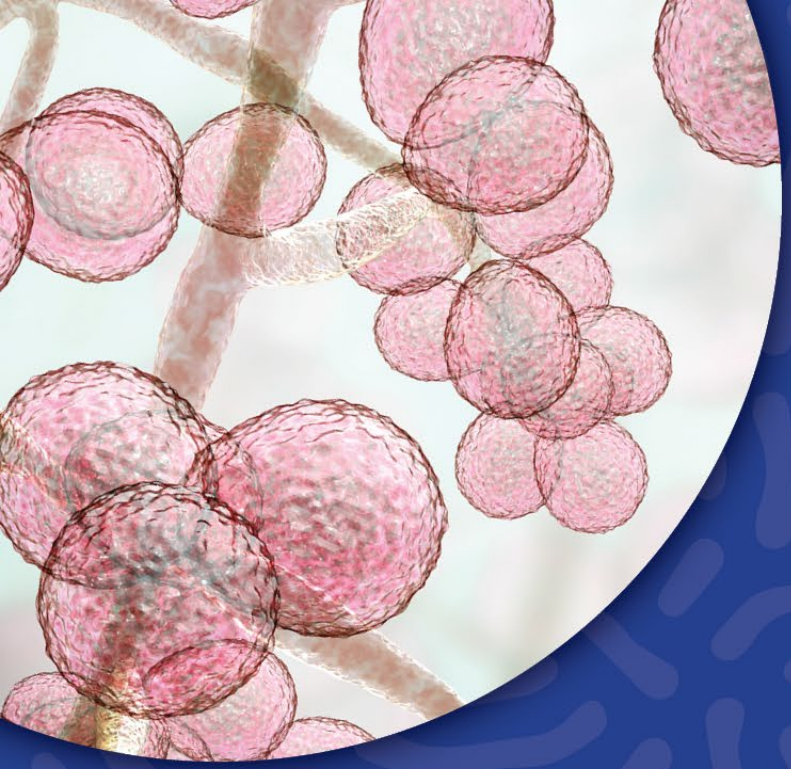
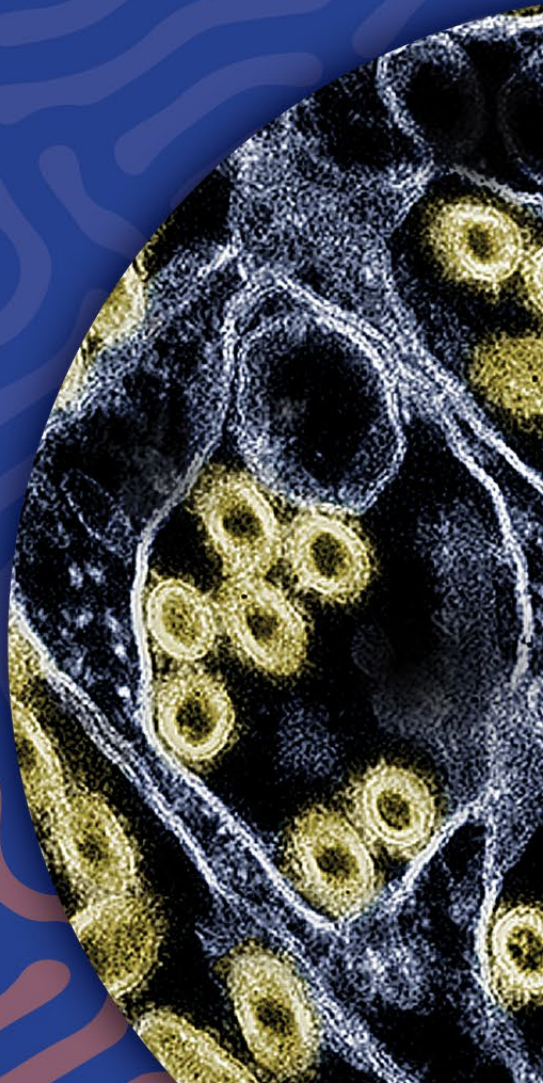


ID LAB CON

APHL

2025

#IDLabCon



#IDLabCon

Optimizing Mycobacteriology Laboratory Operations: Strategies for Supervisor Success

588-509-25 • 5.25 contact hours for this session

Conflict of Interest Statement

No Potential Conflicts of Interest

Speaker: Emily Craig, MS, M(ASCP), Virginia Division of Consolidated Laboratory Services

I have NO conflicts of interest related to this presentation to disclose.

Speaker: Erin Estes, MBA, MLS(ASCP), Association of Public Health Laboratories

I have NO conflicts of interest related to this presentation to disclose.

Speaker: Drew Francis, M(ASCP)CM, Arizona State Public Health Laboratory

I have NO conflicts of interest related to this presentation to disclose.

Speaker: Julie Higashi, MD, PhD, Los Angeles County Public Health Laboratory

I have NO conflicts of interest related to this presentation to disclose.

Conflict of Interest Statement

No Potential Conflicts of Interest

Speaker: Hector Rivas, BS, MPH, PHM, Los Angeles County Public Health Laboratory

I have NO conflicts of interest related to this presentation to disclose.

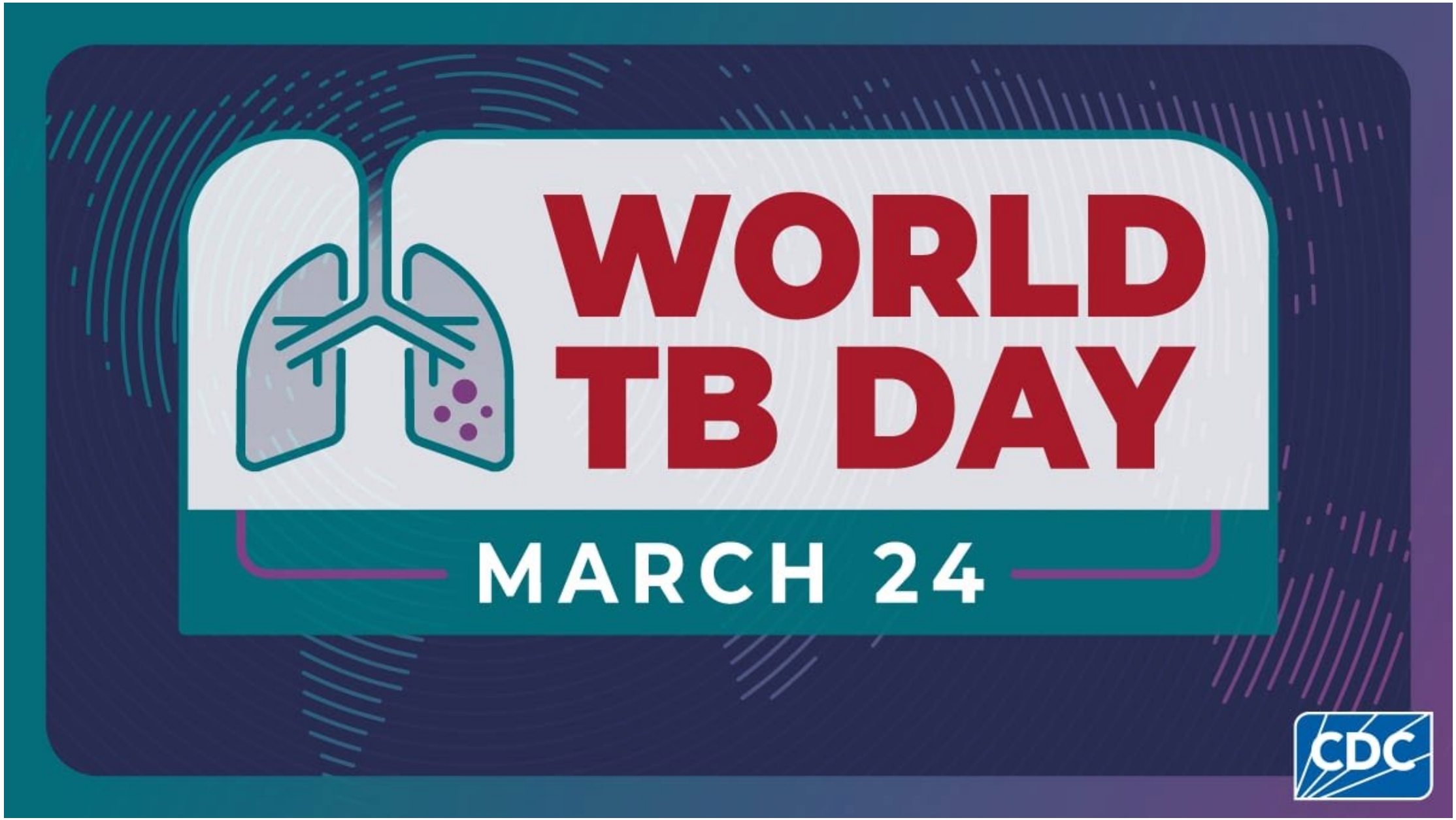
Speaker: Marie-Claire Rowlinson, PhD, D(ABMM), Florida Bureau of Public Health Laboratories

I have NO conflicts of interest related to this presentation to disclose.

Speaker: Angie Schooley, MT(ASCP), Michigan Department of Health and Human Services

I have NO conflicts of interest related to this presentation to disclose.

Table of Contents


WORLD
TB DAY
MARCH 24
CDC


HELLO
MY NAME IS

- Angie Schooley
- Marie-Claire Rowlinson
- Jessica Gentry
- Emily Craig
- Hector Rivas
- Drew Francis
- Julie Higashi
- Monica Youngblood
- Stephanie Swint
- Stephanie Johnston
- Angela Starks
- TB Subcommittee


CDC
U.S. CENTERS FOR DISEASE CONTROL AND PREVENTION
APHL
ASSOCIATION OF PUBLIC HEALTH LABORATORIES™

National Center for HIV, Viral Hepatitis, STD and TB Prevention (NCHHSTP)

Mission: Prevent infections, morbidity, mortality, health inequities, and stigma associated with HIV, viral hepatitis, STIs, and tuberculosis in the United States.

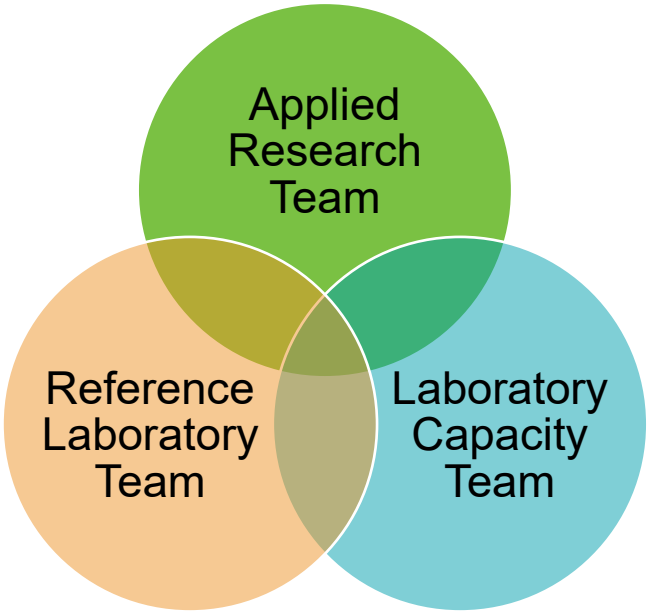
Division of Tuberculosis Elimination (DTBE)

Mission: DTBE promotes health and quality of life by preventing, controlling and eventually eliminating tuberculosis from the United States.

Laboratory Branch

- Dedicated to the elimination of tuberculosis by conducting applied research on *Mycobacterium tuberculosis*, by providing laboratory services to support tuberculosis control and surveillance, and by directly supporting U.S. public health laboratories to increase their capacity to combat tuberculosis.

Laboratory Branch



Systems Group

Quality Manager

Safety Officer

Angela Starks, PhD
Chief

Applied Research Team

- Improve outbreak detection
- Explore novel strategies
- Identify and characterize drug resistance mechanisms

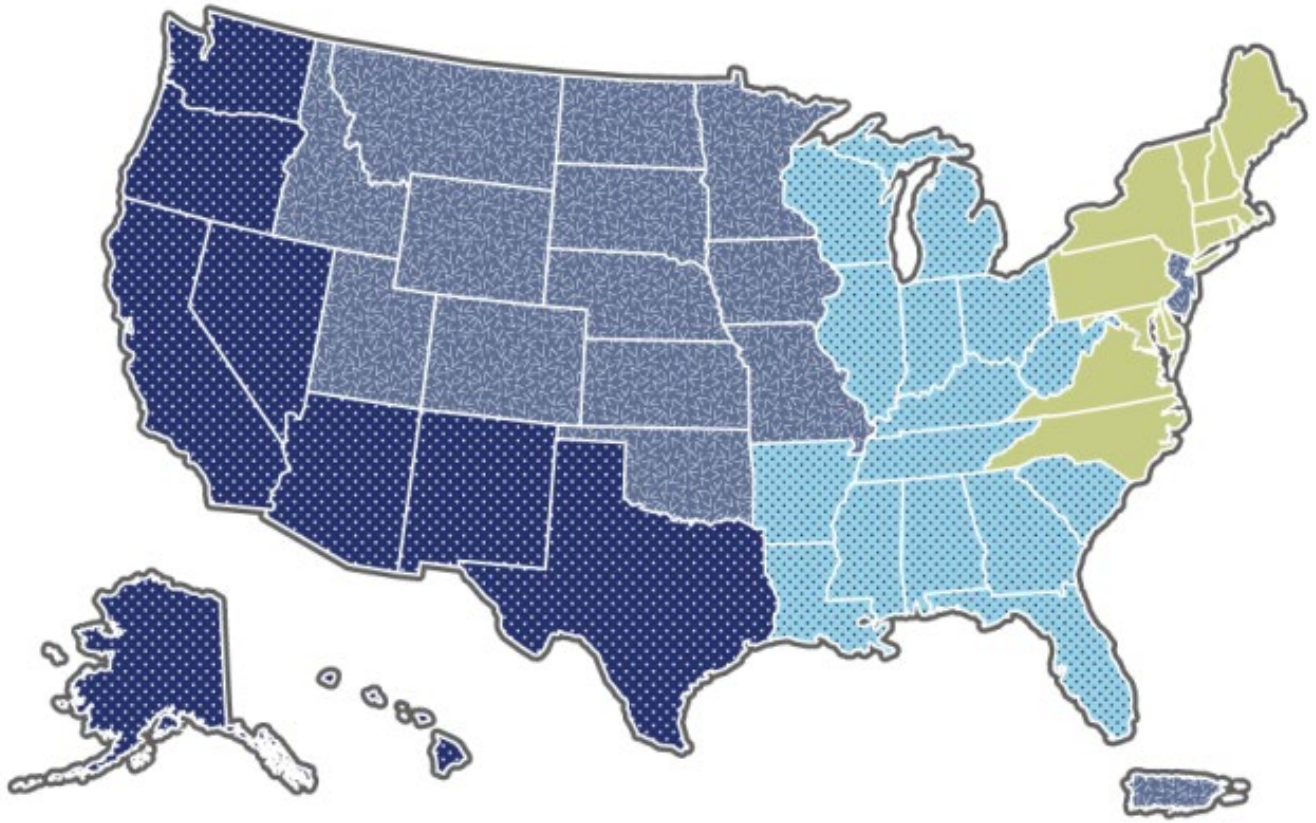
Laboratory Capacity Team

- Oversight of Cooperative Agreements
- Education and Training

Reference Laboratory Team

- Clinical Testing
- Technical Assistance

Laboratory Capacity Team Contact Details



Other Sites:

Stephanie Johnston: Houston, Los Angeles, San Diego, San Francisco, New York City

Stephanie Swint: Philadelphia, District of Columbia

Monica Youngblood: Puerto Rico

Stephanie Johnston
sjohnston@cdc.gov
404-639-5019

Monica Youngblood
myoungblood@cdc.gov
404-718-2079

Cortney Stafford
cstafford@cdc.gov
404-639-3420

Stephanie Swint
sswint@cdc.gov
404-718-7866

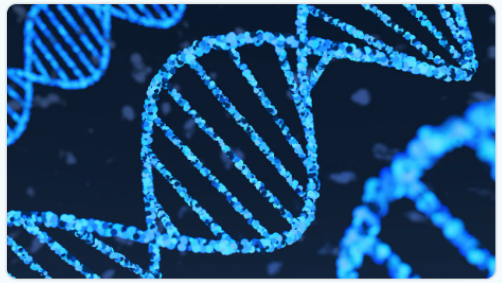
National TB Reference Laboratory

- Drug Susceptibility Testing
- Molecular Detection of Drug Resistance (pre-approval needed)

Molecular Detection of Drug Resistance (MDDR) in Mycobacterium tuberculosis Complex by DNA Sequencing User Guide

WHAT TO KNOW

CDC's Molecular Detection of Drug Resistance (MDDR) service can rapidly identify drug resistance, including multidrug-resistant, *Mycobacterium tuberculosis* complex. This service utilizes DNA sequencing for detection of mutations most frequently associated with resistance to the most effective first and second-line drugs as well as new and repurposed drugs such as bedaquiline, clofazimine, and linezolid.



Other Services

- Model Performance Evaluation Program (MPEP)
- National PHL DST Reference Center for TB

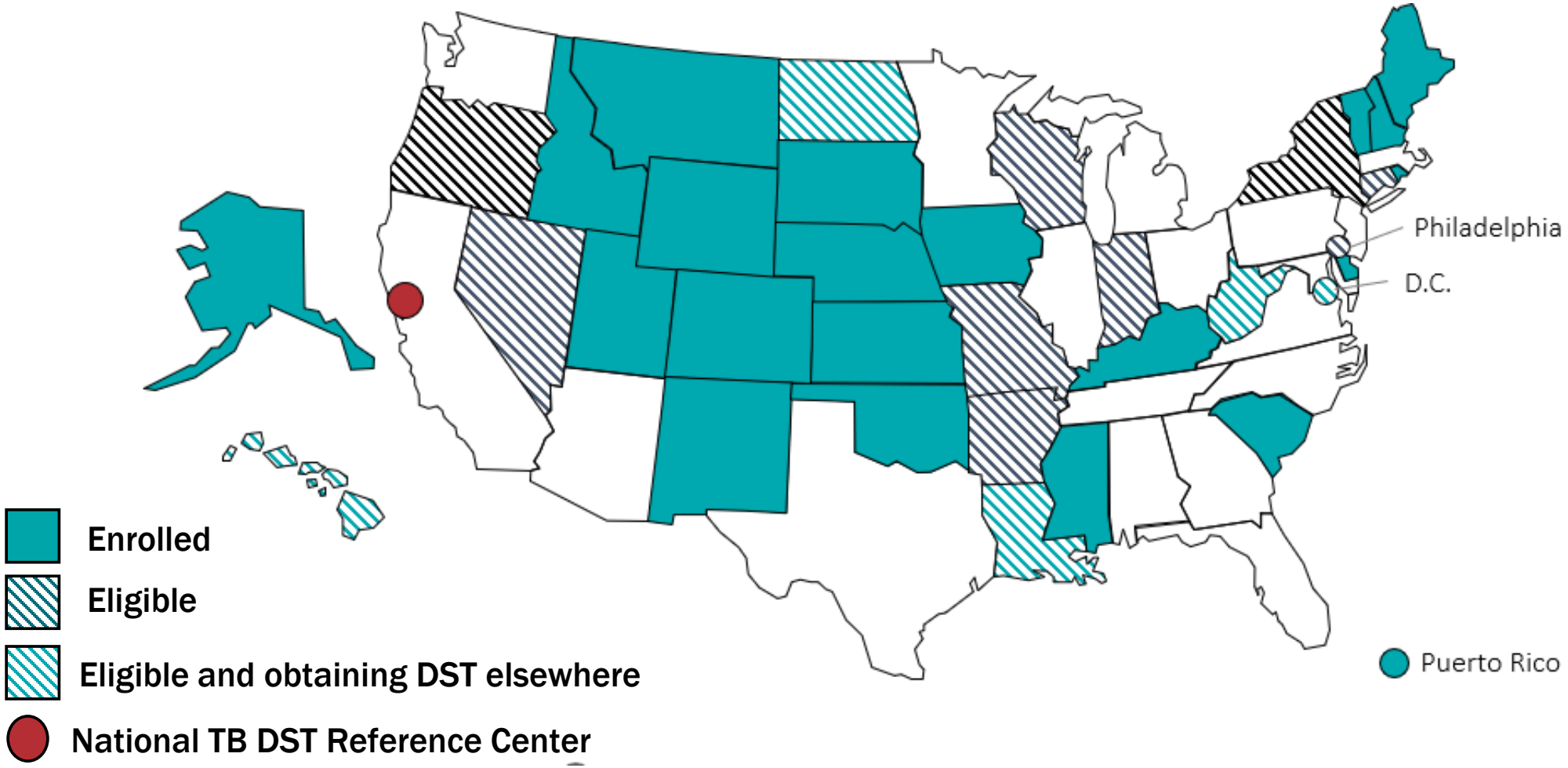
CONTINUITY OF OPERATIONS PLAN TOOLKIT

FOR PUBLIC HEALTH MYCOBACTERIOLOGY LABORATORIES

National PHL DST Reference Center for MTB

- Project objectives
  - To identify drug resistant TB as quickly and reliably as possible
  - To provide PHLs performing TB DST on fewer than 50 TB isolates per year access to high-quality drug susceptibility testing in a shared service model
- Methods
  - WGS: used for molecular DST of isolates and for surveillance
  - tNGS: used for molecular DST of specimens
  - Phenotypic testing using MGIT
    - First-line: rifampin, isoniazid, ethambutol, moxifloxacin
    - Second-line: ethionamide, capreomycin, amikacin, kanamycin, rifabutin, moxifloxacin

National PHL DST Reference Center for TB



2023-2024	FL-DST	SL-DST	PSQ	WGS
Specimens tested	414	67	168	149

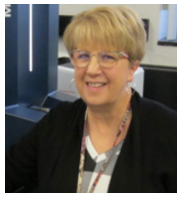
Other APHL and CDC TB Collaborations

- APHL TB Subcommittee
- National Conference on Laboratory Aspects of Tuberculosis
- Diagnostic Principles and Procedures Workshop
- Online Training Modules
- Request for Funding Proposals

TB Subcommittee

Members

Kimberlee Musser (Chair)
Wadsworth Center, NYS DOH



Angie Marie Schooley
Michigan Department of Health and Human Services

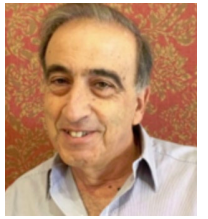
Marie-Claire Rowlinson
Florida Department of Health, Bureau of Public Health Laboratories

Toby Bennett
Rhode Island Department of Health

Zenda Berrada
California Department of Public Health

NEW: Ben Alpers
Texas Department of State Health Services

NTCA Liaison



John Bernardo
Massachusetts DPH/ BU School of Medicine

Clinical Liaison

New: Nancy Wengenack
Mayo Clinic

CDC Liaisons

Michele Owen
NCHHSTP

Angela Starks
Division of Tuberculosis Elimination

Stephanie Johnston
Division of Tuberculosis Elimination

APHL Staff Liaisons

Sarah Buss
Erin Estes

TB Subcommittee Guidance: NGS Technologies

TB: THE NEXT GENERATION (SEQUENCING)

Applications for NGS Technologies in Tuberculosis Testing

Next generation sequencing (NGS) is a large-scale, high-throughput DNA sequencing technology that has powerful applications in the field of tuberculosis (TB) testing. This laboratory method can generate an enormous amount of data in one test—within hours to days—that can be used for a variety of purposes including:

- Comprehensive detection of mutations that predict drug resistance and susceptibility
- Identification of specific strains or species of an organism, such as *Mycobacterium tuberculosis* complex (MTBC)
- Comparison to historic data/genotyping
- High quality analysis to determine relatedness to other cases

During NGS, genomic DNA is sequenced many times and assembled by sophisticated software to form a consensus sequence.

NGS technology is used to perform:

Targeted NGS (tNGS), or amplicon-based NGS, to sequence specific parts of the genome, such as a single gene/loci or group of genes.

Whole genome sequencing (WGS) to sequence the entire genome of an organism.

Metagenomic sequencing to sequence all nucleic acid in a sample, including multiple organisms.

Choosing Between tNGS, WGS and Metagenomic Sequencing

Features	tNGS	WGS	Metagenomic Sequencing
Application	Useful for diagnostic purposes; can be used on primary specimens	Useful for diagnostics and epidemiological/surveillance purposes	Useful for diagnosis of polymicrobial infections or when diagnosis is difficult
Turnaround Time	2–5 days	4–7 days	~5 days
Cost Per Sample *Approximate; cost varies with testing volume.*	$75–200	$50–200	$500
Considerations	• May require less data analysis • Generates data specific to the targets • More challenging assay design and wet lab processes	• Generates large amounts of data, so analysis requires significant computational resources and expertise • Requires large amount of DNA/organism load	• Massively parallel, deep sequencing of all material in a given sample • Complex and computationally intensive genomic data analysis

Resource Requirements & Considerations

Financial	IT Infrastructure	Bioinformatics	Personnel	Instrumentation
Cost per sample, equipment maintenance, contracts, personnel	Data reporting and storage, LIMs	Pipelines, cloud computing	Qualified staff, training and competency	Quality assurance, regular utilization, troubleshooting

APHL Association of Public Health Laboratories
8515 Georgia Avenue, Suite 700, Silver Spring, MD 20910 | www.aphl.org | 240.485.2745 (P) | 240.485.2700 (F)

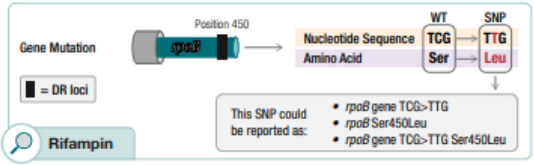
TB: Next Generation Sequencing and Molecular Drug Susceptibility Testing

As laboratories expand their next generation sequencing (NGS) capabilities, it is increasingly possible to use molecular drug susceptibility testing (mDST) to predict *Mycobacterium tuberculosis* (MTB) drug resistance (DR) by identifying key mutations in the MTB genome known to be associated with DR.

This document focuses on resistance associated with changes at the genetic level (i.e., mutations), though other factors can also result in observed resistance, such as intrinsic resistance and expression of efflux pumps.

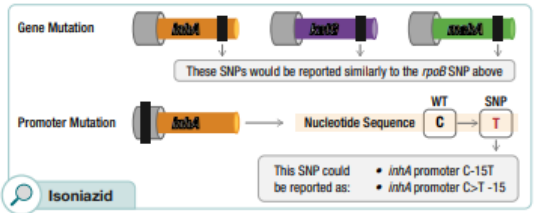
Mutations that confer DR can be simple...

Sometimes resistance to a drug is associated with a single gene or area of a gene.



...or complicated.

Resistance may stem from a mutation within one of several genes or their promoters, making identification more challenging.



Heteroresistance is important to detect.

If heteroresistance is undetected in a patient infected with a mixed population of fluoroquinolone (FQ) susceptible and FQ resistant MTB, FQ treatment may not effectively clear the FQ-resistant subpopulation.

NGS-based methods can be used to detect heteroresistance in MTB.

Drug Resistance Terminology

DST: Drug susceptibility test; there are two categories of tests:

- **Phenotypic DST (pDST):** Growth-based antimicrobial susceptibility (e.g., MGIT, agar proportion)
- **Molecular DST (mDST):** Detection of genetic mutations associated with DR

Discordance: Lack of agreement between laboratory results (e.g., when two pDSTs or a pDST and mDST produce discrepant results)

Heteroresistance: Coexistence of organisms susceptible and resistant to the same MTB drug in a patient

Intrinsic Resistance: Innate ability of a particular species to resist a certain antibiotic or family of antibiotics

Low-level Resistance: Organisms resistant to low-level drug concentrations

Genetic Terminology

Amino Acid: Building blocks of protein

Codon: Three consecutive nucleotides that code for an amino acid

Gene: DNA sequence that encodes specific traits

Locus/Loci: Fixed position on chromosome where a particular gene, genetic marker or mutation is located

Numbering System: Specific location in TB gene or loci where a mutation is located

Promoter: Region within the genome that initiates the expression of a gene; promoters do not code for protein

Indel: Insertion/deletion

Silent Mutation: Change to nucleotide secuence that does not result in a change to the amino acid

SNP: Single nucleotide polymorphism, a variation at a single nucleotide position; the most common type of mutation causing DR in MTB

Variant or Mutation: Alteration in the nucleotide sequence of an organism that may or may not have a phenotypic effect

WT: Wild Type; standard genetic sequence to which mutations are compared (e.g., H37Rv is a commonly used WT reference strain for MTB)

TB Subcommittee White Papers: First Line Drugs

APHL INFECTIOUS DISEASES MAY 2022

Issues in *Mycobacterium tuberculosis* Complex Drug Susceptibility Testing: Rifampin

HIGHLIGHTS

- Rapid and reliable detection of rifampin (RIF) resistance is critical for the diagnosis and treatment of drug resistant and multidrug-resistant tuberculosis (MDR TB).
- Most RIF resistant clinical *Mycobacterium tuberculosis* complex (MTBC) isolates contain mutations in the *rpoB* gene with 96% occurring within the Rifampin Resistance Determining Region (RRDR).
- For the majority of *rpoB* gene mutations, a high correlation with growth-based drug susceptibility testing (DST) is found. Results from molecular assays can be discordant with growth-based testing results in some cases, therefore DNA sequencing should be performed to confirm mutations identified when a non-sequence based molecular assay is utilized.
- A subset of isolates with mutations in the RRDR that are susceptible to RIF using growth-based DST methods (or have only slightly elevated MIC values) are considered to have low-level resistance to RIF.

BACKGROUND

Rifampin (RIF), the most commonly used member of the rifamycin drug class, is the cornerstone of the first-line regimen used for treatment of drug susceptible *Mycobacterium tuberculosis* complex (MTBC) due to its potent bactericidal activity. RIF can kill MTBC rapidly by inhibiting the DNA dependent RNA polymerase β-subunit encoded by the *rpoB* gene. The effectiveness of treatment for tuberculosis depends on many factors including the bacterial strain, its metabolic activity, the bacterial burden, pharmacodynamics of the drugs, the site of infection, and the compliance of the patient. Different drugs in a treatment regimen are believed to act on different populations of bacteria in the lesions. RIF is most effective against the dormant bacteria that have short spurts of metabolic activity or active growth because it can kill within 15-20 minutes unlike other commonly used drugs.[1]

RIF kills MTBC by inhibiting the elongation of messenger RNA, ultimately halting transcription. Mutations in the MTBC *rpoB* gene cause changes in the structure of the β-subunit which can lead to various degrees of sensitivity to RIF.[2–4] The majority of RIF resistant clinical MTBC isolates contain mutations in the *rpoB* gene.

Wild-type strains of MTBC which have not been exposed to RIF are highly susceptible, with minimal inhibitory concentrations (MIC) in the range of 0.125 µg/mL to 0.5 µg/mL in the BACTEC™ MGIT™ 960 system (Becton Dickinson). Resistant strains tend to have much higher MICs, most commonly around 8 µg/mL or as high as 50 µg/mL.[5,6] Therefore, in the majority of cases, a test concentration of 1 µg/mL allows discrimination between the majority of susceptible and resistant strains.[7] However, in March 2021, the World Health Organization (WHO) updated the critical

The World Health Organization (WHO) has published a "Catalogue of mutations in *Mycobacterium tuberculosis* complex and their association with drug resistance" (2021). This is the first document to provide a common, standardized reference for the interpretation of resistance to first and second-line drugs. The catalogue includes 17,000 mutations, their frequency and association with resistance. The mutations are further classified into two tiers. Tier 1 comprises genes considered most likely to contain the resistance mutations. Tier 2 comprises genes that are reasonably likely to contain resistance mutations, with the additional, literature-defined promoter sequences. The WHO document is an important resource with detailed information on numerous mutations associated with drug resistance and may include mutations not specifically addressed in this white paper. We recommend readers to review both documents if they are interested in molecular detection of drug resistance.

APHL MTBC Drug Susceptibility Testing: Rifampin | 1

APHL INFECTIOUS DISEASES APRIL 2022

Issues in *Mycobacterium tuberculosis* Complex Drug Susceptibility Testing: Pyrazinamide

HIGHLIGHTS

- The primary mechanism of PZA resistance is due to mutations in the *pncA* gene and promoter region.
- PZA growth-based DST using automated broth systems have issues with false resistance and reproducibility.
- Some molecular methods may not detect subpopulations of PZA resistant isolates, but WGS could provide this capability.
- Growth-based DST and *pncA* gene and promoter region sequencing together may provide the most reliable prediction of PZA susceptibility or resistance.

BACKGROUND

Pyrazinamide (PZA) is a critical component of first-line drug combination therapy for *Mycobacterium tuberculosis* complex (MTBC) including both susceptible and multidrug-resistant tuberculosis (MDR TB). Inclusion of PZA has shortened the previous 9–12 month chemotherapy regimen to six months.[1–4] PZA has a sterilizing effect due to its significant activity against non-replicating "persister" organisms or semi-dormant, slowly replicating bacilli at acid pH conditions (pH 5.5), killing bacilli that are not eliminated by other TB drugs, such as those found in acidic regions of acute inflammation.[5–10]

Pyrazinamide is a pro-drug which requires conversion to its active form of pyrazinoic acid (POA) by MTBC. Pyrazinamide enters the mycobacterial cell by passive diffusion and is subsequently transformed in the cytoplasm by the protein PncA, a non-essential intracellular nicotinamidase that has pyrazinamidase (PZase) activity, encoded by the *pncA* gene. POA accumulates in the cytoplasm and is actively expelled by a putative efflux pump. Outside of the bacilli, POA is protonated and re-enters the organism where the release of the protons occurs, resulting in an increasingly acidic cytoplasm and the accumulation of POA. This disrupts membrane permeability and transport, resulting in cellular damage.[10–12] While this mechanism of action has been the prevailing theory, others have proposed that POA may not be responsible for acidification of the cytoplasm yet may inhibit target(s) only essential to the bacteria under stress conditions (e.g., hypoxia).[13–16] More recently, Gopal et al. found that POA bound to aspartate decarboxylase, PanD, in the bacterial cell, triggering its degradation and blocking biosynthesis of the essential Coenzyme A.[17] [See Areas of Ongoing Research]

The primary mechanism of PZA resistance is due to mutations in the *pncA* gene and promoter region, identified by several studies in 70-97% of phenotypically PZA resistant isolates.[13,18,19] Mutations in *pncA* and the promoter region affect the catalytic sites of the PZase enzyme and Fe2+ ion

The World Health Organization (WHO) has published a "Catalogue of mutations in *Mycobacterium tuberculosis* complex and their association with drug resistance" (2021). This is the first document to provide a common, standardized reference for the interpretation of resistance to first and second-line drugs. The catalogue includes 17,000 mutations, their frequency and association with resistance. The mutations are further classified into two tiers. Tier 1 comprises genes considered most likely to contain the resistance mutations. Tier 2 comprises genes that are reasonably likely to contain resistance mutations, with the additional, literature-defined promoter sequences. The WHO document is an important resource with detailed information on numerous mutations associated with drug resistance and may include mutations not specifically addressed in this white paper. We recommend readers to review both documents if they are interested in molecular detection of drug resistance.

APHL MTBC Drug Susceptibility Testing: Pyrazinamide | 1

APHL INFECTIOUS DISEASES APRIL 2022

Issues in *Mycobacterium tuberculosis* Complex Drug Susceptibility Testing: Ethambutol

HIGHLIGHTS

- Due to inherent difficulties with growth-based DST for detection of EMB resistance, rapid molecular methods based on the detection of genetic mutations may prove more valuable for determining EMB resistance.[5]
- All EMB monoresistant isolates should be reviewed since EMB monoresistance is rare and is most commonly associated with resistance to INH (up to 96.6% of isolates).[27]
- Mutations at codon 306 of *embB* are the most commonly detected point mutations and confer resistance in 50–70% EMB resistant TB isolates.

BACKGROUND

Ethambutol (EMB) is an antituberculosis drug used as part of the first-line treatment against *Mycobacterium tuberculosis* complex (MTBC) to minimize development of drug resistance to companion first-line drugs, particularly isoniazid (INH).[1,2] EMB is bacteriostatic against actively replicating bacteria but can be bactericidal when serum concentrations are over 10 µg/mL.[3] Research indicates EMB penetrates and accumulates in macrophages—within inflammation sites of human lungs—with measured EMB concentrations up to ten times higher in comparison to those seen in serum or plasma levels.[4]

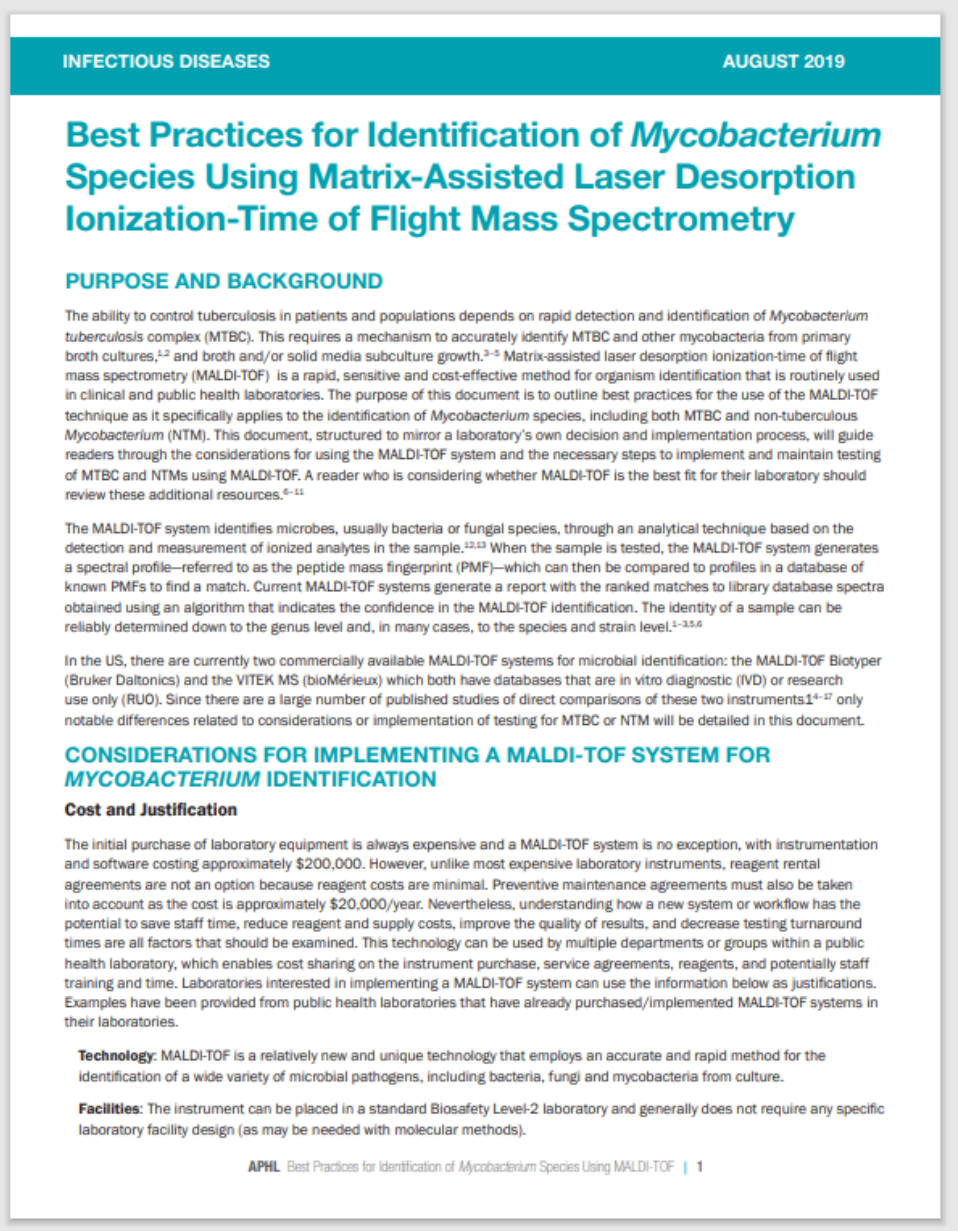
EMB acts through disruption of MTBC cell wall synthesis, targeting and inhibiting the function of the arabinosyl transferases encoded by the *embCAB* operon, comprised of three adjacent genes (*embC*, *embA*, and *embB*) responsible for biosynthesis of the cell wall components arabinogalactan and lipoarabinomannan.[5–7] EMB mainly interrupts polymerization steps in the biosynthesis of the arabinan component of cell wall arabinogalactan.[6,7] This disruption may lead to increased permeability of the cell wall.[1,8]

EMB resistant strains tend to have a minimum inhibitory concentration (MIC) ranging from 7.5 µg/mL to 40 µg/mL.[9–11] The test concentration of 5 µg/mL (using the Mycobacteria Growth Indicator Tube, MGIT) allows discrimination between the majority of susceptible and resistant strains. In addition to traditional growth-based drug susceptibility testing (DST), molecular detection of DNA mutations can provide valuable information to predict drug resistance. While the mechanism of resistance of MTBC to EMB is not well-defined nor genomic targets well-documented,[12] many researchers have focused investigations on the role of the *embCAB* operon, specifically the *embB* gene. Multiple investigators have found that mutations at codon 306 of *embB* are the most commonly detected point mutations, with 50–70% of isolates containing the mutation conferring EMB resistance.[5,9,11,13–16] However, additional mutations in *embB*, as well as mutations in *embC* and *embA*, have also been

The World Health Organization (WHO) has published a "Catalogue of mutations in *Mycobacterium tuberculosis* complex and their association with drug resistance" (2021). This is the first document to provide a common, standardized reference for the interpretation of resistance to first and second-line drugs. The catalogue includes 17,000 mutations, their frequency and association with resistance. The mutations are further classified into two tiers. Tier 1 comprises genes considered most likely to contain the resistance mutations. Tier 2 comprises genes that are reasonably likely to contain resistance mutations, with the additional, literature-defined promoter sequences. The WHO document is an important resource with detailed information on numerous mutations associated with drug resistance and may include mutations not specifically addressed in this white paper. We recommend readers to review both documents if they are interested in molecular detection of drug resistance.

APHL MTBC Drug Susceptibility Testing: Ethambutol | 1

TB Subcommittee Guidance: MALDI-TOF MS



INFECTIOUS DISEASES AUGUST 2019

Best Practices for Identification of *Mycobacterium* Species Using Matrix-Assisted Laser Desorption Ionization-Time of Flight Mass Spectrometry

PURPOSE AND BACKGROUND

The ability to control tuberculosis in patients and populations depends on rapid detection and identification of *Mycobacterium tuberculosis* complex (MTBC). This requires a mechanism to accurately identify MTBC and other mycobacteria from primary broth cultures,[1,2] and broth and/or solid media subculture growth.[3–5] Matrix-assisted laser desorption ionization-time of flight mass spectrometry (MALDI-TOF) is a rapid, sensitive and cost-effective method for organism identification that is routinely used in clinical and public health laboratories. The purpose of this document is to outline best practices for the use of the MALDI-TOF technique as it specifically applies to the identification of *Mycobacterium* species, including both MTBC and non-tuberculous *Mycobacterium* (NTM). This document, structured to mirror a laboratory's own decision and implementation process, will guide readers through the considerations for using the MALDI-TOF system and the necessary steps to implement and maintain testing of MTBC and NTMs using MALDI-TOF. A reader who is considering whether MALDI-TOF is the best fit for their laboratory should review these additional resources.[6–11]

The MALDI-TOF system identifies microbes, usually bacteria or fungal species, through an analytical technique based on the detection and measurement of ionized analytes in the sample.[12,13] When the sample is tested, the MALDI-TOF system generates a spectral profile—referred to as the peptide mass fingerprint (PMF)—which can then be compared to profiles in a database of known PMFs to find a match. Current MALDI-TOF systems generate a report with the ranked matches to library database spectra obtained using an algorithm that indicates the confidence in the MALDI-TOF identification. The identity of a sample can be reliably determined down to the genus level and, in many cases, to the species and strain level.[1–3,5,6]

In the US, there are currently two commercially available MALDI-TOF systems for microbial identification: the MALDI-TOF Biotyper (Bruker Daltonics) and the VITEK MS (bioMérieux) which both have databases that are in vitro diagnostic (IVD) or research use only (RUO). Since there are a large number of published studies of direct comparisons of these two instruments[14–17] only notable differences related to considerations or implementation of testing for MTBC or NTM will be detailed in this document.

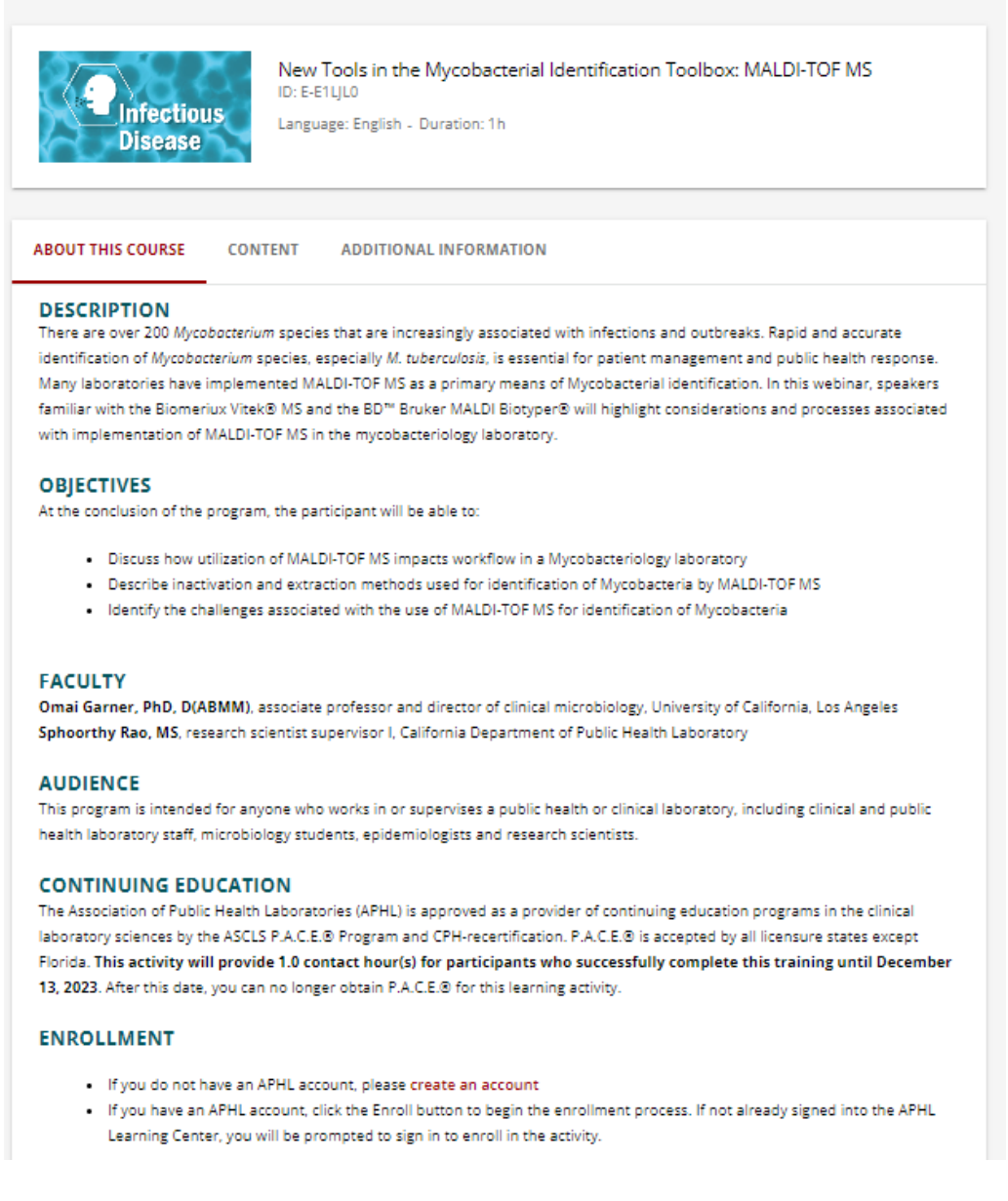
CONSIDERATIONS FOR IMPLEMENTING A MALDI-TOF SYSTEM FOR *MYCOBACTERIUM* IDENTIFICATION

Cost and Justification

The initial purchase of laboratory equipment is always expensive and a MALDI-TOF system is no exception, with instrumentation and software costing approximately $200,000. However, unlike most expensive laboratory instruments, reagent rental agreements are not an option because reagent costs are minimal. Preventive maintenance agreements must also be taken into account as the cost is approximately $20,000/year. Nevertheless, understanding how a new system or workflow has the potential to save staff time, reduce reagent and supply costs, improve the quality of results, and decrease testing turnaround times are all factors that should be examined. This technology can be used by multiple departments or groups within a public health laboratory, which enables cost sharing on the instrument purchase, service agreements, reagents, and potentially staff training and time. Laboratories interested in implementing a MALDI-TOF system can use the information below as justifications. Examples have been provided from public health laboratories that have already purchased/implemented MALDI-TOF systems in their laboratories.

Technology: MALDI-TOF is a relatively new and unique technology that employs an accurate and rapid method for the identification of a wide variety of microbial pathogens, including bacteria, fungi and mycobacteria from culture.

Facilities: The instrument can be placed in a standard Biosafety Level-2 laboratory and generally does not require any specific laboratory facility design (as may be needed with molecular methods).



Infectious Disease

New Tools in the Mycobacterial Identification Toolbox: MALDI-TOF MS
ID: E-E1LJL0

Language: English - Duration: 1h

ABOUT THIS COURSE CONTENT ADDITIONAL INFORMATION

DESCRIPTION

There are over 200 *Mycobacterium* species that are increasingly associated with infections and outbreaks. Rapid and accurate identification of *Mycobacterium* species, especially *M. tuberculosis*, is essential for patient management and public health response. Many laboratories have implemented MALDI-TOF MS as a primary means of Mycobacterial identification. In this webinar, speakers familiar with the Biomeriux Vitek® MS and the BD™ Bruker MALDI Biotyper® will highlight considerations and processes associated with implementation of MALDI-TOF MS in the mycobacteriology laboratory.

OBJECTIVES

At the conclusion of the program, the participant will be able to:

- Discuss how utilization of MALDI-TOF MS impacts workflow in a Mycobacteriology laboratory
- Describe inactivation and extraction methods used for identification of Mycobacteria by MALDI-TOF MS
- Identify the challenges associated with the use of MALDI-TOF MS for identification of Mycobacteria

FACULTY

Omai Garner, PhD, D(ABMM), associate professor and director of clinical microbiology, University of California, Los Angeles
Sphoorthy Rao, MS, research scientist supervisor I, California Department of Public Health Laboratory

AUDIENCE

This program is intended for anyone who works in or supervises a public health or clinical laboratory, including clinical and public health laboratory staff, microbiology students, epidemiologists and research scientists.

CONTINUING EDUCATION

The Association of Public Health Laboratories (APHL) is approved as a provider of continuing education programs in the clinical laboratory sciences by the ASCLS P.A.C.E.® Program and CPH-recertification. P.A.C.E.® is accepted by all licensure states except Florida. **This activity will provide 1.0 contact hour(s) for participants who successfully complete this training until December 13, 2023**. After this date, you can no longer obtain P.A.C.E.® for this learning activity.

ENROLLMENT

- If you do not have an APHL account, please create an account
- If you have an APHL account, click the Enroll button to begin the enrollment process. If not already signed into the APHL Learning Center, you will be prompted to sign in to enroll in the activity.

National Conference on Laboratory Aspects of Tuberculosis

- 2-day conference dedicated to laboratory testing for tuberculosis
- Held every other year, in co-located with the NTCA's National TB Conference
- Data from 2023
  - 8 plenary sessions
  - 3 interactive sessions
  - 121 Attendees
  - 17 Sponsors/Exhibitors
  - 17 Lab focused posters
  - 11.25 contact hours (P.A.C.E.)

Diagnostic Principles and Procedures Workshop

- Annual APHL-DTBE initiative Pre-COVID
- Reframed / reinitiated in 2023 and held at the Alabama Bureau of Clinical Laboratories
  - December 4-7 2023: 19 accepted applicants
  - July 22-25, 2024: 17 accepted applicants
- 4-day training with both wet lab and didactic components
  - MALDI-TOF MS focus for wet lab (previously had been real-time PCR)

Training Modules in the APHL Learning Center

Essentials for the Mycobacteriology Laboratory: Promoting Quality Practices

- TB1 Overview of Tuberculosis
- TB2 Laboratory Safety: Work Practices for Mycobacterium tuberculosis
- TB3 Specimen Collection, Transport, Handling, and Processing
- TB4 AFB Smear Microscopy
- TB5 Overview of Mycobacterial Culture, Identification, and Drug Susceptibility Testing
- TB6 Mycobacterial Culture
- TB7 Identification of Mycobacteria
- TB8 Drug Susceptibility Testing for MTB Complex
- TB9 Molecular Biology 101
- TB10 Molecular Detection and Identification of Mycobacteria
- **TB11 Molecular Detection of Drug Resistance**
- TB12 Landscape and Language of Molecular Diagnostics for TB Drug Resistance
- TB13 Mycobacteriology False-Positive Case Studies

Training Modules in the APHL Learning Center

Understanding Tuberculosis (TB) Laboratory Testing for Public Health Nurses

Free Training Course

The Association of Public Health Laboratories (APHL) is excited to announce a newly developed course, Understanding Tuberculosis Laboratory Testing for Public Health Nurses, now available on our website.

This course will help participants better understand TB laboratory testing workflow, testing methods, and associated results. Being familiar with this information will aid effective and timely communication with the patient, clinician, and testing laboratory.

Preconference Agenda

- Quality Assurance and Quality Control for the Mycobacteriology Laboratory
  - Presentation
  - Interactive Discussion
- Workforce Retention for Laboratories
- Mycobacteriology Testing Considerations and Best Practices
  - Presentation
  - Interactive Discussion
- Partner Relationships (Laboratory, TB Program and Clinicians)


QUESTIONS

Algorithms Activity Groups

Angie Schooley
(Michigan)

Kristin Bauer
(Alabama)

Stephanie Kleinpeter
(Louisiana)

Emily Yang
(Santa Barbara)

Sidhartha Hazari
(San Joaquin)

Marie-Claire Rowlinson
(Florida)

Varvara Kozyreva
(California)

Alicia Terrenzio
(Washington State)

Tania Chiem
(Orange County)

Gerardo Rivera Negron
(Puerto Rico)

Emily Craig
(Virginia)

Miguel Loera
(Tennessee)

Lisa Well
(North Dakota)

Kathryn Phumisithikul
(Fairfax County)

Hector Rivas
(Los Angeles)

Drew Francis
(Arizona)

Laura Louison
(Wisconsin)

Annaya Osinuga
(Delaware)

Jared Hoffman
(Ventura County)

Julie Higashi
(Los Angeles)

Jessica Gentry
(Indiana)

Kathleen Milloy
(Virginia)

Claudia Sandoval
(Texas)

Matthew Schimenti
(Florida)

Jean-Jacques Aucoin
(Louisiana)