Point of Care Molecular Testing

Streamlining Cancer Care from the Anatomic Pathologist's Office

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Medical Oncology

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Speaker Information and Disclosures

Disclosures, Dr. B Sheffield

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Objectives

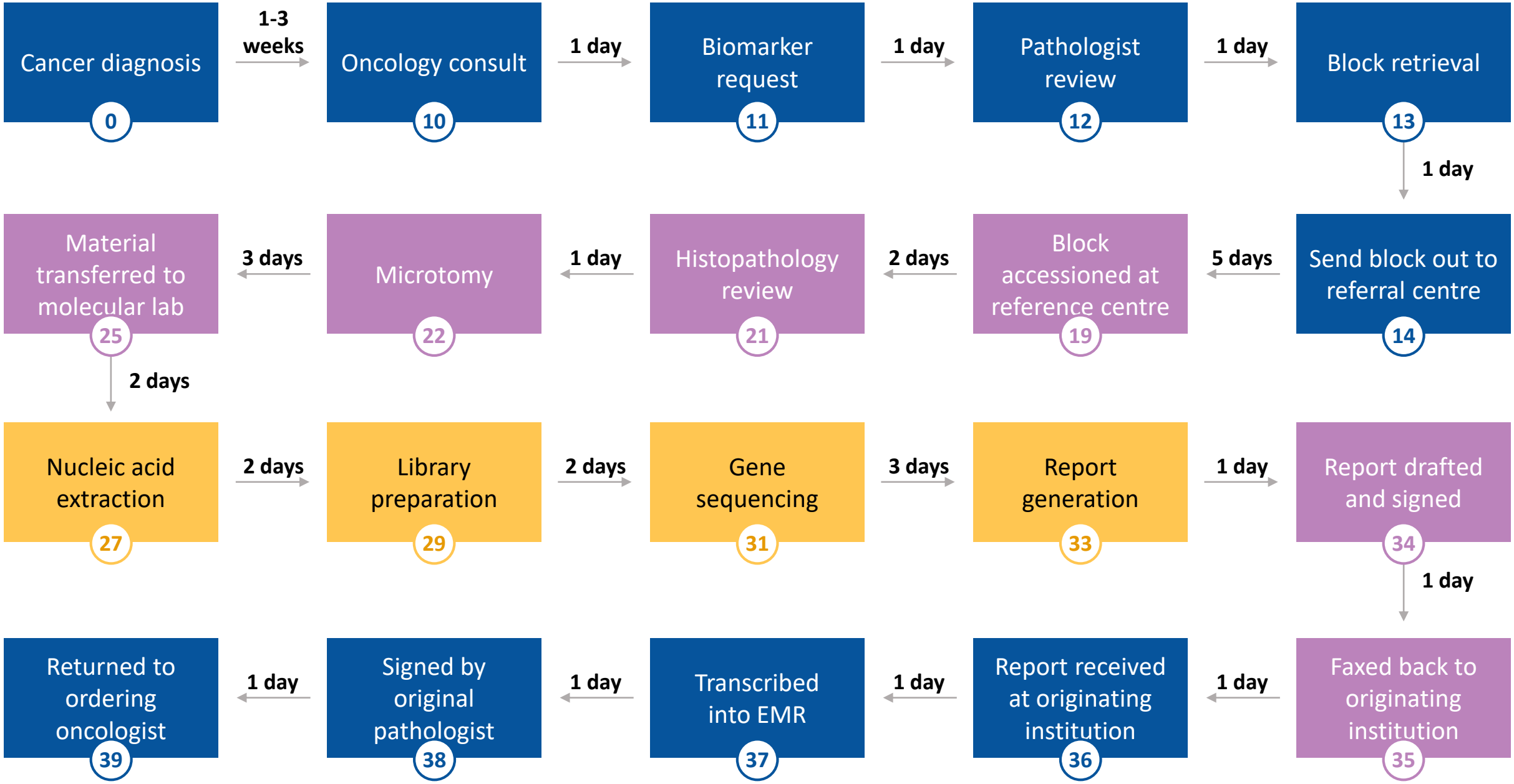
1. Foster an appreciation for the role of ancillary biomarker testing in the treatment of cancer patients.
2. Appreciate how delays in test results can adversely affect cancer care.
3. Identify areas within your own lab or network that impede biomarker results.
4. Explore how existing and novel techniques can help support oncology practice within your centre.

Current state

1. Cancer is diagnosed by an anatomic pathologist

2. Cancer-related testing is requested by a medical oncologist

3. Biomarker testing is performed in a separate molecular facility


Cancer diagnosis
0
1-3 weeks
Oncology consult
10
1 day
Biomarker request
11
1 day
Pathologist review
12
1 day
Block retrieval
13
1 day
Send block out to referral centre
14
5 days
Block accessioned at reference centre
19
2 days
Histopathology review
21
1 day
Microtomy
22
3 days
Material transferred to molecular lab
25
2 days
Nucleic acid extraction
27
2 days
Library preparation
29
2 days
Gene sequencing
31
3 days
Report generation
33
1 day
Report drafted and signed
34
1 day
Faxed back to originating institution
35
1 day
Report received at originating institution
36
1 day
Transcribed into EMR
37
1 day
Signed by original pathologist
38
1 day
Returned to ordering oncologist
39

Net effect

X

Delayed biomarker testing

X

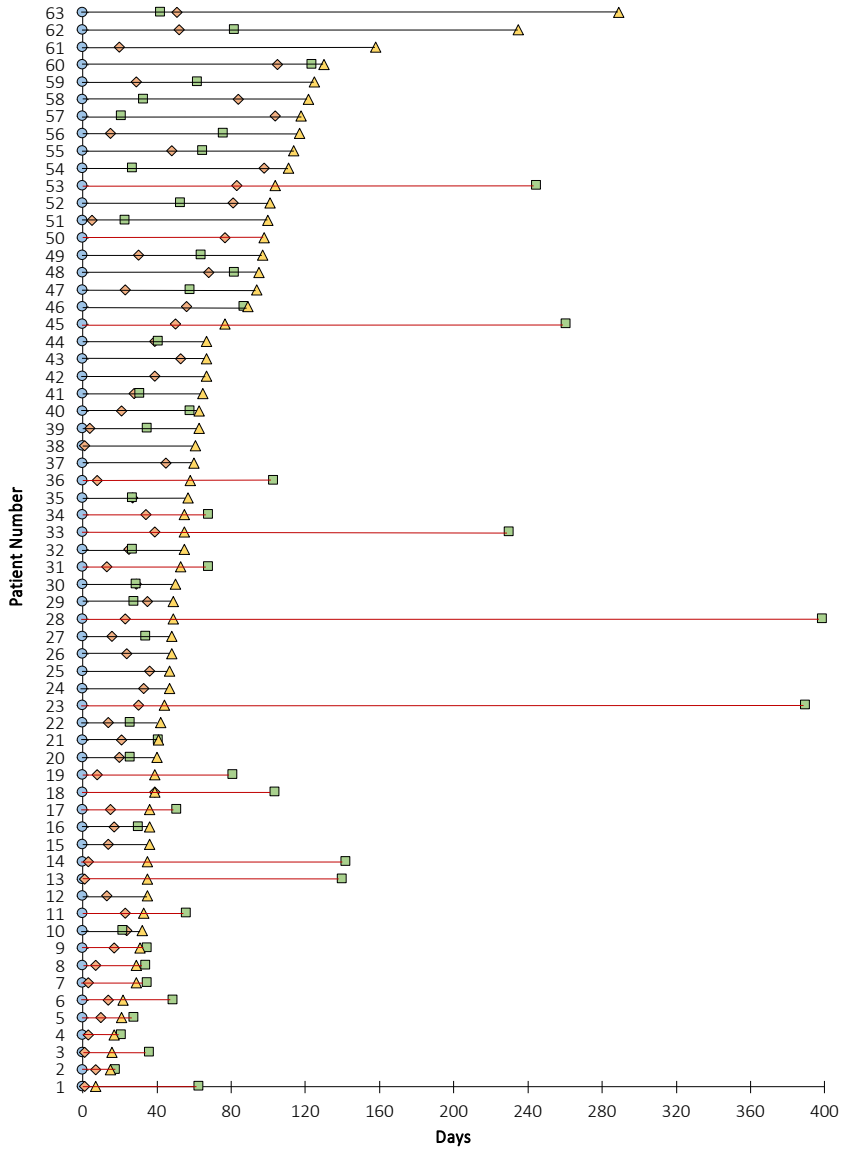
Inefficient use of pathologist / oncologist time

X

Missed treatment opportunities

X

Inappropriate treatment decision

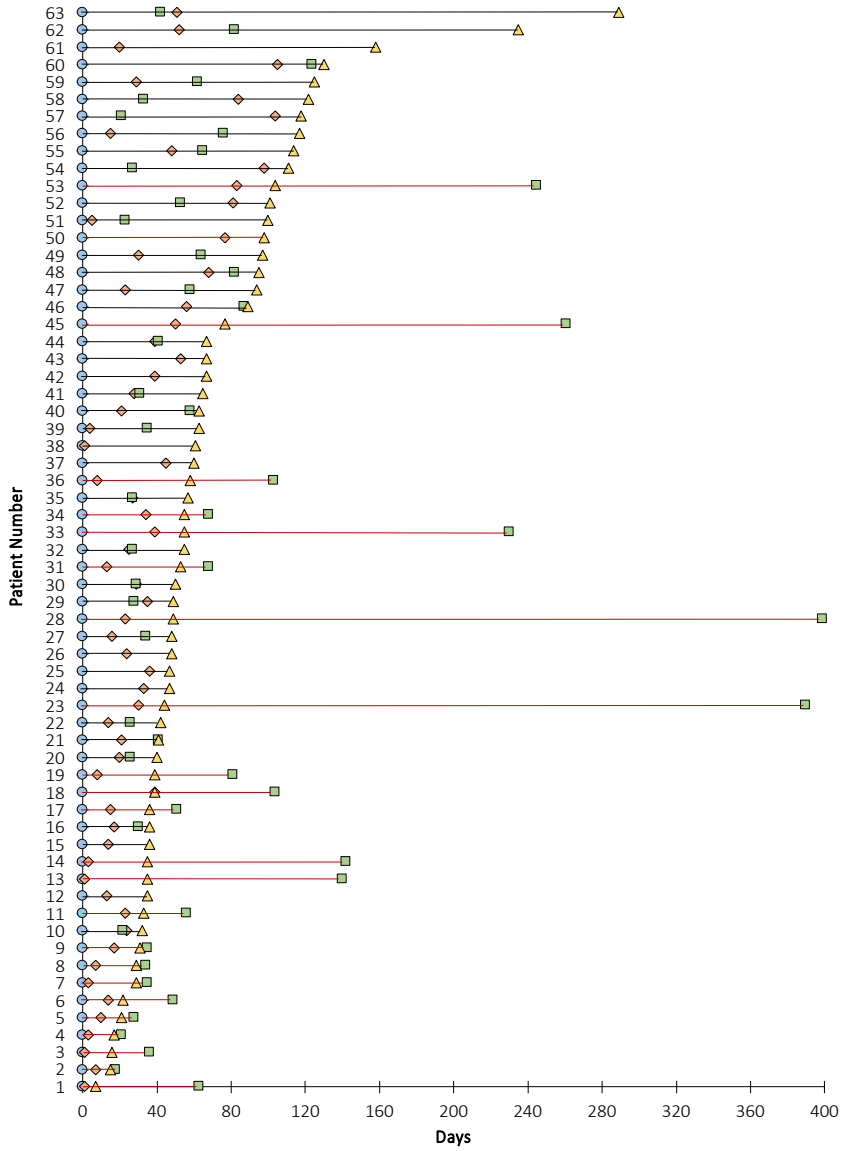


Diagnosis

Oncology Consult Following Diagnosis

Biomarker Report Completion

Treatment



Diagnosis

Oncology Consult Following Diagnosis

Biomarker Report Completion

Treatment

Median turnaround time:
64 days

Biomarkers available at oncology consult:
17%

Consequences of Inefficient Biomarker Testing

The mortality rate of untreated advanced NSCLC is 4% per week[1]

Median life expectancy for stage IV NSCLC is 16 weeks [2]

1. Stewart, D, *et al.* The cost of delaying therapy for advanced non-small cell lung cancer (NSCLC): a population kinetics assessment. 2020 AACR 18(S16):5489.

2. DiStasio, et al. Molecular Testing Turnaround Time for Non-Small Cell Lung Cancer in Routine Clinical Practice Conforms Feasibility of CAP/IASLC/AMP Guideline Recommendations: A Single-center Analysis. *Clinical Lung Cancer* 2017.

COLLEGE of AMERICAN PATHOLOGISTS

IASLC
INTERNATIONAL ASSOCIATION FOR THE STUDY OF LUNG CANCER
Conquering Thoracic Cancers Worldwide

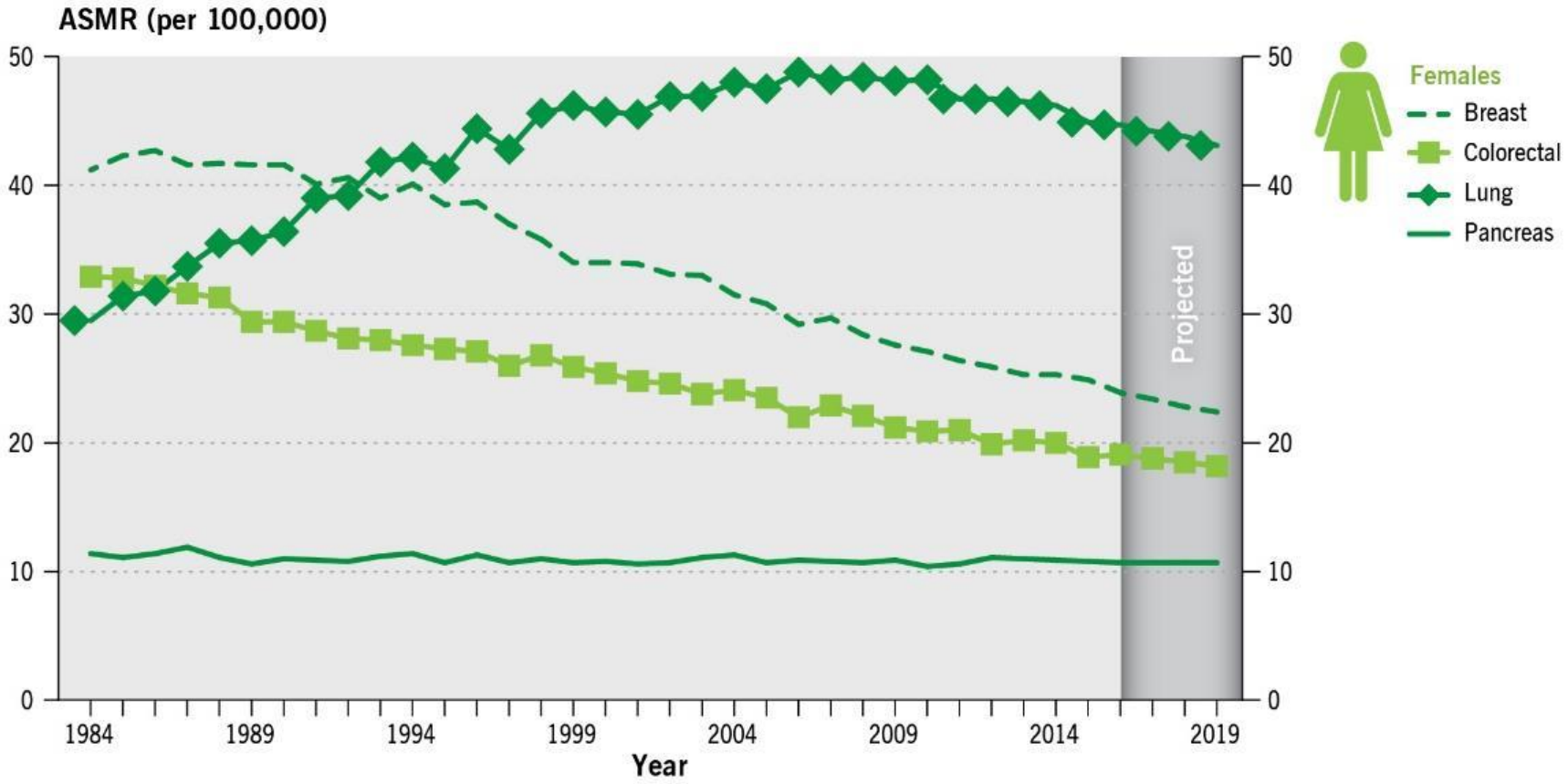
AMP ASSOCIATION FOR MOLECULAR PATHOLOGY

Updated Molecular Testing Guideline for the Selection of Lung Cancer Patients for Treatment With Targeted Tyrosine Kinase Inhibitors

3.2. Expert consensus opinion: Laboratories with average turnaround times beyond two weeks need to make available a more rapid test–either in house or through a reference laboratory–in instances of clinical urgency.
3.1: Expert consensus opinion: EGFR and ALK results should be available within two weeks (10 working days) of receiving the specimen in the testing laboratory.
3.3. Expert consensus opinion: Laboratory departments should establish processes to ensure that specimens that have a final histopathological diagnosis are sent to outside molecular pathology laboratories within 3 working days of receiving requests and to intramural molecular pathology laboratories within 24 hours.

Arch Pathol Lab Med. 2018;142:321–346

FIGURE 2.9 Age-standardized mortality rates (ASMR) for selected* cancers, females, Canada, 1984–2019



Canadian Cancer Society / Société canadienne du cancer

Government of Canada / Gouvernement du Canada

DIAGNOSIS

BREAST (RIGHT, 7 O'CLOCK), NEEDLE BIOPSY:

- INVASIVE DUCTAL CARCINOMA.
- Preliminary grade: 2 (tubules 3, nuclei 2, mitoses 1).
- Biomarkers:
  - ER: POSITIVE (3+ staining in 100% of tumor nuclei; Allred 8).
  - PR: POSITIVE (3+ staining in 100% of tumor nuclei; Allred 8).
  - HER2: negative (IHC 1+).

DIAGNOSIS

BREAST (LEFT, LESION A), NEEDLE BIOPSY:

- **INVASIVE DUCTAL CARCINOMA.**

1. **Preliminary grade: 3 (tubules 3, mitoses 3, nuclei 3).**
2. **Biomarkers:**
   - **ER: negative (no staining present, no internal control present; Allred 0).**
   - **PR: negative (no staining present, no internal control present; Allred 0).**
   - **HER2: negative (IHC 0).**
   - **Ki67: HIGH (nearly 100% tumor cell labelling).**

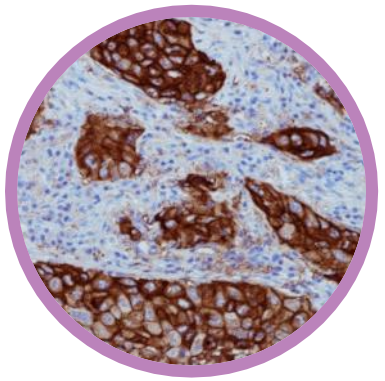
COMMENT: The tumor shows a triple negative (ER-/PR-/HER2-) immunophenotype. No internal control is present for ER and PR stains, repeat testing on a subsequent specimen is recommended. Clinical correlation is required in determining the need for BRCA1/2 testing.

Point of care

For anatomic pathologists

Order

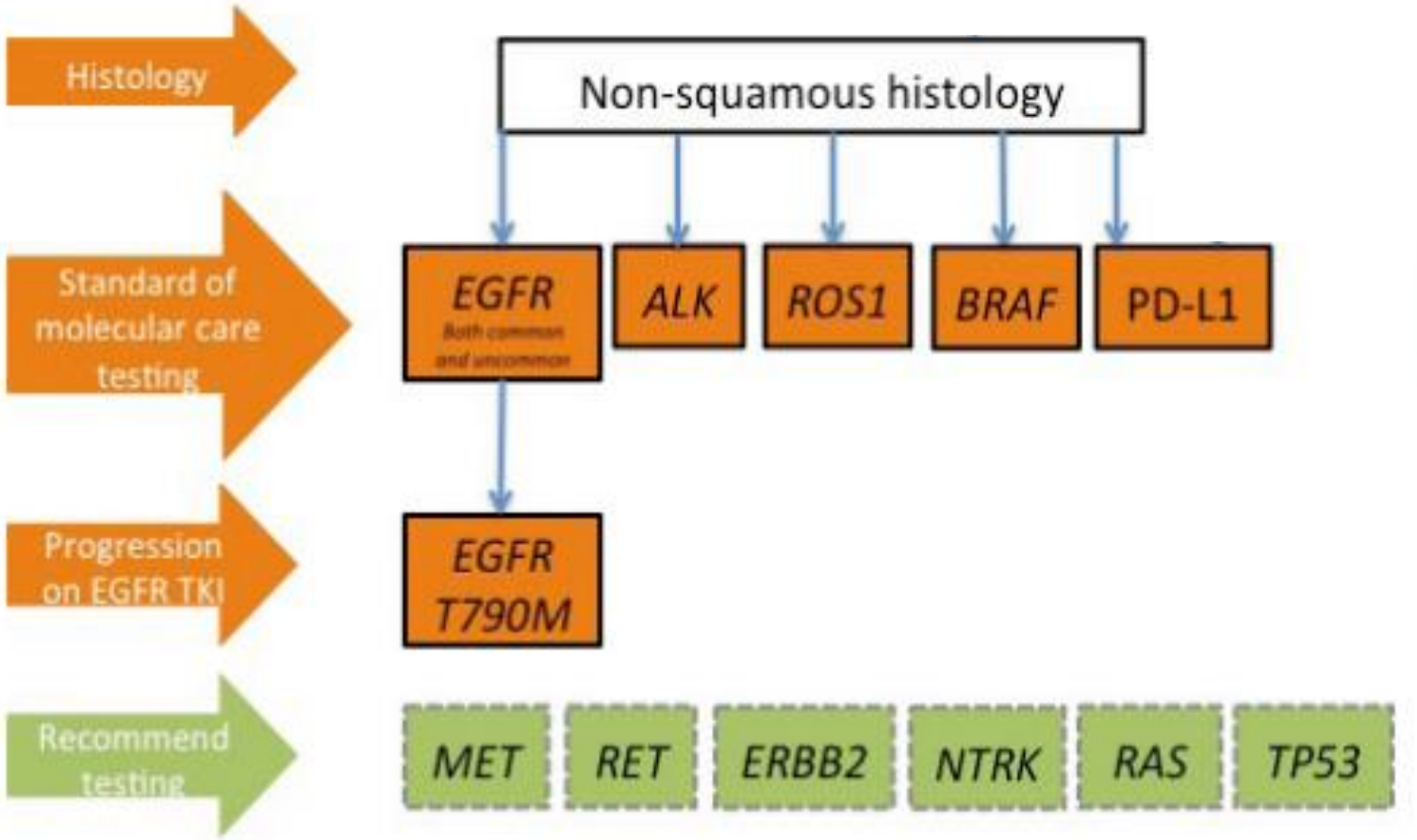
Interpret



Report

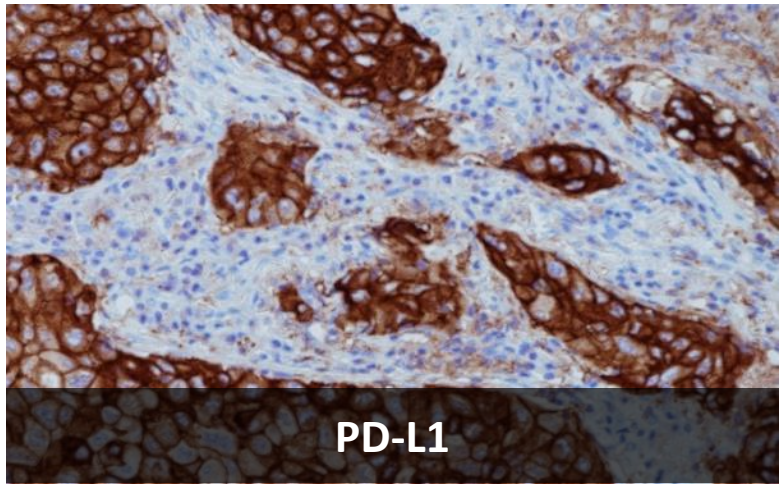
One facility.
One pathologist.
One report.

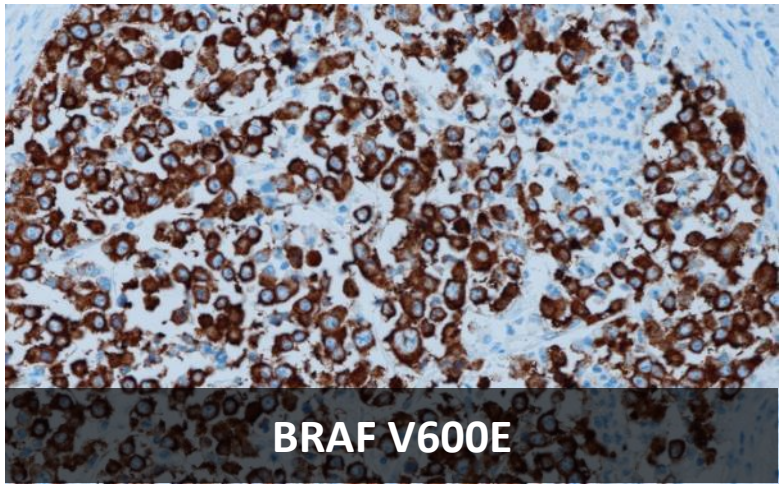
Canadian testing recommendations

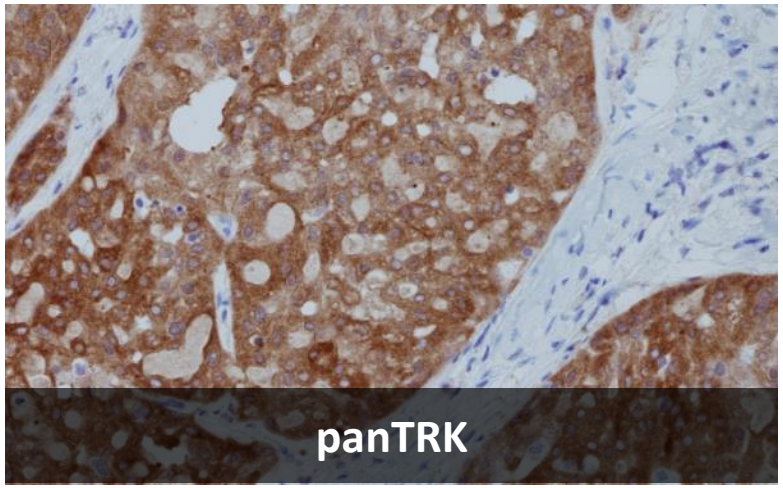


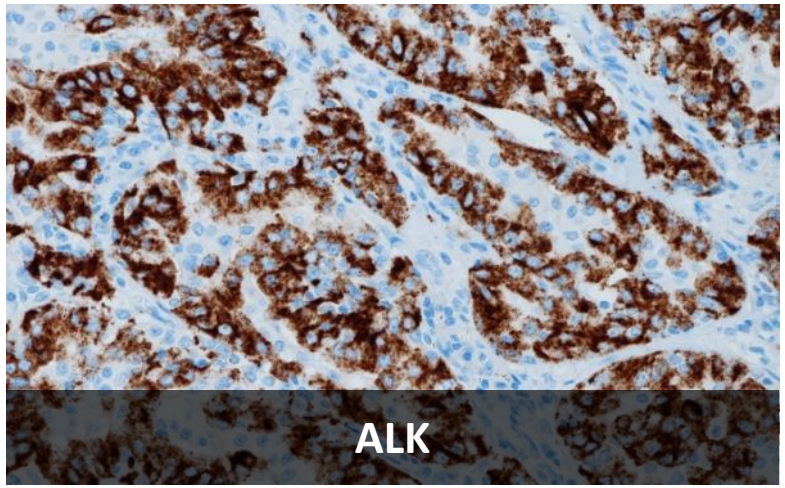
Curr Oncol. 2018 25(1): 73-82.

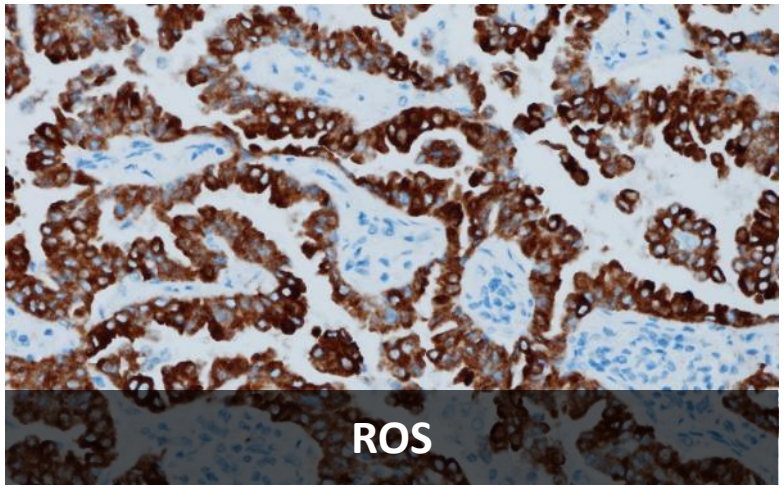
Immunohistochemistry as a Practical Tool in Molecular Pathology





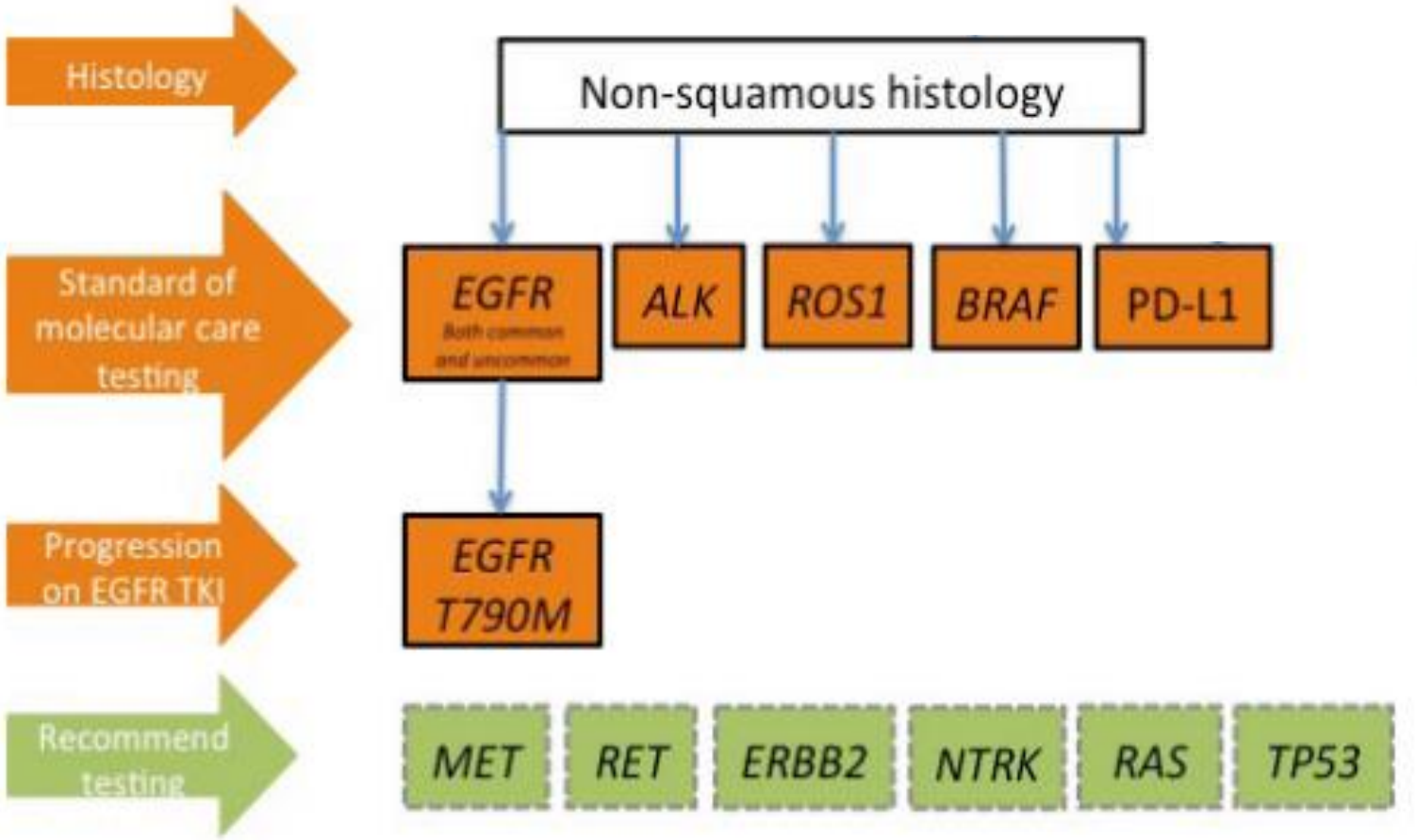






Arch Pathol Lab Med. 2016;140:766–769.

Canadian testing recommendations



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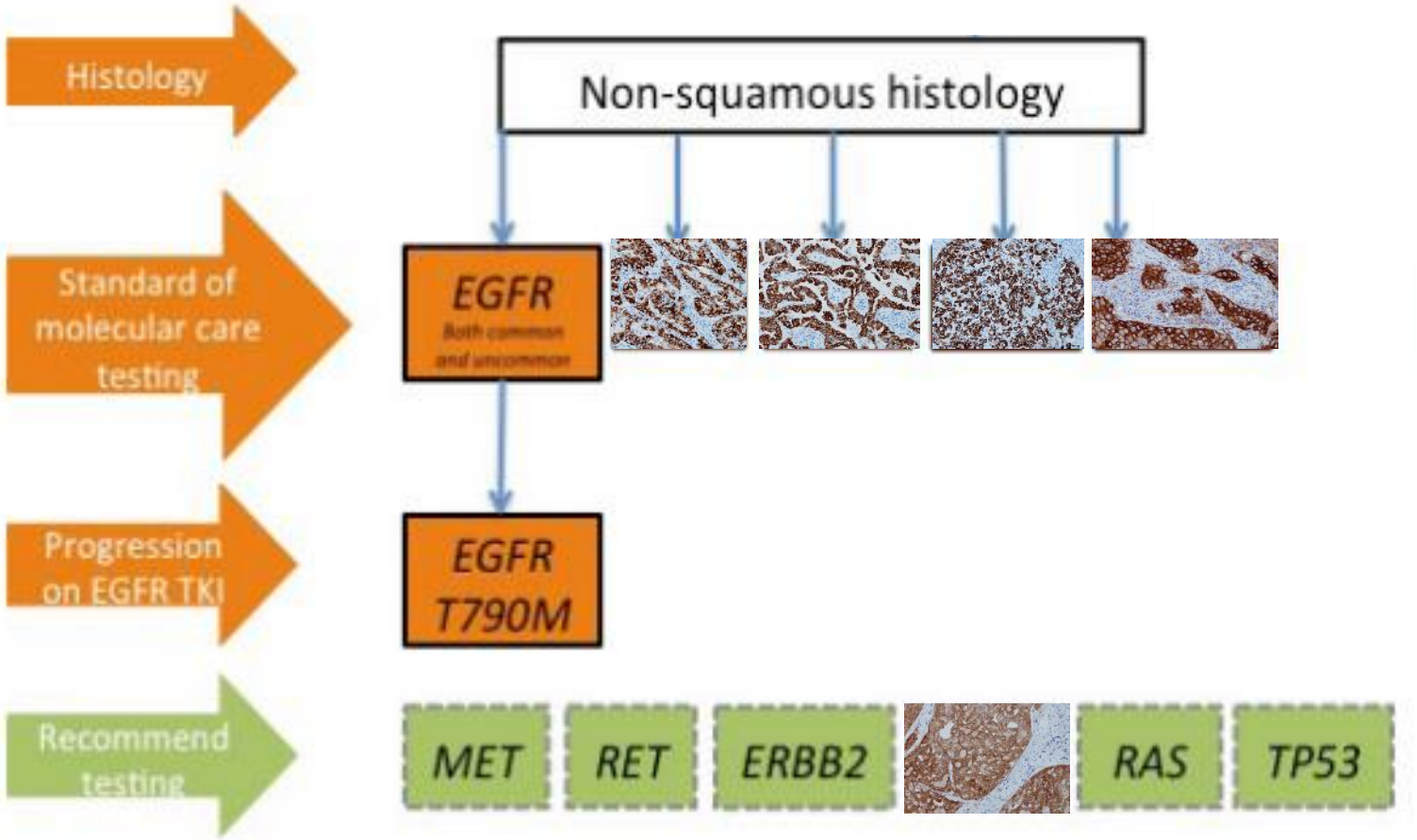

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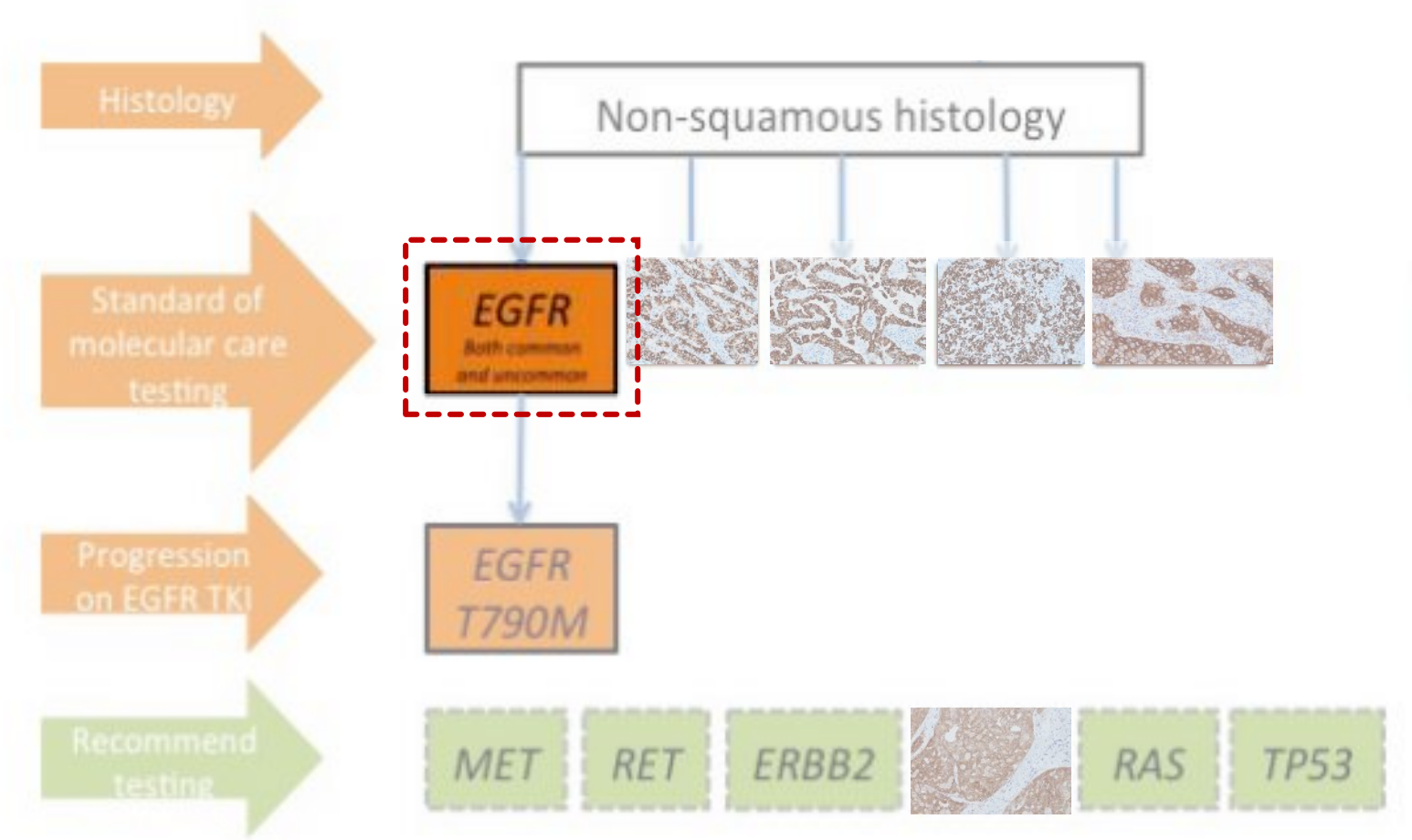

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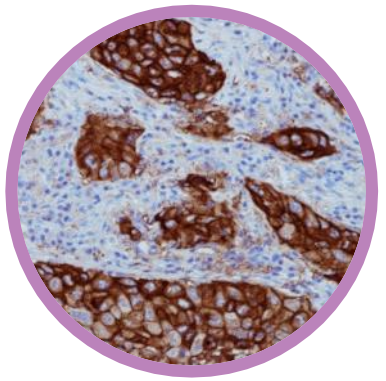
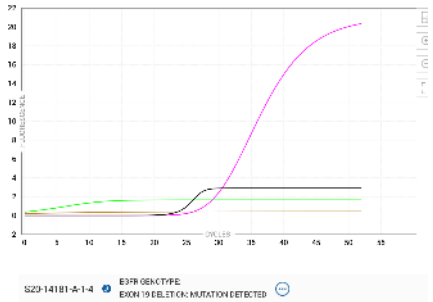
Point of care

For anatomic pathologists

Order

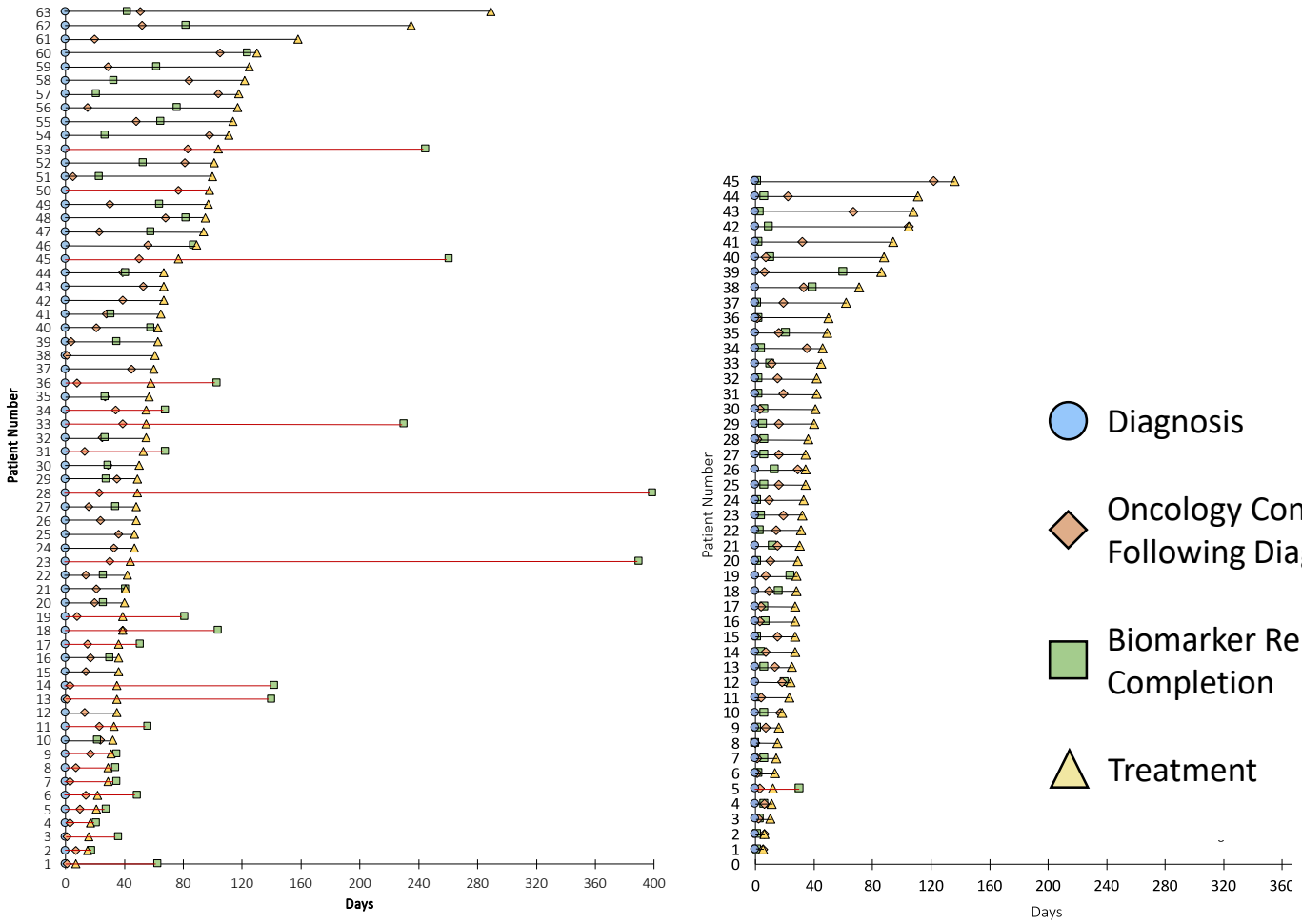
Interpret





Report

One facility.
One pathologist.
One report.



Median Turnaround Time:

~~64 days~~

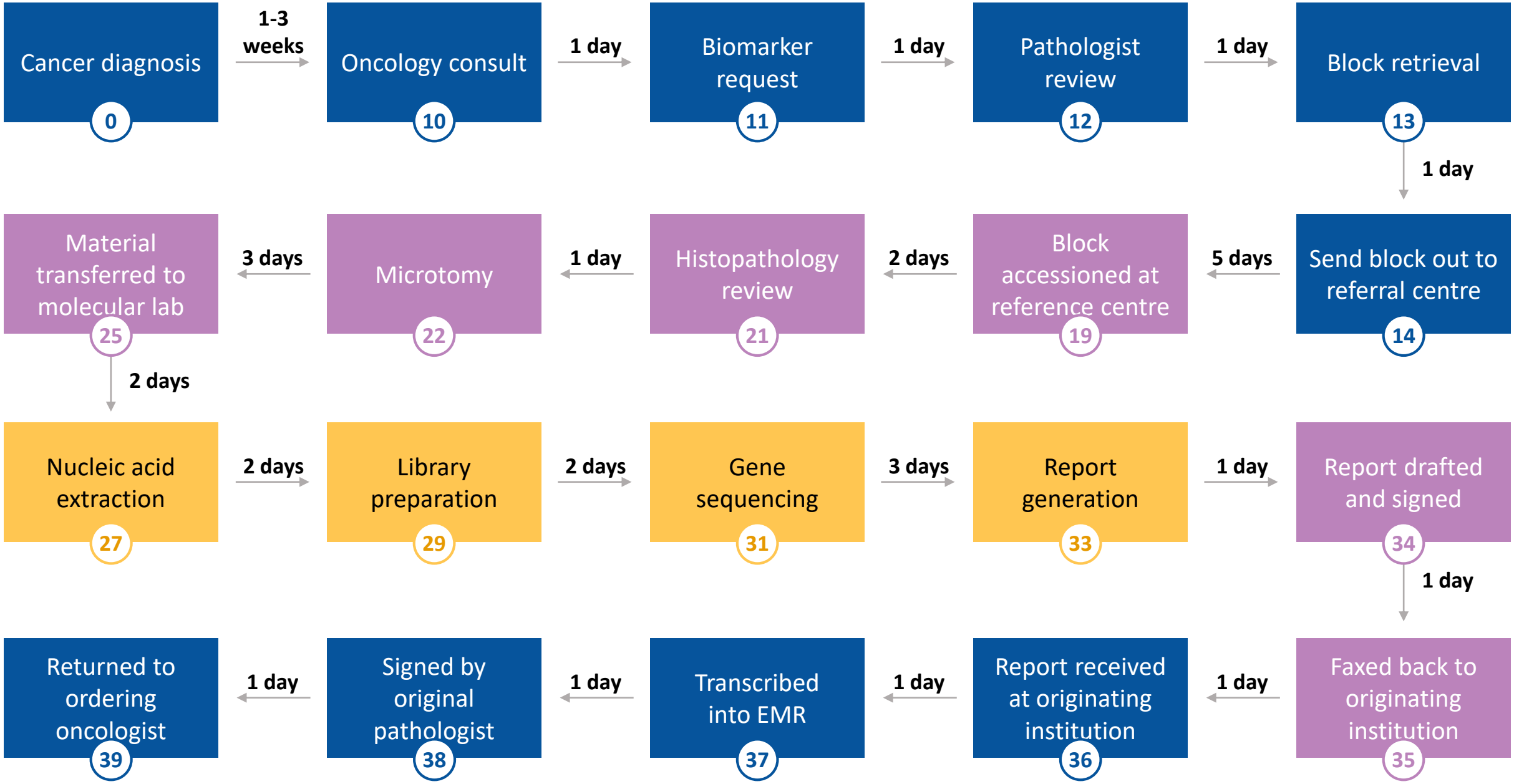
4 days

Biomarkers Available at Oncology Consult:

~~17%~~

94%

Lab Invest. 2020; S1 (100): 1802


Cancer diagnosis
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1-3 weeks
Oncology consult
10
1 day
Biomarker request
11
1 day
Pathologist review
12
1 day
Block retrieval
13
1 day
Send block out to referral centre
14
5 days
Block accessioned at reference centre
19
2 days
Histopathology review
21
1 day
Microtomy
22
3 days
Material transferred to molecular lab
25
2 days
Nucleic acid extraction
27
2 days
Library preparation
29
2 days
Gene sequencing
31
3 days
Report generation
33
1 day
Report drafted and signed
34
1 day
Faxed back to originating institution
35
1 day
Report received at originating institution
36
1 day
Transcribed into EMR
37
1 day
Signed by original pathologist
38
1 day
Returned to ordering oncologist
39

Cancer diagnosis
with biomarkers

Oncology consult

E. LYMPH NODE (STATION 7), BIOPSY:
- POSITIVE FOR METASTATIC NON-SMALL CELL LUNG CARCINOMA.
- Favour adenocarcinoma (TTF1+, p40-).

LUNG BIOMARKERS:

EGFR: POSITIVE (L858R).
- Cellularity: moderate
- Estimated tumor content: 50%

PD-L1: low-level expression (tumor proportion score 1-49%).
- Estimated tumor proportion score: 5%

ALK: negative.
BRAF V600E: negative.
ROS: nevative.

INTERPRETATION: The sample demonstrates an activating mutation in the EGFR gene leading to the p. Leu858Arg protein change. The alteration is amenable to treatment with EGFR tyrosine kinase inhibitor therapy, if clinically indicated.

DIAGNOSIS

A. COLON (RECTOSIGMOID), ANTERIOR RESECTION:
- INVASIVE ADENOCARCINOMA.
1. Moderately differentiated (low-grade).
2. Completely excised.
    - Proximal, distal, and radial margins clear.
    - Please see comment.
3. Carcinoma invades through the muscularis propria, into pericolonic fat.
4. Fifteen lymph nodes are identified.
    - Three tumor deposits are identified.
        - No definite nodal tissue is associated with the deposits.
        - Largest deposit measures 3.5 cm (see comment).
        - pN1c
    - No metastasis is identified within the 15 nodes (0/15).
5. The tumor shows intact (wild-type) expression of MMR proteins.
6. No muation is identified in KRAS, NRAS, or BRAF (see below).

DIAGNOSIS

LYMPH NODE (7), BIOPSY:
- POSITIVE FOR METASTATIC MELANOMA.

COMMENT: The specimen contains malignant epithelioid-appearing cells. Pigment is present, and this is favoured to represent anthracosis. By immunohistochemistry, the lesional cells show strong and diffuse immunoreactivity for SOX10. There is no immunoreactivity identified for TTF1 or p40. The features support a diagnosis of metastatic melanoma. An activating BRAF mutation has been identified (see below).

But what about the rest?

KRAS, MET, ERBB2, RET, NRG1 …

The benefits of NGS in your institution

Comprehensive and actionable results, communicated clearly from one source

Results in one report within days, not weeks

Can be customized to the materials present at your centre: EBUS, surgical, etc.

Cost saving for healthcare system, hospital, and patient

Point of care

Next-generation sequencing (NGS)

Order

Interpret

Report

One facility.
One pathologist.
One report.

Under development

Point of care NGS

Relevant Non-Small Cell Lung Cancer Findings

Gene	Finding	Gene	Finding
ALK	Not detected	NTRK1	Not detected
BRAF	Not detected	NTRK2	Not detected
EGFR	Not detected	NTRK3	Not detected
ERBB2	Not detected	RET	Not detected
KRAS	Not detected	ROS1	Not detected
MET	***MET exon 14 skipping, MET positive***		

Variant Details

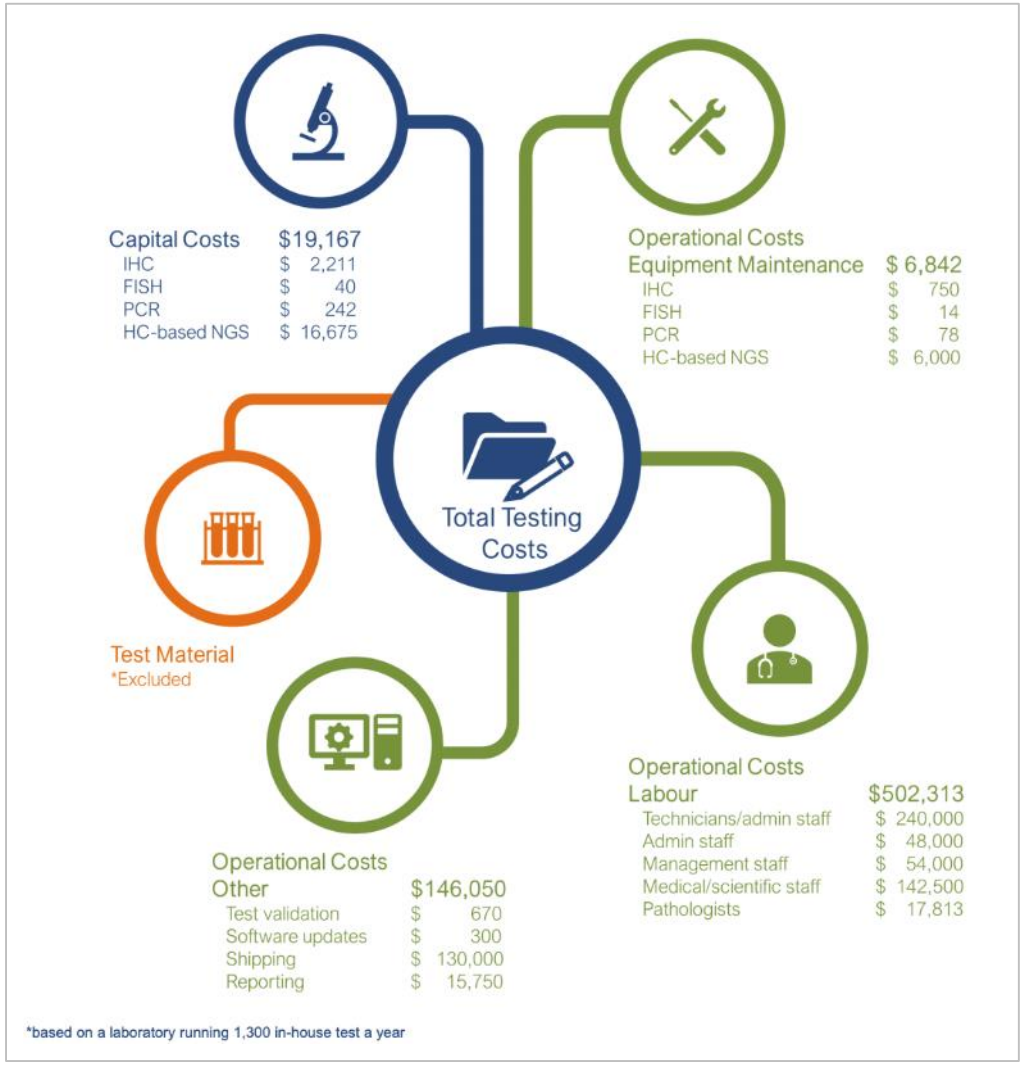
DNA Sequence Variants

Gene	Amino Acid Change	Coding	Variant ID	Locus	Allele Frequency	Transcript	Variant Effect
PIK3CA	p.(E545K)	c.1633G>A	COSM763	chr3:178936091	48.18%	NM_006218.4	missense
MET	p.(?)	c.3082+1G>T	COSM6108462	chr7:116412044	100.00%	NM_001127500.3	unknown
TP53	p.(G245C)	c.733G>T	COSM11081	chr17:7577548	99.65%	NM_000546.5	missense

Gene Fusions (RNA)

Genes	Variant ID	Locus
MET-MET	MET-MET.M13M15.1	chr7:116411708 - chr7:116414935

What's good for patients also saves money



Reduced oncology visits

Reduced number of times a pathologist assesses any given case

Elimination of:

- Extra accessioning
- Additional reporting / transcription
- Shipping

J Med Econ. 2020 Jul 14;1-7.doi: 10.1080/13696998.2020.1789152. Online ahead of print.

Conclusions

1. Anatomic pathologists play a critical role in cancer care – **diagnostics**

2. The role of the pathologist in treatment determination is under appreciated

3. Introducing point of care testing to the pathology lab, including IHC, and NGS can have a deep and meaningful impact on patient care

4. **The role of the pathologist is evolving:**
   The pathologist is more than simply a diagnostician, but a medical expert charged with the task of integrating all available laboratory data to support patients through their journey

Point of Care Molecular Testing
Clinician Perspective

Dr. Parneet K. Cheema, HBSc, MD, MBiotech, FRCPC
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Head of Medical Oncology/Hematology
Head of Cancer research
William Osler Health System

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William Osler Health System

UNIVERSITY OF TORONTO FACULTY OF MEDICINE

Disclosures

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Overview

1

Review the evolving uses of molecular testing in treating patients with cancer, using lung cancer as the example

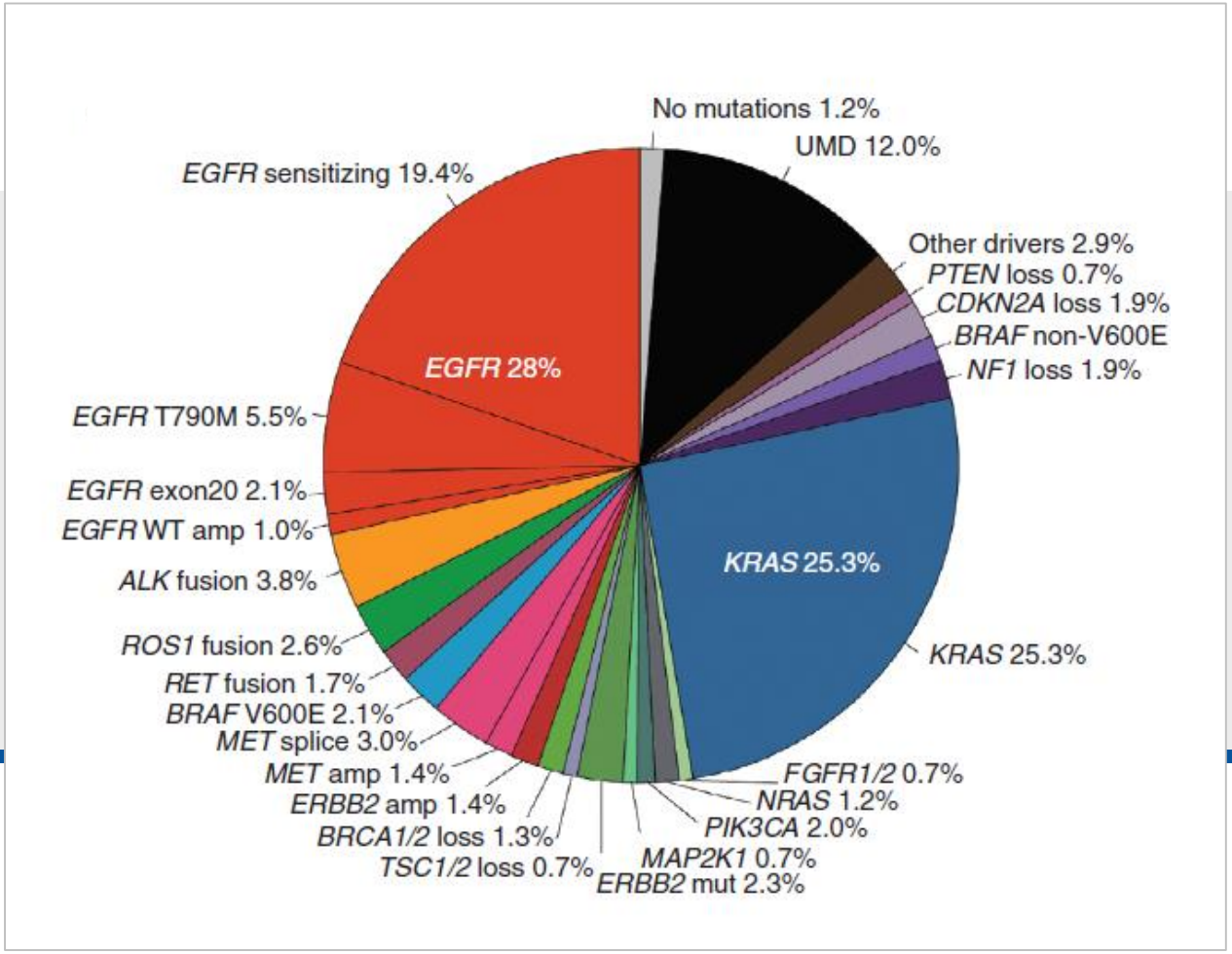
2

Clinical impact of point of care molecular testing

3

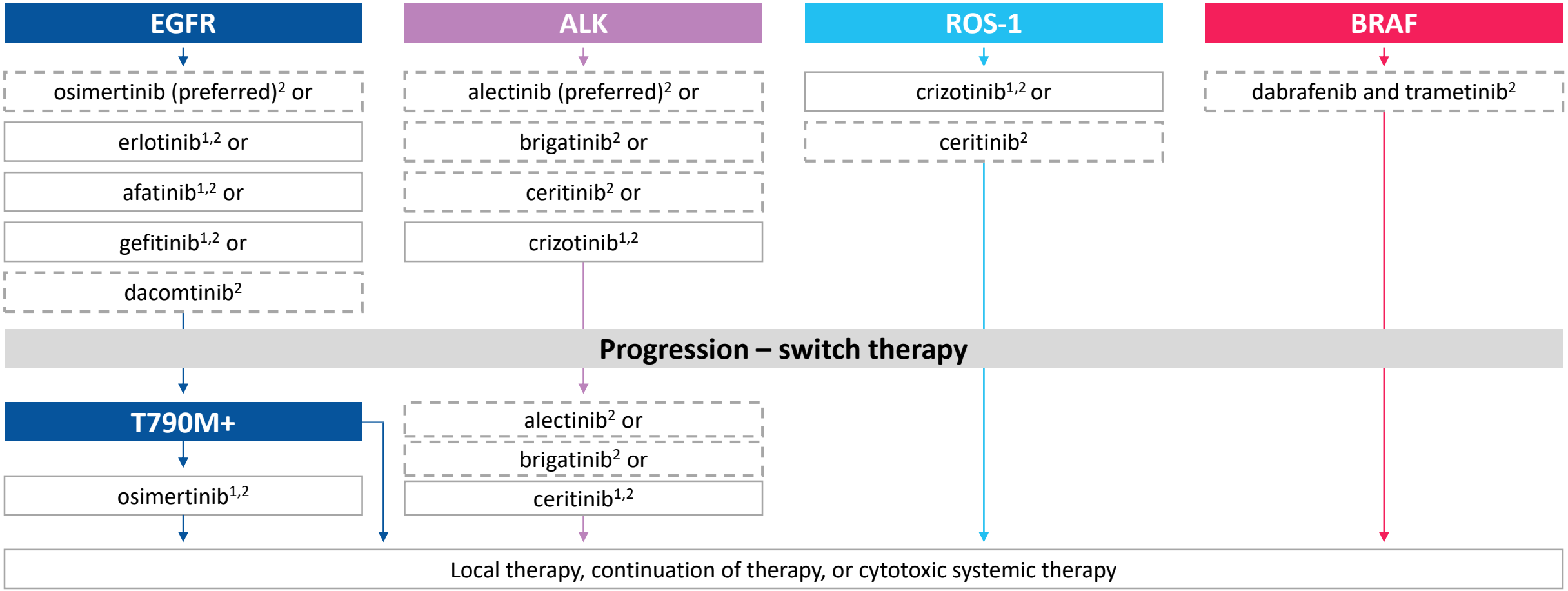
Evolving role of close pathology and molecular oncology collaboration

Molecular profiling is standard of care for patients with advanced NSCLC



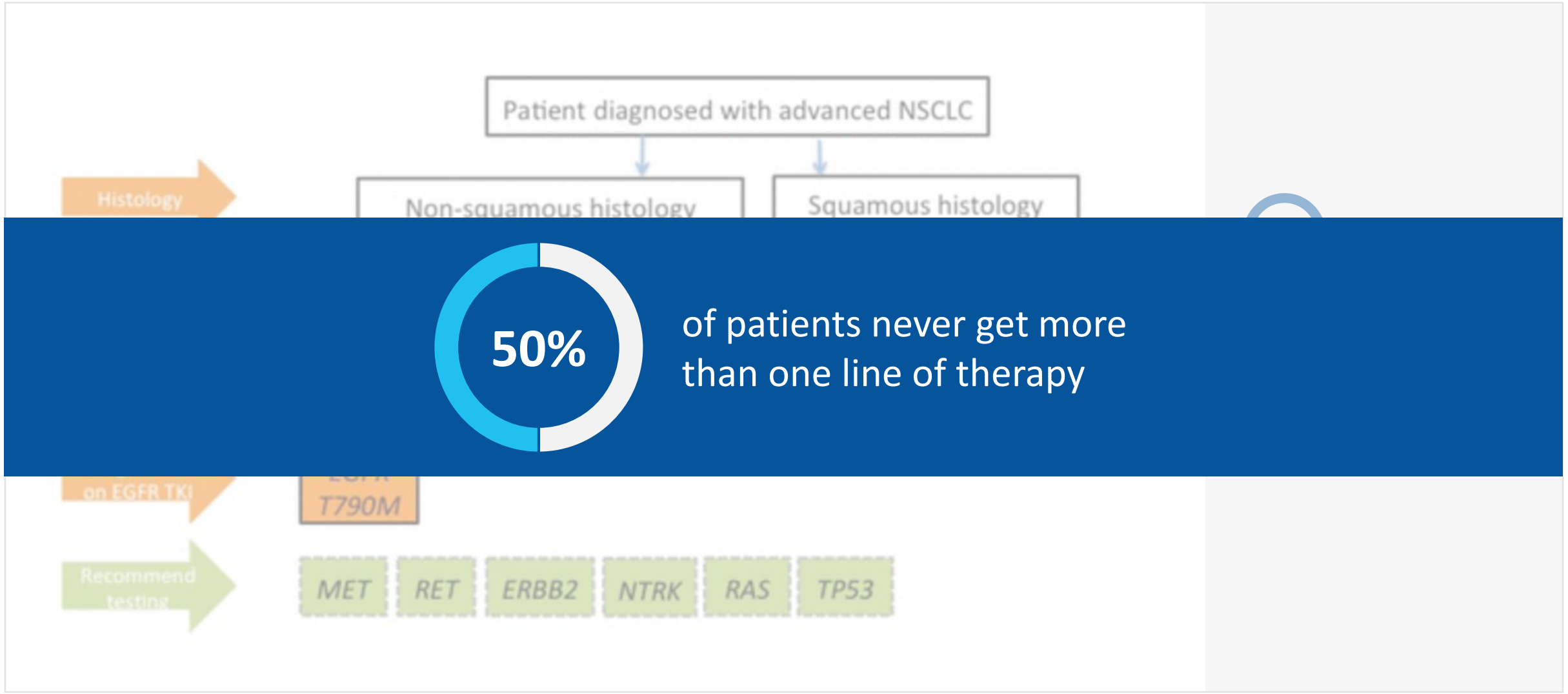
Up to 60% of lung adenocarcinoma have a known oncogenic driver mutation

ASCO & NCCN recommendations for molecular oncogenic driven NSCLC



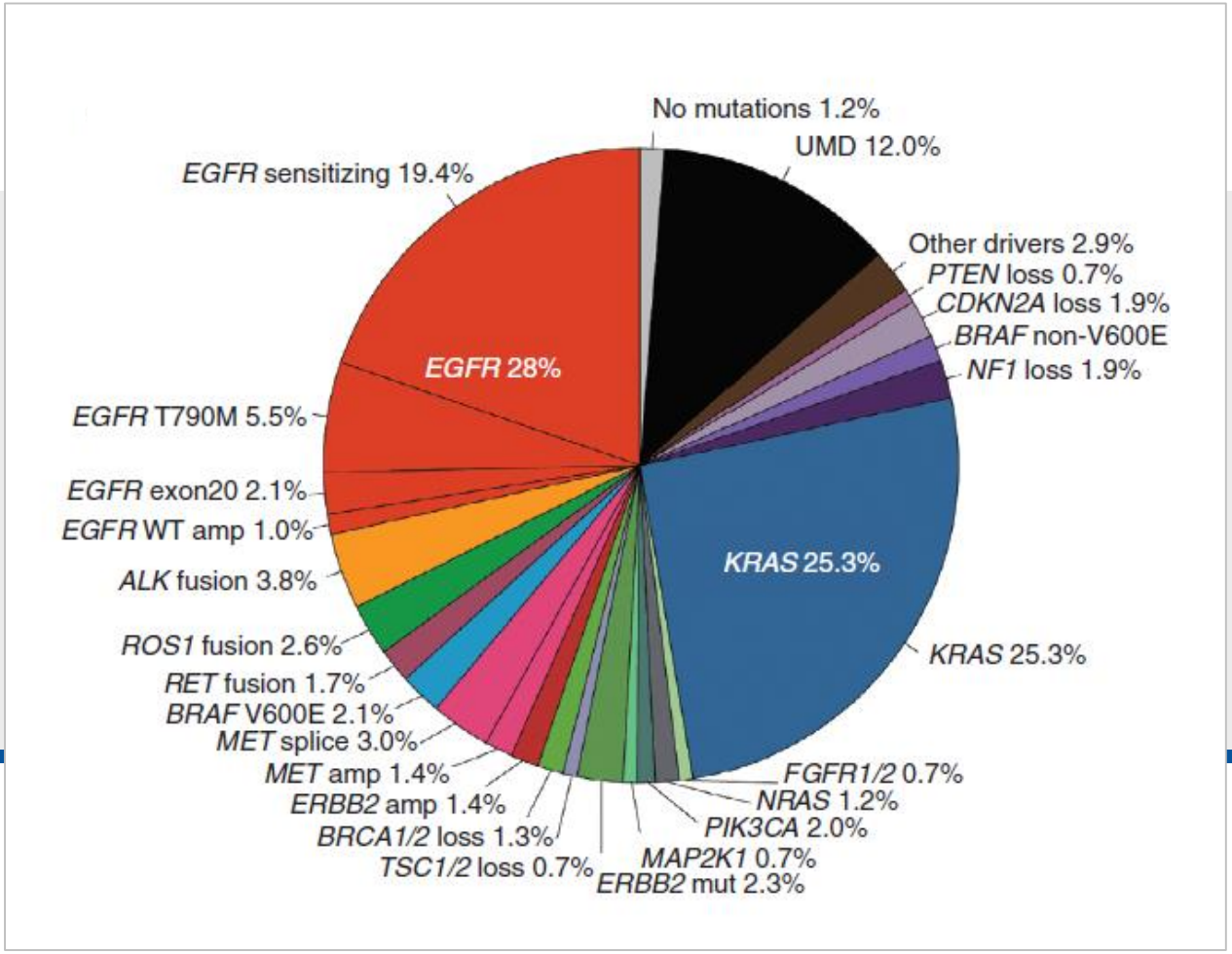
1. Hanna N, Johnson D, Temin S, et al. *J Clin Oncol*. 2017;35(30):3484-3515. (ASCO)
2. Non-small Cell Lung Cancer Version 1.2019. National Comprehensive Cancer Network. https://www.nccn.org/professionals/physician_gls/pdf/nscl.pdf Accessed Nov 15, 2018.

Canadian guidelines on biomarker testing in NSCLC



Melosky B, Blais N, Cheema P, et al. *Curr Oncol.* 2018;25(1):73-82.

Molecular profiling is standard of care for patients with advanced NSCLC



At time of diagnosis

NGS can be more sensitive than other tests

60M, never smoker, adenocarcinoma NSCLC

EGFR negative, ALK negative, PD-L1 1-49%

EGFR Mutational Analysis: No mutation detected, wild-type EGFR allele

High degree of suspicion

NGS can be more sensitive than other tests

RESULTS:

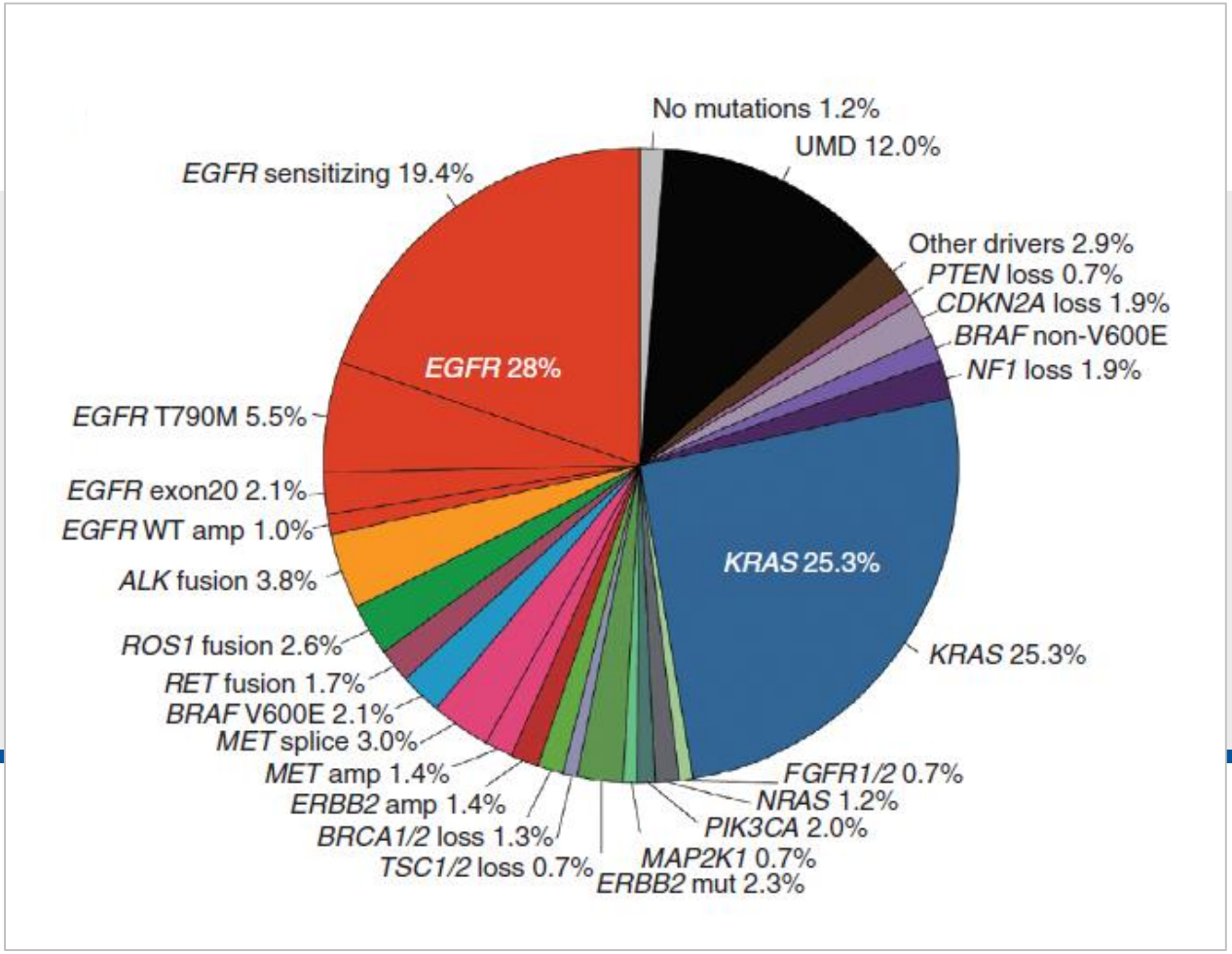
Single nucleotide variants:
EGFR ENSP00000275493.2:p.Gly719Cys (ENST00000275493.2:c.2155G>T)

Insertions/deletions:
No reportable INDELs with known clinical significance were detected.

Copy number variants:
No reportable CNVs with known clinical significance were detected.

INTERPRETATION:
POSITIVE for variant(s) in EGFR.

Molecular profiling in NSCLC is evolving



Reevaluate throughout cancer journey

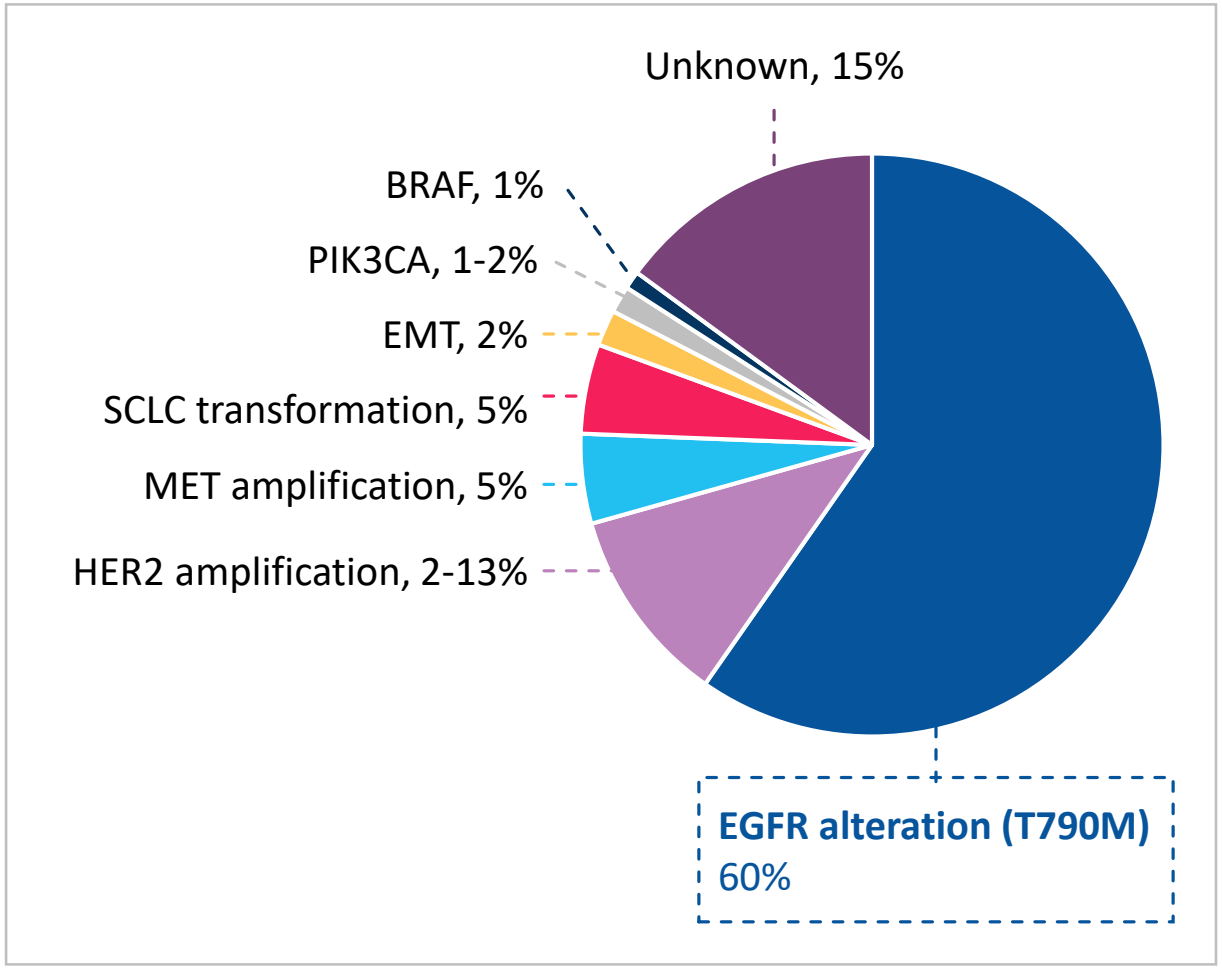
"Resistance mutations"

"Discovery of new mutations"

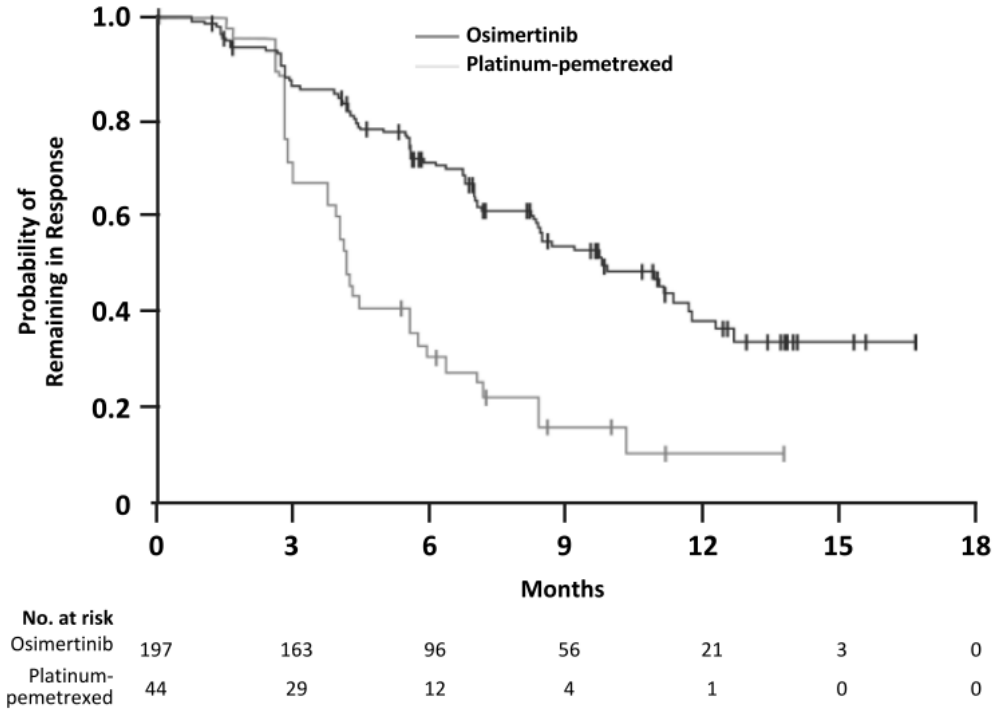
Mechanisms of acquired resistance to 1st/2nd gen EGFR TKIs

The most common acquired resistance mechanisms are[1]:

- Target gene modification (EGFR)
- Alternative pathway activation (HER2, MET, BRAF, PIK3CA)
- Histological or phenotypic transformation (EMT or SCLC)



Targeting T790M resistance mutation with osimertinib in T790M+ NSCLC improved outcomes compared to chemotherapy

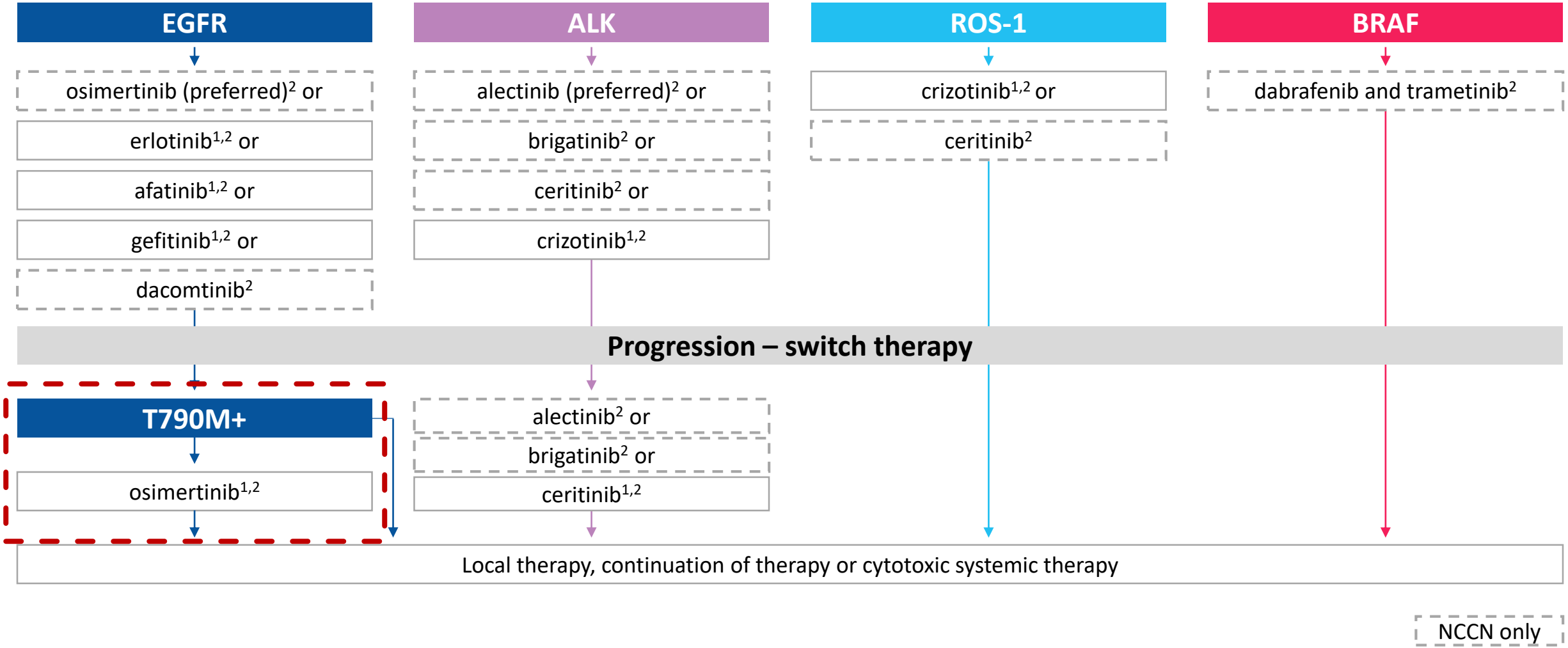


	Progression Free Survival (Months)
Osimertinib	10.1
Platinum-pemetrexed	4.4

Hazard ratio for disease progression or death, 0.30 (95% CI, 0.23–0.41) *P*<0.001

Mok TS et al. N Engl J Med. 2017;376(7):629-640; ESMO ASIA 2019
Population: intent-to-treat
PFS defined as time from randomization until date of objective disease progression or death. Progression included deaths in absence of RECIST progression. Tick Marks indicate censored data;
CI, confidence interval; mPFS, median progression free survival

ASCO and NCCN recommendations for molecular oncogenic-driven NSCLC



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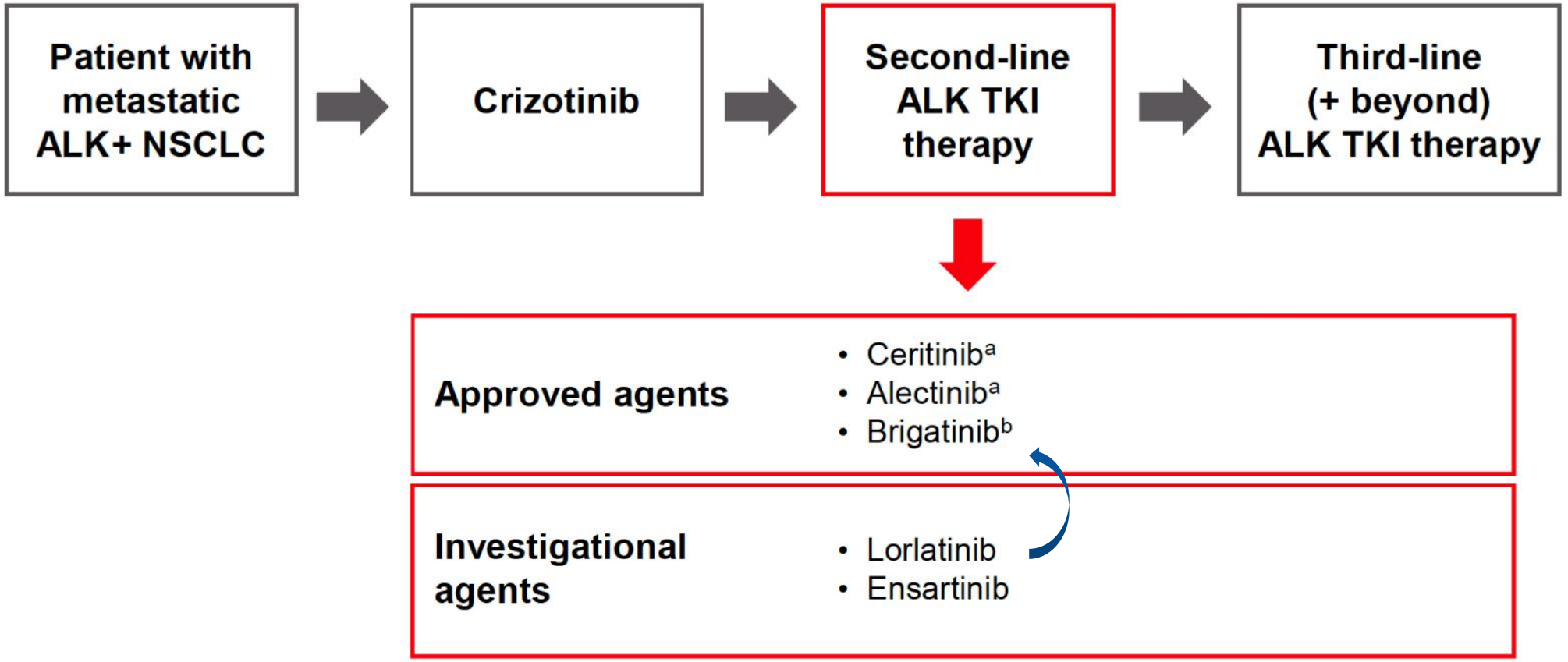
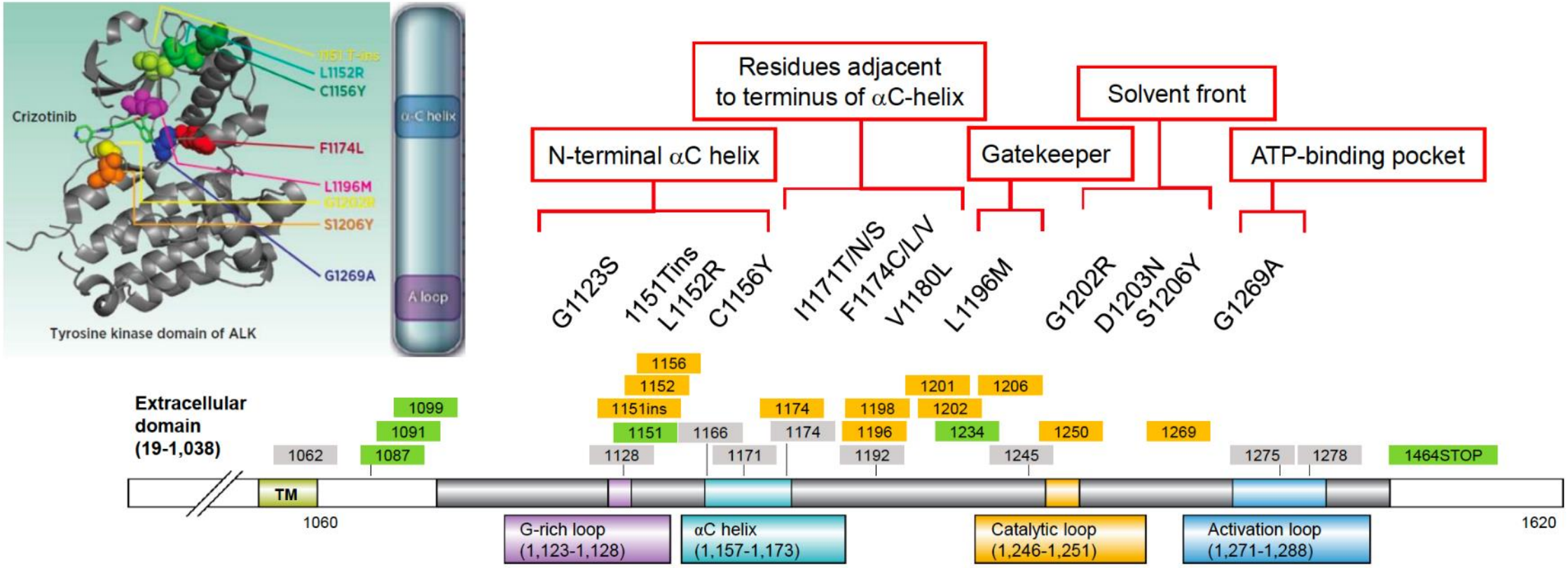
2. Hanna N, Johnson D, Temin S, et al. *J Clin Oncol*. 2017;35(30):3484-3515. (ASCO)

Multiple ALK inhibitors for treatment of ALK+ NSCLC How do you select the right drug for the patient?



[a]Approved in Canada, the European Union, and the United States; [b]Approved in Canada and the United States.
ALK, anaplastic lymphoma kinase; NSCLC, non–small cell lung cancer; TKI, tyrosine kinase inhibitor.

Secondary mutations can arise in the ALK tyrosine kinase domain



ALK, anaplastic lymphoma kinase; ATP, adenosine triphosphate.
Hallberg B, et al. *Nat Rev Cancer*. 2013;13:685-700. Katayama R, et al. *Clin Cancer Res*. 2015;21:2227-2235.

Variations in sensitivities to ALK-resistance mutations

EML4-ALK mutation	Crizotinib	Ceritinib	Alectinib	Brigatinib	Lorlatinib
V1	S	S	S	S	S
C1156Y	I	S	S	S	S
I1171N	I	S	R	S	S
I1171S	I	S	I	S	S
I1171T	I	S	S	S	S
F1174C	I	S	S	S	S
L1196M	R	S	I	S	S
L1198F	S	I	S	S	S
G1202R	R	I	R	I	S
G1202del	I	I	I	I	S
D1203N	I	S	S	S	S
E1210K	S	S	S	S	S
G1269A	I	S	S	No data	S
D1203N + F1174C	R	R	I	I	I
D1203N + E1210K	I	I	I	I	S

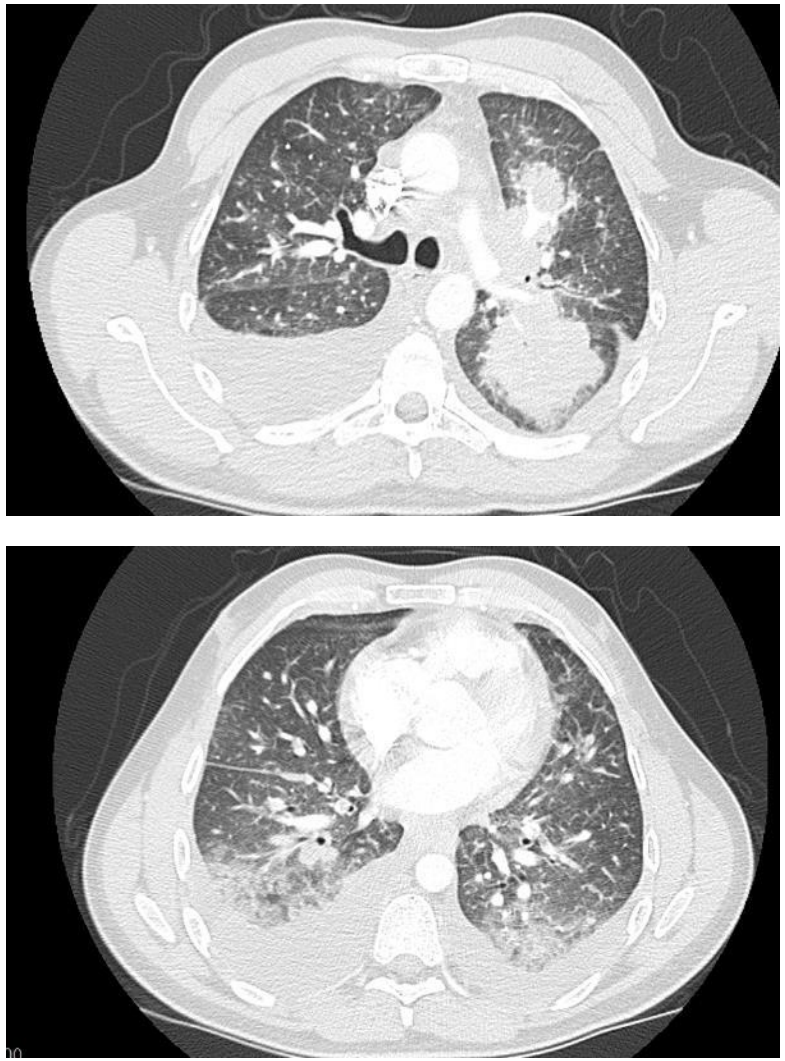
L1198F/C1156Y is lorlatinib resistant but crizotinib sensitive ALK mutation

I, intermediate (IC_{50} > 50 < 200 nmol/L); R, resistant (IC_{50} ≥ 200 nmol/L); S, sensitive (IC_{50} ≤ 50 nmol/L)

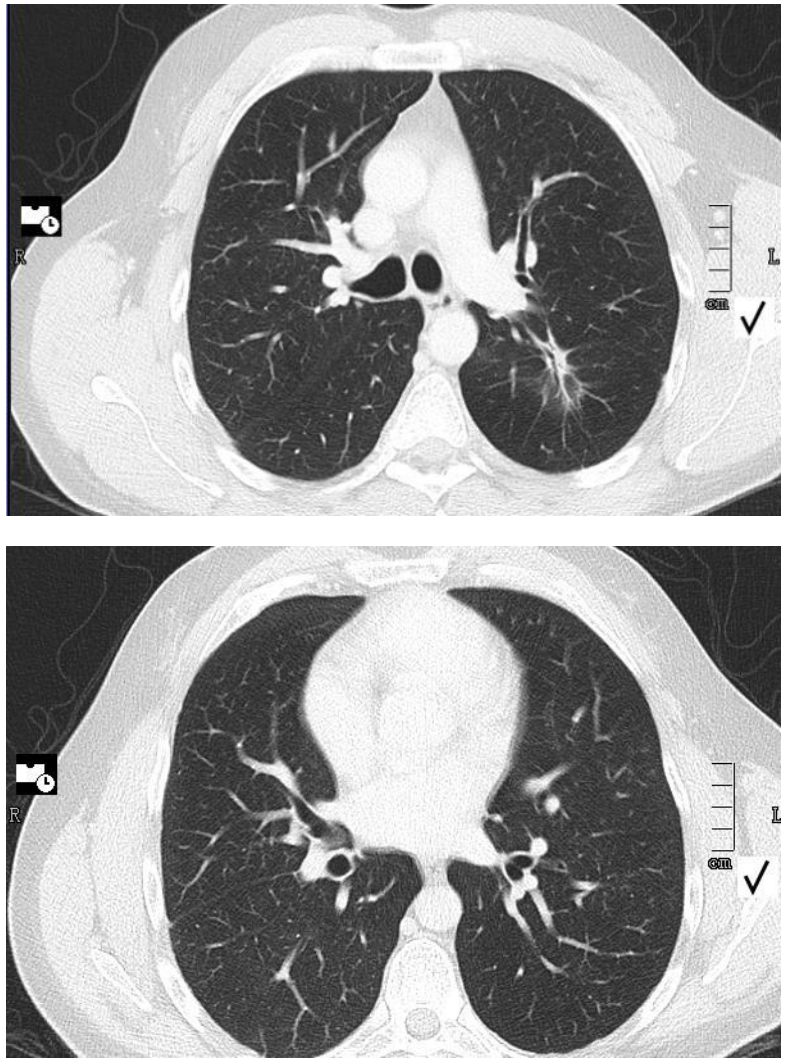
Should we be rebiopsing patients for resistance mutations??

Bui KT, Cooper WA, Kao S, Boyer M.Targeted Molecular Treatments in Non-Small Cell Lung Cancer: A Clinical Guide for Oncologists. J Clin Med. 2018 Jul 31;7(8). Gainor JF, Dardaei L, Yoda S, et al. Molecular Mechanisms of Resistance to First- and Second-Generation ALK Inhibitors in ALK-Rearranged Lung Cancer. Cancer Discov. 2016 Oct;6(10):1118-1133.

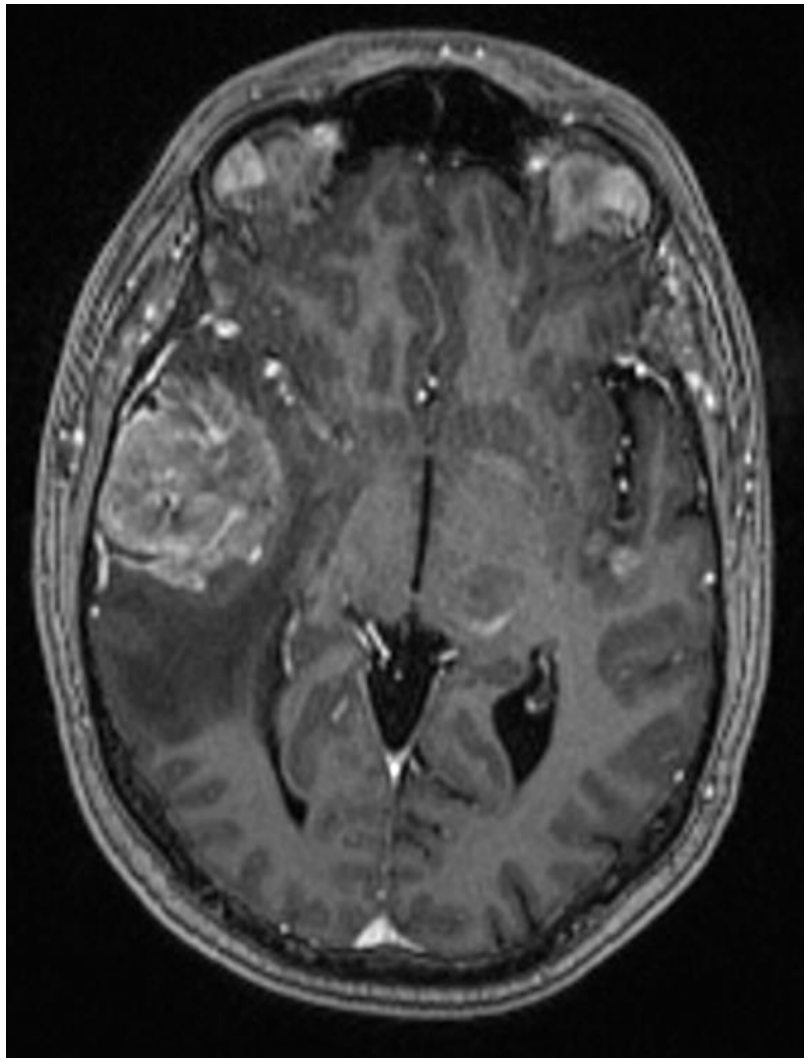
35M with ROS1+ NSCLC on crizotinib



March 2016

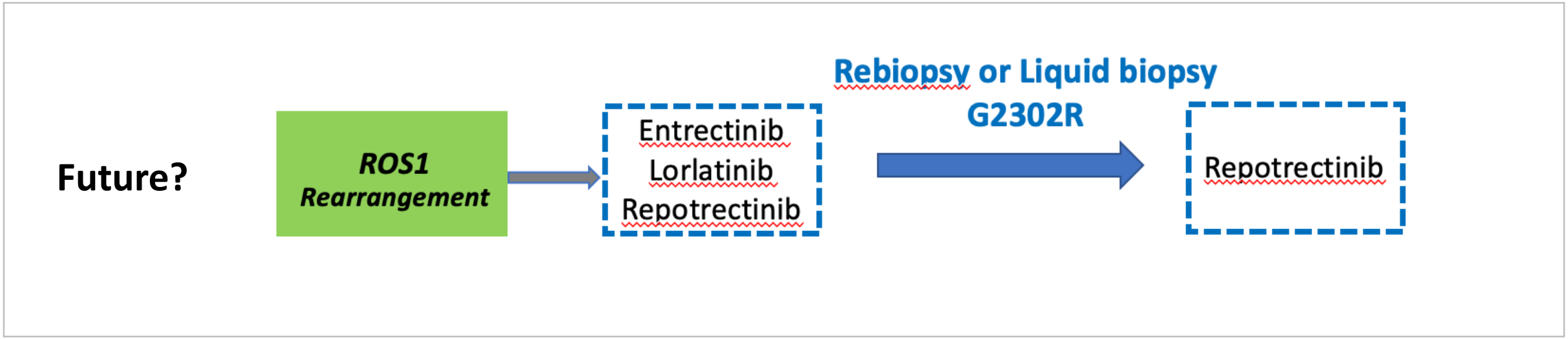


October 2017



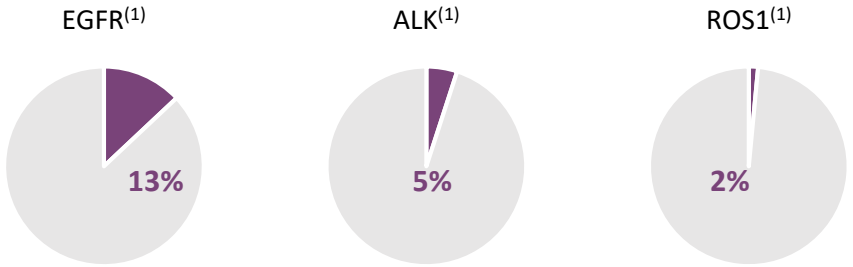
October 2017

ROS1, NSCLC, and evolving role of NGS?

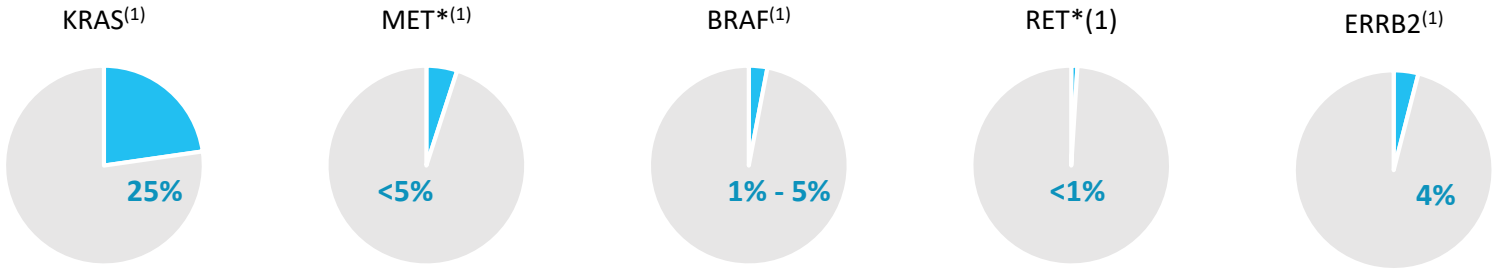


Balancing limited tissue with the growing number of mutations to be tested

Recommended for routine assessment[2]



Recommended for further characterization as all have corresponding drugs in development[2]



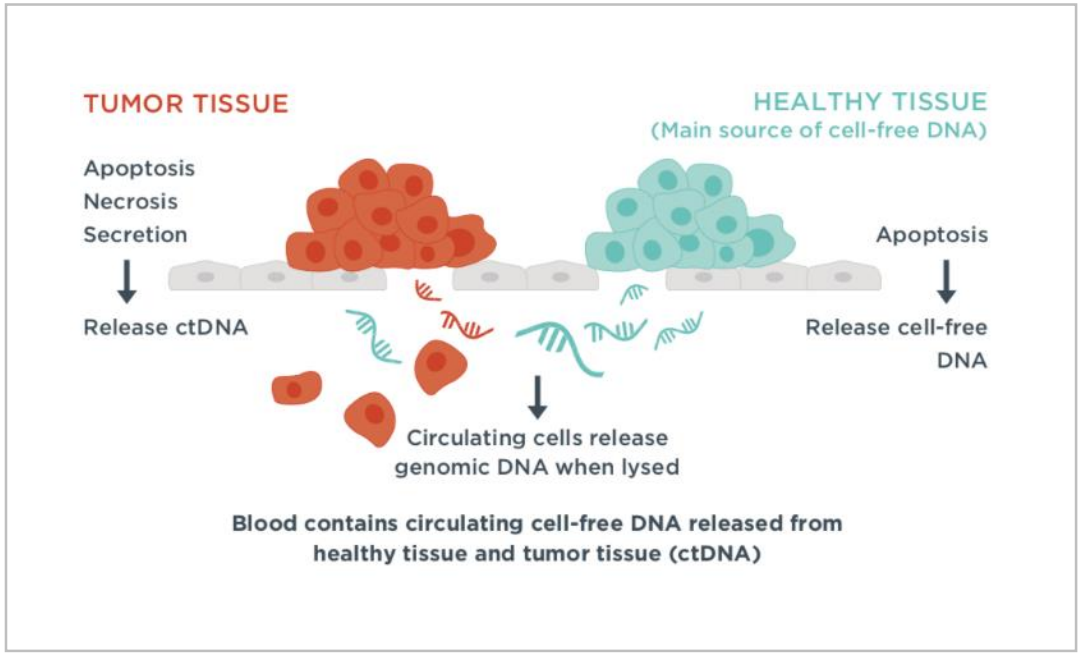
Reported positivity rates in NSCLC

"A new responsibility for pathologists … is to manage small specimens strategically so there is sufficient tissue preserved for molecular studies."[3]

*Next generation sequencing preferred for detection, according to CAP/IASLC/AMP[5]

1. Salgia R. *Future Oncol* 2015; 11(3):489-500. 2. Daoud A, Chu QS. *Front. Oncol.* 2017; 7:222. 3. Travis WD, Brambilla E, Nogushi M, et al. *Arch Pathol Lab Med* 2013; 137:668–684. 4. Lindeman NI, Cagle PT, Beasley MB, et al. *J Thorac Oncol.* 2013;8(7):823–859. 5. Lindeman NI, Cagle PT, Aisner DL, et al. *J Mol Diagn*. 2018; 20(2):129-159. (CAP/IASLC/AMP)

Role of plasma based NGS advancing access to broad molecular testing



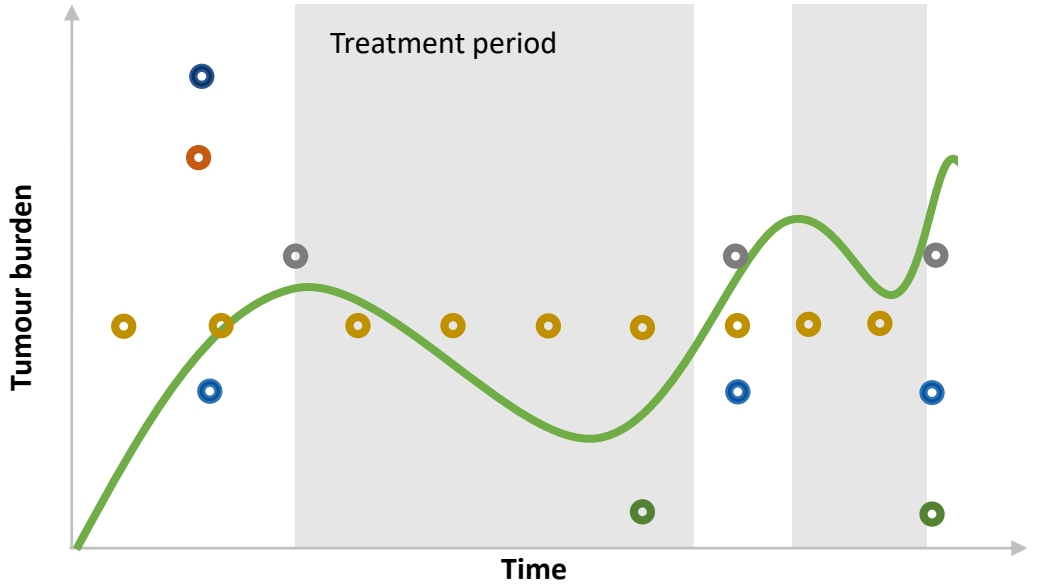
- **30%** of patients have **inadequate tumour** tissue for molecular analysis at diagnosis
- **Repeat biopsies** are **not feasible ~20%** of patients with advanced NSCLC
- **~25% repeat biopsies fail** to yield sufficient material for genomic analysis

Blood-based NGS has the potential to overcome some of the limitations associated with tissue collection and testing, which may enable clinicians to offer more effective personalised therapies

Potential clinical applications of liquid biopsy and circulating DNA

Liquid biopsy is a **non-invasive**, easily repeatable sampling approach that collects peripheral blood containing cfDNA for analysis.[1]

ctDNA is an established surrogate marker for monitoring disease burden and anticancer therapy response and has many other **possible clinical applications**.[2,3]



Assessment of molecular heterogeneity

Diagnosis

Prognosis

Monitoring

Identification of genetic determinants for targeted therapy

Resistance characterization

cfDNA, cell free DNA; ctDNA, circulating tumour DNA; DNA, deoxyribonucleic acid

1. Malapelle U, et al. *Transl Lung Cancer Res* 2016;5(5):505-10.
2. Heitzer E, et al. *Clin Chem* 2015;61(1):112-23.
3. Busser B, et al. *Biomed Res Int* 2017;5986129:1-8.
4. Lim C, Sekhon HS, Cutz JC, et al. *Curr Oncol.* 2017; 24(2):103-110

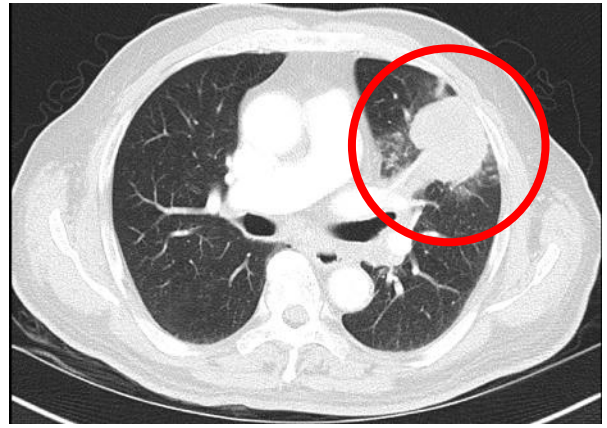
Optimal state – point of care molecular testing

Cancer diagnosis
with biomarkers

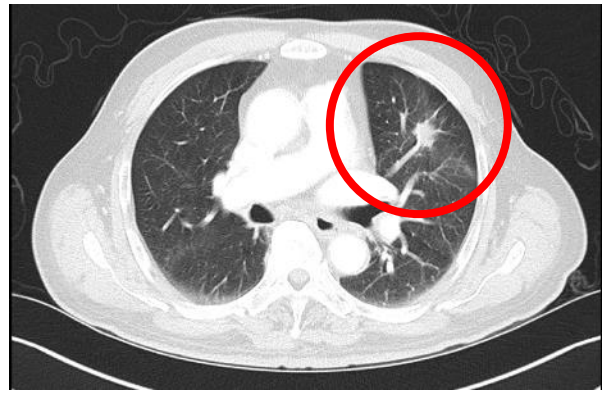
Oncology consult

In-house biomarker testing prevented missed opportunity for treatment

- Diagnosed w/ squamous cell NSCLC but was a non-smoker
- EGFR testing <24 hours of seeing Oncologist
- **EGFR L858R + mutation found**
- In 3 business days from seeing oncologist, patient was on targeted treatment

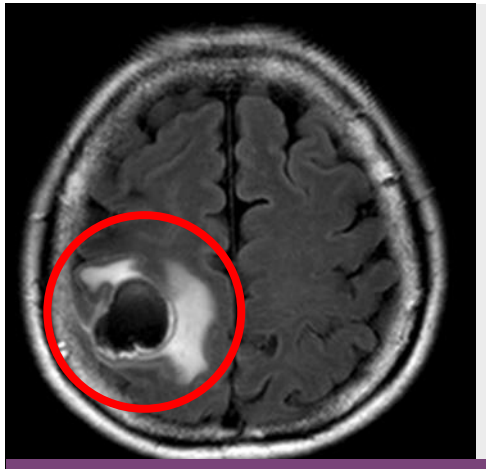


10 days later…



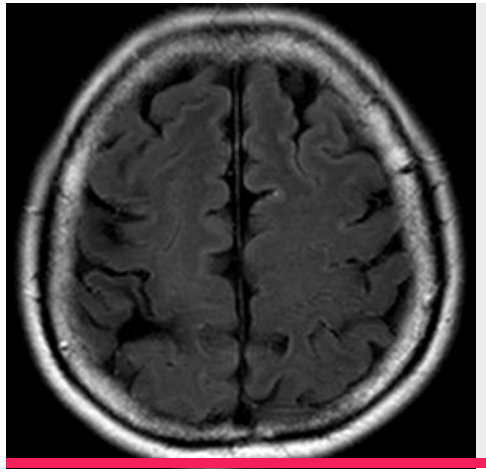
At this timepoint, with sending testing out, patient would have still been waiting for biomarker results

Timely biomarker results allows for appropriate treatment



55F with ALK + NSCLC

Started on targeted therapy instead of radiation to the whole brain +/- surgery



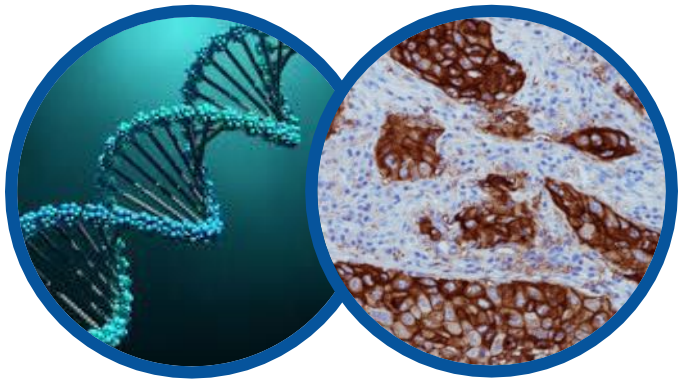
17 months after starting targeted therapy, complete response to brain lesion

No radiation or surgery was done

Point of care molecular testing

Order

Interpret



Report

One facility.
One pathologist.
One report.

One report for diagnostic and molecular results optimizes treatment selection

1. 72F Asian, life-time non smoker

2. Malignant pleural effusion, pulmonary metastases

3. **Adenocarcinoma:**
   - Driver mutations: EGFR/ALK/ROS1 negative
   - Biomarkers: PDL1 >50%

Genomic Alterations Identified

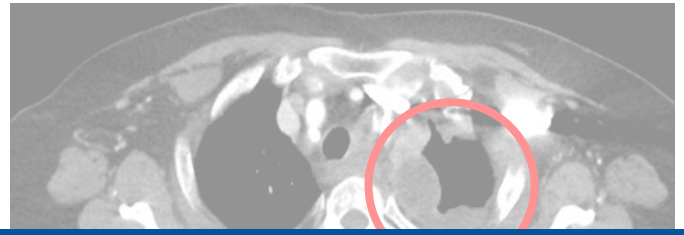
Gene	Alteration
TP53	E17fs*23
TP53	R273H
MET	splice site 3022_3028+14del21

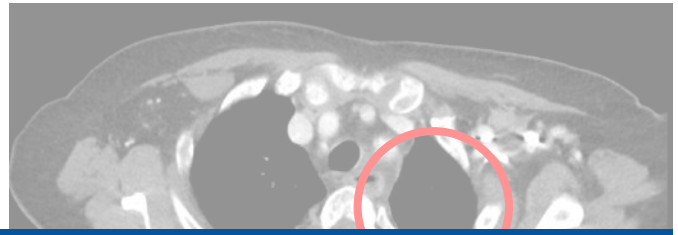
Variants of Unknown Significance Identified

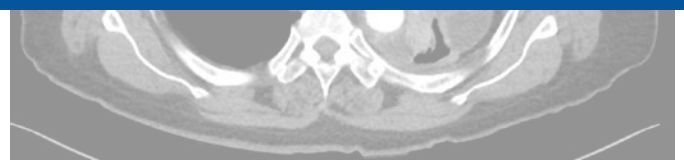
Gene	Alteration
ERBB2	I740S
CDK4	K22Q

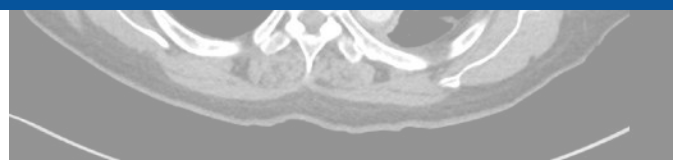
Patient would get immunotherapy based on this information

Point of care NGS is needed to offer most effective therapy for patients









of patients do NOT get to a subsequent therapy

Baseline

3 months later

Case – impact of piecemeal broad molecular testing results

1. 46F, life-time nonsmoker history presents with persistent cough -> hemoptysis

2. Imaging shows large lung mass, mediastinal lymphadenopathy, bone metastases, and 1.1 cm brain metastasis; non squamous NSCLC

3. EGFR-/ALK-/PD-L1 > 50%

Treatment:

- Platinum doublet x 2 cycles
- Switched to pembrolizumab x 3 months, progression with new malignant pericardial effusion, new bone lesions, and increasing mediastinal adenopathy.
- Referred to Osler for clinical trials
- On presentation: in wheelchair, ECOG 2, on oxygen
- Plan: liquid NGS biopsy, repeat EBUS bx for inclusion into clinical trial

Molecular report

TUMOR TYPE: LUNG NON-SMALL CELL LUNG CARCINOMA (NOS)

Genomic Alterations Identified[†]

RET KIF5B-RET fusion

CDK4 amplification – equivocal[*]

TP53 E285K

On selpercatinib

One report of diagnostics and biomarkers

Move away from addendums

DIAGNOSIS

A. LIVER, EUS BIOPSY:
- **POSITIVE FOR METASTATIC NON-SMALL CELL CARCINOMA.**

B. LYMPH NODE (7), EUS BIOPSY:
- **POSITIVE FOR METASTATIC NON-SMALL CELL CARCINOMA.**
- **Favour pulmonary adenocarcinoma.**

LUNG BIOMARKERS:

EGFR:	POSITIVE (exon 20 insertion) - Cellularity: low - Estimated tumor content: 10% - Please see comment.
PD-L1:	low-level expression (tumor proportion score 1-49%). - Estimated tumor proportion score: Please see comment.
ALK:	negative.
BRAF V600E:	negative.
ROS:	negative.

COMMENT: The tumor shows an activating EGFR exon 20 insertion. This type of activating mutation may show an attenuated response to EGFR inhibitors compared to more classical activating mutations.

Interpretation by oncologists needs to be considered

Genomic Alterations Identified

Gene	Alteration
TP53	E17fs*23
TP53	R273H
MET	splice site 3022_3028+14del21

Variants of Unknown Significance Identified

Gene	Alteration
ERBB2	I740S
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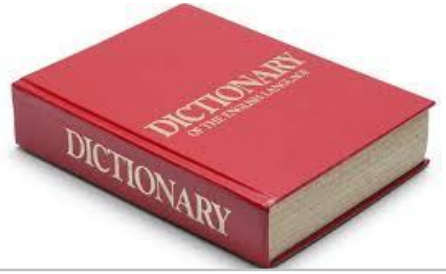

Communication of medical oncology and the lab

Relevant Non-Small Cell Lung Cancer Findings

Gene	Finding	Gene	Finding
ALK	Not detected	NTRK1	Not detected
BRAF	Not detected	NTRK2	Not detected
EGFR	Not detected	NTRK3	Not detected
ERBB2	Not detected	RET	Not detected
KRAS	Not detected	ROS1	Not detected
MET	***MET exon 14 skipping, MET positive***		

How do you treat this EGFR mutation?

1

***EGFR* c.2369C>T**

2

***EGFR* g. 7:55249071C>T**

3

EGFR T790M mutation

Compatible with language of clinical trials for targeted therapies

Driver mutations/alterations and evolving targets with multiple promising agents

EGFR	EGFR T790 M	ALK	ROS1
Osimertinib/ Afatinib/Gefitinib	Osimertinib	Alectinib, Lorlatinib, Certinib, Brigatinib, Ensartinib, Crizotinib	Criztotinib, Lorlatinib, Repotrectinib, Entrectinib

BRAF V600E	NTRK	RET	MET exon 14 skipping
Dabrafenib/Trametinib	Larotrectinib, Entrectinib	Selpercatinib, Pralsetinib	Capmatinib, Tepotinib, Crizotinib

Up and coming targeted therapies for the following drivers

- KRAS G12C
- Exon 20 insertion
- HER2 mutations/amplifications
- NRG1

Summary

1. **Timely molecular testing in oncology is critical for treatment decisions**
   Providing the diagnosis without complete molecular information can lead to delays in treatment or patients receiving suboptimal treatment or no treatment at all.

2. In house testing is an option to improves turn around time for cancer programs.

3. Introducing **point of care** testing to the pathology lab, including IHC, and NGS can have a deep and meaningful impact on patient care.

4. The relationship of the medical oncologist and pathologist is evolving, and increased collaboration is required to optimize outcomes of patients.

5. The collaboration starts in the lab!

Thank you

Please visit our exhibit for more information or to speak with a representative