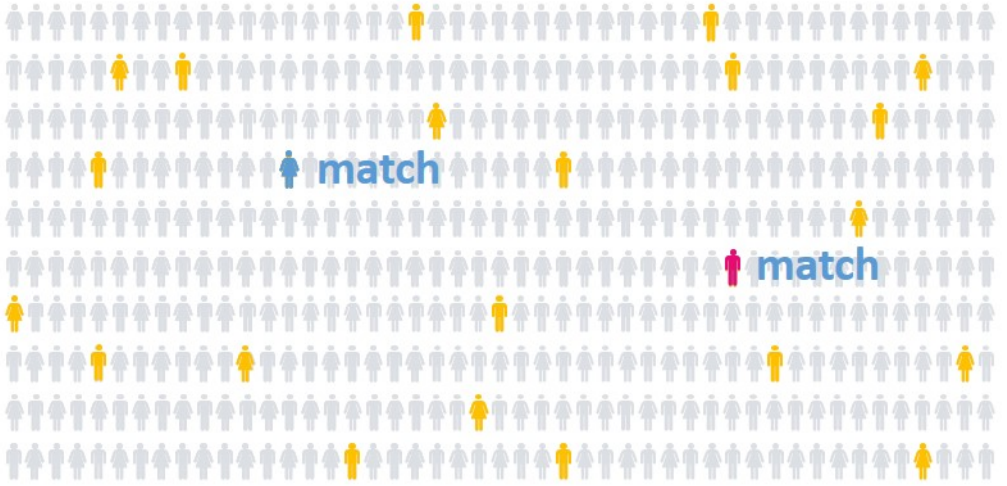
- PGx_m: pharmacokinetic
  - What the body does to the drug
    - Absorption
    - Distribution
    - Metabolism
    - Excretion
- PGx_t: targeted therapy
  - Presence/absence of therapeutic target
  - Response or lack of response
  - Resistance
  - Local or distant recurrence

Human Cancer - Precision Medicine

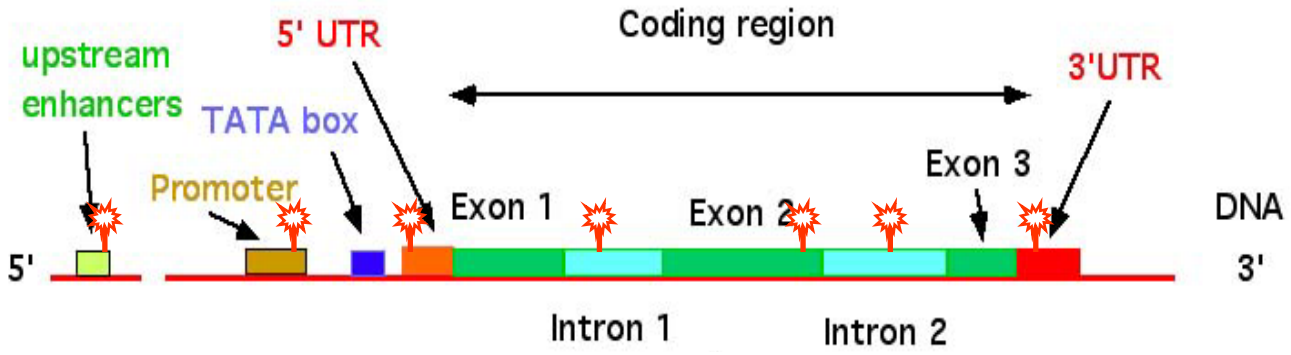
- PGx_m: pharmacokinetic
  - Polymorphisms
  - Not typically disease causing mutations
  - Ex. Irinotecan and UGT1A1



- PGx_t: targeted therapy
  - Mostly mutations in disease causing genes
  - Includes driver and passenger mutations
  - Germline vs somatic variants



Human Cancer - Precision Medicine PGx_m



Genetic polymorphisms

Spectrum of Drug Metabolism

Poor (PM)

Intermediate (IM)

Extensive (EM)

Ultra-Rapid (UM)

Human Cancer – Targeted Therapy (PGX_t)

1960s

EGF identified

1970s

Overexpression of HER1/EGFR reported in cancer cells

Monoclonals identified

1980s

Small molecule TK inhibitors identified

HER1/HER2 overexpression correlates with poor prognosis

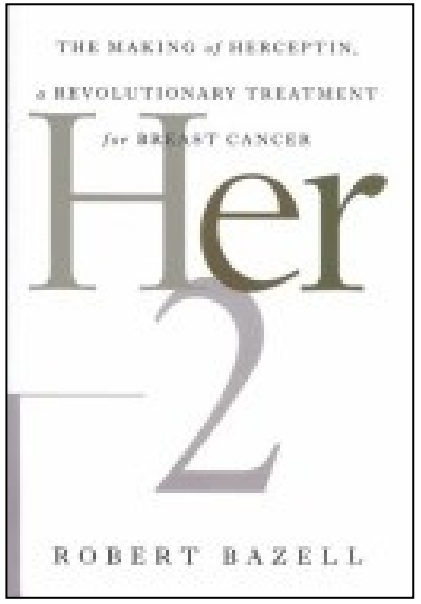
1990s

First HER-targeted MAb, trastuzumab (Herceptin®) approved for HER2+ MBC

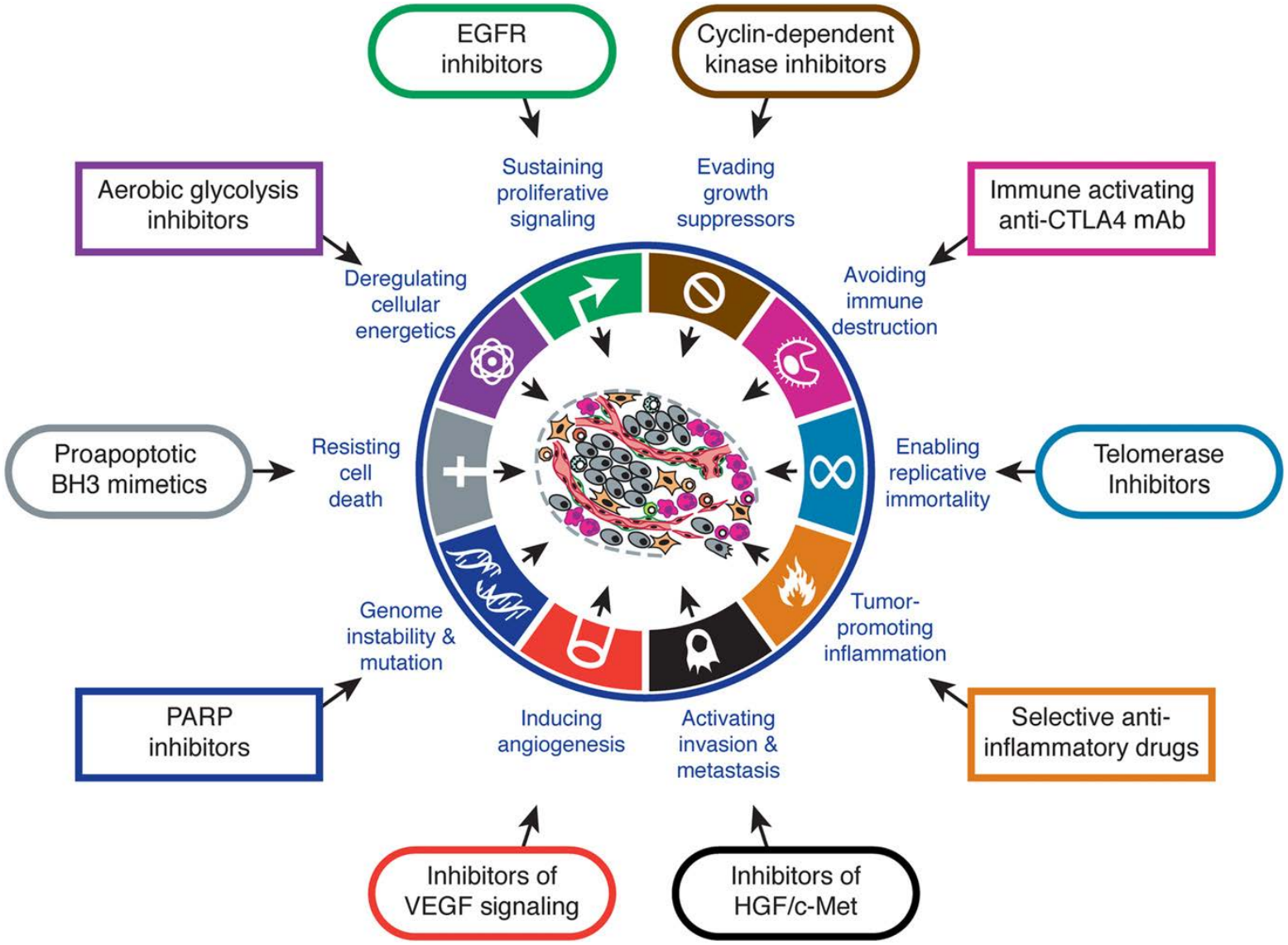
2000s

Gefitinib (Iressa®) approved as monotherapy for patients with locally advanced or metastatic NSCLC

HER-targeted therapies being explored in wide range of human cancers



Human Cancer – Targeted Therapy (PGX_t)

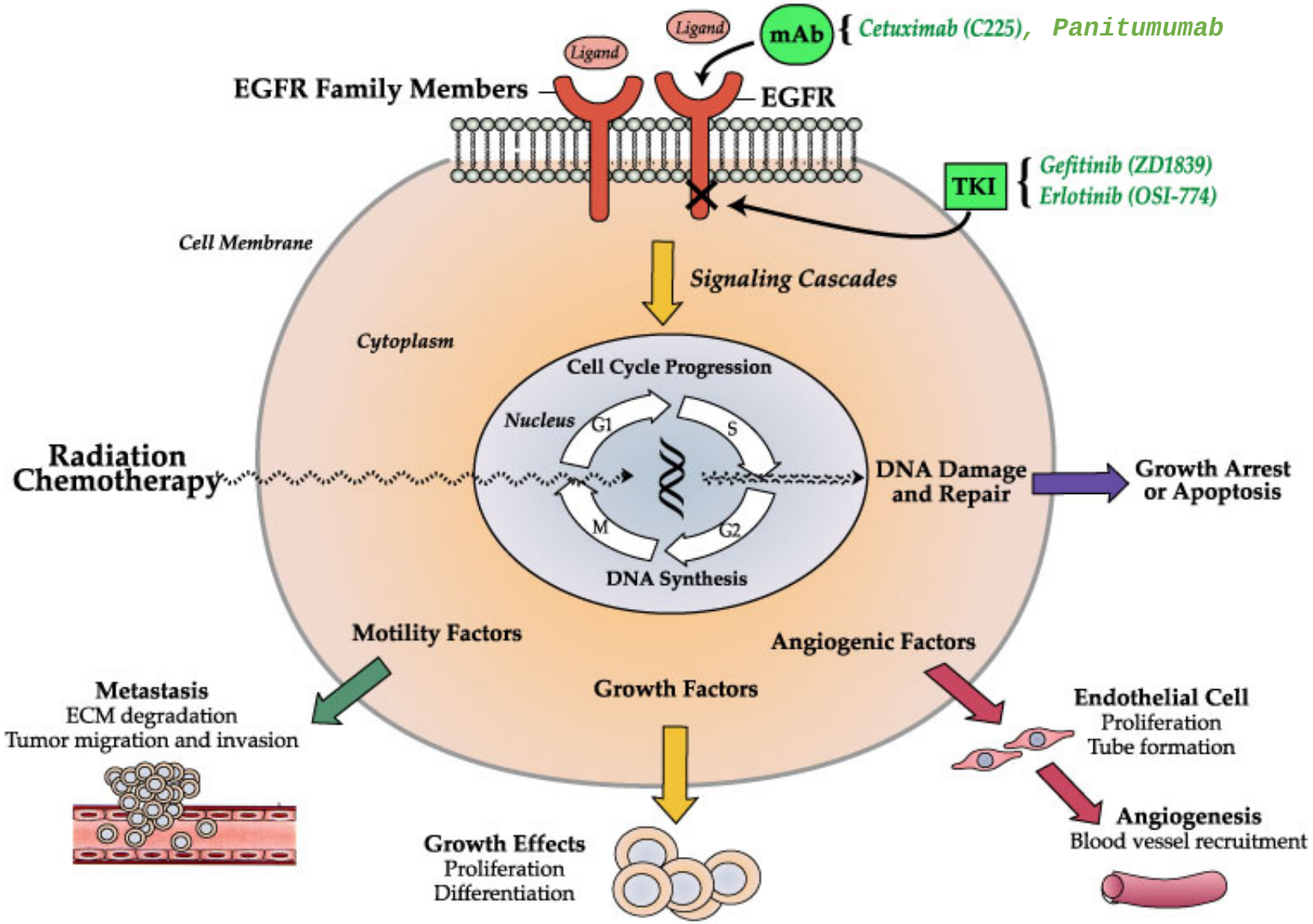


Hanahan et al. *Cell*, 144:646–74, 2011.

Human Cancer – Targeted Therapy (PGX_t)

- BCR-ABL1
  - Imatinib (Gleevec) for CML
- HER2 amplification
  - Trastuzumab (Herceptin) for breast cancer
- KRAS point mutation
  - Cetuximab and Panitumimab for colon cancer
- EGFR point mutation and/or amplification
  - Iressa, Tarceva for lung cancer

EGFR and Targeted Therapies



VOLUME 26 · NUMBER 10 · APRIL 1 2008

JOURNAL OF CLINICAL ONCOLOGY

ORIGINAL REPORT

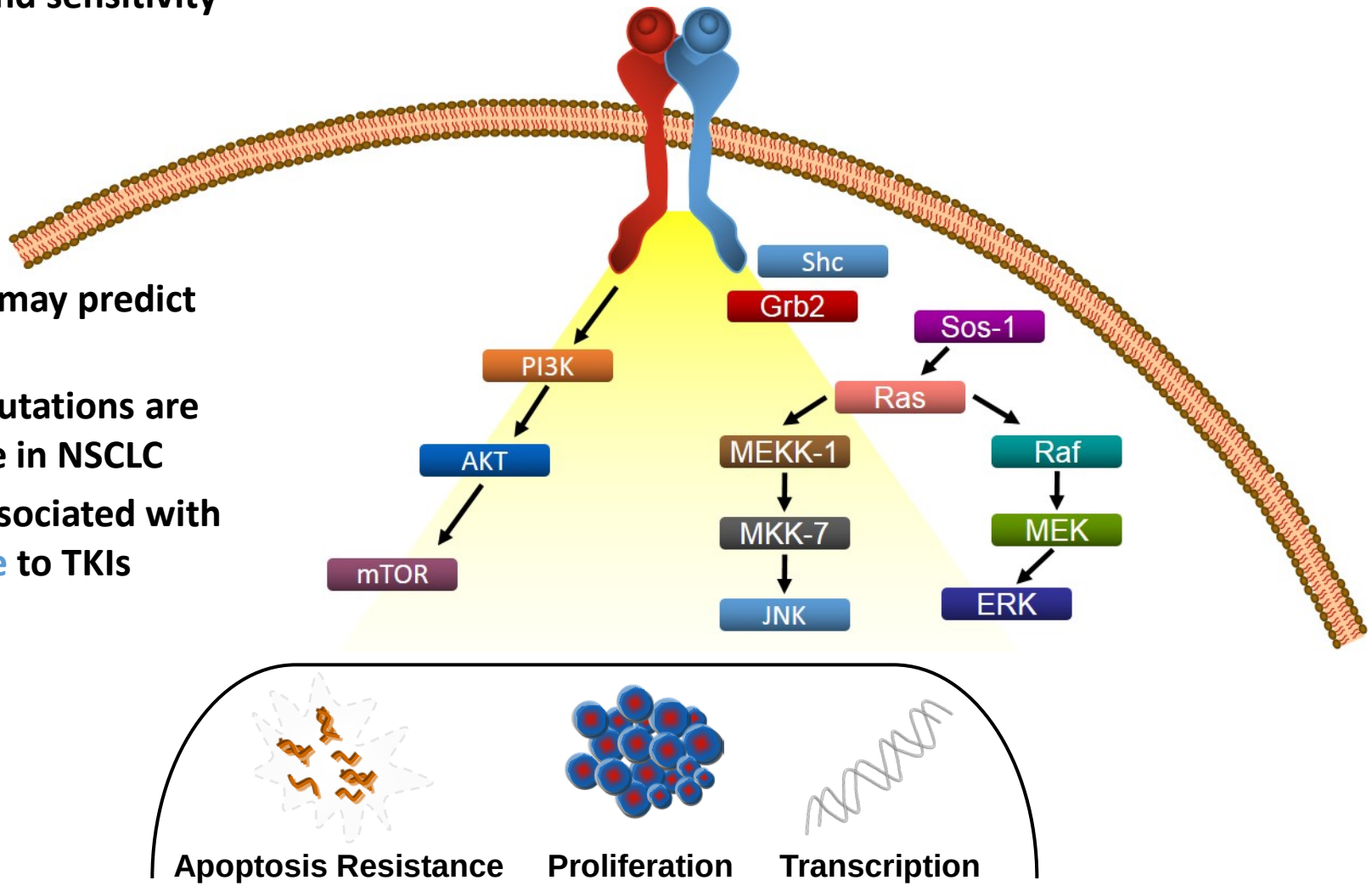
Wild-Type *KRAS* Is Required for Panitumumab Efficacy in Patients With Metastatic Colorectal Cancer

Rafael G. Amado, Michael Wolf, Marc Peeters, Eric Van Cutsem, Salvatore Siena, Daniel J. Freeman, Todd Juan, Robert Sikorski, Sid Suggs, Robert Radinsky, Scott D. Patterson, and David D. Chang

Targeting the EGFR Pathway in NSCLC

- **EGFR mutations and sensitivity to TKIs**
  - **Exon 19 deletion**
  - **Exon 21 (L858R)**
  - **Exon 18 (G719X)**
- **Exon 20 insertion may predict resistance to TKIs**
- **EGFR and KRAS mutations are mutually exclusive in NSCLC**
- **KRAS mutation associated with primary resistance to TKIs**

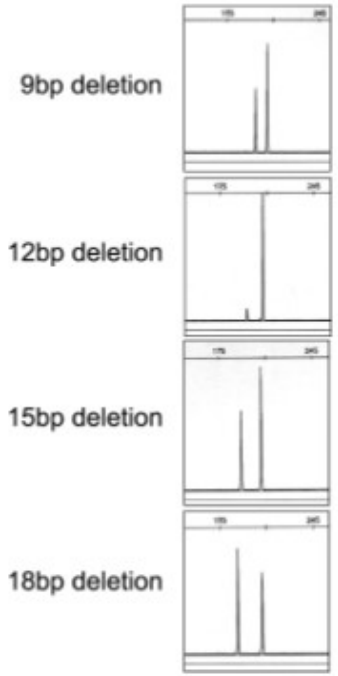
Gefitinib (Iressa) first EGFR-TK inhibitor to be approved for use in NSCLC



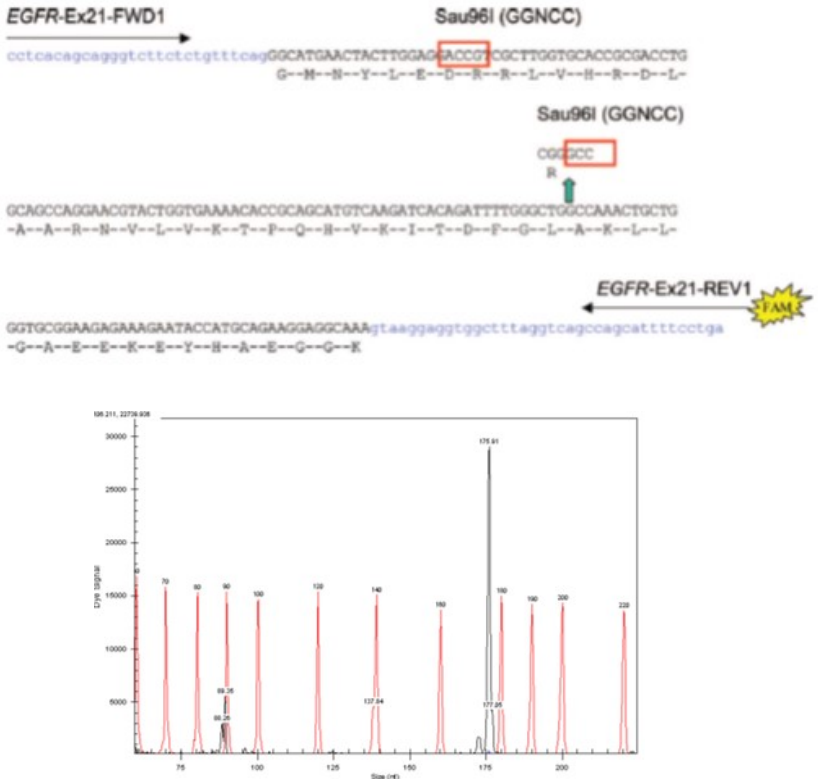
The Role of Molecular Dx in Oncology

(Somatic Mutation Detection)

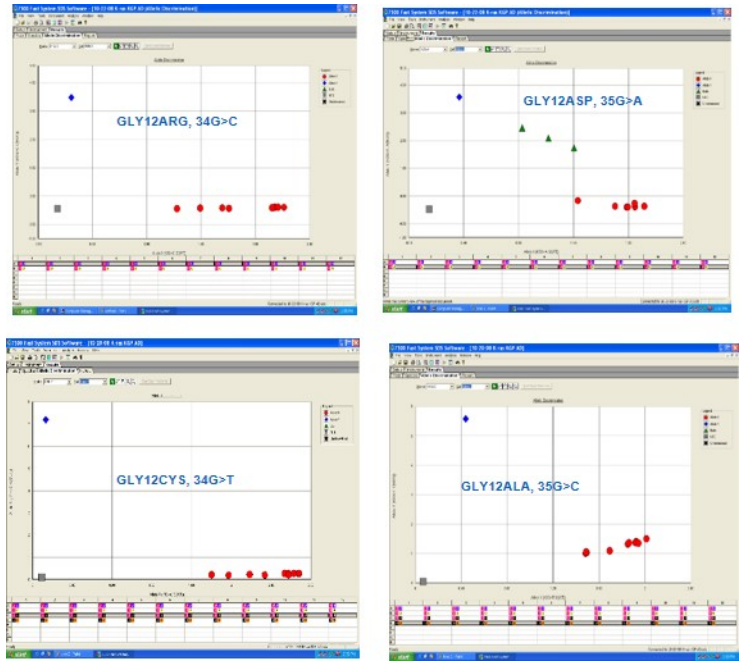
EGFR Exon 19 Deletion Analysis



EGFR Exon 21 SNP (L858R) Analysis



KRAS Analysis x7



The Role of Molecular Dx in Oncology

(Sanger Sequencing - Somatic Mutation Detection)

1 ctccgggctg tcccagctcg gcaagcgctg cccaggtcct ggggtggtgg cagccagcgg
61 gagcaggaaa ggaagcat**g**t tcccaggctg cccacgcctc tgggtcctgg tggtcttggg
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901 cctggagcag aaccatcat**a** aggtctcagc catcaccctt gtcagtgcta catccactac
961 cgcaaatatg actgtgggcc cagagggaaa gtggatcata tc**ttctct**ca ccccaaaaca

The Role of Molecular Dx in Oncology
(Somatic Mutation Detection)

- Single gene assays
- Single variants
- Labor intensive
- Costly
- Algorithms for testing
- Increasing demand
- Increasing numbers of genes and variants

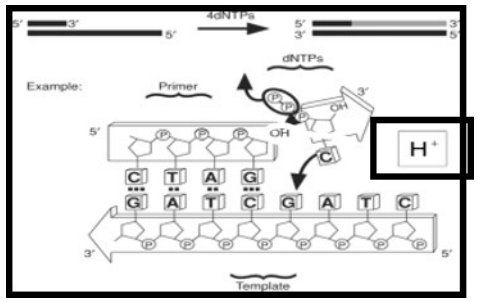
The Role of Molecular Dx in Oncology

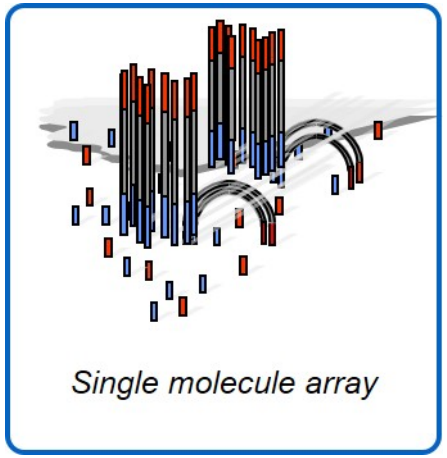
(Next Generation or Massively Parallel Sequencing - Somatic Mutation Detection)

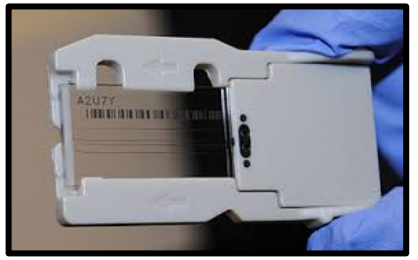
- Low quantities of DNA
- Multiple genes (10-380 genes) simultaneously
- Each DNA fragment sequenced 100’s-1,000’s x
- Multiple patients’ samples (8-10) simultaneously

The Role of Molecular Dx in Oncology

(NGS - Somatic Mutation Detection)







Somatic Mutation Analysis (NGS)

Ion Torrent Cancer Hotspot v2 gene panel (CHPv2) (50)

ABL1	***EGFR***	***GNAS***	***KRAS***	***PTPN11***
AKT1	***ERBB2***	***GNAQ***	***MET***	***RB1***
ALK	***ERBB4***	***HNF1A***	***MLH1***	***RET***
APC	***EZH2***	***HRAS***	***MPL***	***SMAD4***
ATM	***FBXW7***	***IDH1***	***NOTCH1***	***SMARCB1***
BRAF	***FGFR1***	***IDH2***	***NPM1***	***SMO***
CDH1	***FGFR2***	***JAK2***	***NRAS***	***SRC***
CDKN2A	***FGFR3***	***JAK3***	***PDGFRA***	***STK11***
CSF1R	***FLT3***	***KDR***	***PIK3CA***	***TP53***
CTNNB1	***GNA11***	***KIT***	***PTEN***	***VHL***

(207 amplicons, >20kb, 10ng DNA input)

Routine Use of the Ion Torrent AmpliSeq™ Cancer Hotspot Panel for Identification of Clinically Actionable Somatic Mutations. Clin Chem Lab Med Dec 2013;13:1-8.

SPECIAL ARTICLE

Guidelines for Validation of Next-Generation Sequencing Based Oncology Panels: A Joint Consensus Recommendation of the Association for Molecular Pathology and College of American Pathologists

(**J Molec Diagn 2017**)

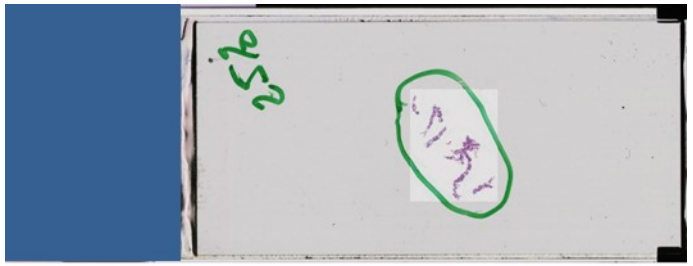
Lawrence J. Jennings, Maria E. Arcila, Christopher Corless, Suzanne Kamel-Reid, Ira M. Lubin, John Pfeifer, Robyn L. Temple-Smolkin, Karl V. Voelkerding, and Marina N. Nikiforova

The Role of Molecular Dx in Oncology

(NGS - Somatic Mutation Detection)

Molecular testing ordered by surgical pathologist
2 H&E and 10 USS

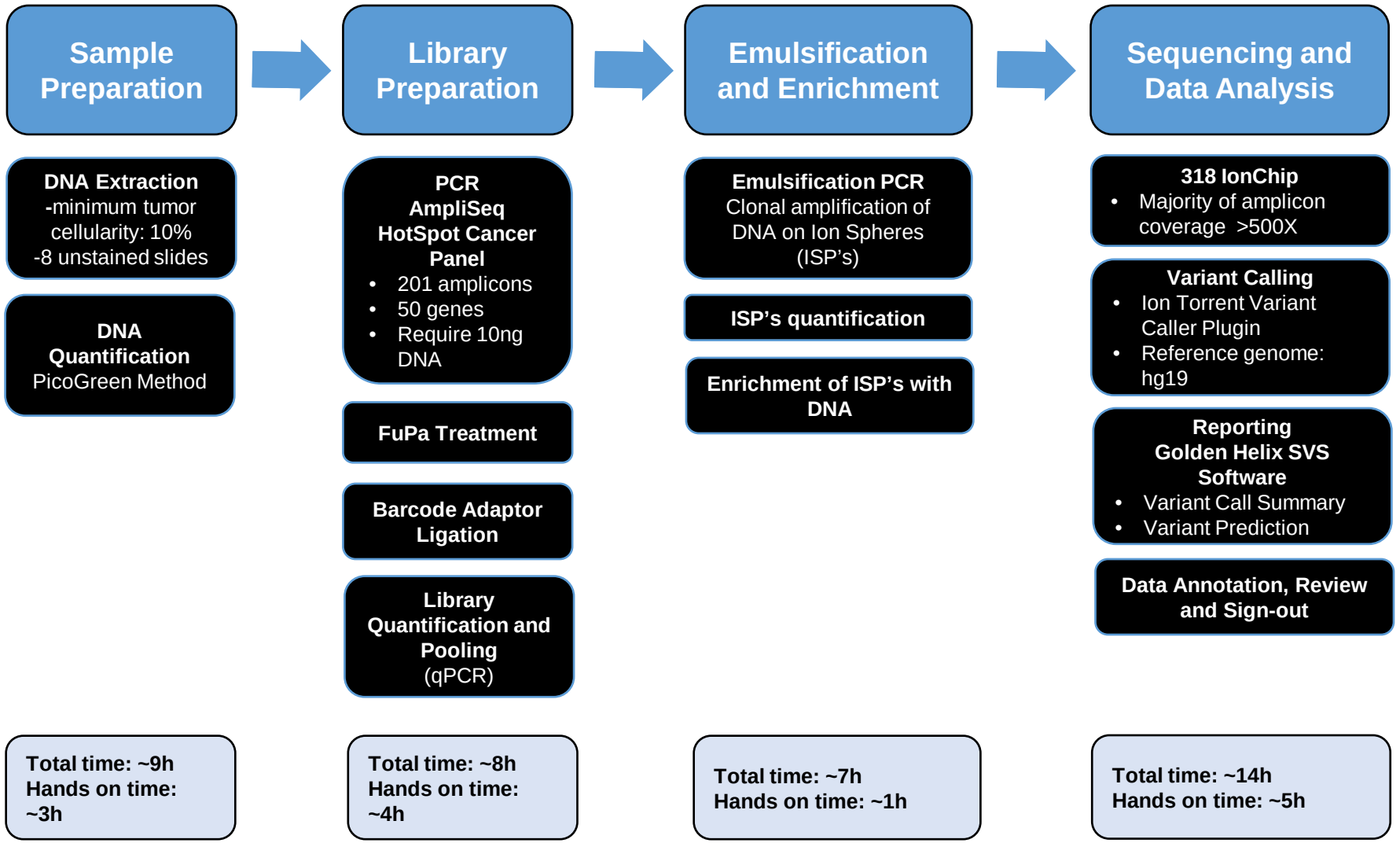
MG Pathologist review of H&E for adequacy and % tumor



1 H&E and 2 USS to FISH lab to hold for additional testing if needed

DNA/RNA extracted from USS in molecular laboratory

To NGS or Not To NGS



To NGS or Not To NGS

Complexity of Somatic Mutation Analysis

- Clinically actionable (sensitizing or resistance) and FDA approved application
- Clinically actionable but off label (drug not approved for tumor type, maybe for compassionate use)
- Clinically actionable to select clinical trial
- Not actionable but therapeutics in the pipeline

To NGS or Not To NGS

Complexity of Somatic Mutation Analysis

- How many genes and which ones do we really need to test
- Which mutations are most important
- Which combinations of mutations may be important
- Are we treating the 5-20% tumor cells with mutation or the 80-95% without
- What about the 10% of cases that are wild type
- Regulatory and reimbursement issues

ABSTRACT (1998) - **DNA STAT**

Gregory J. Tsongalis

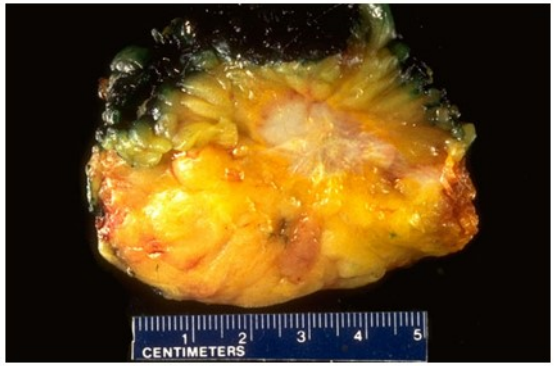
Introduction. Rapid advances in molecular biology techniques over the past few years have resulted in a transition of these technologies from the research laboratory to the clinical laboratory and in the near future to the bedside. Following in the footsteps of other more established clinical diagnostic technologies, nucleic acid testing is becoming automated and very routine for the evaluation of hematologic, infectious, and genetic diseases. One disadvantage of these new technologies has been the inability for rapid turn around times, a clinical assay attribute crucial for the critically ill patient. While a STAT designation is unbecoming of nucleic acid based tests, new methods for performing DNA/RNA extraction, amplification and detection have reduced the turn around times for these assays dramatically. The aim of this study is to demonstrate some of the time savings in performing nucleic acid tests based on currently available technologies with respect to assays suitable for the critical care patient.

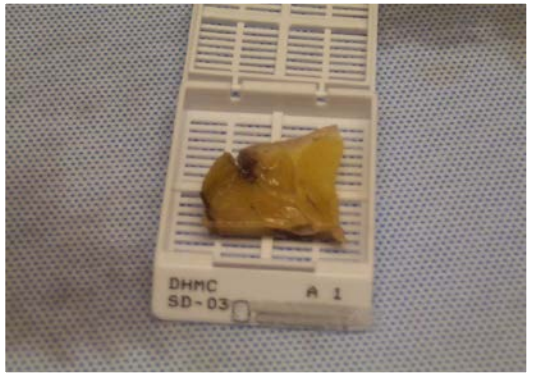
Methods. Random whole blood specimens which were submitted for CBCs were received from Hematology. DNA extraction was performed using the Puregene Kit (Gentra Systems, Minneapolis, MN) according to the recommendations of the manufacturer. Multiple PCR assays were evaluated for different target sequences, including human genomic targets and microbial targets in a time study to optimize amplification efficiency and turn around times. Detection methods included agarose and polyacrylamide gel electrophoresis, liquid hybridization assays, and fragment size analysis using an automated DNA sequencing system (OpenGene, Visible Genetics, Toronto, Canada).

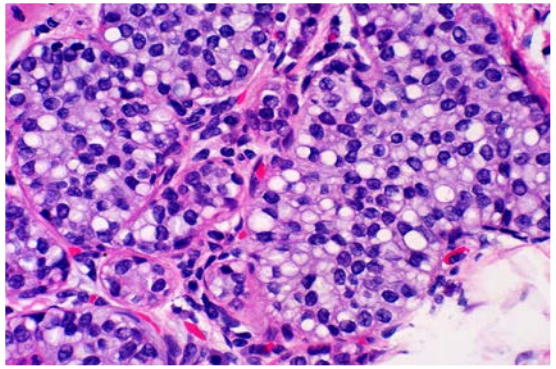
Results. Using rapid column extraction protocols, DNA suitable for PCR amplification can be isolated from whole blood specimens in less than 30 minutes. While PCR amplification times are most often target dependent, newer thermal cyclers can speed this process to less than two hours. Detection by gel electrophoresis, liquid hybridization and/or automated DNA sequencer analysis can also be accomplished within two to three hours. Thus, a completed molecular diagnostic assay for the qualitative detection of a target sequence can be accomplished with an approximately five hour turn around time.

Conclusions. In this study, we demonstrate the feasibility of a STAT nucleic acid based test. Using modified protocols and newer technologies, we are able to detect the presence of a target sequence within five hours. While five hours may not seem appropriate for a STAT designation with respect to more traditional automated clinical diagnostic assays, this is extremely rapid for a molecular based assay. However, with respect to the critical care patient, our ability to detect the presence of a microbial pathogen within a few hours versus a few days may prove crucial to decreasing morbidity and mortality of these patients. In addition, continued advances in these technologies such as DNA chip based assays and highly automated instrumentation will continue to drive turn around times downward while maintaining extraordinarily high sensitivities and specificities.

Human Cancer – The Diagnosis







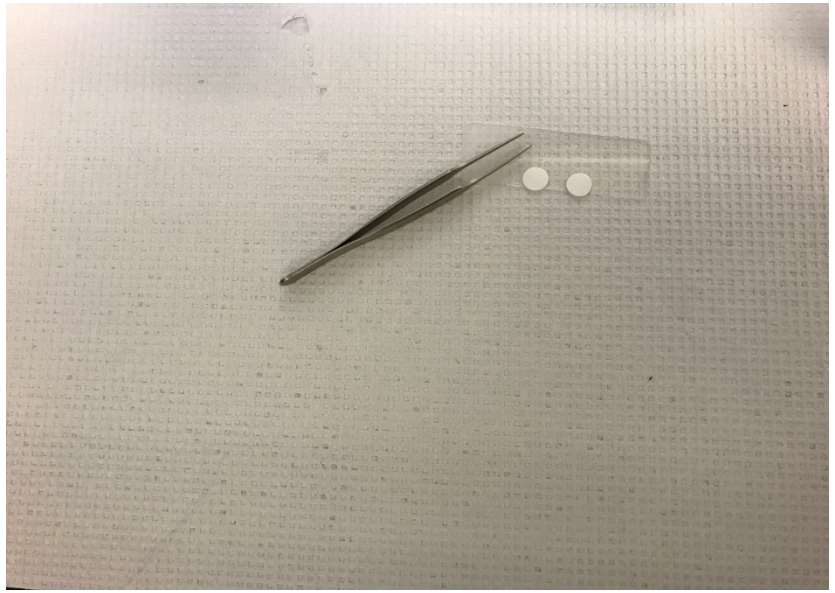
STAT DNA Testing for Oncology?

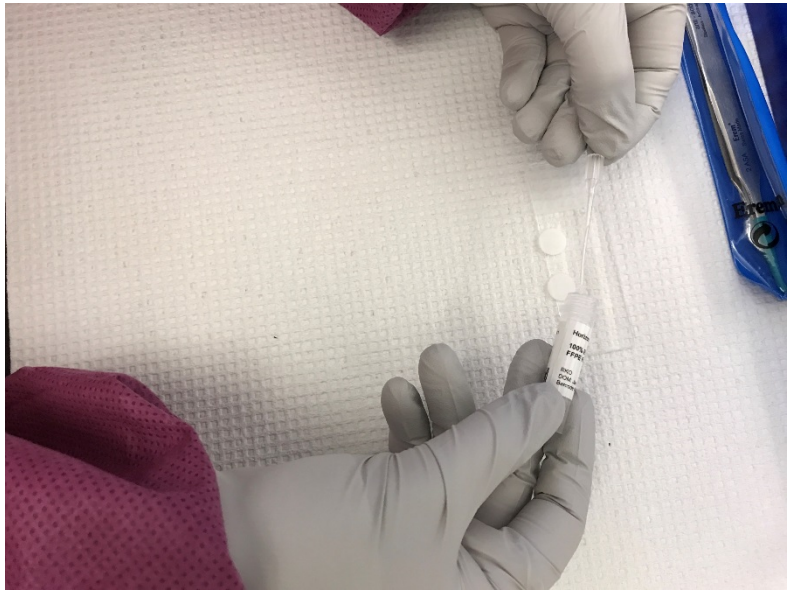
- Clinical utility
- Complex specimen (FFPE tissue)
- Assay performance
- TAT
- Data analysis

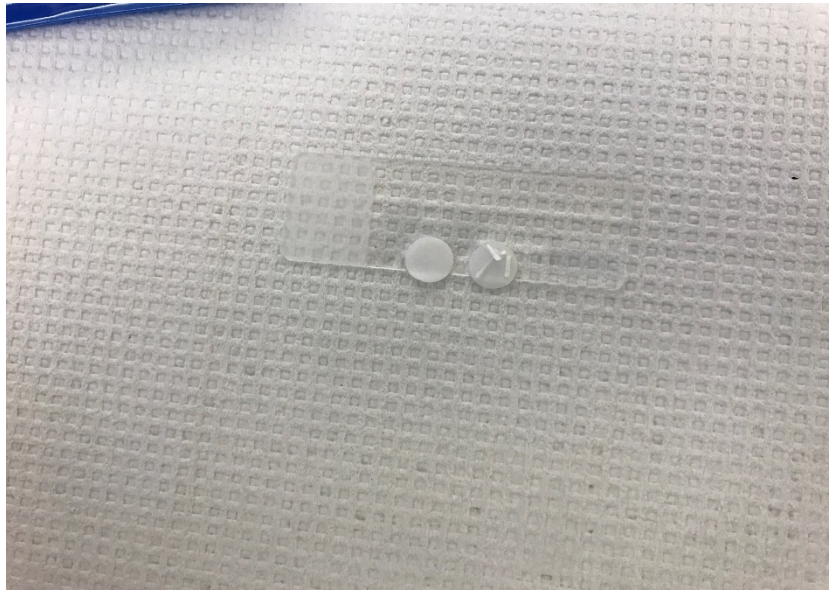
STAT DNA Testing for Oncology

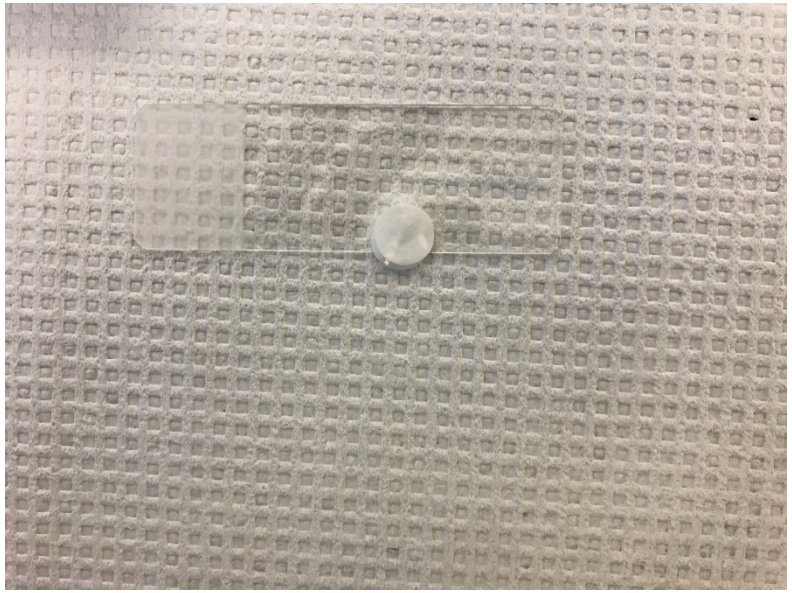
Simplifying FFPE Somatic Mutation Testing

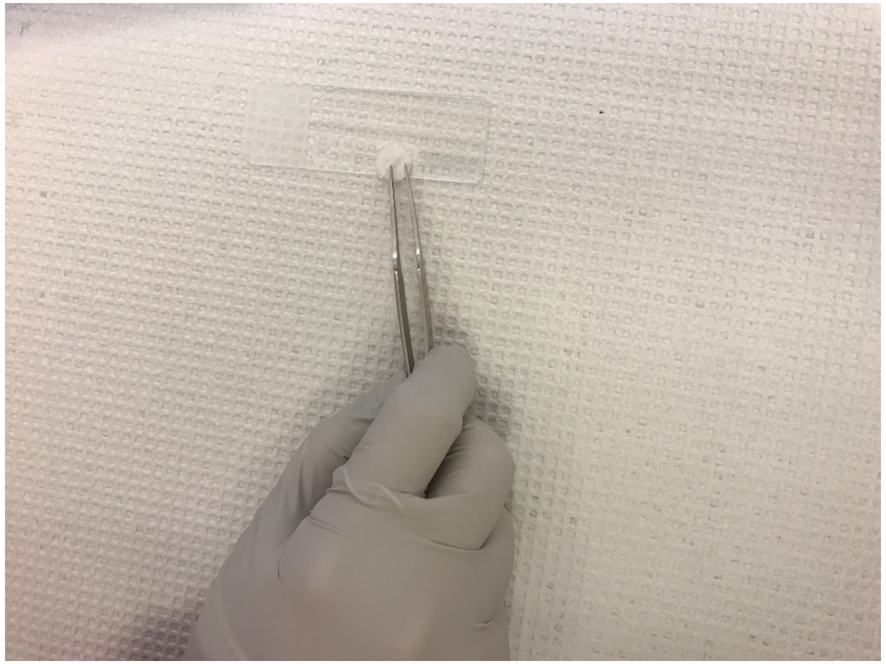
M. Rabie Al-Turkmani, PhD and Kelley Godwin, BS

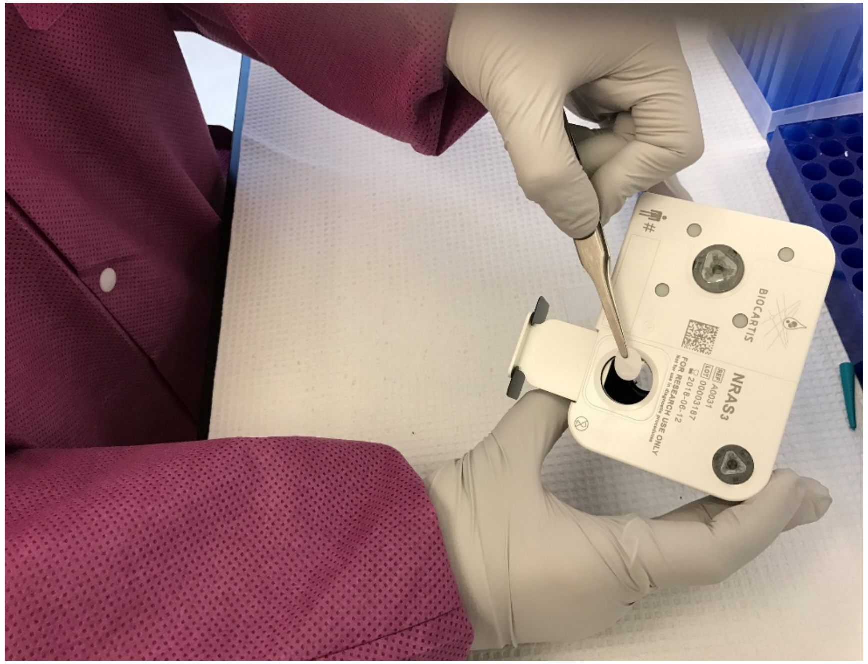




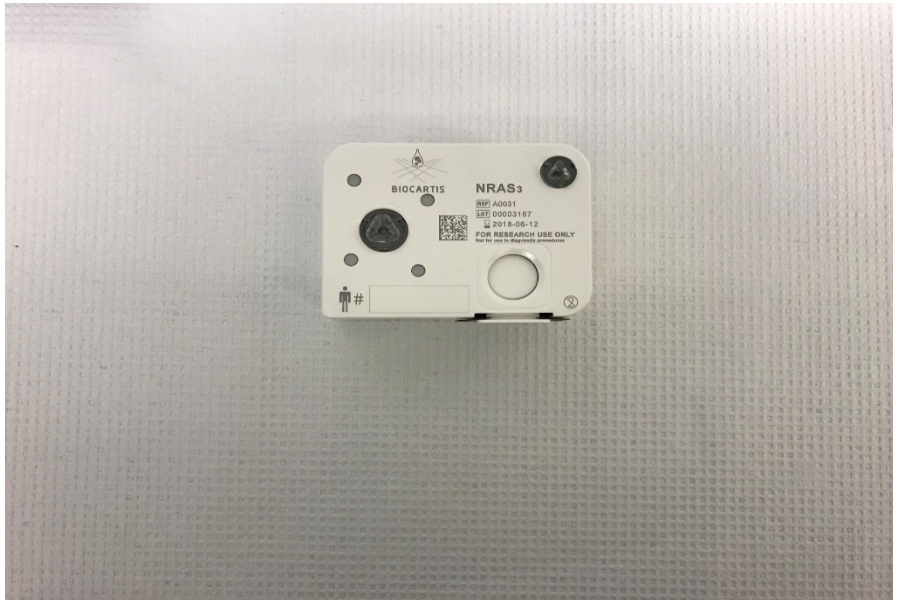









BIOCARTIS
NRAS3
REF A0031
LOT 00003187
2018-06-12
FOR RESEARCH USE ONLY
Not for use in diagnostic procedures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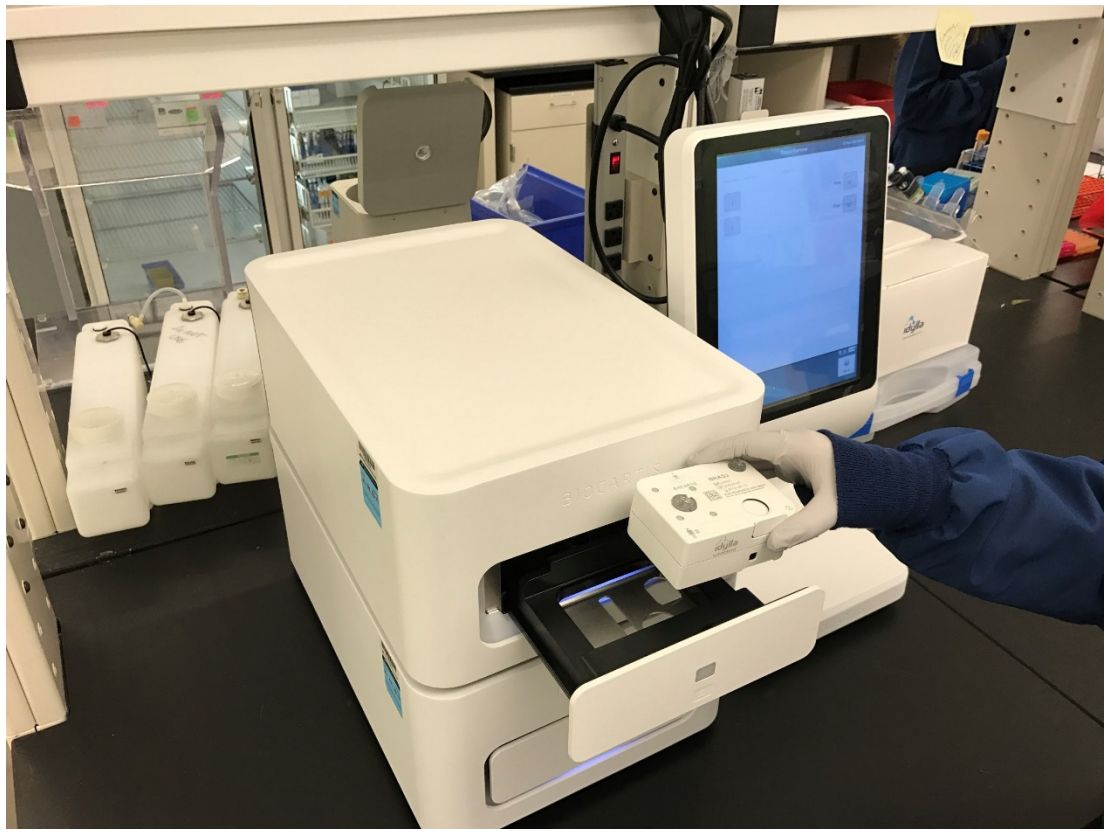
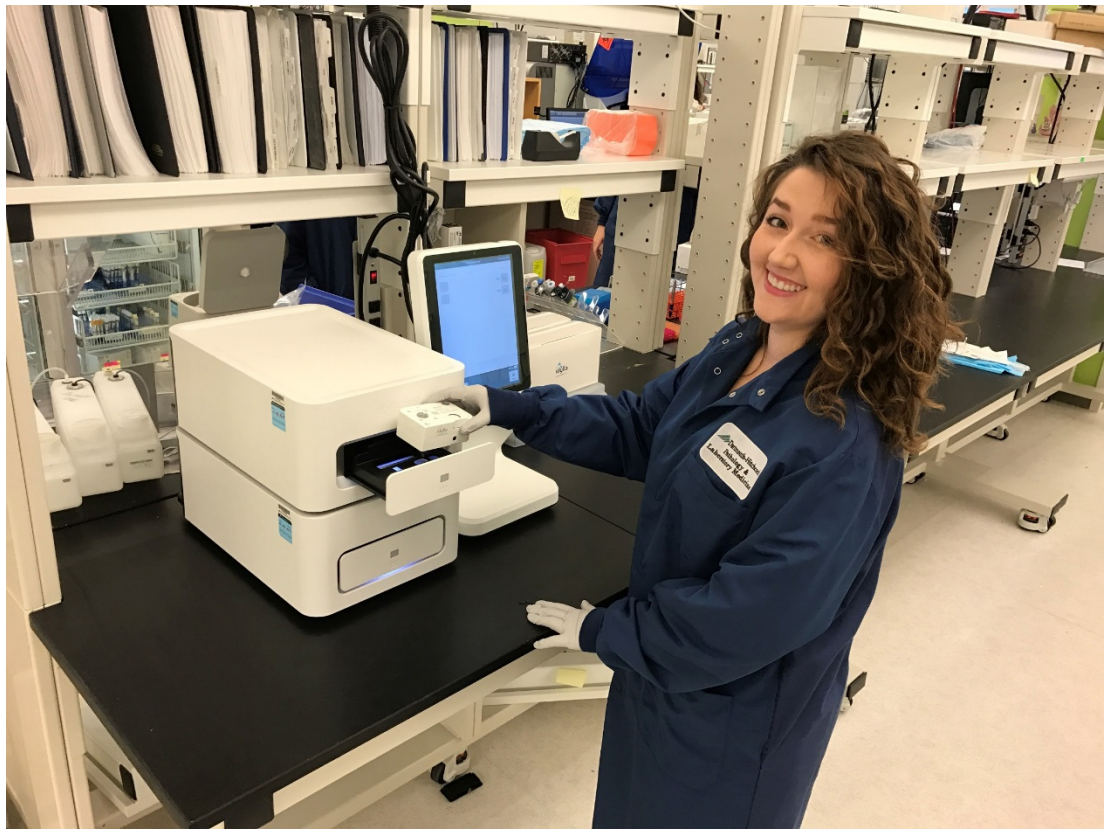
2018-06-12
FOR RESEARCH USE ONLY





Testing Steps Within the Cartridge

Lysis Chamber

Liquification buffer/lysis buffer

High intensity focused ultrasound

Heating

Enzymatic

Extraction Chamber

DNA fixed on silica membrane

Washing steps

Elution step

PCR Unit

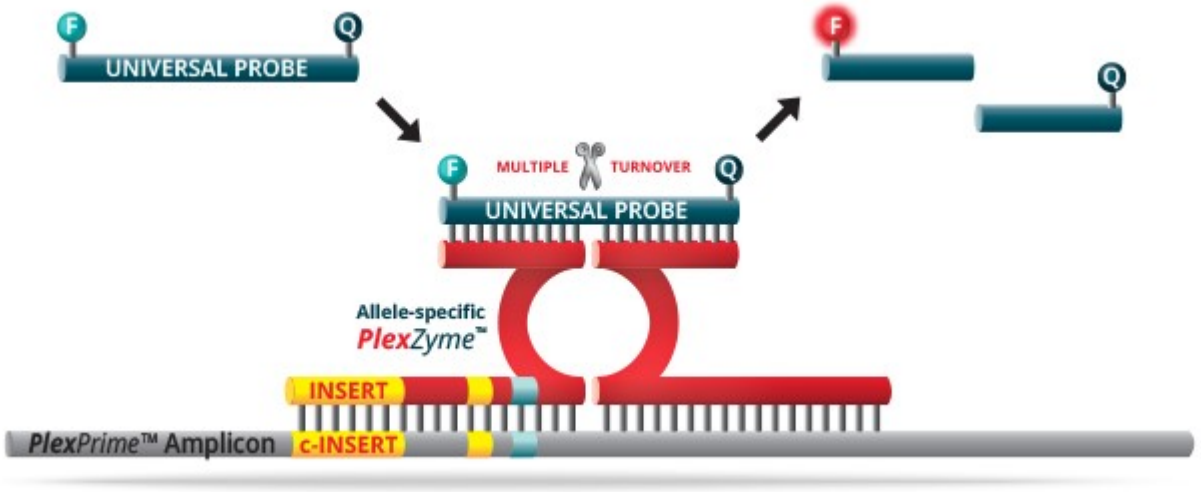
5 independent chambers

6 fluorophores per chamber

Data Analysis

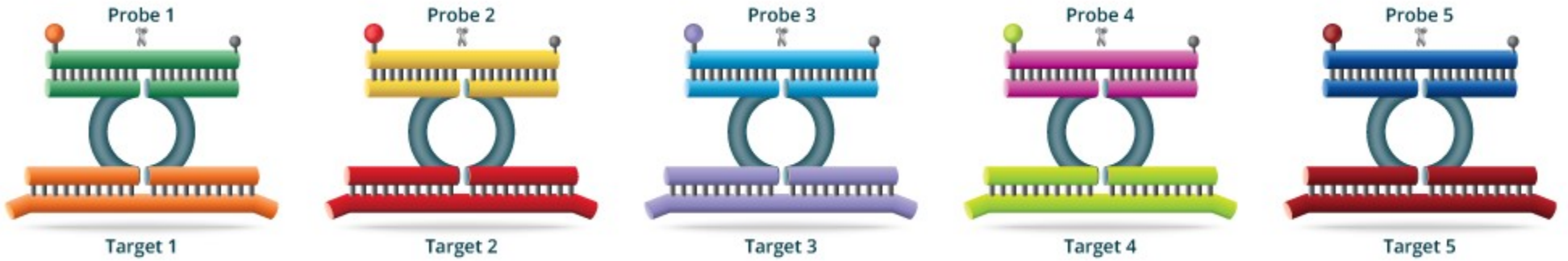
Automated

PlexPrimer® / PlexZyme®



Amplicons are detected in real time by an allele-specific ***PlexZyme®***.

PlexPrimer® / PlexZyme®



In a multiplex reaction, the universal probes are labelled with different fluorophores so that fluorescence signal corresponding to detection of each target sequence can be monitored simultaneously in real time. The highly multiplex nature of ***Plex****Zyme*® enzymes can maximize the outputs of qPCR instruments.

Idylla Assays Evaluated

- Idylla KRAS Mutation Assay
  - 21 mutations in KRAS exon 2, 3, and 4

- Idylla NRAS-BRAF-EGFR S492R Mutation Assay ($NRAS_3$)
  - 25 mutations in NRAS exon 2, 3, 4, BRAF exon 15, and EGFR exon 12

Validation of Cartridge Based Assays

- Limit of Detection – obtain Horizon FFPE controls that contain cell lines with varying allele frequencies for mutations (ideally 10% or less) and run 5-10 cartridges on the same sample.
- Precision – use the data from the LOD studies in #1 to show that the results are reproducible from run to run and operator to operator.
- Accuracy – using purchased control FFPE material or previously tested patient samples, run 5-10 samples and assess concordance with previous method.
- ****include different types of variants that assay tests for*****

Samples Analyzed

- Colorectal cancer FFPE tissue samples with mutation in *KRAS* (n=17), *NRAS* (n=5), or *BRAF* (n=12) were analyzed (total = 34).
- 10 colorectal cancer tissue samples with no mutation.
- 9 horizon control samples in triplicate (27).
- A single 10 µm FFPE tissue section was used (total of 4 sections and 2 H&E slides obtained from each sample).
- Results were compared against those previously obtained by NGS using the AmpliSeq 50-gene Cancer Hotspot Panel.

KRAS Results

Sample	Tumor Content (%)	NGS	Idylla
1	10	c.34G>T, p.G12C	c.34G>T, p.G12C
2	25	c.34G>T, p.G12C	c.34G>T, p.G12C
3	75	c.35G>A, p.G12D	c.35G>A, p.G12D
4	70	c.35G>A, p.G12D	c.35G>A, p.G12D
5	40	c.35G>A, p.G12D	c.35G>A, p.G12D
6	30	c.35G>T, p.G12V	c.35G>T, p.G12V
7	60	c.35G>T, p.G12V	c.35G>T, p.G12V
8	80	c.35G>T, p.G12V	c.35G>T, p.G12V
9	25	c.38G>A, p.G13D	c.38G>A, p.G13D
10	40	c.38G>A, p.G13D	c.38G>A, p.G13D
11	50	c.38G>A, p.G13D	c.38G>A, p.G13D
12	50	c.38G>A, p.G13D	c.38G>A, p.G13D
13	40	c.181C>A, p.Q61K	**c.181C>A** / c.180_181 delinsAA, **p.Q61K**
14	50	c.182A>G, p.Q61R	**c.182A>G** / c.182A>T, **p.Q61R**/L
15	75	c.182A>G, p.Q61R	**c.182A>G** / c.182A>T, **p.Q61R**/L
16	40	c.436G>A, p.A146T	c.436G>C/ **c.436G>A**/ c.437 C>T, **p.A146**P/**T**/V
17	50	c.436G>A, p.A146T	c.436G>C/ **c.436G>A**/ c.437 C>T, **p.A146**P/**T**/V

NRAS Results

Sample	Tumor Content (%)	NGS	Idylla
1	85	c.35G>T, p.G12V	**c.35G>T**, c.35G>T**, p.G12**A/**V**
2	70	c.37G>C, p.G13R	**c.37G>C**/ c.38G>T, **p.G13R**/V
3	50	c.183A>C, p.Q61H	**c.183A>C**; c.183A>T , **p.Q61H**
4	40	c.183A>T, p.Q61H	c.183A>C; **c.183A>T , p.Q61H**
5	80	c.183A>T, p.Q61H	c.183A>C; **c.183A>T , p.Q61H**

BRAF Results

Sample	Tumor Content (%)	NGS	Idylla
1	60	c.1799T>A, p.V600E	**c.1799T>A**; c.1799_1800delinsAA/c.1799_1800delinsAC, **p.V600E**/D
2	50	c.1799T>C, p.V600E	**c.1799T>A**; c.1799_1800delinsAA/c.1799_1800delinsAC, **p.V600E**/D
3	50	c.1799T>A, p.V600E	**c.1799T>A**; c.1799_1800delinsAA/c.1799_1800delinsAC, **p.V600E**/D
4	60	c.1799T>A, p.V600E	**c.1799T>A**; c.1799_1800delinsAA/c.1799_1800delinsAC, **p.V600E**/D
5	50	c.1799T>A, p.V600E	**c.1799T>A**; c.1799_1800delinsAA/c.1799_1800delinsAC, **p.V600E**/D
6	50	c.1799T>A, p.V600E	**c.1799T>A**; c.1799_1800delinsAA/c.1799_1800delinsAC, **p.V600E**/D
7	60	c.1799T>A, p.V600E	**c.1799T>A**; c.1799_1800delinsAA/c.1799_1800delinsAC, **p.V600E**/D
8	20	c.1799T>A, p.V600E	**c.1799T>A**; c.1799_1800delinsAA/c.1799_1800delinsAC, **p.V600E**/D
9	50	c.1799T>A, p.V600E	**c.1799T>A**; c.1799_1800delinsAA/c.1799_1800delinsAC, **p.V600E**/D
10	70	c.1799T>A, p.V600E	**c.1799T>A**; c.1799_1800delinsAA/c.1799_1800delinsAC, **p.V600E**/D
11	75	c.1799T>A, p.V600E	**c.1799T>A**; c.1799_1800delinsAA/c.1799_1800delinsAC, **p.V600E**/D
12	30	c.1799T>A, p.V600E	**c.1799T>A**; c.1799_1800delinsAA/c.1799_1800delinsAC, **p.V600E**/D

Horizon Control Results

Mutation	Tumor Content (%)	Repeats	Idylla
KRAS G12V	50	3	c.35G>T, p.G12V
KRAS G13D	50	3	c.38G>A, p.G13D
KRAS Q61H	50	3	c.183A>C / c.183A>T, p.Q61H
KRAS A146 T	50	3	c.436G>C/ c.436G>A/ c.437 C>T, p.A146P/T/V
NRAS Q61H	50	3	c.183A>C, p.Q61H
NRAS Q61L	50	3	c.182A>T, p.Q61L
NRAS Q61R	50	3	c.182A>G, p.Q61R
BRAF V600E	50	3	c.1798T>A; c.1799_1800delinsAA/c.1799_1800delinsAC, p.V600E/D
BRAF V600R	50	3	c.1798_1799 delinsAA/c.1798_1799delinsAG, p.V600K/R

A

KRAS GENOTYPE	MUTATION DETECTED IN KRAS CODON 13
Mutation	G13D
Protein	p.Gly13Asp
Nucleotide Change	c.38G>A

B

NRAS GENOTYPE	MUTATION DETECTED IN NRAS CODON 61
Mutation	Q61H
Protein	p.Gln61His
Nucleotide Change	c.183A>C ; c.183A>T
BRAF GENOTYPE	NO MUTATION DETECTED IN BRAF CODON 600
EGFR GENOTYPE	NO MUTATION DETECTED IN EGFR CODON 492

C

NRAS GENOTYPE	NO MUTATION DETECTED IN NRAS CODON 12,13,59,61,117,146
BRAF GENOTYPE	MUTATION DETECTED IN BRAF CODON 600
Mutation	V600E/D
Protein	p.Val600Glu / p.Val600Asp
Nucleotide Change	c.1799T>A ; c.1799_1800delinsAA / c.1799_1800delinsAC
EGFR GENOTYPE	NO MUTATION DETECTED IN EGFR CODON 492

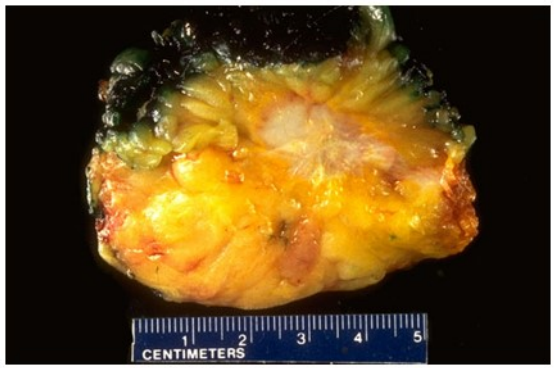
KRAS Detection in Colonic Tumors by DNA Extraction From FTA Paper: The Molecular Touch-Prep

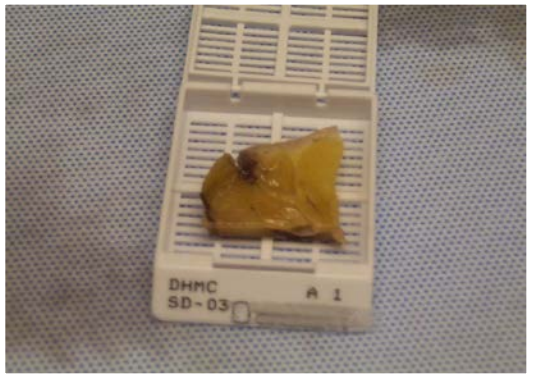
Melissa L. Petras, Joel A. Lefferts, Brian P. Ward, Arief A. Suriawinata, Gregory J. Tsongalis

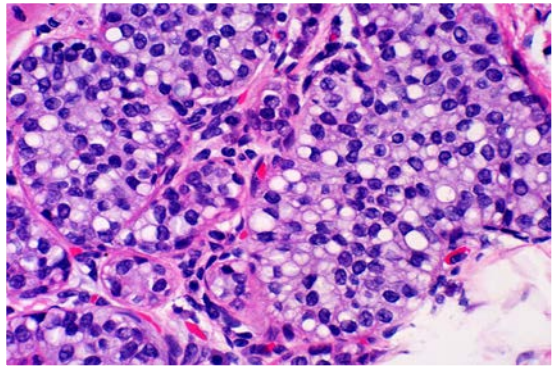
Diagnostic Molecular Pathology. 20(4):189–193, DEC 2011
DOI: 10.1097/PDM.0b013e318211d554

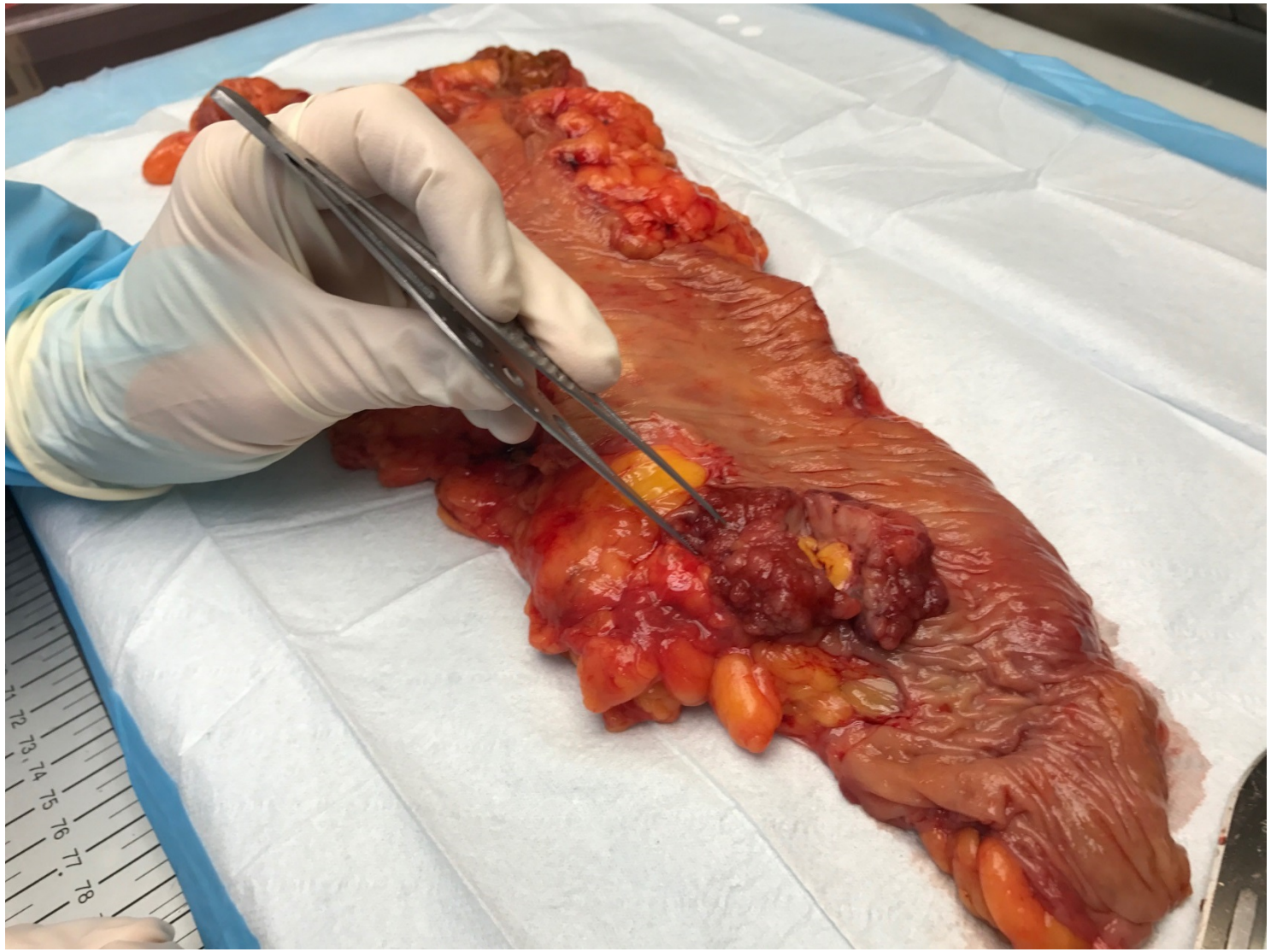
PMID: 22089345
Issn Print: 1052-9551
Publication Date: 2011/12/01

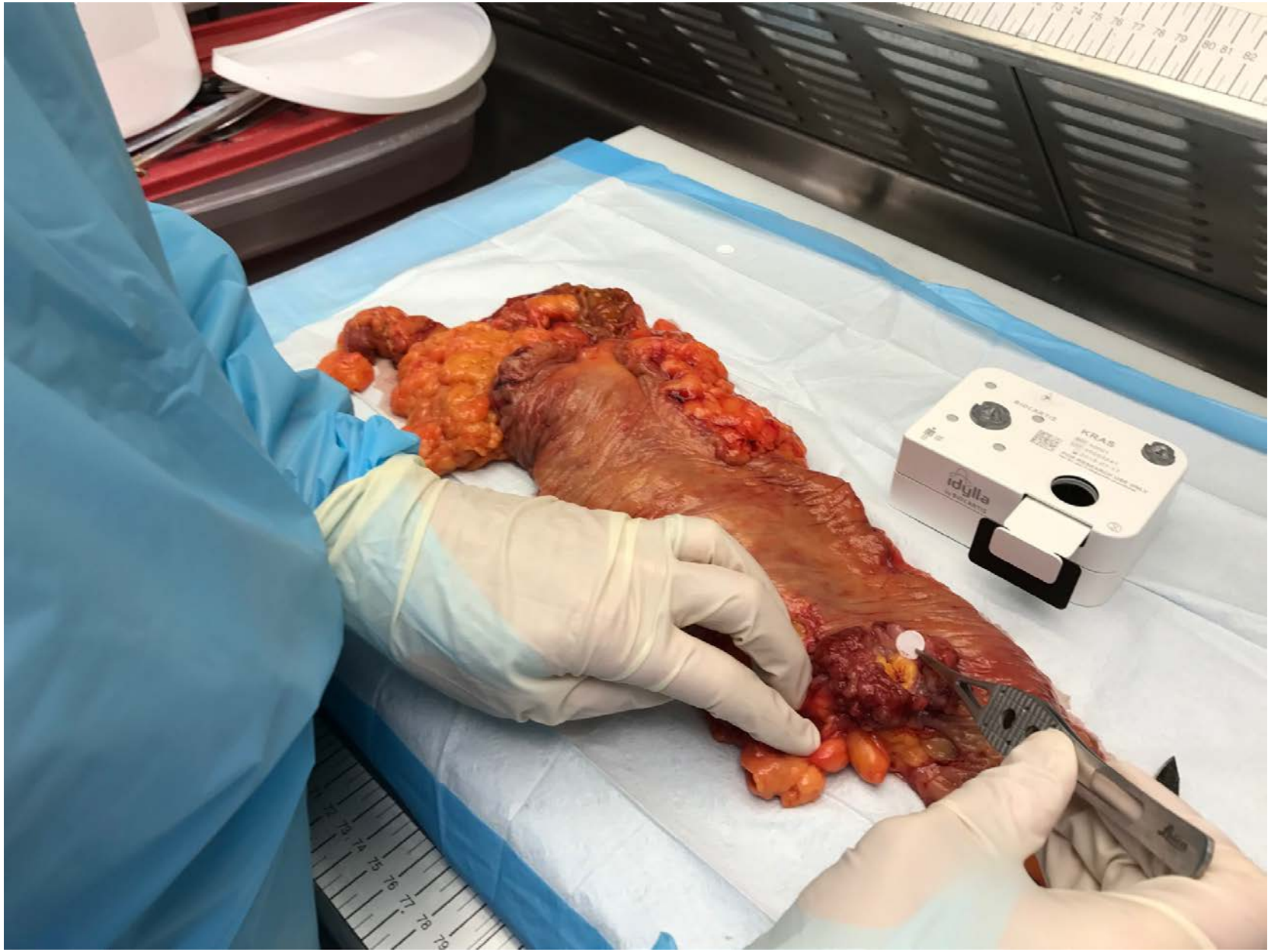
Human Cancer – The Diagnosis










KRAS
idylla


KRAS
idylla

Potential of STAT Somatic Mutation Testing at Resection

M. Rabie Al-Turkmani, Shannon N. Schutz, Gregory J. Tsongalis

Clinical Chemistry 64:5

STAT DNA Testing for Oncology

- Robust performance
- Rapid TAT
- Ease of use
- Molecular touch prep
- Targeted mutations but ALL are actionable
- Potential for liquid bx analysis

Clinical Genomics and Advanced Technology (CGAT)

CGAT- Fellows
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M. Rabie Al-Turkmani

Precision Summer Interns
Rachel Barney – UVM
Sarah Benware – UNH
Jamie Dinulos – Dartmouth
Mackenzie Keegan – Northeastern
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Greg Tsongalis
Wendy Wells

Please note:

The Biocartis Idylla™ instrument and console are approved for IVD use while the oncology cartridges are for research use only in the United States.

NGS technologies are for research use only.