Interpace Diagnostics

PRESENTATION, DECEMBER 2017

INTERPACE™ DIAGNOSTICS
GUIDING TREATMENT. DELIVERING INNOVATION.

NASDAQ
IDXG

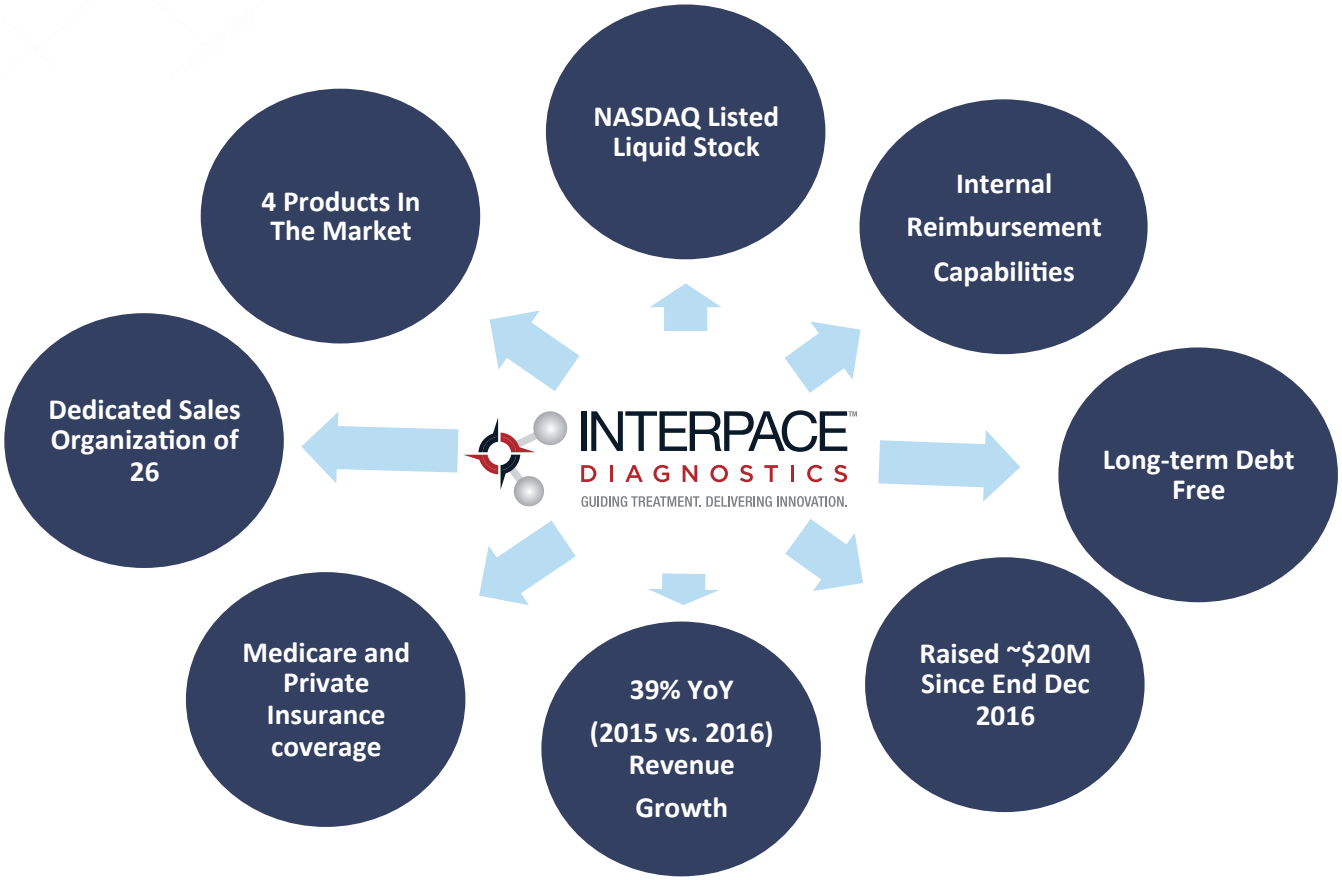
Forward Looking Statements

This Investor Presentation contains forward-looking statements within the meaning of Section 27A of the Securities Act of 1933, Section 21E of the Securities Exchange Act of 1934 and the Private Securities Litigation Reform Act of 1995, relating to Interpace Diagnostic Group Inc.'s ("the Company") future financial and operating performance. The Company has attempted to identify forward looking statements by terminology including "believes," "estimates," "anticipates," "expects," "plans," "projects," "intends," "potential," "may," "could," "might," "will," "should," "approximately" or other words that convey uncertainty of future events or outcomes to identify these forward-looking statements. These statements are based on current expectations, assumptions and uncertainties involving judgments about, among other things, future economic, competitive and market conditions and future business decisions, all of which are difficult or impossible to predict accurately and many of which are beyond the Company's control. These statements also involve known and unknown risks, uncertainties and other factors that may cause the Company's actual results to be materially different from those expressed or implied by any forward-looking statement. Known and unknown risks, uncertainties and other factors include, but are not limited to, the Company's ability to adequately finance the business, its ability to restructure its liabilities and other obligations, the market's acceptance of its molecular diagnostic tests, its ability to retain or secure reimbursement, its ability to secure additional business and generate higher profit margins through sales of its molecular diagnostic tests, in-licensing or other means, projections of future revenues, growth, gross profit and anticipated internal rate of return on investments and our ability to maintain our NASDAQ listing. Additionally, all forward-looking statements are subject to the risk factors detailed from time to time in the Company's filings with the Securities and Exchange Commission (SEC), including without limitation, the Annual Report on Form 10-K filed with the SEC on March 31, 2017 and the amendment on Form 10-K/A filed on April 28, 2017, the Company's Quarterly Reports on Form 10-Q filed with the SEC on May 12, 2017, August 10, 2017, and November 13, 2017, and the Company's Registration Statement on Form S-1 (333-218140, the "registration statement"). Because of these and other risks, uncertainties and assumptions, undue reliance should not be placed on these forward-looking statements. In addition, these statements speak only as of the date of hereof and, except as may be required by law, the Company undertakes no obligation to revise or update publicly any forward-looking statements for any reason. The Company has filed the Registration Statement (including a preliminary prospectus) with the Securities and Exchange Commission (SEC) for the offering for which this Investor Presentation relates. Before you invest, you should read the preliminary prospectus contained in the Company's Registration Statement and other documents the Company has filed with the SEC for more complete information about the Company and this offering. The preliminary prospectus and the Registration Statement may be accessed through the SEC's website at www.sec.gov. Alternatively, the Company, any underwriter or any dealer participating in the offering will arrange to send you the preliminary prospectus if you request it through Maxim Group LLC, 405 Lexington Ave, New York, NY 10174, Attn: Prospectus Department or by Tel: (800) 724-0761. This Investor Presentation contains statistics and other data that has been obtained from or compiled from information made available by third parties service providers. The Company has not independently verified such statistics or data.

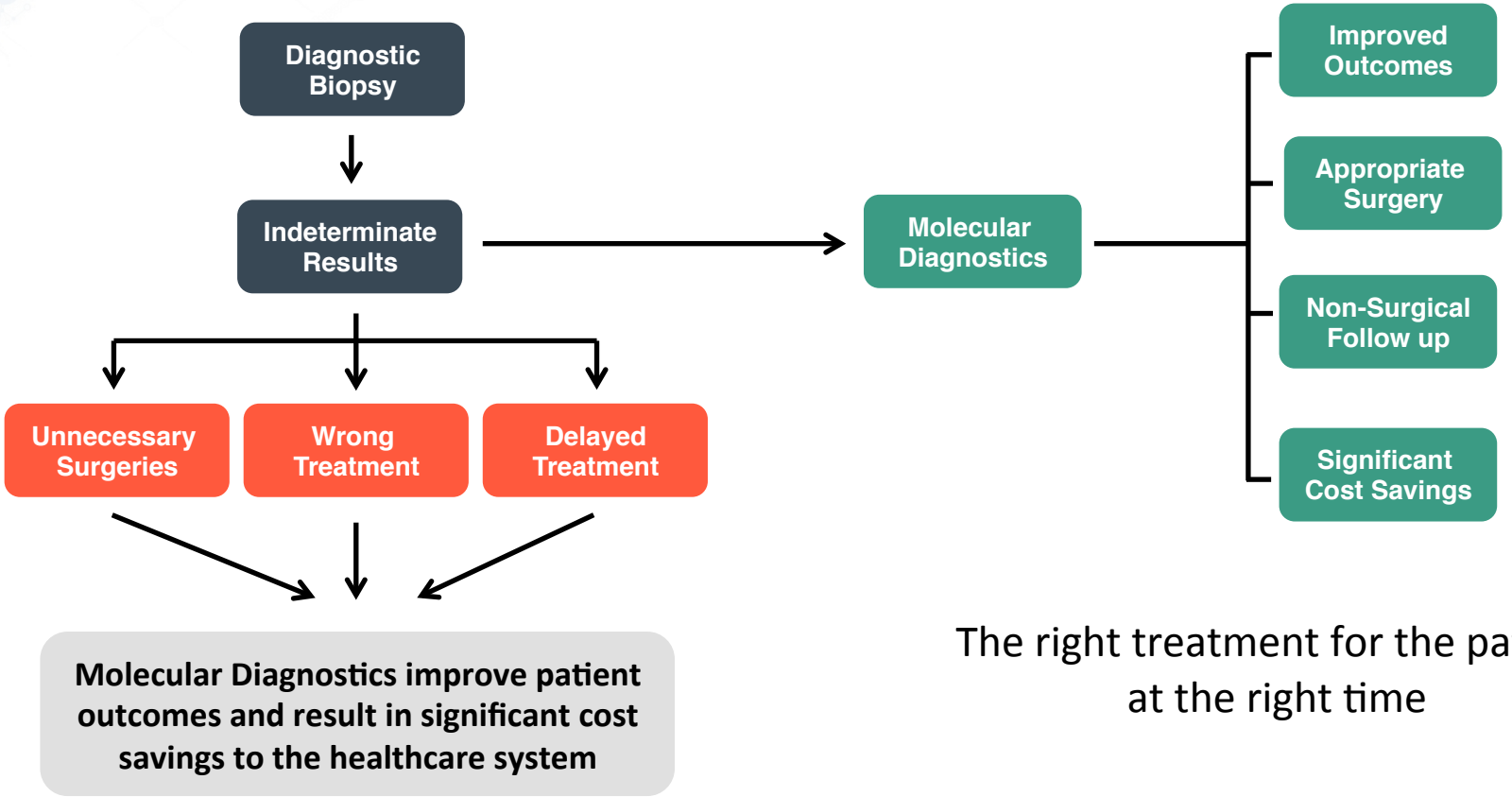
Our Mission

We are a fully integrated “commercial” company that provides molecular and diagnostic tests and pathology services to evaluate the risk of cancer by leveraging the latest technology in personalized medicine for better informed clinical decisions and improved patient management.

Investment Highlights



Inconclusive Biopsy: Uncertainty Drives Need for Molecular Testing



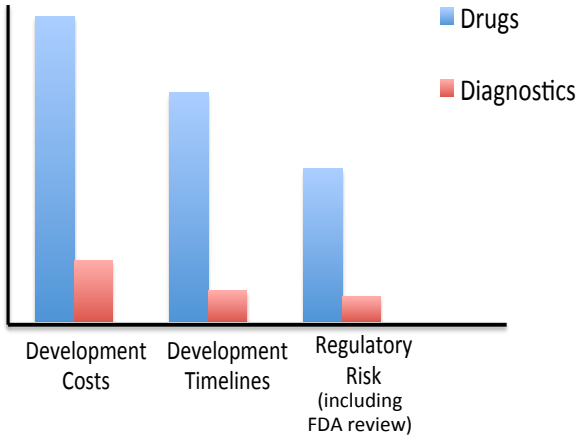
The right treatment for the patient at the right time

Attractive Molecular Business Model

De-Risked Business Model

Versus Drugs, Diagnostics have:

- Lower Developmental Costs
- Lower Developmental Risk
- Faster time to market
- Lower regulatory hurdles



Strong Guideline Support

2014 American Thyroid Association Revised Guidelines
Molecular Diagnostics tests should be considered for suspicion of malignancy or indeterminate.

2013 NCCN Guidelines
Molecular Diagnostics recommended testing on some indeterminate cytologies to minimize unnecessary surgeries

2016 ASGE Guidelines
Recommendation no. 4 suggests use of PancraGen should be considered when cysts are indeterminate based on cytology

Current Pancreatic Cysts Guidelines
Sendai guidelines 2012 and ACG guidelines 2007 strongly favor surgical resection because of the inability of first-line tests to predict biological behavior and aggressiveness.

Interpace Diagnostics' Product Portfolio

PancraGEN™
PANCREATIC CANCER RISK CLASSIFIER

- Risk-Stratifies Pancreatic Cysts
- Over 71M Lives Covered
- Integrated Molecular Pathology test

$300M-$370M*
Pancreatic Cysts

BarreGEN™
ESOPHAGEAL CANCER RISK CLASSIFIER

- Risk-Stratifies Barrett's Esophagus
- Soft Launch Began In 2017
- Integrated Molecular Pathology test

$1.5B-$2B*
Barrett's Esophagus

ThyGenX®
THYROID ONCOGENE PANEL

- NGS panel for Thyroid Cancer
- Over 250M Lives Covered
- Rules In Thyroid Cancer

$350M*
Thyroid Nodules

ThyraMIR™
THYROID miRNA CLASSIFIER

- Micro-RNA Classifier
- Over 250M Lives Covered
- Rules Out Thyroid Cancer

$350M*
Thyroid Nodules

MVPdx

- Lung Cancer: Metastatic vs. Primary
- Medicare Coverage in place
- Integrated Molecular Pathology test

Up to $90M*
MVP Lung

* Total Market opportunity, Company Estimates, www.endocrineweb.com, C. Hur *et al. Gastroenterology*, (May 21, 2012).

Pricing & Reimbursement

PANCRAGEN
PANCREATIC CANCER RISK CLASSIFIER

List Price	$4,000
Averaged Realized Revenue	$2,600
Now Billed Under Molecular Code	81479
Covered Lives	+71 million

ThyGenX™ THYROID ONCOGENE PANEL
ThyraMIR™ THYROID miRNA CLASSIFIER

List Price	$1,675	$4,000
Averaged Realized Revenue	$1,100	$2,000
Now Billed Under Molecular Code	81445	81479
Covered Lives	+200 million	+200 million

MVPdX

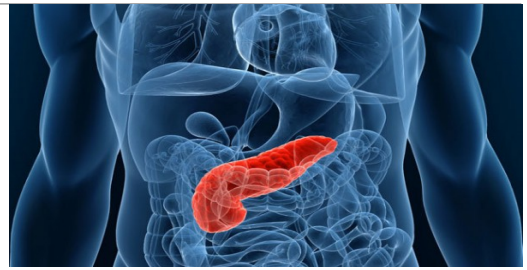
List Price	$4,500
Averaged Realized Revenue	$2,600
Now Billed Under Molecular Code	81479
Covered Lives	+90 million

PANCRAGEN™

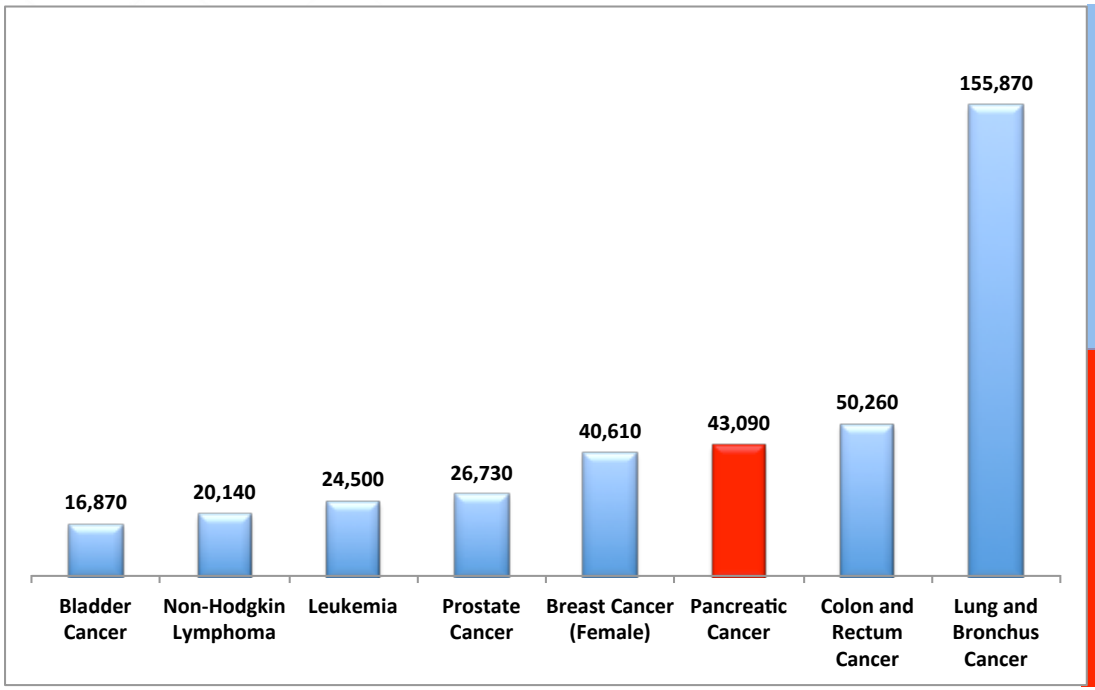
PANCREATIC CANCER RISK CLASSIFIER

PANCREATIC CYSTS CANCER RISK

POWERED BY PathFinderTG®



Pancreatic Cancer



Number of New US Cases in 2017:
43,090

Number of US Deaths in 2016:
43,090

- The Third Leading U.S. Cancer Killer
- 5-year survival rate 7.2%

https://seer.cancer.gov/statfacts/more.html

INTERPACE DIAGNOSTICS
GUIDING TREATMENT. DELIVERING INNOVATION.

PancraGEN®
Integrated Molecular Pathology

Results published in leading GI journal, Endoscopy

	PancraGen® % [95% CI]	Sendai 2012 model % [95%CI]	P value for PancraGen® vs. Sendai 2012 model
NPV	97.2 [95.1-98.6]	97 [93.7-98.9]	0.88
PPV	57.9 [47.3-68.0]	20.8 [16.2-25.9]	<0.0001

PancraGEN can identify non-progressors as well as 2012 International Sendai Guidelines, however it's significantly better at identifying progressors

Eluri et al. Am J Gastroenterol 2015; 110:828–834; doi: 10.1038/ajg.2015.152 (9)

PANCRAGEN™ PANCREATIC CANCER RISK CLASSIFIER

INTERPACE DIAGNOSTICS
GUIDING TREATMENT. DELIVERING INNOVATION.

Significant Clinical Evidence

5 Clinical Validation Papers Published

4 Clinical Utility Papers Published

1 Registry Study published

5 Cost Benefit Studies Published

Budget Impact Model

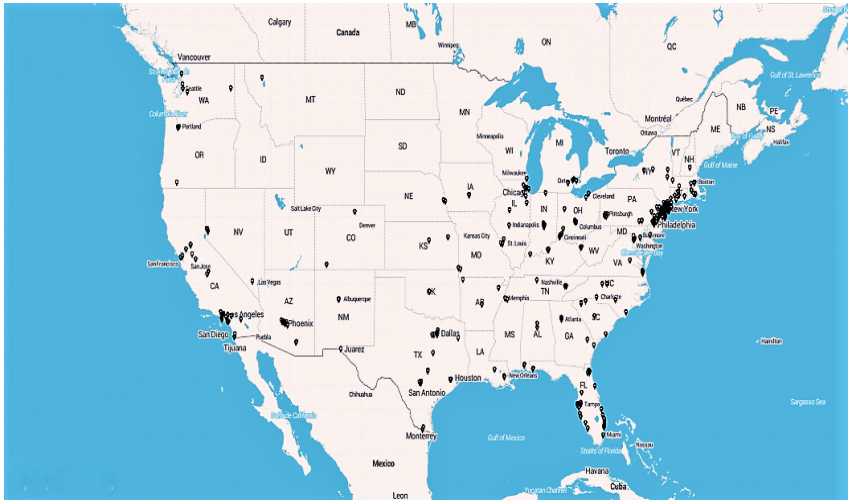
Sendai guidelines 2012 and ACG guidelines 2007 strongly favor surgical resection because of the inability of first-line tests to predict biological behavior and aggressiveness.

PancraGEN establishes a new standard for the prognosis and diagnosis of pancreatic cysts

1 Al-Haddad MA, Kowalski T, Siddiqui A, Mertz HR, Mallat D, Haddad N, Malhotra N, Sadowski B, Lybik MJ, Patel SN, Okoh E, Rosenkranz L,Karasik M, Golioto M, Linder J, Catalano MF. Endoscopy. 2015 Feb;47(2):136-46. doi: 10.1055/s-0034-1390742

PANCRAGEN™ PANCREATIC CANCER RISK CLASSIFIER

PancraGEN® Adoption & Growth



- ✓ 250 Physicians and Hospitals
- ✓ Over 30,000 tests performed
- ✓ International Distribution

BlueCross BlueShield

TRICARE

HUMANA

HIGHMARK Blue Cross Blue Shield

NOVITAS SOLUTIONS

Medicare

Multiplan

Galaxy Health Network

ChoiceCare NETWORK

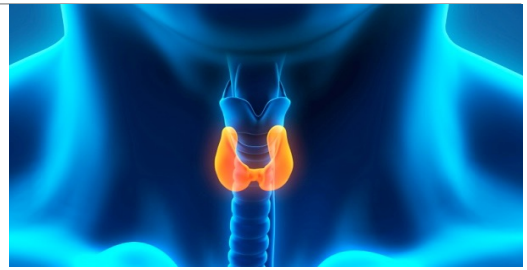
ThyGenX®

THYROID ONCOGENE PANEL

ThyraMIR™

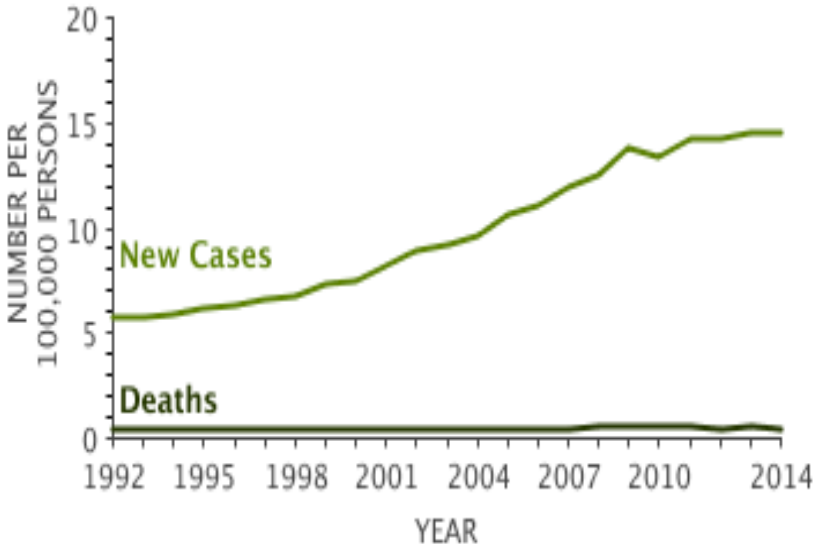
THYROID miRNA CLASSIFIER

ENDOCRINE ONCOLOGY



Thyroid Nodules

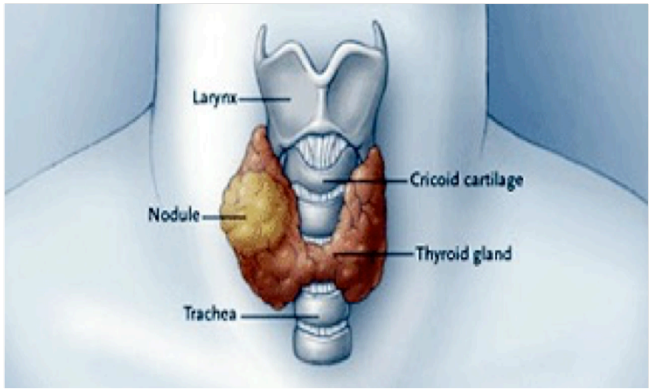
Thyroid Cancer Incidence*



- ~10-18M U.S. adults have Nodules^
- Estimated 525,00 thyroid Fine Needle Aspirates (FNAs) per year in the U.S. and growing

56,870 estimated new cases of thyroid cancer in 2017.*

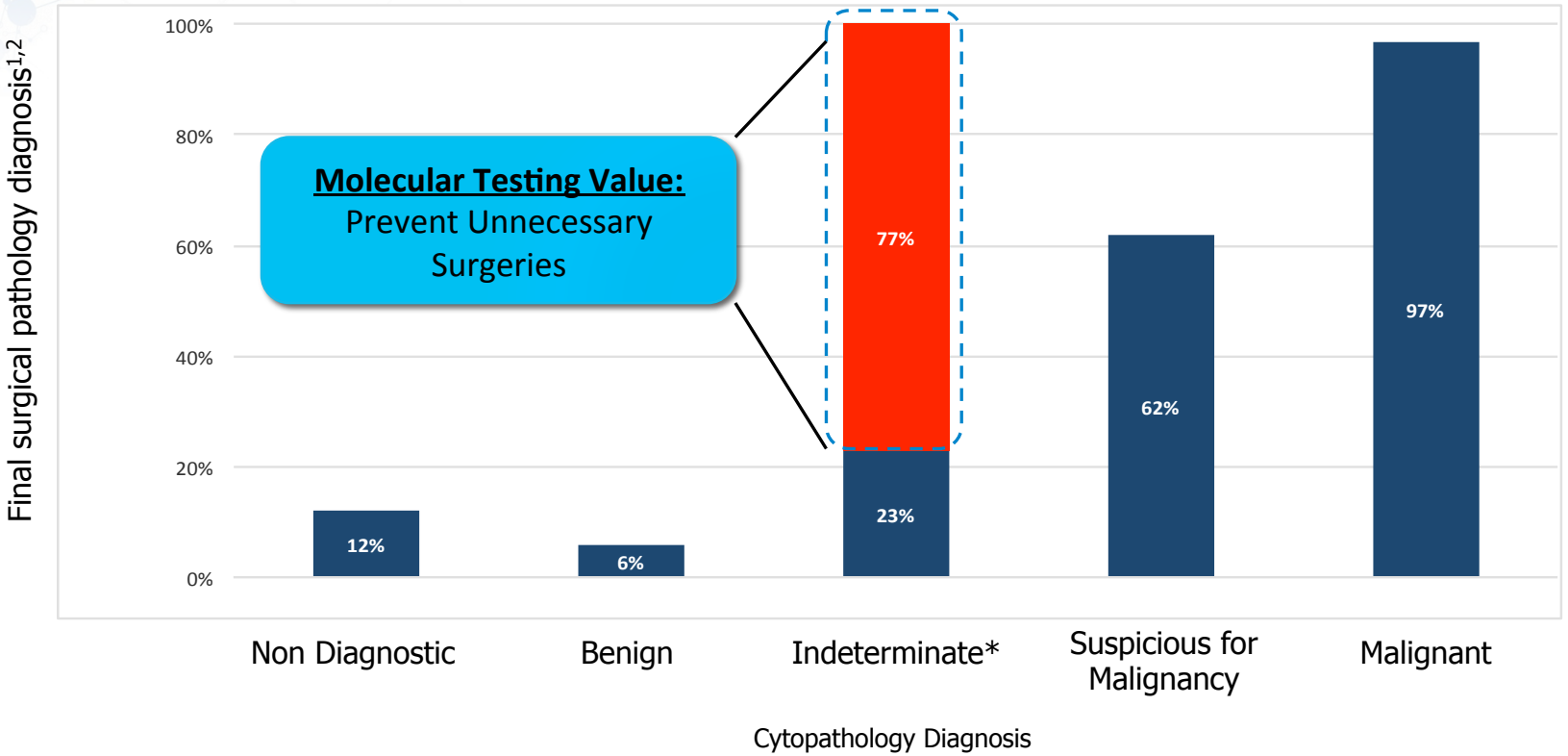
Prevalence of This Cancer: In 2014, there were an estimated 726,646 people living with thyroid cancer in the United States.*



^American Cancer Society http://www.cancernetwork.com/thyroid-cancer/thyroid-nodules-when-biopsy^

* https://seer.cancer.gov/statfacts/html/thyro.html

Clinical Dilemma of Indeterminate Thyroid Nodules



*__Indeterminate__ (Follicular Lesion2) includes Atypia of Undetermined Significance (AUS)/Follicular Lesion of Undetermined Significance(FLUS) and (suspicious for) Hürthle/Follicular Neoplasm

1 Gharib H, et al. Endoc Pract 2010.
2 Wang CC, et al. Thyroid 2011.

Data Comparison with Market Leader

Test performance [95% CI]	Combined mutation & miRNA testing (ThyGenX + ThyraMIR)*	Mutation testing alone (ThyGenX)*	Gene expression classifier with mRNA testing^ (Afirma®)
PPV (%)	74% [58-86]	81% [54-96]	47% [40-55] 37% [23-52]
NPV (%)	94 %[85-98]	64 %[47-79]	93% [86-97] 94% [79-99]

ThyGenX™ and ThyraMIR™ combination testing can accurately "Rule in" and "Rule out" the risk of malignancy

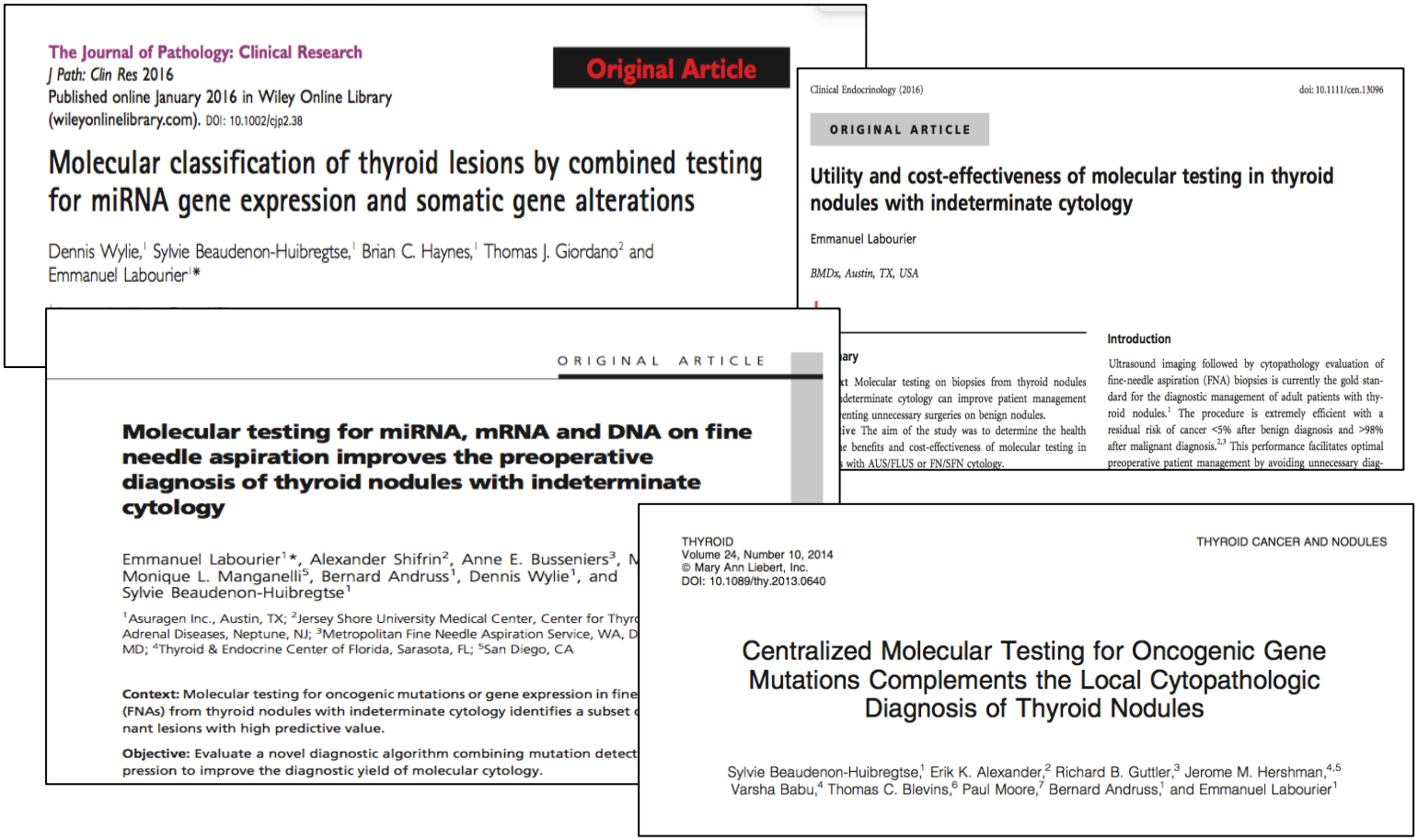
ThyraMIR™ measures the expression of 10 microRNAs and, and the combination with ThyGenX™, yields both high NPV and high PPV

Only commercial test that can be performed from FNA and/or Cytology Slides

ThyGenX™ and ThyraMIR™ combination testing addresses a unmet clinical need for more actionable information in the management of indeterminate thyroid nodules

*Labourier et al. JCEM 2015.
^Alexander et al. NJEM 2012

Multiple Publications Support Clinical Validity and Utility of ThyGenX® / ThyraMIR®



The Journal of Pathology: Clinical Research
J Path: Clin Res 2016
Published online January 2016 in Wiley Online Library (wileyonlinelibrary.com). DOI: 10.1002/cjp2.38

Original Article

Molecular classification of thyroid lesions by combined testing for miRNA gene expression and somatic gene alterations

Dennis Wylie,[1] Sylvie Beaudenon-Huibregtse,[1] Brian C. Haynes,[1] Thomas J. Giordano[2] and Emmanuel Labourier[1]*

Clinical Endocrinology (2016) doi: 10.1111/cen.13096

ORIGINAL ARTICLE

Utility and cost-effectiveness of molecular testing in thyroid nodules with indeterminate cytology

Emmanuel Labourier

BMDx, Austin, TX, USA

Introduction

Ultrasound imaging followed by cytopathology evaluation of fine-needle aspiration (FNA) biopsies is currently the gold standard for the diagnostic management of adult patients with thyroid nodules.[1] The procedure is extremely efficient with a residual risk of cancer <5% after benign diagnosis and >98% after malignant diagnosis.[2,3] This performance facilitates optimal preoperative patient management by avoiding unnecessary diag-

ORIGINAL ARTICLE

Molecular testing for miRNA, mRNA and DNA on fine needle aspiration improves the preoperative diagnosis of thyroid nodules with indeterminate cytology

Emmanuel Labourier[1]*, Alexander Shifrin[2], Anne E. Busseniers[3], M
Monique L. Manganelli[5], Bernard Andruss[1], Dennis Wylie[1], and
Sylvie Beaudenon-Huibregtse[1]

[1]Asuragen Inc., Austin, TX; [2]Jersey Shore University Medical Center, Center for Thyr
Adrenal Diseases, Neptune, NJ; [3]Metropolitan Fine Needle Aspiration Service, WA, D
MD; [4]Thyroid & Endocrine Center of Florida, Sarasota, FL; [5]San Diego, CA

Context: Molecular testing for oncogenic mutations or gene expression in fine
(FNAs) from thyroid nodules with indeterminate cytology identifies a subset
nant lesions with high predictive value.

Objective: Evaluate a novel diagnostic algorithm combining mutation detect
pression to improve the diagnostic yield of molecular cytology.

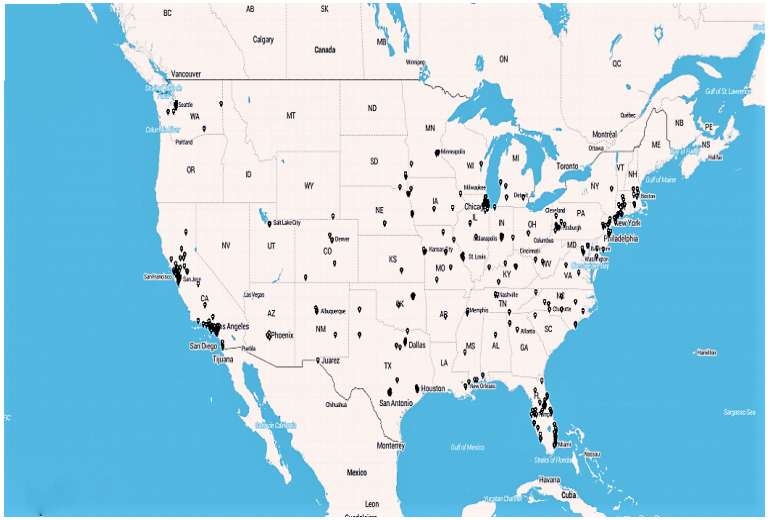
THYROID
Volume 24, Number 10, 2014
© Mary Ann Liebert, Inc.
DOI: 10.1089/thy.2013.0640

THYROID CANCER AND NODULES

Centralized Molecular Testing for Oncogenic Gene Mutations Complements the Local Cytopathologic Diagnosis of Thyroid Nodules

Sylvie Beaudenon-Huibregtse,[1] Erik K. Alexander,[2] Richard B. Guttler,[3] Jerome M. Hershman,[4,5] Varsha Babu,[4] Thomas C. Blevins,[6] Paul Moore,[7] Bernard Andruss,[1] and Emmanuel Labourier[1]

ThyGenX®/ ThyraMIR® Strong Adoption & Growth



- ✓ 400 Physicians and Hospitals
- ✓ Over 15,000 tests performed
- ✓ LabCorp Partnership
- ✓ International Distribution

UnitedHealthcare

Aetna

KAISER PERMANENTE®

BlueCross BlueShield

Oxford Health Plans

Cigna

NOVITAS SOLUTIONS

Medicare

GEISINGER HEALTH SYSTEM
REDEFINING BOUNDARIES

CareFirst

EmblemHealth

LabCorp
Laboratory Corporation of America

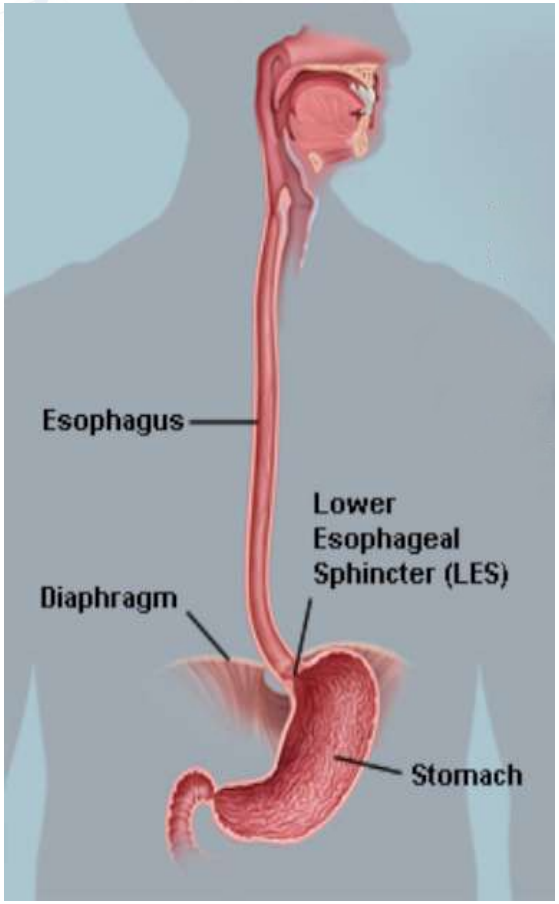
BarreGEN™

ESOPHAGEAL CANCER RISK CLASSIFIER

BARRETT'S ESOPHAGUS RISK OF PROGRESSION TO CANCER

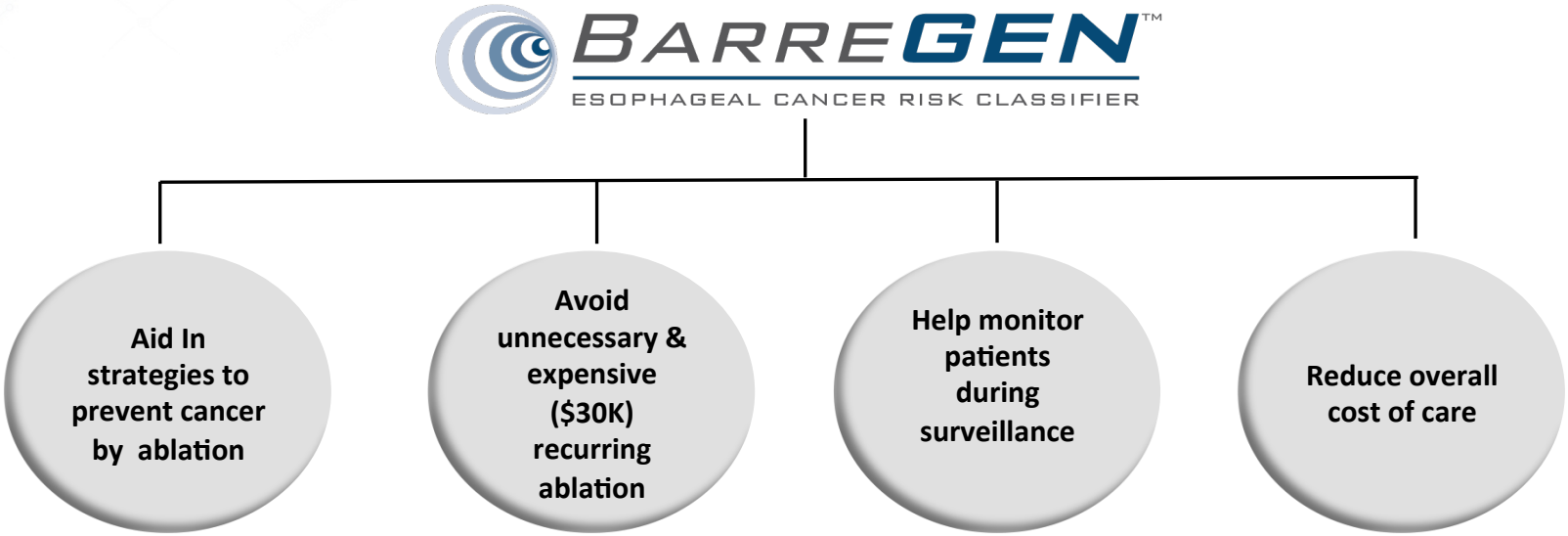
POWERED BY PathFinderTG®

What is Barrett's Esophagus?



- Gastroesophageal reflux very common (10-20% US adults)
- 6% progress to Barrett's Esophagus **(~3.3 million adults)**
- Barrett's Esophagus precedes esophageal cancer (EAC) infrequently (1-3%)
- Ablation (Barrx) has emerged as a treatment and prevention strategy
- Current tests cannot predict which patients will progress to Esophageal Cancer – one of the most lethal cancers – creating a high unmet need for a molecular diagnostic test

How May BarreGEN® Be Useful?

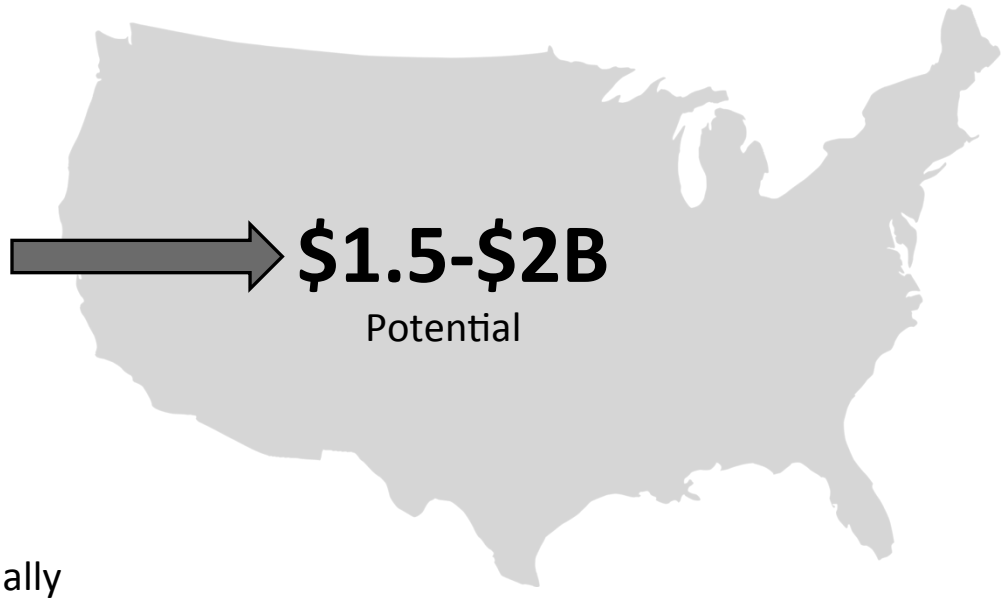


BarreGEN can allow for more personalized management of Barrett's patients

BarreGEN® for Barrett's Esophagus

~3.3M
Adults

~850,000
Endoscopic screens Annually

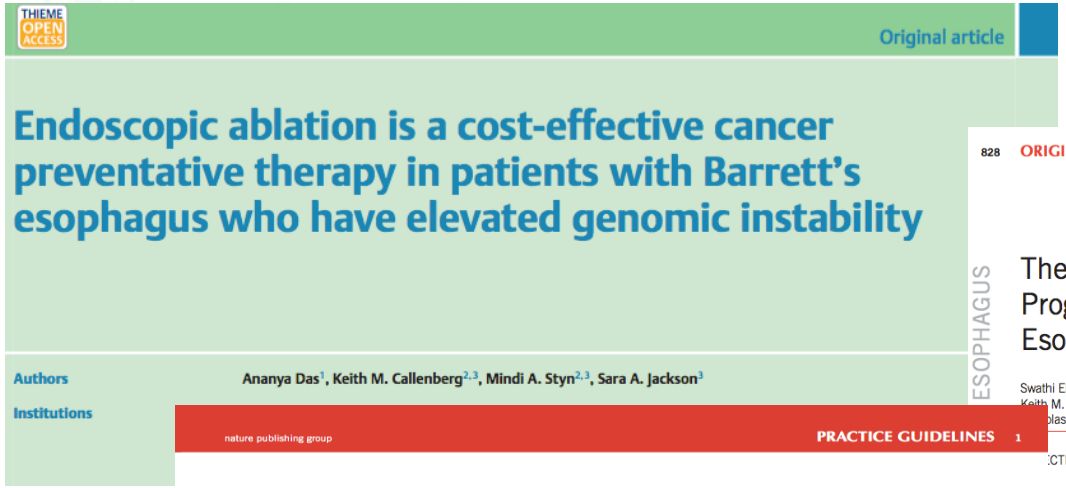


$1.5-$2B
Potential

PathfinderTG® platform
BarreGEN Clinical Experience Program (CEP) launched September 1st, 2016

Source: Indications and Outcomes of Gastrointestinal Endoscopy, 2009

Multiple Publications Support Clinical Validity and Utility of BarreGEN®



THIEME OPEN ACCESS

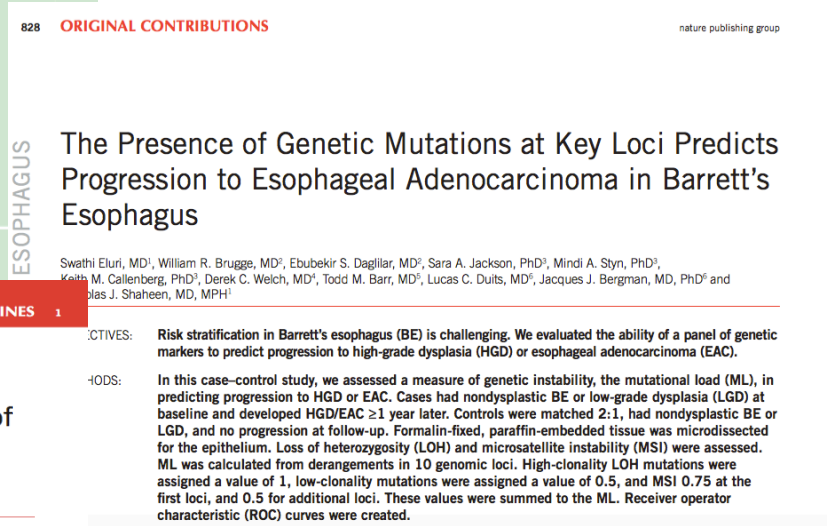
Original article

Endoscopic ablation is a cost-effective cancer preventative therapy in patients with Barrett's esophagus who have elevated genomic instability

Authors

Institutions

Ananya Das[1], Keith M. Callenberg[2,3], Mindi A. Styn[2,3], Sara A. Jackson[3]



828 ORIGINAL CONTRIBUTIONS nature publishing group

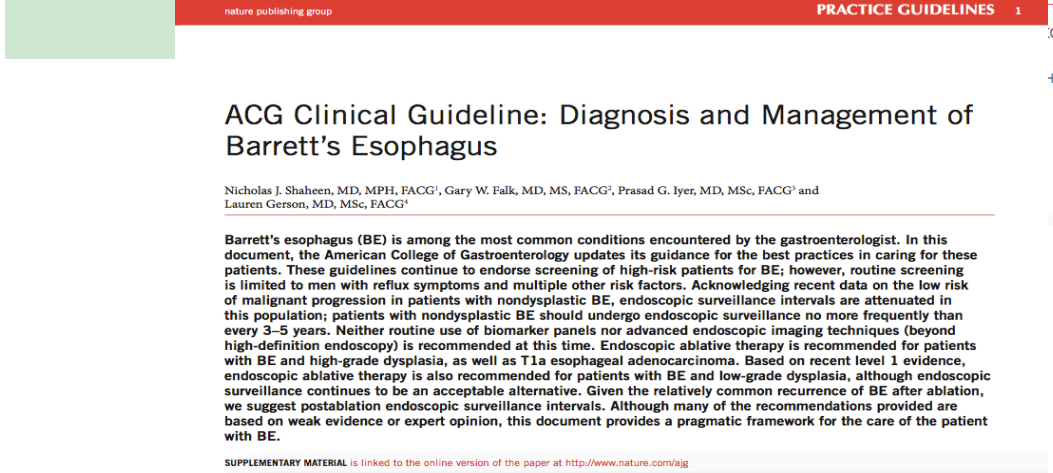
ESOPHAGUS

The Presence of Genetic Mutations at Key Loci Predicts Progression to Esophageal Adenocarcinoma in Barrett's Esophagus

Swathi Eluri, MD[1], William R. Brugge, MD[2], Ebubekir S. Daglilar, MD[2], Sara A. Jackson, PhD[3], Mindi A. Styn, PhD[3], Keith M. Callenberg, PhD[3], Derek C. Welch, MD[4], Todd M. Barr, MD[5], Lucas C. Duits, MD[6], Jacques J. Bergman, MD, PhD[6] and Nicholas J. Shaheen, MD, MPH[1]

CTIVES: **Risk stratification in Barrett's esophagus (BE) is challenging. We evaluated the ability of a panel of genetic markers to predict progression to high-grade dysplasia (HGD) or esophageal adenocarcinoma (EAC).**

HODS: **In this case–control study, we assessed a measure of genetic instability, the mutational load (ML), in predicting progression to HGD or EAC. Cases had nondysplastic BE or low-grade dysplasia (LGD) at baseline and developed HGD/EAC ≥1 year later. Controls were matched 2:1, had nondysplastic BE or LGD, and no progression at follow-up. Formalin-fixed, paraffin-embedded tissue was microdissected for the epithelium. Loss of heterozygosity (LOH) and microsatellite instability (MSI) were assessed. ML was calculated from derangements in 10 genomic loci. High-clonality LOH mutations were assigned a value of 1, low-clonality mutations were assigned a value of 0.5, and MSI 0.75 at the first loci, and 0.5 for additional loci. These values were summed to the ML. Receiver operator characteristic (ROC) curves were created.**



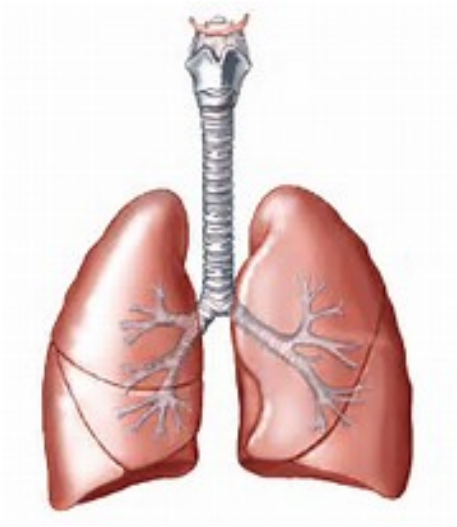
nature publishing group PRACTICE GUIDELINES 1

ACG Clinical Guideline: Diagnosis and Management of Barrett's Esophagus

Nicholas J. Shaheen, MD, MPH, FACG[1], Gary W. Falk, MD, MS, FACG[2], Prasad G. Iyer, MD, MSc, FACG[3] and Lauren Gerson, MD, MSc, FACG[4]

Barrett's esophagus (BE) is among the most common conditions encountered by the gastroenterologist. In this document, the American College of Gastroenterology updates its guidance for the best practices in caring for these patients. These guidelines continue to endorse screening of high-risk patients for BE; however, routine screening is limited to men with reflux symptoms and multiple other risk factors. Acknowledging recent data on the low risk of malignant progression in patients with nondysplastic BE, endoscopic surveillance intervals are attenuated in this population; patients with nondysplastic BE should undergo endoscopic surveillance no more frequently than every 3–5 years. Neither routine use of biomarker panels nor advanced endoscopic imaging techniques (beyond high-definition endoscopy) is recommended at this time. Endoscopic ablative therapy is recommended for patients with BE and high-grade dysplasia, as well as T1a esophageal adenocarcinoma. Based on recent level 1 evidence, endoscopic ablative therapy is also recommended for patients with BE and low-grade dysplasia, although endoscopic surveillance continues to be an acceptable alternative. Given the relatively common recurrence of BE after ablation, we suggest postablation endoscopic surveillance intervals. Although many of the recommendations provided are based on weak evidence or expert opinion, this document provides a pragmatic framework for the care of the patient with BE.

SUPPLEMENTARY MATERIAL is linked to the online version of the paper at http://www.nature.com/ajg



MVPdx

METASTASIS VS. NEW PRIMARY CANCER - LUNG

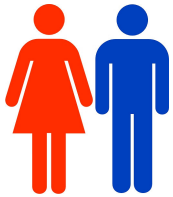
POWERED BY PathFinderTG®

AMERICAN LUNG ASSOCIATION®

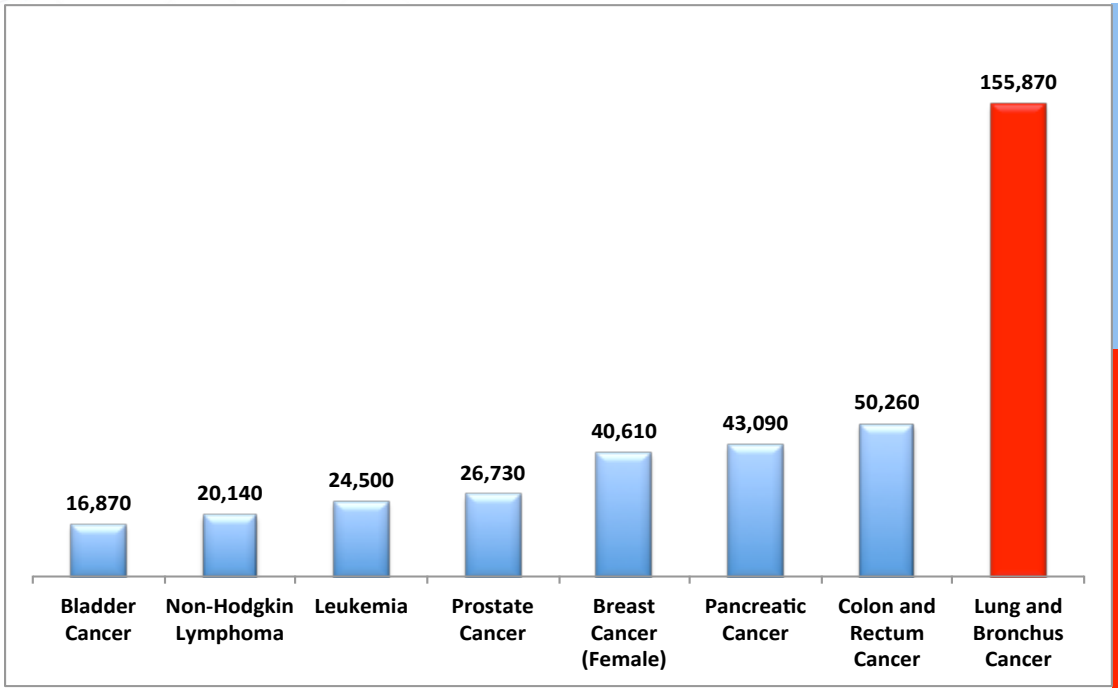
222,500 Newly Diagnosed Cases/Year

No. 1 Leading Cause of Death in Men & Women

No. 2 New Cancer Incidence in Both Men & Women



Lung Cancer Statistics



Number of New US Cases in 2017: 222,500

Number of US Deaths in 2017: 155,870

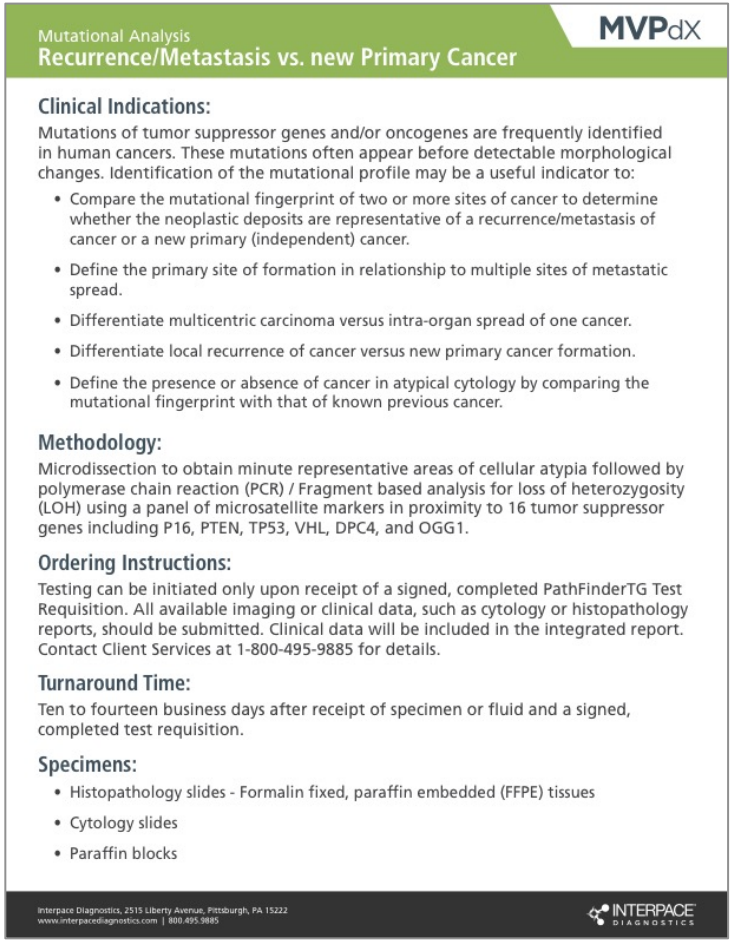
- Leading U.S. Cancer Killer
- 5-year survival rate 18.1%

https://seer.cancer.gov/statfacts/more.html

MVPdx (Lung Cancer)

Clinical Indications

- **Compare the mutational fingerprint of two or more cancer sites to determine recurrence/metastasis or new primary (independent) cancer**
  - Define Primary site of formation in relationship to multiple metastatic spread
  - Differentiate multi-centric carcinoma versus intra-organ spread of one cancer
  - Differentiate local reoccurrence versus intra-organ cancer formation



Mutational Analysis
MVPdX
Recurrence/Metastasis vs. new Primary Cancer

Clinical Indications:

Mutations of tumor suppressor genes and/or oncogenes are frequently identified in human cancers. These mutations often appear before detectable morphological changes. Identification of the mutational profile may be a useful indicator to:

- Compare the mutational fingerprint of two or more sites of cancer to determine whether the neoplastic deposits are representative of a recurrence/metastasis of cancer or a new primary (independent) cancer.
- Define the primary site of formation in relationship to multiple sites of metastatic spread.
- Differentiate multicentric carcinoma versus intra-organ spread of one cancer.
- Differentiate local recurrence of cancer versus new primary cancer formation.
- Define the presence or absence of cancer in atypical cytology by comparing the mutational fingerprint with that of known previous cancer.

Methodology:

Microdissection to obtain minute representative areas of cellular atypia followed by polymerase chain reaction (PCR) / Fragment based analysis for loss of heterozygosity (LOH) using a panel of microsatellite markers in proximity to 16 tumor suppressor genes including P16, PTEN, TP53, VHL, DPC4, and OGG1.

Ordering Instructions:

Testing can be initiated only upon receipt of a signed, completed PathFinderTG Test Requisition. All available imaging or clinical data, such as cytology or histopathology reports, should be submitted. Clinical data will be included in the integrated report. Contact Client Services at 1-800-495-9885 for details.

Turnaround Time:

Ten to fourteen business days after receipt of specimen or fluid and a signed, completed test requisition.

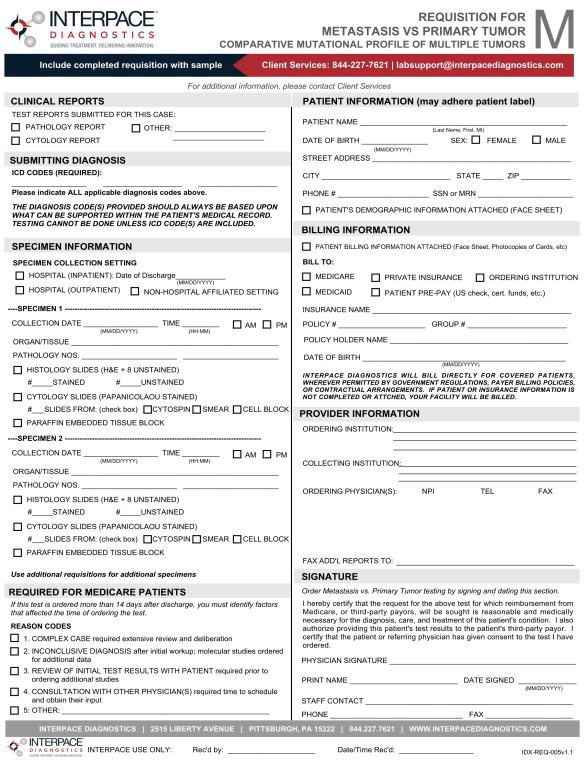
Specimens:

- Histopathology slides - Formalin fixed, paraffin embedded (FFPE) tissues
- Cytology slides
- Paraffin blocks

Interpace Diagnostics, 2515 Liberty Avenue, Pittsburgh, PA 15222
www.interpacediagnostics.com | 800.495.9885
INTERPACE DIAGNOSTICS

* Based on Current Medicare Reimbursement

MVPdx Lung
600+ Tests Performed To Date



INTERPACE DIAGNOSTICS

REQUISITION FOR
METASTASIS VS PRIMARY TUMOR
COMPARATIVE MUTATIONAL PROFILE OF MULTIPLE TUMORS

Include completed requisition with sample | Client Services: 844-227-7621 | labsupport@interpacediagnostics.com

For additional information, please contact Client Services

CLINICAL REPORTS

TEST REPORTS SUBMITTED FOR THIS CASE:
☐ PATHOLOGY REPORT ☐ OTHER: _____
☐ CYTOLOGY REPORT _____

SUBMITTING DIAGNOSIS

ICD CODES (REQUIRED): _____ _____

Please indicate ALL applicable diagnosis codes above.

THE DIAGNOSIS CODE(S) PROVIDED SHOULD ALWAYS BE BASED UPON WHAT CAN BE SUPPORTED WITHIN THE PATIENT'S MEDICAL RECORD. TESTING CANNOT BE DONE UNLESS ICD CODE(S) ARE INCLUDED.

SPECIMEN INFORMATION

SPECIMEN COLLECTION SETTING
☐ HOSPITAL (INPATIENT): Date of Discharge _____ (MM/DD/YYYY)
☐ HOSPITAL (OUTPATIENT) ☐ NON-HOSPITAL AFFILIATED SETTING

----SPECIMEN 1 ----------
COLLECTION DATE _____ (MM/DD/YYYY) TIME _____ (HH:MM) ☐ AM ☐ PM
ORGAN/TISSUE _____
PATHOLOGY NOS. _____ _____
☐ HISTOLOGY SLIDES (H&E + 8 UNSTAINED)
#____STAINED #____UNSTAINED
☐ CYTOLOGY SLIDES (PAPANICOLAOU STAINED)
#___SLIDES FROM: (check box) ☐CYTOSPIN ☐SMEAR ☐CELL BLOCK
☐ PARAFFIN EMBEDDED TISSUE BLOCK

----SPECIMEN 2 ----------
COLLECTION DATE _____ (MM/DD/YYYY) TIME _____ (HH:MM) ☐ AM ☐ PM
ORGAN/TISSUE _____
PATHOLOGY NOS. _____ _____
☐ HISTOLOGY SLIDES (H&E + 8 UNSTAINED)
#____STAINED #____UNSTAINED
☐ CYTOLOGY SLIDES (PAPANICOLAOU STAINED)
#___SLIDES FROM: (check box) ☐CYTOSPIN ☐SMEAR ☐CELL BLOCK
☐ PARAFFIN EMBEDDED TISSUE BLOCK

Use additional requisitions for additional specimens

REQUIRED FOR MEDICARE PATIENTS

If this test is ordered more than 14 days after discharge, you must identify factors that affected the time of ordering the test.

REASON CODES
☐ 1. COMPLEX CASE required extensive review and deliberation
☐ 2. INCONCLUSIVE DIAGNOSIS after initial workup; molecular studies ordered for additional data
☐ 3. REVIEW OF INITIAL TEST RESULTS WITH PATIENT required prior to ordering additional studies
☐ 4. CONSULTATION WITH OTHER PHYSICIAN(S) required time to schedule and obtain their input
☐ 5: OTHER: _____

PATIENT INFORMATION (may adhere patient label)

PATIENT NAME _____ (Last Name, First, MI)
DATE OF BIRTH _____ (MM/DD/YYYY) SEX: ☐ FEMALE ☐ MALE
STREET ADDRESS _____
CITY _____ STATE ____ ZIP _____
PHONE # _____ SSN or MRN _____
☐ PATIENT'S DEMOGRAPHIC INFORMATION ATTACHED (FACE SHEET)

BILLING INFORMATION

☐ PATIENT BILLING INFORMATION ATTACHED (Face Sheet, Photocopies of Cards, etc)
BILL TO:
☐ MEDICARE ☐ PRIVATE INSURANCE ☐ ORDERING INSTITUTION
☐ MEDICAID ☐ PATIENT PRE-PAY (US check, cert. funds, etc.)
INSURANCE NAME _____
POLICY # _____ GROUP # _____
POLICY HOLDER NAME _____
DATE OF BIRTH _____ (MM/DD/YYYY)

INTERPACE DIAGNOSTICS WILL BILL DIRECTLY FOR COVERED PATIENTS, WHEREVER PERMITTED BY GOVERNMENT REGULATIONS, PAYER BILLING POLICIES, OR CONTRACTUAL ARRANGEMENTS. IF PATIENT OR INSURANCE INFORMATION IS NOT COMPLETED OR ATTCHED, YOUR FACILITY WILL BE BILLED.

PROVIDER INFORMATION

ORDERING INSTITUTION: _____
COLLECTING INSTITUTION: _____
ORDERING PHYSICIAN(S): NPI TEL FAX
FAX ADD'L REPORTS TO: _____

SIGNATURE

Order Metastasis vs. Primary Tumor testing by signing and dating this section.

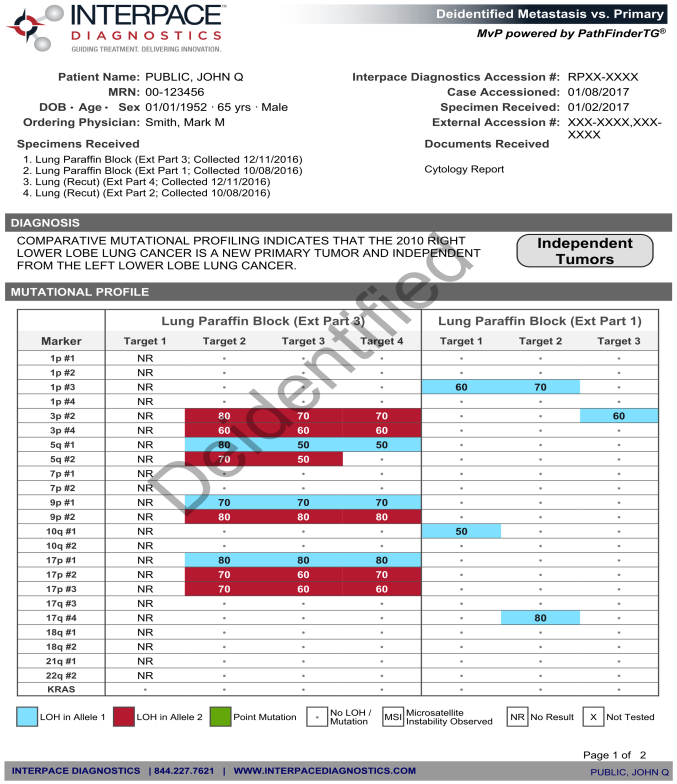
I hereby certify that the request for the above test for which reimbursement from Medicare, or third-party payors, will be sought is reasonable and medically necessary for the diagnosis, care, and treatment of this patient's condition. I also authorize providing this patient's test results to the patient's third-party payor. I certify that the patient or referring physician has given consent to the test I have ordered.

PHYSICIAN SIGNATURE _____
PRINT NAME _____ DATE SIGNED _____ (MM/DD/YYYY)
STAFF CONTACT _____
PHONE _____ FAX _____

INTERPACE DIAGNOSTICS | 2515 LIBERTY AVENUE | PITTSBURGH, PA 15222 | 844.227.7621 | WWW.INTERPACEDIAGNOSTICS.COM

INTERPACE DIAGNOSTICS INTERPACE USE ONLY: Rec'd by: _____ Date/Time Rec'd: _____ IDX-REQ-005v1.1

Requisition



INTERPACE DIAGNOSTICS
GUIDING TREATMENT. DELIVERING INNOVATION.

Deidentified Metastasis vs. Primary
MvP powered by PathFinderTG®

Patient Name: PUBLIC, JOHN Q
MRN: 00-123456
DOB · Age · Sex 01/01/1952 · 65 yrs · Male
Ordering Physician: Smith, Mark M

Interpace Diagnostics Accession #: RPXX-XXXX
Case Accessioned: 01/08/2017
Specimen Received: 01/02/2017
External Accession #: XXX-XXXX,XXX-XXXX

Specimens Received
1. Lung Paraffin Block (Ext Part 3; Collected 12/11/2016)
2. Lung Paraffin Block (Ext Part 1; Collected 10/08/2016)
3. Lung (Recut) (Ext Part 4; Collected 12/11/2016)
4. Lung (Recut) (Ext Part 2; Collected 10/08/2016)

Documents Received
Cytology Report

DIAGNOSIS

COMPARATIVE MUTATIONAL PROFILING INDICATES THAT THE 2010 RIGHT LOWER LOBE LUNG CANCER IS A NEW PRIMARY TUMOR AND INDEPENDENT FROM THE LEFT LOWER LOBE LUNG CANCER.

Independent Tumors

Deidentified

MUTATIONAL PROFILE

	Lung Paraffin Block (Ext Part 3)				Lung Paraffin Block (Ext Part 1)		
Marker	Target 1	Target 2	Target 3	Target 4	Target 1	Target 2	Target 3
1p #1	NR	-	-	-	-	-	-
1p #2	NR	-		-	-	-	-
1p #3	NR	-		-	60	70	-
1p #4	NR	-		-	-	-	-
3p #2	NR	80	70	70	-	-	60
3p #4	NR	60	60	60	-	-	-
5q #1	NR	80	50	50	-	-	-
5q #2	NR	70	50	-	-	-	-
7p #1	NR		-	-	-	-	-
7p #2	NR	-	-	-	-	-	-
9p #1	NR	70	70	70	-	-	-
9p #2	NR	80	80	80	-	-	-
10q #1	NR	-	-	-	50	-	-
10q #2	NR	-	-	-	-	-	-
17p #1	NR	80	80	80	-	-	-
17p #2	NR	70	60	70	-	-	-
17p #3	NR	70	60	60	-	-	-
17q #3	NR	-	-	-	-	-	-
17q #4	NR	-	-	-	-	80	-
18q #1	NR	-	-	-	-	-	-
18q #2	NR	-	-	-	-	-	-
21q #1	NR	-	-	-	-	-	-
22q #2	NR	-	-	-	-	-	-
KRAS	-	-	-	-	-	-	-

LOH in Allele 1 | LOH in Allele 2 | Point Mutation | - No LOH / Mutation | MSI Microsatellite Instability Observed | NR No Result | X Not Tested

INTERPACE DIAGNOSTICS | 844.227.7621 | WWW.INTERPACEDIAGNOSTICS.COM PUBLIC, JOHN Q

Sample Report

MVPdx Supportive Publications

GENERAL THORACIC

Comparative Mutational Profiling in the Assessment of Lung Lesions: Should It Be the Standard of Care?

Susan D. Moffatt-Bruce, MD, PhD, Patrick Ross, MD, PhD, Marino E. Leon, MD, Gang He, MD, Sydney D. Finkelstein, MD, Alexandru M. Vaida, MD, O. Hans Iwenofu, MD, Wendy L. Frankel, MD, and Charles L. Hitchcock, MD, PhD

Division of Cardiothoracic Surgery, Department of Surgery, and Division of Thoracic Pathology, Department of Pathology, Ohio State University, Columbus, Ohio; and RedPath Integrated Pathology, Pittsburgh, Pennsylvania

Background. **Discerning primary versus metastatic lung lesions is problematic. Comparative mutational profiling (CMP) involves genetic and point mutation analysis of lesions to facilitate this. We sought to review our experience in cases of two lung lesions or head and neck cancer and lung lesions to determine whether a significantly clinical problem existed, what standard processes were in place to address it, and whether a new diagnostic standard was required.**

Methods. **Between January 1, 2007, and October 31, 2008, CMP was used in 24 cases of two lung lesions or a head and neck cancer and lung lesion. Routine hematoxylin and eosin stain examination and immunohistochemistry were performed as appropriate. The CMP involved DNA sequencing for specific oncogene point mutations and a panel of allelic imbalance markers. Metastatic cancer required demonstration of concordant mutations affecting the same allele copy in different cancer deposits.**

Results. **The patient mean age was 62 years; there were 13 men and 11 women. The cases involved two lung lesions (n = 13) or a head and neck cancer and a lung lesion (n = 11). Standard pathology examination was unable to discriminate the lesions, and they were subsequently differentiated by CMP. Fifteen discordant CMP results were interpreted as independent primaries; 9 cases were concordant, consistent with metastatic disease.**

Conclusions. **Discerning primary versus metastatic disease when dealing with lung lesions is a clinically significant problem. Comparative mutational profiling was found to be useful and reliable to assess the relatedness of multiple cancer lesions when routine pathology assessment was unable to.**

(Ann Thorac Surg 2010;90:388–97)

Thoracic surgeons are often faced with trying to discern whether lung masses are primary or metastatic processes when a patient has two lung lesions or another neoplastic process and a lung lesion. This differentiation is absolutely essential to planning patient care and prognosticating patient outcomes. Currently, tissue specimens undergo routine staining with hematoxylin and eosin staining and immunohistochemistry (IHC) staining of cytoplasmic, nuclear, and cell surface antigens to discern the histogenesis of each lesion. However, when these techniques are unable to differentiate between a primary and a metastatic process, comparative mutational profiling (CMP) is appropriate. We sought to determine whether discerning lung masses as primary or metastatic was a clinical problem, what standard processes were in place to address it, and whether a new diagnostic standard was required.

Beyreuther first described synchronous lung lesions in 1924 [1]. The actual frequency of multiple lung lesions varies from 0.8% to 14.5%, depending on whether it is calculated from a cancer registry, autopsy series, or surgical series [2]. The criteria of Martini and Melamed [3] currently being used to resolve this problem is more subjective than objective in that it is based on tumor morphology, location, presence or absence of carcinoma in situ, vascular invasion, metastases, and other empirical features and not on objective molecular genetic features. Recent data have suggested that an aggressive surgical approach is well justified and safe for patients with truly synchronous primary lung cancers, further emphasizing the need for diagnostic accuracy and often a multidisciplinary approach to patient management [4–9].

Owing to common predisposing risk factors, lung cancer and head and neck cancer often coexist. Additionally, the risk of developing a subsequent malignancy is very high for lung cancer patients, with and without prior primary malignancies [10]. Synchronous tumors, defined as neoplasms that present within 6 months, and metachronous tumors, defined as those presenting more than 6 months after the index tumor, must be differentiated from metastatic processes originating from the head and neck primary [11, 12]. The diagnostic dilemma com-

Accepted for publication March 16, 2010.

Address correspondence to Dr Moffatt-Bruce, Ohio State University Medical Center, N935 Doan Hall, 410 W 10th Ave, Columbus, OH 43210; e-mail: susan.moffatt-bruce@osumc.edu.

Published by Elsevier Inc

0003-4975/$36.00
doi:10.1016/j.athoracsur.2010.03.059

Translational Oncology
www.transonc.com

Volume 10 Number xx Month 2017 pp. 442–449 442

Utility of Genomic Analysis in Differentiating Synchronous and Metachronous Lung Adenocarcinomas from Primary Adenocarcinomas with Intrapulmonary Metastasis[1,2]

Jad Saab, Hamid Zia, Susan Mathew, Michael Kluk, Navneet Narula and Helen Fernandes

Department of Pathology and Laboratory Medicine, New York-Presbyterian Hospital-Weill Cornell Medicine, New York, NY 10065

Abstract

Distinguishing synchronous and metachronous primary lung adenocarcinomas from adenocarcinomas with intrapulmonary metastasis is essential for optimal patient management. In this study, multiple lung adenocarcinomas occurring in the same patient were evaluated using comprehensive histopathologic evaluation supplemented with molecular analysis. The cohort included 18 patients with a total of 52 lung adenocarcinomas. Eleven patients had a new diagnosis of multiple adenocarcinomas in the same lobe (n = 5) or different lobe (n = 6). Seven patients had a history of lung cancer and developed multiple new tumors. The final diagnosis was made in resection specimens (n = 49), fine needle aspiration (n = 2), and biopsy (n = 1). Adenocarcinomas were non-mucinous, and histopathologic comparison of tumors was performed. All tumors save for one were subjected to *ALK* gene rearrangement testing and targeted Next Generation Sequencing (NGS). Using clinical, radiologic, and morphologic features, a confident conclusion favoring synchronous/metachronous or metastatic disease was made in 65% of patients. Cases that proved challenging included ones with more than three tumors showing overlapping growth patterns and lacking a predominant lepidic component. Genomic signatures unique to each tumor were helpful in determining the relationship of multiple carcinomas in 72% of patients. Collectively, morphologic and genomic data proved to be of greater value and achieved a conclusive diagnosis in 94% of patients. Assessment of the genomic profiles of multiple lung adenocarcinomas complements the histological findings, enabling a more comprehensive assessment of synchronous, metachronous, and metastatic lesions in most patients, thereby improving staging accuracy. Targeted NGS can identify genetic alterations with therapeutic implications.

Translational Oncology (2017) 10, 442–449

Introduction

Lung cancer remains the leading cause of cancer-related death in both men and women in the United States with an estimated 221,000 new diagnoses and 158,000 deaths in 2015 [1]. Of all the subtypes of lung carcinoma, adenocarcinoma has shown a dramatic rise in incidence worldwide and currently accounts for approximately half of all newly diagnosed primary lung malignancies [2–4]. Although most patients are diagnosed with a single primary lung adenocarcinoma, the frequency of identifying two or more adenocarcinomas at presentation is not uncommon and has an estimated incidence ranging from 1% to 8% [5–13]. Depending on the stage at

Address all correspondence to: Helen Fernandes, PhD, Department of Pathology and Cell Biology, Laboratory of Personalized Genomic Medicine, Columbia University Medical Center, New York, NY 10032.
E-mail: hf2340@cumc.columbia.edu
[1] Conflict of Interest and Disclosure: The authors have no disclosures.
[2] Funding: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.
Received 15 December 2016; Revised 15 February 2017; Accepted 23 February 2017

http://dx.doi.org/10.1016/j.tranon.2017.02.009

Review Article

Microsatellite alteration in multiple primary lung cancer

Cheng Shen, Xin Wang, Long Tian, Guowei Che

Department of Thoracic Surgery, West-China Hospital, Sichuan University, Chengdu 610041, China
Correspondence to: Prof. Guowei Che. Department of Thoracic Surgery, West-China Hospital, Sichuan University, Chengdu 610041, China. Email: cheguowei_hx@aliyun.com.

Abstract: Patients with pulmonary neoplasms have an increased risk for developing a second tumor of the lung, either at the same time or different times. It is important to determine if the second tumor represents an independent primary tumor or recurrence/metastasis, because it will significantly change the management and prognosis. Microsatellite instability (MSI) and loss of heterozygosity (LOH) represents molecular disorders acquired by the cell during neoplastic transformation. Both are associated with genetic instability. Functional silencing of tumour suppressor genes may be the consequence of genomic instability, particularly of the globally occurring LOH phenomenon. Numerous studies have confirmed the role of MSI/LOH at both the early and the late stages of multiple primary lung cancer. This paper reviews the published literatures focused on the role of MSI/LOH significance in multiple primary lung cancer. Additionally, a new method based on the allelic variations at polymorphic microsatellite markers was offered that it does not rely on collection of normal tissue, performed with minimal tumor sample, and will complement clinical criteria for diagnostic discrimination between multiple primary cancers versus solitary metastatic diseases.

Keywords: Microsatellite instability (MSI); loss of heterozygosity (LOH); multiple primary lung cancer

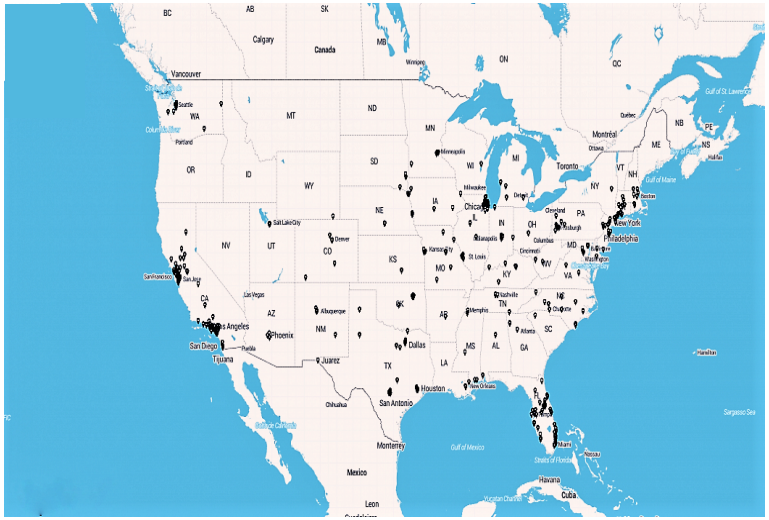
Submitted May 09, 2014. Accepted for publication Aug 28, 2014.
doi: 10.3978/j.issn.2072-1439.2014.09.14
View this article at: http://dx.doi.org/10.3978/j.issn.2072-1439.2014.09.14

Introduction

Lung cancer is the most common cause of cancer death in both males and females in the world. In clinical practice, it is not common to encounter patients with multiple anatomically isolate but histologically similar lung tumors. The incidence of this condition has been reported to range from 0.2% to 2.0% in patients with primary lung cancer (1-3). Coexisting lung cancers in a patient can be characterized as either synchronous or metachronous. The incidence of synchronous lung carcinomas is variably reported between 1% and 16% (4). Metachronous primary lung cancers are likely more common, representing 40-60% of all patients with multiple lung cancers (5). Two different theories have been proposed to explain the multifocality of lung tumors by Slaughter and his colleges. The first is that multifocal tumors arise separately from anatomically distinct malignant progenitor cells that independently undergo different genetic alterations, leading to neoplastic transformation. The second is that these tumors are of monoclonal origin, arising from a single malignant cell that forms a neoplasm, which metastasizes to other regions of the lung parenchyma (6). This phenomenon has been related to the chronic exposure of the bronchial tree to carcinogens through a so-called "field cancerization" process (7). Distinguishing between these two possible mechanisms for the development of multifocal lesions has important surgical, therapeutic, and prognostic implications.

Molecular analysis of microsatellite markers is a method that has been used to assess clonality (8). Microsatellite markers, or tandem simple sequence repeats (SSR), are abundant across genomes and show high levels of polymorphism. Genomic microsatellites, iterations of 1-6 bp nucleotide motifs, have been detected in the genomes of every organism analyzed so far, and are often found at frequencies much higher than would be predicted purely on the grounds of base composition (9). Microsatellite alterations include microsatellite instability (MSI) and loss of heterozygosity (LOH). The relevance of genomic imbalance is further underscored by the association between

MVPdX Adoption



- ✓ 5 Beta Hospitals – Northeast
- ✓ Over 600 tests performed

UnitedHealthcare

Aetna

NOVITAS SOLUTIONS

Medicare

BlueCross BlueShield

Saint Barnabas Medical Center

RWJBarnabas HEALTH

fallonhealth

Independence

Cigna

HIGHMARK

Horizon

Horizon Blue Cross Blue Shield of New Jersey

FINANCIAL AND CORPORATE REVIEW

Select Financial Information

(In US Million)

Income Statement	2016	2015	Q3 2017	Q3 2016	Q3 2017 YTD	Q3 2016 YTD
Revenue	$13.1	$9.4	$4.2	$3.3	$11.5	$10.0
Gross Profit	$6.4	$2.5	$2.1	$1.5	$5.8	$5.1
Total Operating Expenses	$12.9	$42.9	$5.2	$8.0	$8.8	$18.3
Loss from Continuing Ops	($8.4)	($31.1)	($3.4)	($7.2)	($7.8)	($14.7)
Balance Sheet						
Cash Balance (End of Period)	$0.6	$8.3	$11.7	$1.7		
Accounts Payable and Accrued Expenses	$12.1	$9.9	$7.0	$11.4		
Stockholders' Equity	$6.5	$13.0	$36.4	($1.5)		

Source: SEC Filings, 10-K

Patent Portfolio and Proprietary Assets

Thyroid	**2 US patents pending, 5 ex-US Patents Pending**
Pancreas	**5 patents Issued**
Lung	**Proprietary Algorithm**
Barrett's Esophagus	**2 patents Pending**
Proprietary	**Extensive experience in managing extremely low quality, fixative treated clinical specimens.** **Lab information management system that extracts results from database and allows efficient integration of molecular and clinical results.** **Proprietary Algorithm for ThyraMIR Classifier.** **Proprietary extraction and micro dissection, methodology from slides, Buffer and FFPE samples.**

Experienced Management Team

Jack Stover President, CEO	ONCONOVA THERAPEUTICS; antares pharma; sicor; GYNÉTICS Practise better Medicine
Greg Richard Chief Commercial Officer	Aetna; Quest Diagnostics; LabCorp
Syd Finklestein, MD Chief Scientific Officer	REDPATH integrated pathology; UPMC University of Pittsburgh Medical Center
Jim Early VP CFO	AbGenomics International; Synageva BioPharma Dedicated to Rare Diseases
Glenn Gershon Sr. VP. Operations	Abbott Diagnostics; Bayer Diagnostics; athena diagnostics
Alidad Mireskandari, Ph.D. VP Business Development	JSGENETICS; NOMURA; BNP PARIBAS

Recent Accomplishments

Corporate

- Revenue Growth 39% Y-O-Y
- Raised over $14M in Equity
- Converted $9M In Secured Notes into Stock
- Eliminated milestone payments, liens & royalties related to 2014 RedPath Assets Acquisition
- Reduced Cash Burn $20M Y-O-Y 2015 to 2016

Commercial

- AETNA National Contract
- Cigna Coverage
- Oxford Coverage
- UnitedHealth Coverage
- NY State Approval For ThyGenX
- Launched International Distributions
- EU Patent Granted for ThyraMIR
- LabCorp Co-Promotion
- + 250M Thyroid lives covered
- +71M Pancreas Lives covered
- Lab Services Agreement signed with Einstein Medical Center June 5th,2017
- Lab Services Hoag Hospital, CA

Clinical

- Launched new Biliary product October 3rd, 2016
- Launched AccuCEA™ Insights August 1st, 2016
- Launched PanDNA March, 2017
- Launched BarreGen® Clinical Experience Program (CEP) September 1st, 2016, and Soft launched BarreGen at select sites
- Launched Cytopathology Services October 1st, 2016
- Launched Cytology Slides as primary specimen October1st, 2016
- Launched TERT marker of aggressiveness June 1st, 2017

2017/18
Key Drivers of Growth

Internal Drivers

- Contract with Aetna & United Healthcare (Thyroid)
- Launch Lung Vertical MVPdx
- Expanding Sales Force
- Launch of ThyGenX V2 and TERT Marker of Aggressiveness
- New PancraGEN studies (GI)
- Additional Lab Services Contracts

External Drivers

- **Selective Product Acquisitions and Alliances**
- **Strategic Partnership For BarreGEN**
- **Leverage New LabCorp Contract**

Why Invest in IDXG?

Significant Financial Progress

- Strong, growing revenue (39% YoY 2015 to 2016) and reimbursement
- NASDAQ listing fully compliant
- Eliminated all of our secured debt and liens and eliminated royalty & milestone obligations from 2014 purchase of RedPath Assets
- Demonstrated liquidity with our stock trading volume
- $21M cash raised in last 6 months

New Clinical and Product Development

- ThyGenX V2 development underway
- Thyroid registry launched
- Completed development of Lung Vertical MVPdx
- Multi-Center Thyroid study launched
- Barrett's CEP/Soft launch underway
- Multiple presentations at key Scientific meetings planned for 2017
- PancraGEN: 3 clinical utility studies underway

Growing Commercial Capabilities

- 26 Person Internal Sales Organization
- In-house billing, reimbursement and collections expertise
- Marketing and product launch expertise
- AETNA & United Health approvals
- All products covered by Medicare
- 250+ million lives covered for Thyroid test,71+ million lives covered for PancraGEN test
- Launched MVPdx Lung 3Q 2017

A SIGNIFICANT PARTNERING OPPORTUNITY FOR BARREGEN

Thank You