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ABSTRACT WRITING

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12th June 2026

BHP
BOTSWANA HARVARD HEALTH PARTNERSHIP

A Generous Group of Mentors – ABSTRACT MENTORSHIP PROGRAM

PRESENTATION OUTLINE

01 Communicating your research

02 Summarising your work (Abstract)

03 How to write an abstract

04 Types of Abstracts

05 Structuring your abstract

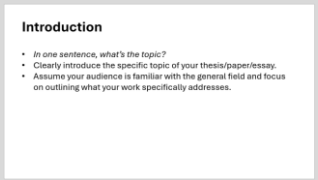
06 Tips about getting accepted into Major Conference

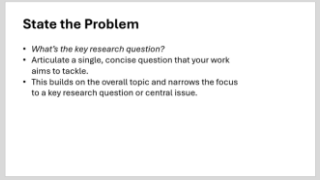
07 Discuss examples (Volunteers)

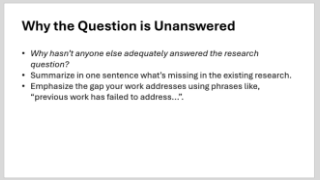
08 Abstract mentorship program

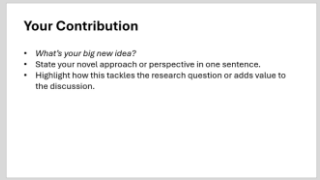
How to write an abstract

How to Write an Abstract



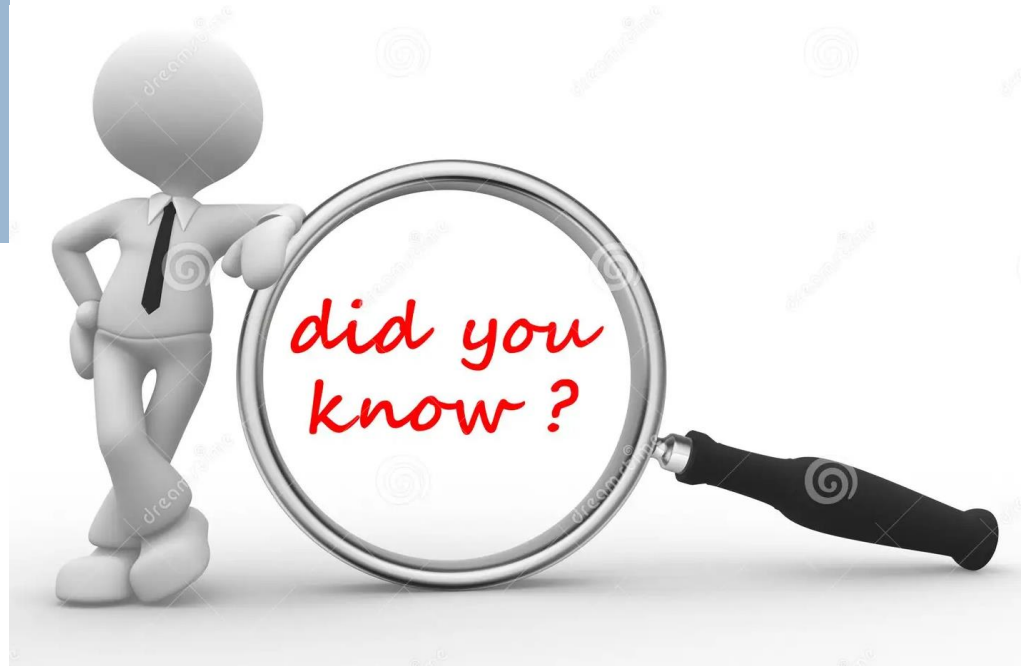






for your Research Paper

How to Write an Abstract for your Research Paper



- Did you know that many people decide whether or not to read your entire research paper based on the **abstract alone**?
- Make sure yours packs a punch!

How to Write an Abstract for your Research Paper

The key trick is to plan your **argument in six sentences**, and then use these to structure the entire thesis/paper/essay. The six sentences are:

1. **Introduction**
   Start strong and set the context
2. **State the Problem**
   Define the research question clearly
3. **Gap in Knowledge**
   Why hasn't this been solved yet?
4. **Your Approach**
   How you tackled the problem
5. **Methodology**
   What research methods did you use?
6. **Impact**
   Highlight significance of your findings

The “science story” inside a strong abstract

BACKGROUND
Why does this matter? What is the gap?

OBJECTIVE
What exactly did we aim to test/describe?

METHODS
Who, where, design, measures and analysis?

RESULTS
Denominator + main outcome + effect estimates.

CONCLUSION
Answer + implication, no new data.

Sentence-level teaching prompts

Opening sentence	Start with a focused public-health or scientific problem, not a generic global statement.
Gap sentence	Signal what remains uncertain, unresolved or under-measured.
Objective	Use “We aimed to…” and make it match the results.
Methods	Include design, setting, participants, outcome and analysis in compressed form.
Results	Lead with the primary result and include numbers.
Conclusion	State what the findings support; avoid “proves” unless the design truly warrants it.

Introduction

- *In one sentence, what's the topic?*
- Clearly introduce the specific topic of your thesis/paper/essay.
- Assume your audience is familiar with the general field and focus on outlining what your work specifically addresses.

State the Problem

- *What's the key research question?*
- Articulate a single, concise question that your work aims to tackle.
- This builds on the overall topic and narrows the focus to a key research question or central issue.

Why the Question is Unanswered

- *Why hasn't anyone else adequately answered the research question?*
- Summarize in one sentence what's missing in the existing research.
- Emphasize the gap your work addresses using phrases like, "previous work has failed to address…".

Your Contribution

- *What's your big new idea?*
- State your novel approach or perspective in one sentence.
- Highlight how this tackles the research question or adds value to the discussion.

Research Approach

- *How did you address the research question?*
- Briefly explain your methods or strategy.
- Focus on the overall approach (e.g., experiments, software development, case studies) without delving into extensive detail.

Impact and Implications

- *What's the key impact of your research?*
- Summarize the broader implications, conclusions, or why your findings matter.
- Address why others should care and how your work contributes to the field or answers the central question.

Purpose of an Abstract

Think of your abstract as a multi-purpose tool:

1. **Snapshot for Readers**
    - **Quick Overview:** Provides the gist of your research in minutes.
    - **Decision-Making Tool:** Helps readers decide if your paper aligns with their interests.
2. **Discoverability Beacon**
    - **Keywords:** Increases chances of appearing in relevant searches.
    - **Indexing:** Enhances visibility within journals and databases.

Crafting a Research Paper Abstract

A well-written abstract should include:

- **Research Problem:** The question or gap your study addresses.
- **Methodology:** Summarize your approach (e.g., experiments, surveys).
- **Main Results:** Key findings or discoveries.
- **Conclusion/Significance:** The impact of your work on the field.

Tips:

- Be specific and self-contained.
- Adhere to word count limits (typically 150–250 words).

Types of abstracts & Structure of abstracts

Types of Abstracts

Descriptive

Purpose: Outlines the main purpose, scope, and focus of your research.
Omissions: Does NOT include specific results, conclusions, or recommendations.
Best For: Papers where the research question and methodology are the most interesting, or when the findings are complex.

Informative

Purpose: Acts as a mini version of your paper, including all major elements.
Includes: Problem statement, methods, key results, conclusions, and potential implications.
Best For: Empirical studies with straightforward results and clear conclusions.

How to Write Each Type

1. **Descriptive Abstract:**
    - State the research problem/question
    - Briefly outline your methods
    - Highlight the type of data examined or the theoretical perspective used
    - Indicate the scope or focus of the research

2. **Informative Abstract**
    - All the elements of the descriptive abstract
    - Succinctly present your key findings
    - Conclude with significance or recommendations

Is an Informative Abstract Suitable for All Types of Research Papers?

No. Here's why:

- **Length:** Informative abstracts can become lengthy. Some publications have stricter word limits.
- **Complexity:** Papers with nuanced results or less conclusive findings might be better served by a descriptive abstract that focuses on the motivation of the research.
- **Audience:** Consider who the likely readers are. A highly specialized audience might appreciate a detailed informative abstract, while a wider readership might find a descriptive abstract less intimidating.

Structuring an Abstract

A typical structure includes:

1. **Problem Statement:** What question or gap does your research address?
2. **Methodology:** Your approach to answering the question.
3. **Results:** The most important findings.
4. **Conclusion/Significance:** Implications for the field.

Tip: Consider leading with groundbreaking results or unique methods to capture interest.

Key Components to Include

- **Purpose:** The "why" of your research. What were you aiming to achieve?
- **Method:** How did you go about answering your question or exploring the issue?
- **Results:** What did you find? (This should be the most substantial part of your abstract)
- **Significance:** Why does your research matter? What does it contribute to the field?

Tips:

- **Adapt Based on Type:** A descriptive abstract might omit specific results, while an informative one must have them.
- **Word Count Matters:** Strive for conciseness. Avoid wordiness or unnecessary jargon.

Key Components to Include

1

Write it Last
It's often easier to write a strong abstract once you've finished your paper.

2

Keywords
Choose keywords that accurately reflect your research focus to increase discoverability in databases.

3

Get Feedback
Ask a peer, instructor, or mentor to read your abstract.

4

Revise, Revise
Don't settle for your first draft. Refine your word choices for clarity and maximum impact within the word limit.

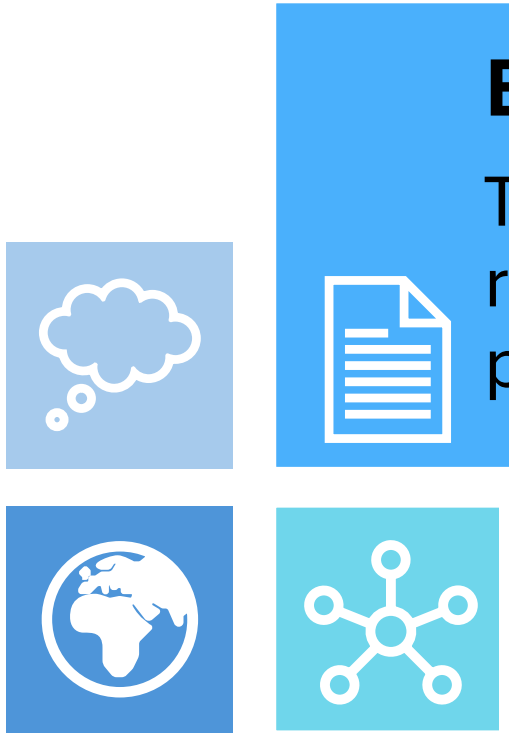
Common Pitfalls to Avoid

Common Pitfalls to Avoid

Too Much Jargon

Keep language accessible to your audience.

Common Pitfalls to Avoid



Excessive Detail

The abstract should entice readers to explore the full paper.

Common Pitfalls to Avoid

Vagueness

Be specific about your research.

Common Pitfalls to Avoid

New Material

Don’t introduce ideas not covered in the paper.

Common weak abstract moves - and how to fix them

1 Too broad

Weak:

“HIV is a major global problem...”

Fix

Use a setting-specific, study-specific opening.

2 Vague methods

Weak:

“We analyzed data...”

Fix

Name the design, population, outcome and model.

3 Results without numbers

Weak:

“There was a significant association...”

Fix

Give n, %, effect estimate and 95% CI when possible.

4 Overclaiming conclusion

Weak:

“This proves the intervention works...”

Fix

Match causal language to design and precision.

Evaluating Abstract Quality

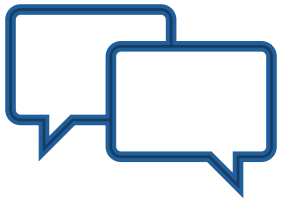
Ask yourself these key questions:

- **Does it accurately reflect the paper?** Anyone reading your abstract should have a clear idea of what your full paper will contain, with no misleading promises.
- **Is it self-contained?** The abstract should make sense on its own, without forcing the reader to consult your paper for clarification.
- **Is it engaging?** Does your opening sentence grab attention? Does the abstract leave the reader curious to learn more?
- **Is it free of errors?** A polished abstract signals attention to detail and professionalism. Proofread carefully!
- **Does it follow guidelines?** Adherence to word limits and any formatting requirements mandated by your instructor or publisher is essential.

How Abstract Quality Impacts Reception

- **Gatekeeper:** Researchers will often decide whether to read your full paper based on the abstract alone. A weak abstract means lost potential readers.
- **Search Optimizer:** Your abstract, with its keywords, plays a crucial role in whether your paper surfaces when people search databases relevant to your field.
- **First Impression:** The abstract sets the reader's expectations for the entire paper. A well-crafted abstract creates a positive anticipation for your work.

Feedback and Revisions



1. Get feedback from people with varying levels of familiarity with your topic.

2. Ask your reviewers: Did the abstract make you want to read the full paper? Were any parts confusing or overly technical?

3. Don't be afraid to significantly revise your abstract based on feedback.

Examples

Example 1: Structured epidemiology abstract

Moyo et al., JAIDS 2025 - concurrent sexual partnerships and incident HIV infection in Botswana

Predictors of Concurrent Sexual Partnerships and Association With Recent HIV Infection in a Large Population-Based Survey in Botswana

Sikhulile Moyo, MSc, MPH, PhD,[a,b,c,d,e] Etienne K. Yankinda, MD, MS,[a,f] Kathleen E. Hurwitz, ScD,[g] Kara Bennett, MSc,[g] Unoda A. Chakalisa, MD,[a] Tendani Gaolathe, MD,[a] Lillian Okui, MD, MPH,[a,f] Jean Leidner, MSc,[h] Coulson Kgathi, BS,[a] Tumalano Sekoto, BSc,[a] Erik van Widenfelt, BS,[a] M. Lendsey Melton, MSc,[b] Refeletswe Lebelonyane, MD,[i] Simani Gaseitsiwe, PhD,[a,b] Mompati O. Mmalane, MD,[a] Vlad Novitsky, MD, PhD,[b,j] Joseph M. Makhema, MBcHB,[a,b] Scott L. Dryden-Peterson, MD,[k] Molly P. Holme, MSc,[b] Max Essex, DVM, PhD,[b] and Shahin Lockman, MD, MSc[a,b,k]

B **Background:** Multiple concurrent sexual partnerships (MCP) may drive new HIV infections. We investigated the association between MCP and recent or incident HIV infection in a cluster-randomized HIV prevention trial that followed a population-based HIV incidence cohort across 30 communities in Botswana.

M **Methods:** We used structured questionnaires to evaluate MCP for prior 12 months, defined as either (1) MCP per UNAIDS definition or (2) concurrent sexual relationship per survey questions. Recent HIV infection was determined using an avidity assay-based algorithm or seroconversion within 2 years, and incident infection was determined through annual HIV testing for up to 3 years. We estimated prevalence ratios (PRs) and 95% confidence intervals (CIs) for MCP predictors using univariate and multivariable Poisson regression with log-link and fixed effects for matched pairs.

R **Results:** We included 11,965 (94.9%) of the 12,610 participants in the Botswana Combination Prevention Project with sexual history data. Among 9363 sexually active persons in prior 12 months, 2770 (29.6%) were engaged in MCP. Factors independently associated with MCP included male sex (aPR = 1.57; 95% CI: 1.45 to 1.71), age < 25 years (aPR = 1.25, 95% CI: 1.01 to 1.56), alcohol consumption ≥ 2 times/week (aPR = 1.38, 95% CI: 1.26 to 1.51), transactional sex (aPR = 1.69, 95% CI: 1.49 to 1.92), having a partner with MCP (aPR = 1.82, 95% CI: 1.65 to 2.02), and intergenerational sex (partners 10 years younger: aPR = 1.16, 95% CI: 1.06 to 1.28 or 10 years older: aPR = 1.32, 95% CI: 1.15 to 1.51). Reporting prior MCP was associated with HIV seroconversion during follow-up (aPR= 1.28, 95% CI: 1.05 to 1.57) but not with prevalent or recent HIV infection at baseline.

C **Conclusions:** MCP was common and associated with incident HIV infection. People reporting MCP may benefit from pre-exposure prophylaxis.

Key Words: concurrent sexual partnerships, recent HIV infection, HIV seroconversion

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From the [a]Botswana Harvard Health Partnership, Gaborone, Botswana; [b]Department of Immunology and Infectious Diseases, Harvard T.H Chan School of Public Health, Boston, MA; [c]Department of Pathology, Division of Virology, Faculty of Medicine and Health Sciences, Stellenbosch University, Cape Town, South Africa; [d]School of Health Systems and Public Health, University of Pretoria, Pretoria, South Africa; [e]School of Allied Health Professions, Faculty of Health Sciences, niversity of Botswana, Gaborone, Botswana [f]Department of Strategic Information, Botswana–University of Maryland School of Medicine Health Initiative, Gaborone, Botswana; [g]Target RWE, Durham, NC; [h]Goodtables Data Consulting, Norman, OK; [i]Botswana-Rutgers Partnership for Health, Gaborone, Botswana; [j]Division of Infectious Diseases, Department of Medicine, Brown University Alpert Medical School, Providence, RI; and [k]Division of Infectious Diseases, Brigham and Women's Hospital, Boston, MA.

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Teaching features to point out

B Problem + rationale: MCP may drive new HIV infections.

M Design + definitions: survey data, recent/incident infection, Poisson regression.

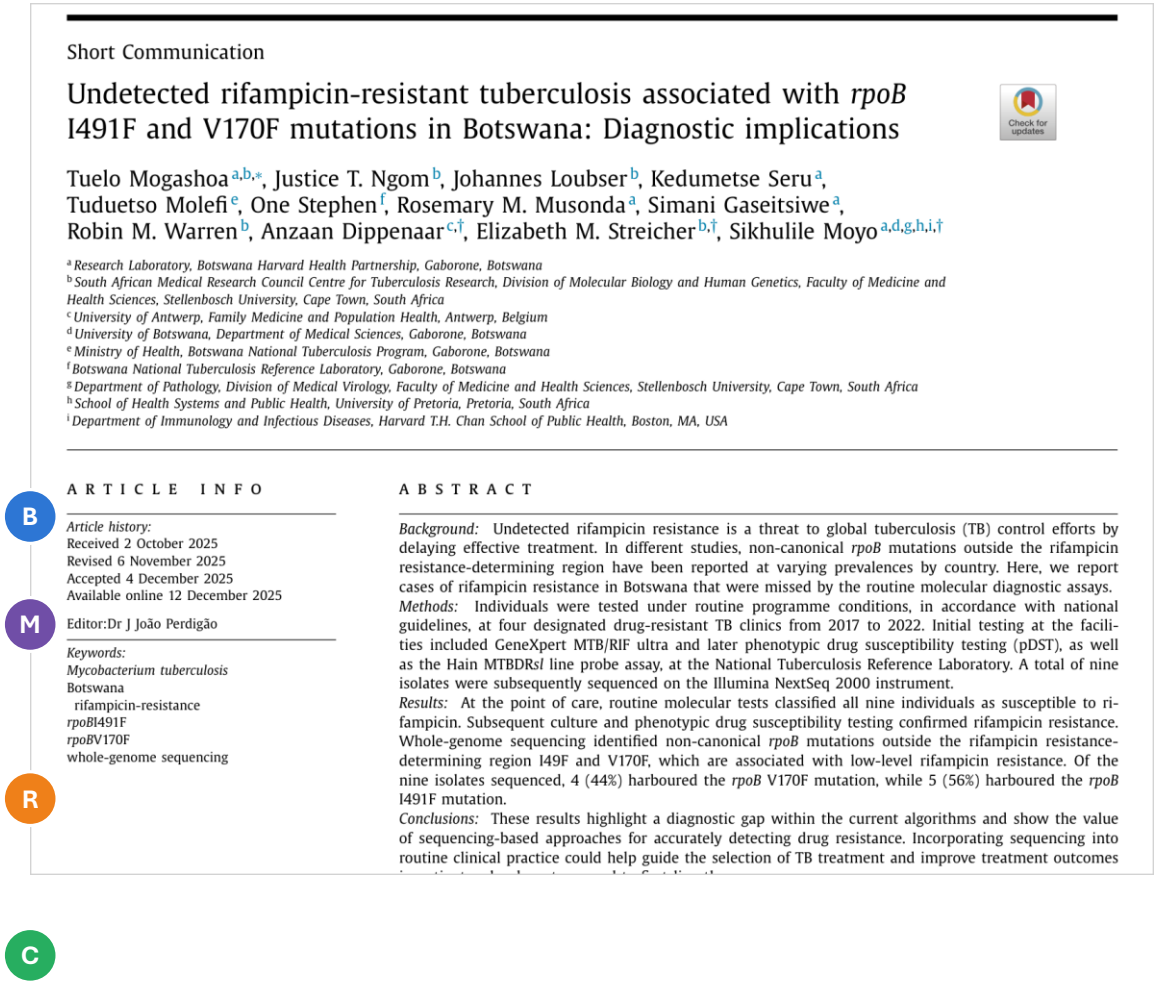
R Results with denominators: n=11,965; MCP prevalence; adjusted PRs + CIs.

C Actionable implication: people reporting MCP may benefit from PrEP.

Question! Which result most directly answers the objective, and which results are supporting predictors?

Example 2: Short communication with diagnostic implications

Tuelo/Mogashoa et al., JGAR 2026 - missed rifampicin resistance in Botswana



Short Communication

Undetected rifampicin-resistant tuberculosis associated with *rpoB* I491F and V170F mutations in Botswana: Diagnostic implications

Tuelo Mogashoa[a,b,*], Justice T. Ngom[b], Johannes Loubser[b], Kedumetse Seru[a], Tuduetso Molefi[e], One Stephen[f], Rosemary M. Musonda[a], Simani Gaseitsiwe[a], Robin M. Warren[b], Anzaan Dippenaar[c,†], Elizabeth M. Streicher[b,†], Sikhulile Moyo[a,d,g,h,i,†]

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*rpoB*I491F
*rpoB*V170F
whole-genome sequencing

ABSTRACT

Background: Undetected rifampicin resistance is a threat to global tuberculosis (TB) control efforts by delaying effective treatment. In different studies, non-canonical *rpoB* mutations outside the rifampicin resistance-determining region have been reported at varying prevalences by country. Here, we report cases of rifampicin resistance in Botswana that were missed by the routine molecular diagnostic assays.
Methods: Individuals were tested under routine programme conditions, in accordance with national guidelines, at four designated drug-resistant TB clinics from 2017 to 2022. Initial testing at the facilities included GeneXpert MTB/RIF ultra and later phenotypic drug susceptibility testing (pDST), as well as the Hain MTBDR*sl* line probe assay, at the National Tuberculosis Reference Laboratory. A total of nine isolates were subsequently sequenced on the Illumina NextSeq 2000 instrument.
Results: At the point of care, routine molecular tests classified all nine individuals as susceptible to rifampicin. Subsequent culture and phenotypic drug susceptibility testing confirmed rifampicin resistance. Whole-genome sequencing identified non-canonical *rpoB* mutations outside the rifampicin resistance-determining region I49F and V170F, which are associated with low-level rifampicin resistance. Of the nine isolates sequenced, 4 (44%) harboured the *rpoB* V170F mutation, while 5 (56%) harboured the *rpoB* I491F mutation.
Conclusions: These results highlight a diagnostic gap within the current algorithms and show the value of sequencing-based approaches for accurately detecting drug resistance. Incorporating sequencing into routine clinical practice could help guide the selection of TB treatment and improve treatment outcomes

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Teaching features to point out

B Opening frames a diagnostic gap and why it matters for TB control.

M Methods specify routine program testing, pDST and sequencing.

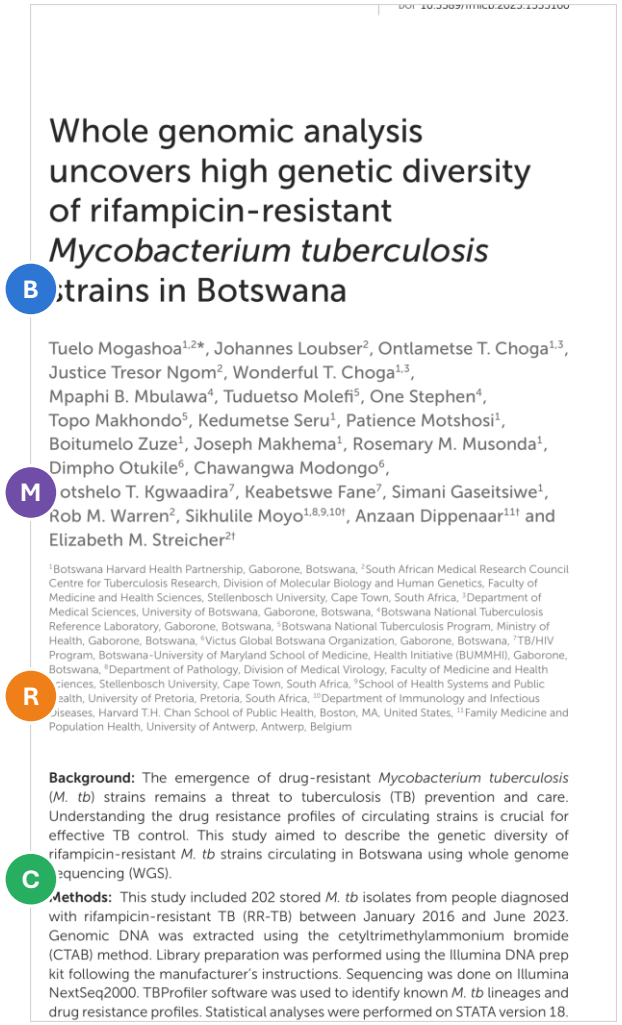
R Results state the contradiction: routine tests susceptible, pDST/WGS resistant.

C Conclusion translates findings into an algorithm/sequencing implication.

How does the abstract move from a small sample to a broader diagnostic implication without overclaiming?

Example 3: Full original research abstract

Mogashoa et al., Frontiers in Microbiology 2025 - genomic diversity of RR-TB strains in Botswana



Whole genomic analysis uncovers high genetic diversity of rifampicin-resistant *Mycobacterium tuberculosis* strains in Botswana

Tuelo Mogashoa[1,2]*, Johannes Loubser[2], Ontlametse T. Choga[1,3], Justice Tresor Ngom[2], Wonderful T. Choga[1,3], Mpaphi B. Mbulawa[4], Tuduetso Molefi[5], One Stephen[4], Topo Makhondo[5], Kedumetse Seru[1], Patience Motshosi[1], Boitumelo Zuze[1], Joseph Makhema[1], Rosemary M. Musonda[1], Dimpho Otukile[6], Chawangwa Modongo[6], Motshelo T. Kgwaadira[7], Keabetswe Fane[7], Simani Gaseitsiwe[1], Rob M. Warren[2], Sikhulile Moyo[1,8,9,10†], Anzaan Dippenaar[11†] and Elizabeth M. Streicher[2†]

[1]Botswana Harvard Health Partnership, Gaborone, Botswana, [2]South African Medical Research Council Centre for Tuberculosis Research, Division of Molecular Biology and Human Genetics, Faculty of Medicine and Health Sciences, Stellenbosch University, Cape Town, South Africa, [3]Department of Medical Sciences, University of Botswana, Gaborone, Botswana, [4]Botswana National Tuberculosis Reference Laboratory, Gaborone, Botswana, [5]Botswana National Tuberculosis Program, Ministry of Health, Gaborone, Botswana, [6]Victus Global Botswana Organization, Gaborone, Botswana, [7]TB/HIV Program, Botswana-University of Maryland School of Medicine, Health Initiative (BUMMHI), Gaborone, Botswana, [8]Department of Pathology, Division of Medical Virology, Faculty of Medicine and Health Sciences, Stellenbosch University, Cape Town, South Africa, [9]School of Health Systems and Public Health, University of Pretoria, Pretoria, South Africa, [10]Department of Immunology and Infectious Diseases, Harvard T.H. Chan School of Public Health, Boston, MA, United States, [11]Family Medicine and Population Health, University of Antwerp, Antwerp, Belgium

Background: The emergence of drug-resistant *Mycobacterium tuberculosis* (*M. tb*) strains remains a threat to tuberculosis (TB) prevention and care. Understanding the drug resistance profiles of circulating strains is crucial for effective TB control. This study aimed to describe the genetic diversity of rifampicin-resistant *M. tb* strains circulating in Botswana using whole genome sequencing (WGS).

Methods: This study included 202 stored *M. tb* isolates from people diagnosed with rifampicin-resistant TB (RR-TB) between January 2016 and June 2023. Genomic DNA was extracted using the cetyltrimethylammonium bromide (CTAB) method. Library preparation was performed using the Illumina DNA prep kit following the manufacturer's instructions. Sequencing was done on Illumina NextSeq2000. TBProfiler software was used to identify known *M. tb* lineages and drug resistance profiles. Statistical analyses were performed on STATA version 18.

Teaching features to point out

B Problem and objective: drug-resistant M. tuberculosis; describe genetic diversity.

M Methods compressed well: isolates, dates, DNA extraction, sequencing, TBProfiler, STATA.

R Dense numeric results: resistance categories, lineages and key mutations.

C Conclusion repeats only the core interpretation and setting value.

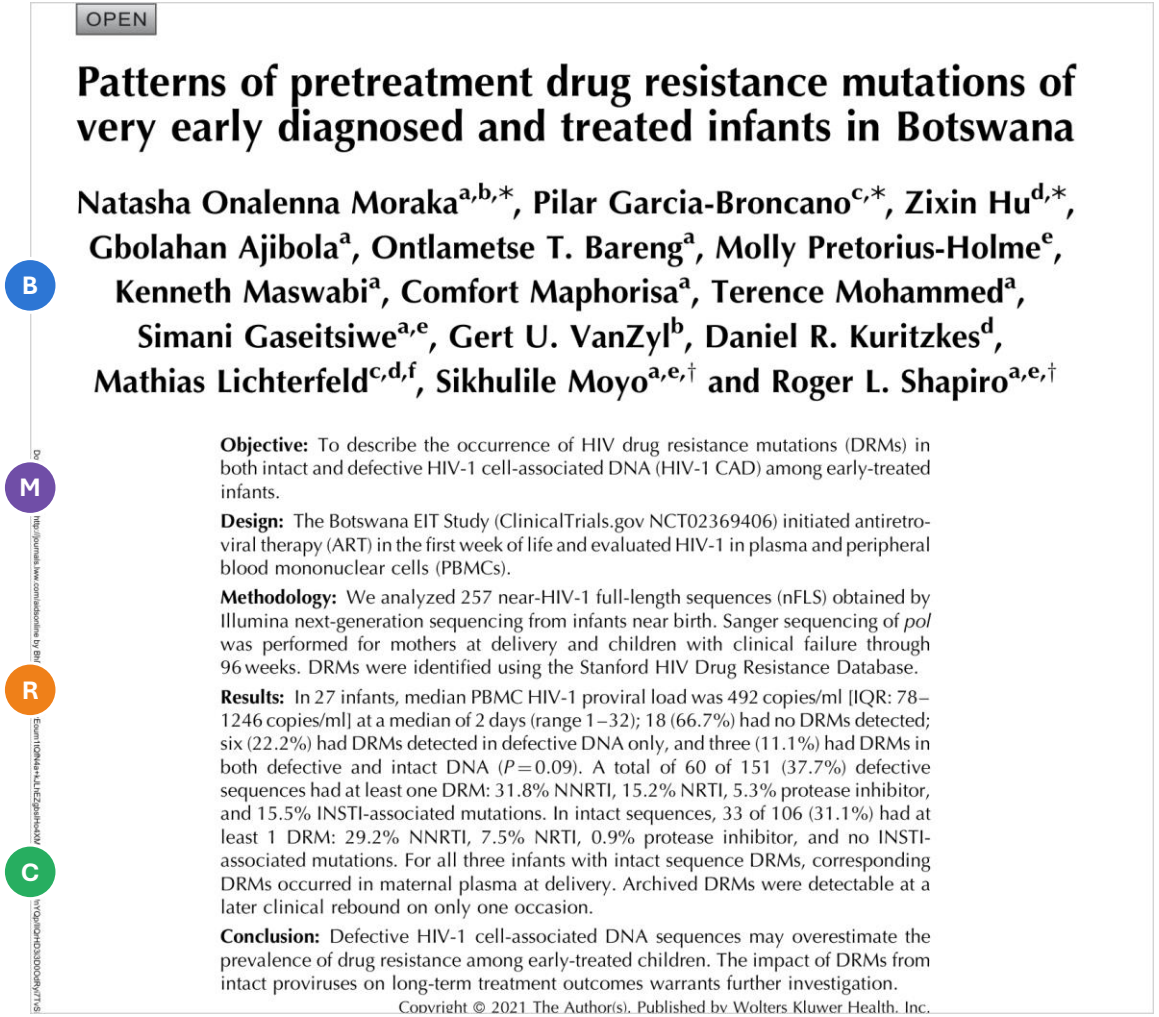
Ask the class Teaching question: When results are dense, what should stay in the abstract and what belongs in the paper?

B Background/gap M Methods R Results C Conclusion

Screenshots fixed; labels/callouts editable.

Example 4: Laboratory + clinical HIV reservoir abstract

Moraka et al., AIDS 2021 - drug resistance mutations in early-treated infants



OPEN

Patterns of pretreatment drug resistance mutations of very early diagnosed and treated infants in Botswana

Natasha Onalenna Moraka[a,b,*], Pilar Garcia-Broncano[c,*], Zixin Hu[d,*], Gbolahan Ajibola[a], Ontlametse T. Bareng[a], Molly Pretorius-Holme[e], Kenneth Maswabi[a], Comfort Maphorisa[a], Terence Mohammed[a], Simani Gaseitsiwe[a,e], Gert U. VanZyl[b], Daniel R. Kuritzkes[d], Mathias Lichterfeld[c,d,f], Sikhulile Moyo[a,e,†] and Roger L. Shapiro[a,e,†]

Objective: To describe the occurrence of HIV drug resistance mutations (DRMs) in both intact and defective HIV-1 cell-associated DNA (HIV-1 CAD) among early-treated infants.

Design: The Botswana EIT Study (ClinicalTrials.gov NCT02369406) initiated antiretroviral therapy (ART) in the first week of life and evaluated HIV-1 in plasma and peripheral blood mononuclear cells (PBMCs).

Methodology: We analyzed 257 near-HIV-1 full-length sequences (nFLS) obtained by Illumina next-generation sequencing from infants near birth. Sanger sequencing of *pol* was performed for mothers at delivery and children with clinical failure through 96 weeks. DRMs were identified using the Stanford HIV Drug Resistance Database.

Results: In 27 infants, median PBMC HIV-1 proviral load was 492 copies/ml [IQR: 78–1246 copies/ml] at a median of 2 days (range 1–32); 18 (66.7%) had no DRMs detected; six (22.2%) had DRMs detected in defective DNA only, and three (11.1%) had DRMs in both defective and intact DNA ($P = 0.09$). A total of 60 of 151 (37.7%) defective sequences had at least one DRM: 31.8% NNRTI, 15.2% NRTI, 5.3% protease inhibitor, and 15.5% INSTI-associated mutations. In intact sequences, 33 of 106 (31.1%) had at least 1 DRM: 29.2% NNRTI, 7.5% NRTI, 0.9% protease inhibitor, and no INSTI-associated mutations. For all three infants with intact sequence DRMs, corresponding DRMs occurred in maternal plasma at delivery. Archived DRMs were detectable at a later clinical rebound on only one occasion.

Conclusion: Defective HIV-1 cell-associated DNA sequences may overestimate the prevalence of drug resistance among early-treated children. The impact of DRMs from intact proviruses on long-term treatment outcomes warrants further investigation.

B

M

R

C

Teaching features to point out

B Objective is concise and descriptive: occurrence of DRMs in intact and defective HIV-1 DNA.

M Methods anchor the cohort and sequencing approach.

R Results separate defective vs intact DNA and link mutations to maternal plasma.

C Conclusion is cautious: defective sequences may overestimate resistance prevalence.

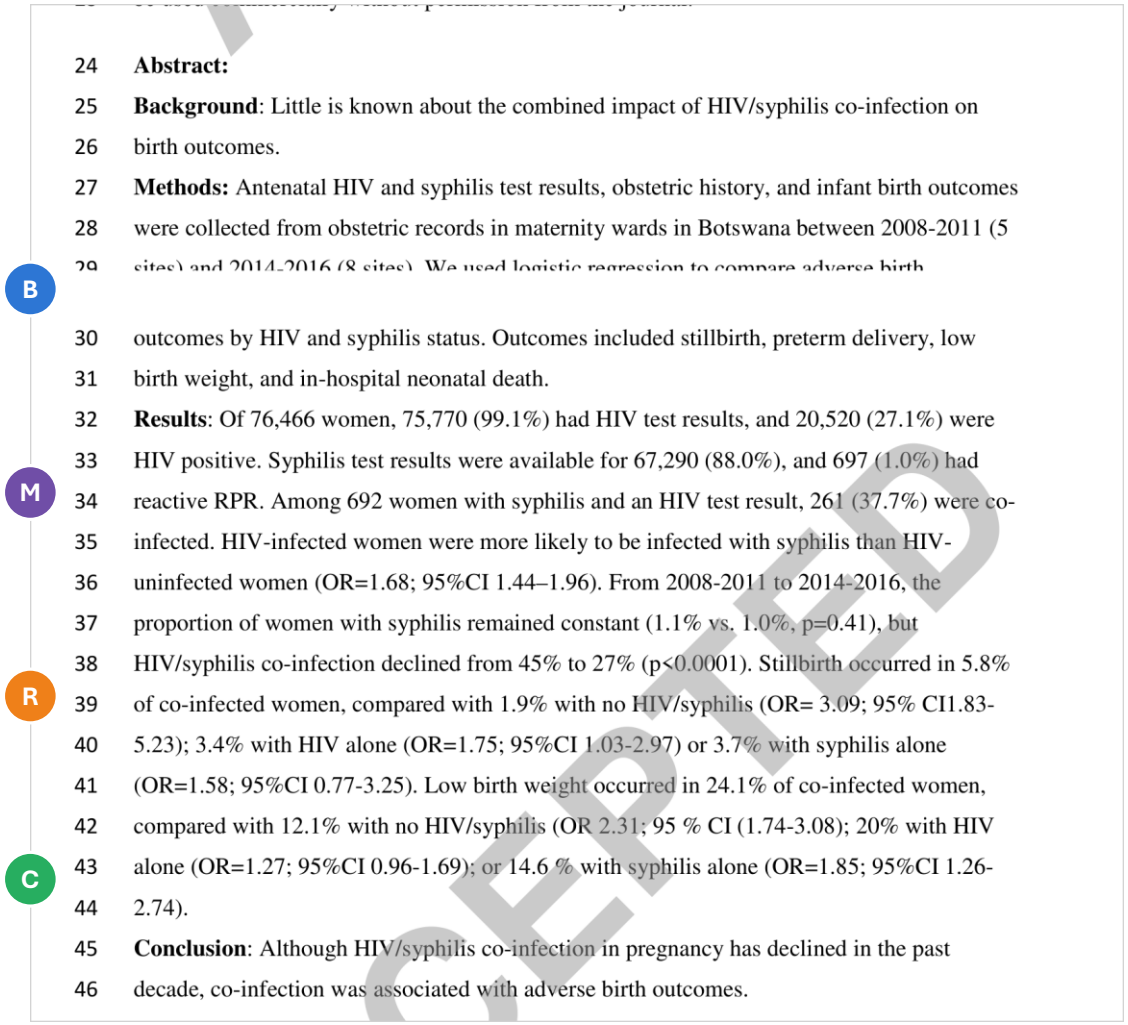
Ask the class Teaching question: How does the conclusion acknowledge assay interpretation rather than just reporting prevalence?

B Background/gap M Methods R Results C Conclusion

Screenshots fixed; labels/callouts editable.

Example 5: Secondary data analysis with public-health outcomes

Shava et al., JAIDS 2019 - HIV/syphilis co-infection and adverse birth outcomes in Botswana



Abstract:

Background: Little is known about the combined impact of HIV/syphilis co-infection on birth outcomes.

Methods: Antenatal HIV and syphilis test results, obstetric history, and infant birth outcomes were collected from obstetric records in maternity wards in Botswana between 2008-2011 (5 sites) and 2014-2016 (8 sites). We used logistic regression to compare adverse birth outcomes by HIV and syphilis status. Outcomes included stillbirth, preterm delivery, low birth weight, and in-hospital neonatal death.

Results: Of 76,466 women, 75,770 (99.1%) had HIV test results, and 20,520 (27.1%) were HIV positive. Syphilis test results were available for 67,290 (88.0%), and 697 (1.0%) had reactive RPR. Among 692 women with syphilis and an HIV test result, 261 (37.7%) were co-infected. HIV-infected women were more likely to be infected with syphilis than HIV-uninfected women (OR=1.68; 95%CI 1.44–1.96). From 2008-2011 to 2014-2016, the proportion of women with syphilis remained constant (1.1% vs. 1.0%, p=0.41), but HIV/syphilis co-infection declined from 45% to 27% (p<0.0001). Stillbirth occurred in 5.8% of co-infected women, compared with 1.9% with no HIV/syphilis (OR= 3.09; 95% CI1.83-5.23); 3.4% with HIV alone (OR=1.75; 95%CI 1.03-2.97) or 3.7% with syphilis alone (OR=1.58; 95%CI 0.77-3.25). Low birth weight occurred in 24.1% of co-infected women, compared with 12.1% with no HIV/syphilis (OR 2.31; 95 % CI (1.74-3.08); 20% with HIV alone (OR=1.27; 95%CI 0.96-1.69); or 14.6 % with syphilis alone (OR=1.85; 95%CI 1.26-2.74).

Conclusion: Although HIV/syphilis co-infection in pregnancy has declined in the past decade, co-infection was associated with adverse birth outcomes.

Teaching features to point out

B Background is short and gap-focused: combined impact is little known.

M Methods identify records, years, sites, outcomes and logistic regression.

R Results use denominators, trend, and ORs for stillbirth/low birth weight.

C Conclusion balances decline in co-infection with persisting adverse outcomes.

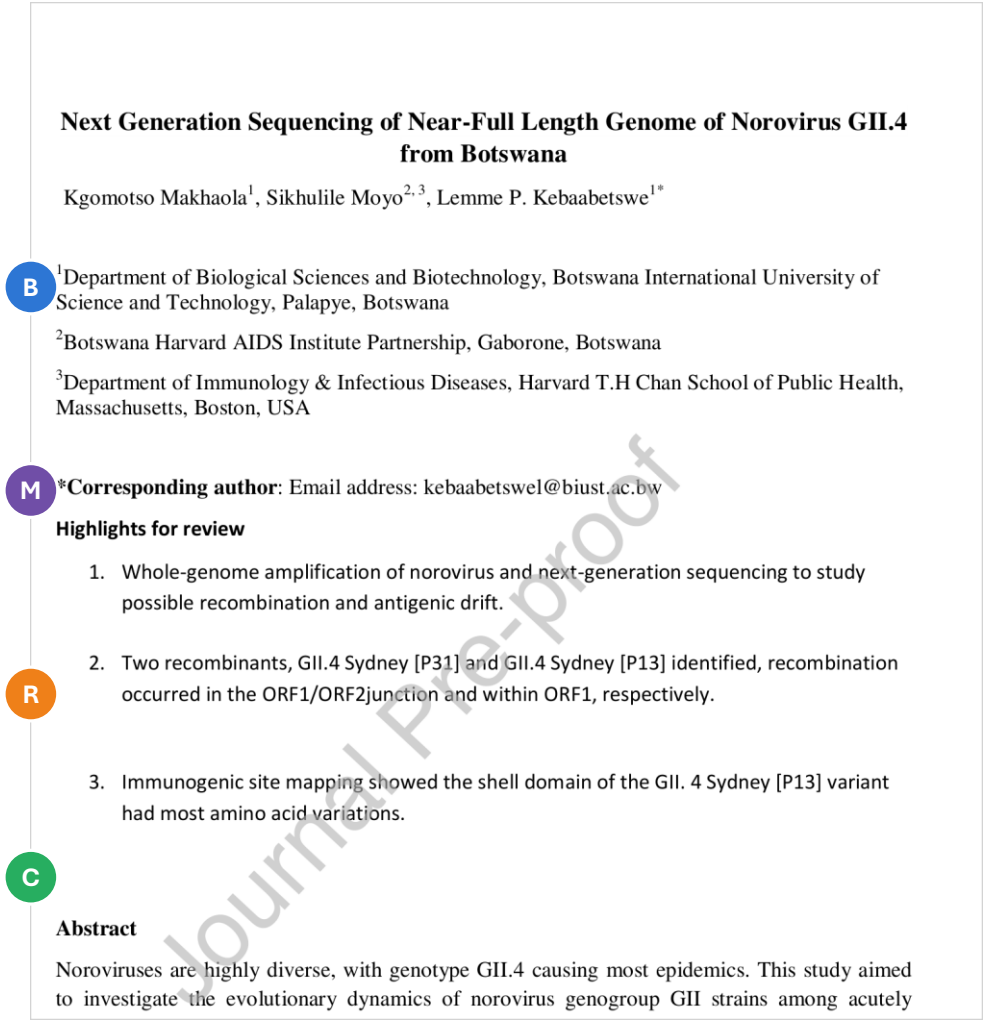
Ask the class Teaching question: How do denominators help the reader trust the result?

B Background/gap M Methods R Results C Conclusion

Screenshots fixed; labels/callouts editable.

Example 6: Unstructured virology abstract with novelty signal

Makhaola, Moyo & Kebaabetswe, Virus Research 2021 - near-full-length norovirus genomes from Botswana



Next Generation Sequencing of Near-Full Length Genome of Norovirus GII.4 from Botswana

Kgomotso Makhaola[1], Sikhulile Moyo[2,3], Lemme P. Kebaabetswe[1*]

B [1]Department of Biological Sciences and Biotechnology, Botswana International University of Science and Technology, Palapye, Botswana

[2]Botswana Harvard AIDS Institute Partnership, Gaborone, Botswana

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Highlights for review

1. Whole-genome amplification of norovirus and next-generation sequencing to study possible recombination and antigenic drift.
2. Two recombinants, GII.4 Sydney [P31] and GII.4 Sydney [P13] identified, recombination occurred in the ORF1/ORF2junction and within ORF1, respectively.
3. Immunogenic site mapping showed the shell domain of the GII. 4 Sydney [P13] variant had most amino acid variations.

R

C

Abstract

Noroviruses are highly diverse, with genotype GII.4 causing most epidemics. This study aimed to investigate the evolutionary dynamics of norovirus genogroup GII strains among acutely

Teaching features to point out

B Starts with pathogen diversity and objective rather than a generic disease statement.

M Methods are brief but specific: RT-PCR, whole genome amplification, nanopore sequencing.

R Results emphasize recombination, antigenic drift and mutations in epitopes.

C Novelty sentence: first description/first sequences in Botswana.

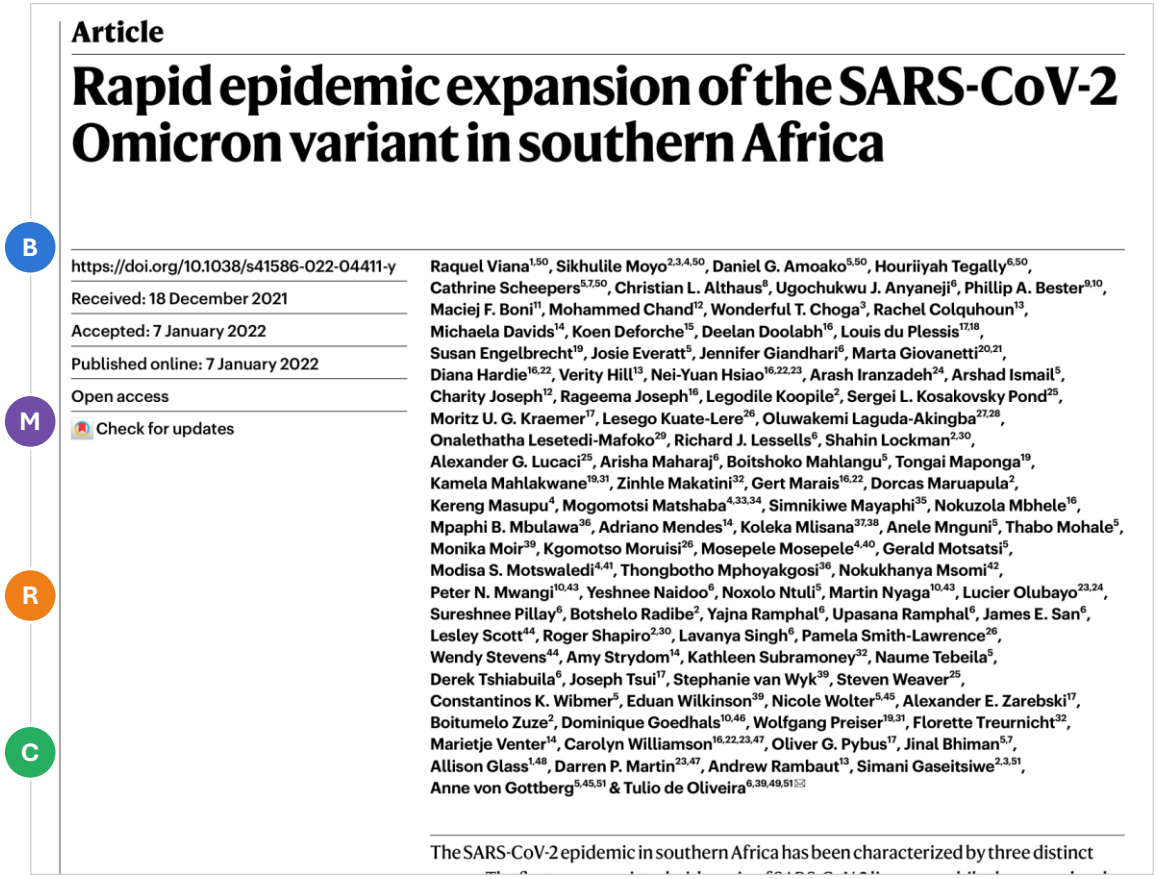
Ask the class Teaching question: Where is the novelty claim, and is it supported by the results?

B Background/gap M Methods R Results C Conclusion

Screenshots fixed; labels/callouts editable.

Example 7: High-impact unstructured Nature abstract

Viana, Moyo et al., Nature 2022 - rapid expansion of the SARS-CoV-2 Omicron variant



Article

Rapid epidemic expansion of the SARS-CoV-2 Omicron variant in southern Africa

B

https://doi.org/10.1038/s41586-022-04411-y

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Open access

M

Check for updates

Raquel Viana[1,50], Sikhulile Moyo[2,3,4,50], Daniel G. Amoako[5,50], Houriiyah Tegally[6,50], Cathrine Scheepers[5,7,50], Christian L. Althaus[8], Ugochukwu J. Anyaneji[6], Phillip A. Bester[9,10], Maciej F. Boni[11], Mohammed Chand[12], Wonderful T. Choga[3], Rachel Colquhoun[13], Michaela Davids[14], Koen Deforche[15], Deelan Doolabh[16], Louis du Plessis[17,18], Susan Engelbrecht[19], Josie Everatt[5], Jennifer Giandhari[6], Marta Giovanetti[20,21], Diana Hardie[16,22], Verity Hill[13], Nei-Yuan Hsiao[16,22,23], Arash Iranzadeh[24], Arshad Ismail[5], Charity Joseph[12], Rageema Joseph[16], Legodile Koopile[2], Sergei L. Kosakovsky Pond[25], Moritz U. G. Kraemer[17], Lesego Kuate-Lere[26], Oluwakemi Laguda-Akingba[27,28], Onalethatha Lesetedi-Mafoko[29], Richard J. Lessells[6], Shahin Lockman[2,30], Alexander G. Lucaci[25], Arisha Maharaj[6], Boitshoko Mahlangu[5], Tongai Maponga[19], Kamela Mahlakwane[19,31], Zinhle Makatini[32], Gert Marais[16,22], Dorcas Maruapula[2], Kereng Masupu[4], Mogomotsi Matshaba[4,33,34], Simnikiwe Mayaphi[35], Nokuzola Mbhele[16], Mpaphi B. Mbulawa[36], Adriano Mendes[14], Koleka Mlisana[37,38], Anele Mnguni[5], Thabo Mohale[5], Monika Moir[39], Kgomotso Moruisi[26], Mosepele Mosepele[4,40], Gerald Motsatsi[5], Modisa S. Motswaledi[4,41], Thongbotho Mphoyakgosi[36], Nokukhanya Msomi[42], Peter N. Mwangi[10,43], Yeshnee Naidoo[6], Noxolo Ntuli[5], Martin Nyaga[10,43], Lucier Olubayo[23,24], Sureshnee Pillay[6], Botshelo Radibe[2], Yajna Ramphal[6], Upasana Ramphal[6], James E. San[6], Lesley Scott[44], Roger Shapiro[2,30], Lavanya Singh[6], Pamela Smith-Lawrence[26], Wendy Stevens[44], Amy Strydom[14], Kathleen Subramoney[32], Naume Tebeila[5], Derek Tshiabuila[6], Joseph Tsui[17], Stephanie van Wyk[39], Steven Weaver[25], Constantinos K. Wibmer[5], Eduan Wilkinson[39], Nicole Wolter[5,45], Alexander E. Zarebski[17], Boitumelo Zuze[2], Dominique Goedhals[10,46], Wolfgang Preiser[19,31], Florette Treurnicht[32], Marietje Venter[14], Carolyn Williamson[16,22,23,47], Oliver G. Pybus[17], Jinal Bhiman[5,7], Allison Glass[1,48], Darren P. Martin[23,47], Andrew Rambaut[13], Simani Gaseitsiwe[2,3,51], Anne von Gottberg[5,45,51] & Tulio de Oliveira[6,39,49,51]✉

R

The SARS-CoV-2 epidemic in southern Africa has been characterized by three distinct

C

Teaching features to point out

B Narrative context: previous waves, detection of a new variant, and rapid designation.

R Core findings: genomic profile and early transmission dynamics.

C Implication: rapid spread in populations with high immunity.

M Teaching point: unstructured does not mean unorganized - the logic is still visible.

Ask the class Teaching question: How does this abstract create urgency while staying scientific?

M Methods R Results C Conclusion B Narrative

Screenshots fixed; labels/callouts editable.

Example 8: Letter format - when there is no formal abstract

Shava/Izu/Gaolathe et al., Journal of Infection 2023 - AZD1222 safety and COVID-19 incidence in Botswana

Journal of Infection

journal homepage: www.elsevier.com/locate/jinf

Letter to the Editor

Safety and incidence of COVID-19 following ChAdOx1(AZD1222) COVID-19 vaccination in Botswana

B

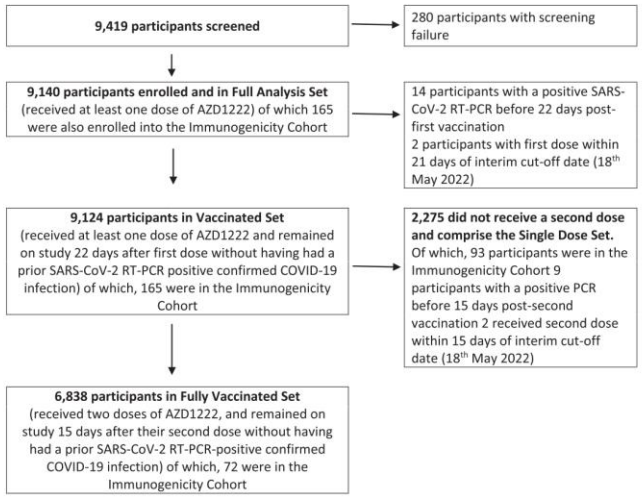
Dear editor,

A recent publication in this journal by Nisar *et al.*[1] assessed the effectiveness of various COVID-19 vaccines in Pakistan using a test-negative case-control design. They report moderate effectiveness of various vaccines and higher effectiveness mRNA vaccines compared with inactivated vaccines. Although they assessed the impact of concurrent medical conditions, they were only a few cases of immunosuppression contributing to < 3% of the study population and approximately 87% of the participants received Sinopharm and Sinovac, and 1% received the Oxford/AstraZeneca vaccine. AstraZeneca (AZD1222), formerly called ChAdOx1, previously demonstrated robust immunogenicity after a single dose with favorable safety profiles.[2,3] Despite Botswana's high HIV prevalence, treatment has been hugely successful with over 90% of all adults living with HIV currently receiving antiretroviral therapy (ART).[4] There are no data on the safety and effectiveness of AZD1222 in this high HIV prevalence setting.

M

We conducted a single-arm, open-label interventional multi-site study (D8111C00013/ESR-21–21311) to monitor vaccine safety and the occurrence of symptomatic COVID-19 infections, hospitalizations and deaths among individuals vaccinated with AZD1222 between September 15, 2021, and May 18, 2022, at five sites in Botswana. The study was approved by the Health and Research Development Committee (HRDC#00936). All participants provided written informed consent.

We screened 9419 and enrolled 9140 participants (Fig. 1). Of the participants included in this study, 9124 (99.8%) were receiving at least one dose of AZD1222 and remaining in the study 22 days after the first dose without having a SARS-CoV-2 RT-PCR positive confirmed COVID-19 infection, Vaccinated Set (VS); 2275 of these par-



Teaching features to point out

B Letter opening: cites recent paper, then states the local knowledge gap.

M Study description appears in narrative form instead of a Methods heading.

R Visual method: participant-flow diagram carries much of the design information.

C Teaching point: for letters, students still need problem -> method -> key result -> implication.

Ask the class Teaching question: If converting this letter into a structured abstract, what would become Background, Methods, Results and Conclusion?

B Background/gap M Methods C Conclusion R Visual

Screenshots fixed; labels/callouts editable.

High Rates of TB in the first six months of Dolutegravir-based ART in Botswana

Lucy Mupfumi[1], Sikhulile Moyo[2], Ava Avalos[3], Lesedi Bawley[3], Kaelo Seatla[1], Shin Sanghyuk[4], Ishmael Kasvosve[5], Nicola Zetola[6,7], Simani Gaseitsiwe[1,3] and the BEAT Cohort Study Team

1. Botswana-Harvard AIDS Institute Partnership, Gaborone, Botswana; Department of Medical Laboratory Sciences, Faculty of Health Sciences, University of Botswana, Gaborone, Botswana
2. Botswana-Harvard AIDS Institute Partnership, Gaborone, Botswana; Department of Immunology & Infectious Diseases, Harvard TH Chan School of Public Health, Boston, MA, USA
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5. Department of Medical Laboratory Sciences, Faculty of Health Sciences, University of Botswana, Gaborone, Botswana
6. Botswana-University of Pennsylvania (BUP) Partnership, Gaborone, Botswana; Infectious Diseases Division, University of Pennsylvania, PA, USA

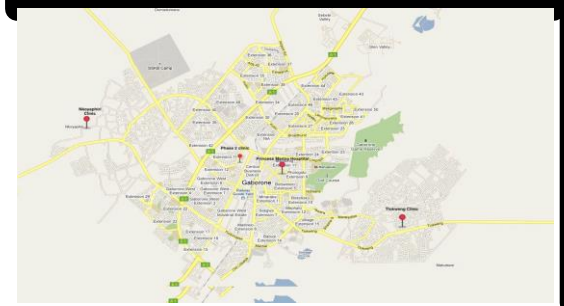
DELTAS AFRICA SCIENTIFIC MEETING
Dakar, Senegal July 15–17, 2019

BACKGROUND

- TB drives the high mortality rate characteristic of ART programmes in sub-Saharan Africa
- The incidence of TB in the "Test and Treat" era has not been reported.

METHODS

- Data was extracted from electronic and manual patients records from 4 clinics in Gaborone, Botswana
- These sites were part of the operational cohort to determine DTG treatment efficacy and monitor drug resistance
- Wilcoxon rank sum and Kruskal Wallis was used to compare differences in baseline covariates
- Kaplan-Meir plots and Cox regression was used to determine time to TB diagnosis and risk factors of incident TB.



RESULTS

- 737 patients initiating ART at 4 clinics in Gaborone, Botswana (March 2016-June 2018)
  60% (441) female
  Baseline CD4 count: 387 cells/ul
  (Q1,Q3: 219, 577)
  Prevalent TB: 10/737 (1%)
- Follow-up time: 247 d (119-391)
- **Incident TB: 18/676: IR 3.82/100py (95% CI: 2.41-6.06)**
  Time to TB diagnosis: 52 days (17-116)

11/18 (61%) within 3 months IR: 7.31 (95% CI: 4.25- 12.59)
89% (16/18) within 6 months of ART

Table 1. Baseline characteristics of all patients

Characteristic	N=737
Age (Q1, Q3)	34 (28, 41)
Female (%)	441 (60)
CD4 count* (cells/ul) (%)	
<50	16 (7)
50-99	9 (4)
100-199	25 (11)
200-349	44 (20)
350-499	47 (22)
>500	78 (36)
ART regimen initiated on (%)	
TRU/DTG	725 (98)
ABC/3TC/DTG	12 (2)

*CD4 count available for 219 patients

Time to TB diagnosis and role of immune recovery

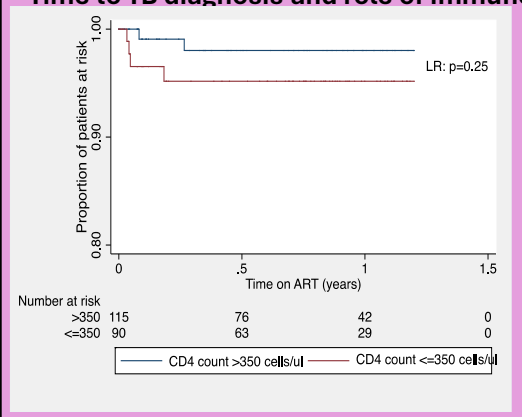


Figure 1: Time to incident TB in patients on DTG-based ART

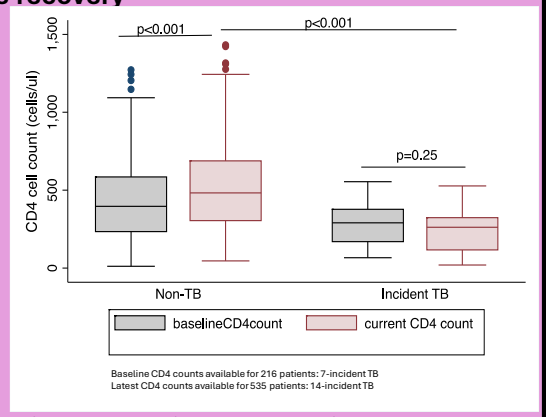


Figure 2: Change in CD4 counts during follow-up
Current CD4: CD4 count at latest clinic review [median 330 days (Q1:Q3 183, 737) post ART initiation]

Risk factors for TB in the first year of ART

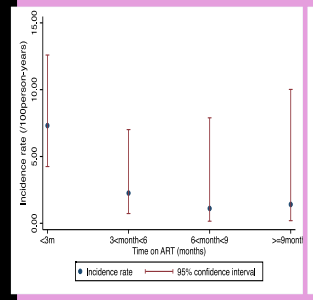


Figure 3: Change in incident TB with time on ART

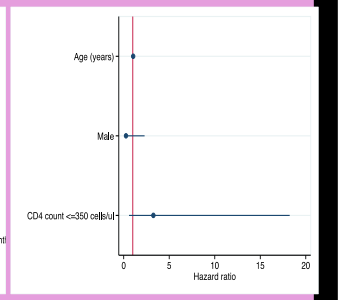


Figure 4: Risk factors for incident TB

CONCLUSION

- **High incidence within first 3 months of ART**
  *** likely reflects missed TB diagnosis**
- **Possible role of poor immune reconstitution and low CD4 count at ART initiation**
- **Need for better strategies to screen for TB in antiretroviral programs in sub-Saharan Africa**
  ***critical as countries implement "Treat-all" strategies**

ACKNOWLEDGMENTS

We wish to thank the patients whose data was used for this analysis, the Botswana Ministry of Health and BEAT Cohort Study Team at Botswana Harvard AIDS Institute Partnership. LM is funded by the sub-Saharan African Network for TB/HIV Research Excellence

PLOS ONE

OPEN ACCESS PEER-REVIEWED

RESEARCH ARTICLE

Immunological non-response and low hemoglobin levels are predictors of incident tuberculosis among HIV-infected individuals on Truvada-based therapy in Botswana

Lucy Mupfumi, Sikhulile Moyo, Kesaobaka Molebatsi, Prisca K. Thami, Motswedi Anderson, Tuelo Mogashoa, Thato Iketleng, Joseph Makhema, Ric Marlink, Ishmael Kasvosve, Max Essex, Rosemary M. Musonda, Simani Gaseitsiwe

Background

There is a high burden of tuberculosis (TB) in HIV antiretroviral programmes in Africa. However, few studies have looked at predictors of incident TB while on Truvada-based combination antiretroviral therapy (cART) regimens.

Methods

We estimated TB incidence among individuals enrolled into an observational cohort evaluating the efficacy and tolerability of Truvada-based cART in Gaborone, Botswana between 2008 and 2011. We used Cox proportional hazards regressions to determine predictors of incident TB.

Results

Of 300 participants enrolled, 45 (15%) had a diagnosis of TB at baseline. During 428 person-years (py) of follow-up, the incidence rate of TB was 3.04/100py (95% CI, 1.69–5.06), with 60% of the cases occurring within 3 months of ART initiation. Incident cases had low baseline CD4+ T cell counts (153cells/mm^3 [Q1, Q3: 82, 242]; p = 0.69) and hemoglobin levels (9.2g/dl [Q1, Q3: 8.5,10.1]; p<0.01). In univariate analysis, low BMI (HR = 0.73; 95% CI 0.58–0.91; p = 0.01) and hemoglobin levels <8 g/dl (HR = 10.84; 95%CI: 2.99–40.06; p<0.01) were risk factors for TB. Time-to-incident TB diagnosis was significantly reduced in patients with poor immunological recovery (p = 0.04). There was no association between baseline viral load and risk of TB (HR = 1.75; 95%CI: 0.70–4.37).

Conclusion

Low hemoglobin levels prior to initiation of ART are significant predictors of incident tuberculosis. Therefore, there is potential utility of iron biomarkers to identify patients at risk of TB prior to initiation on ART. Furthermore, additional strategies are required for patients with poor immunological recovery to reduce excess risk of TB while on ART.

Whole genome sequencing-based profiling of rifampicin-resistant Mycobacterium tuberculosis strains by HIV status in Botswana

Abstract Text (max 350 words)

Background: Whole genome sequencing technology (WGS) provides more comprehensive drug resistance profiles which could guide choice of treatment. The objective of this study was to utilize WGS technology to characterize *Mycobacterium tuberculosis* (*Mtb*) isolates and evaluate the association of rifampicin-resistant-TB with HIV acquisition in Botswana.

Methods: A retrospective cohort study was conducted on 133 individuals diagnosed with rifampicin-resistant-TB from 2016-2021. Genomic DNA was extracted from isolates cultured on *Löwenstein-Jensen* media using the cetyltrimethylammonium bromide(CTAB) method. Library preparation was performed with the Illumina DNA prep kit and loaded on Illumina NextSeq 2000 platform. TBProfiler(v4.4.2) was used to identify and delineate *M.tb* lineages and drug resistance profiles according to WHO mutations catalogue. Logistic regression models were used to identify predictive factors of HIV in people with rifampicin-resistant-TB.

Results: In this cohort, 73/133(55%) were people living with HIV (PLWH). The median age of PLWH was 41 (Q1, Q3: 32, 48) years, which were older than people not living with HIV (PNLWH) (median age: 29 (Q1, Q3: 25, 47)(p=0.01). A total of 106/133 were successfully sequenced of which 64 were PLWH and 42 were PNLWH. Amongst 106 sequences, 61, 27, 16 and 2 belonged to lineage4, lineage1, lineage2 and lineage3(all from PLWH), respectively. For rifampicin, *rpoB*_p.S450L(n=28) and *rpoB*_p.H445L(n = 23) and were detected while for isoniazid, *katG*_p.S315T was found in 50/106. Mutations associated with anti-TB drugs were stratified by HIV status in Table 1. Mutation *rpoB*_c.1303_1305delGAC was only observed among PLWH(n=3). Mono-rifampicin-resistant-TB was associated with living with HIV (unadjusted odd ratio:3.3, 95% confidence intervals:1.2-8.9(p=0.02)) when compared to those diagnosed with multidrug resistant-TB and pre-extensively drug resistant-TB.

> Viruses. 2024 May 1;16(5):720. doi: 10.3390/v16050720.

Low-Level Viremia among Adults Living with HIV on Dolutegravir-Based First-Line Antiretroviral Therapy Is a Predictor of Virological Failure in Botswana

Ontlametse T Bareng [1 2], Sikhulile Moyo [1 3 4 5], Mbatshi Mudanga [6], Kagiso Sebina [6], Catherine K Koofhethile [1 3], Wonderful T Choga [1 2], Natasha O Moraka [1 2], Dorcas Maruapula [1], Irene Gobe [2], Modisa S Motswaledi [2], Rosemary Musonda [1], Bornapate Nkomo [7], Dinah Ramaabya [7], Tony Chebani [7], Penny Makuruetsa [7], Joseph Makhema [1 3], Roger Shapiro [1 3], Shahin Lockman [1 3 8], Simani Gaseitsiwe [1 3]

Affiliations + expand

PMID: 38793602 PMCID: PMC11125697 DOI: 10.3390/v16050720

Abstract

We evaluated subsequent virologic outcomes in individuals experiencing low-level virem ia (LLV) on dolutegravir (DTG)-based first-line antiretroviral therapy (ART) in Botswana. We used a national dataset from 50,742 adults who initiated on DTG-based first-line ART from June 2016-December 2022. Individuals with at least two viral load (VL) measurements post three months on DTG-based first-line ART were evaluated for first and subsequent episodes of LLV (VL:51-999 copies/mL). LLV was sub-categorized as low-LLV (51-200 copies/mL), medium-LLV (201-400 copies/mL) and high-LLV (401-999 copies/mL). The study outcome was virologic failure (VF) (VL ≥ 1000 copies/mL): virologic non-suppression defined as single-VF and confirmed-VF defined as two-consecutive VF measurements after an initial VL < 1000 copies/mL. Cox regression analysis identified predictive factors of subsequent VF. The prevalence of LLV was only statistically different at timepoints >6-12 (2.8%) and >12-24 (3.9%) (p-value < 0.01). LLV was strongly associated with both virologic non-suppression (adjusted hazards ratio [aHR] = 2.6; 95% CI: 2.2-3.3, p-value ≤ 0.001) and confirmed VF (aHR = 2.5; 95% CI: 2.4-2.7, p-value ≤ 0.001) compared to initially virally suppressed PLWH. High-LLV (HR = 3.3; 95% CI: 2.9-3.6) and persistent-LLV (HR = 6.6; 95% CI: 4.9-8.9) were associated with an increased hazard for virologic non-suppression than low-LLV and a single-LLV episode, respectively. In a national cohort of PLWH on DTG-based first-line ART, LLV > 400 copies/mL and persistent-LLV had a stronger association with VF. Frequent VL testing and adherence support are warranted for individuals with VL > 50 copies/mL.

Keywords: Botswana; dolutegravir-based first line antiretroviral therapy; low level viremia; people living with HIV.

> J Glob Antimicrob Resist. 2022 Dec:31:128-134. doi: 10.1016/j.jgar.2022.08.008. Epub 2022 Aug 13.

Doravirine-associated resistance mutations in antiretroviral therapy naïve and experienced adults with HIV-1 subtype C infection in Botswana

Ontlametse T Bareng [1], Sekgabo Seselamarumo [2], Kaelo K Seatla [1], Wonderful T Choga [3], Blessing Bakae [4], Dorcas Maruapula [2], Nametso Kelentse [1], Natasha O Moraka [5], Baitshepi Mokaleng [1], Patrick T Mokgethi [2], Tsotlhe R Ditlhako [4], Molly Pretorius-Holme [6], Mpaphi B Mbulawa [7], Refeletswe Lebelonyane [7], Ebi Celestin Bile [8], Tendani Gaolathe [4], Roger Shapiro [9], Joseph M Makhema [9], Shahin Lockman [10], Max Essex [9], Vlad Novitsky [9], Sununguko W Mpoloka [11], Sikhulile Moyo [9], Simani Gaseitsiwe [12]

Affiliations + expand

PMID: 35973671 PMCID: PMC9750894 DOI: 10.1016/j.jgar.2022.08.008

Abstract

Objectives: There are limited data on the prevalence of doravirine (DOR)-associated drug resistance mutations in people with HIV (PWH) in Botswana. This cross-sectional, retrospective study aimed to explore the prevalence of DOR-associated resistance mutations among ART-naïve and -experienced PWH in Botswana enrolled in the population-based Botswana Combination Prevention Project (BCPP).

Methods: A total of 6078 HIV-1C pol sequences were analysed for DOR-associated resistance mutations using the Stanford HIV drug resistance database, and their levels were predicted according to the Stanford DRM penalty scores and resistance interpretation. Virologic failure was defined as HIV-1 RNA load (VL) >400 copies/mL.

Results: Among 6078 PWH, 5999 (99%) had known ART status, and 4529/5999 (79%) were on ART at time of sampling. The suppression rate among ART-experienced was 4517/4729 (96%). The overall prevalence of any DOR-associated resistance mutations was 181/1473 (12.3% [95% confidence interval {CI}: 10.7-14.1]); by ART status: 42/212 (19.8% [95% CI: 14.7-25.4]) among ART-failing individuals (VL ≥400 copies/mL) and 139/1261 (11.0% [95% CI: 9.3-12.9]) among ART-naïve individuals (P < 0.01). Intermediate DOR-associated resistance mutations were observed in 106/1261 (7.8% [95% CI: 6.9-10.1]) in ART-naïve individuals and 29/212 (13.7% [95% CI: 9.4-8.5]) among ART-experienced participants (P < 0.01). High-level DOR-associated resistance mutations were observed in 33/1261 (2.6% [95% CI: 1.8-3.7]) among ART-naïve and 13/212 (6.1% [95% CI: 3.6-10.8]) among ART-failing PWH (P < 0.01). PWH failing ART with at least one EFV/NVP-associated resistance mutation had high prevalence 13/67 (19.4%) of high-level DOR-associated resistance mutations.

Conclusion: DOR-associated mutations were rare (11.0%) among ART-naive PWH but present in 62.7% of Botswana individuals who failed NNRTI-based ART with at least one EFV/NVP-associated resistance mutation. Testing for HIV drug resistance should underpin the use of DOR in PWH who have taken first-generation NNRTIs.

Keywords: Antiretroviral (ART) experienced; Antiretroviral (ART) naive; Botswana; Doravirine; Drug resistance mutations; HIV-1C.

AIDS. 2024 Nov 4. doi: 10.1097/QAD.0000000000004055. Online ahead of print.

Predictors of HIV seroconversion in Botswana: machine learning analysis in a representative, population-based HIV incidence cohort

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Abstract

Objective: To identify predictors of HIV acquisition in Botswana.

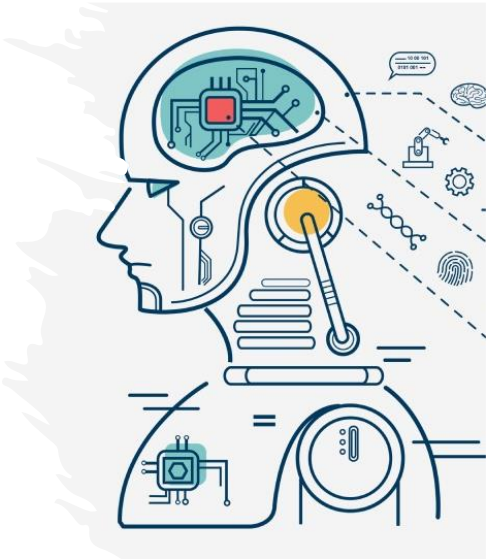
Design: We applied machine learning approaches to identify HIV risk predictors using existing data from a large, well-characterized HIV incidence cohort.

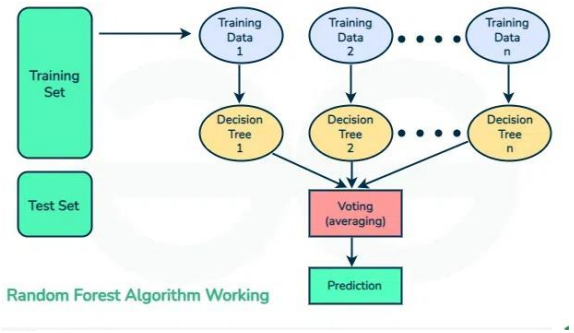
Methods: We applied machine learning (randomForestSRC) to analyze data from a large population-based HIV incidence cohort enrolled in a cluster-randomized HIV prevention trial in 30 communities across Botswana. We sought to identify the most important risk factors for HIV acquisition, starting with 110 potential predictors.

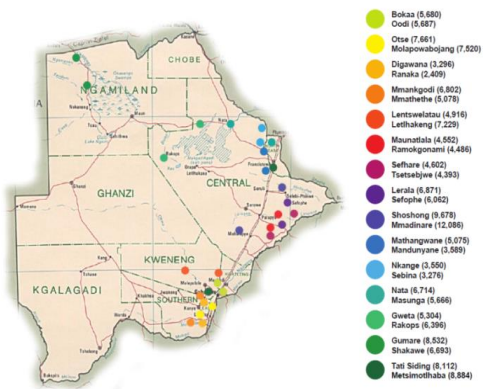
Results: During a median 29-month follow-up of 8,551 HIV-negative adults, 147 (1.7%) acquired HIV. Our machine learning analysis found that for females, the most important variables for predicting HIV acquisition were the use of injectable hormonal contraception, frequency of sex in the prior 3 months with the most recent partner and residing in a community with HIV prevalence of 29% or higher. For the small proportion (0.3%) of females who had all three risk factors, their estimated probability of acquiring HIV during 29 months of follow-up was 34% (approximate annual incidence of 14%). For males, non-long-term relationships with the most recent partner and community HIV prevalence of 34% or higher were the most important HIV risk predictors. The 6% of males who had both risk factors had a 5.1% probability of acquiring HIV during the follow-up period (approximate annual incidence of 2.1%).

Conclusions: Machine learning approaches allowed us to analyze a large number of variables to efficiently identify key factors strongly predictive of HIV risk. These factors could help target HIV prevention interventions in Botswana.

Clinical trials registration: NCT01965470.

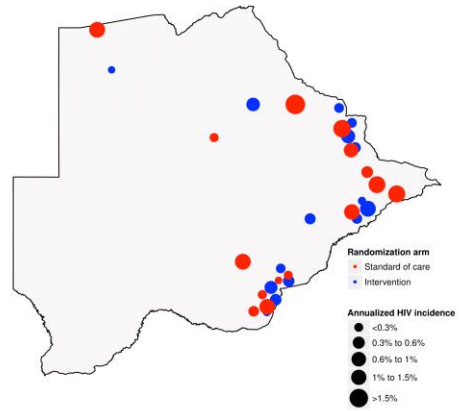






We applied machine learning approaches to identify HIV risk predictors using existing data from a large, well-characterized HIV incidence cohort (Ya Tsie Trial).

Applied **randomForestSRC** starting with 110 potential predictors.



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The Challenge of Multiple Concurrent Partnerships

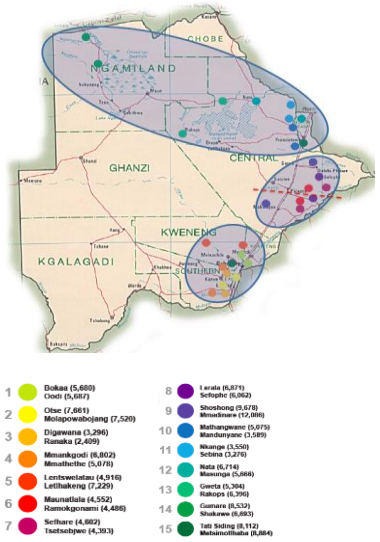
AIDS Behav (2007) 11:822–830
DOI 10.1007/s10461-006-9203-6

ORIGINAL PAPER

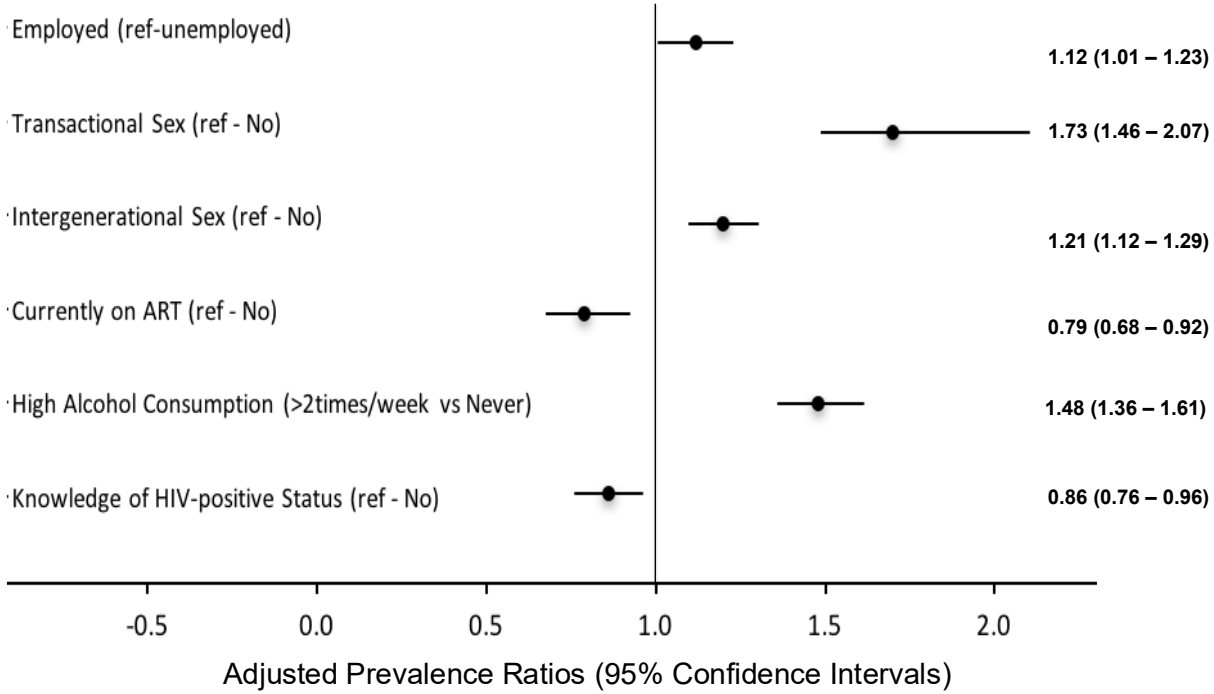
"A Bull Cannot be Contained in a Single Kraal": Concurrent Sexual Partnerships in Botswana

Marion W. Carter · Joan Marie Kraft · Todd Koppenhaver · Christine Galavotti · Thierry H. Roels · Peter H. Kilmarx · Boga Fidzani

Abstract To inform efforts to curb HIV in Botswana, we describe sexual concurrency and related norms and behaviors among a sample of 807 Batswana age 15–49 years who participated in a 2003 population-based survey. Of 546 sexually active respondents, 23% reported ever having a concurrent sexual partnership with any of the last three partners from the last 12 months. Multivariate analysis found that men and youth (age <25 years), and non-religious people were more likely than their respective counterparts to report concurrency. Respondents reporting concurrency were more likely than those not, to have norms that support multiple partnerships and report low self-efficacy to be faithful to one partner. However, a majority of both groups reported believing that fidelity is important and that they would be looked down upon by family and friends if discovered to have multiple partnerships. The findings suggest that concurrency in Botswana is not uncommon, and yet may not be generally acceptable.



Adjusted PR (95% CI) for predictors of Multiple and Concurrent sexual Partnerships



Moyo et al 2024 *Under Review (JAIDS)*

The Challenge of Multiple Concurrent Partnerships

ABSTRACT

***Background**: Multiple concurrent sexual partnerships (MCP) may drive new HIV infections. We investigated the association of MCP and recent or incident HIV infection in a cluster-randomized HIV prevention trial that followed a population-based HIV incidence cohort across 30 communities in Botswana.*

***Methods**: We used structured questionnaires to evaluate MCP over prior 12 months, defined as either (i) MCP per UNAIDS definition or (ii) concurrent sexual relationship per survey questions. Recent HIV infection was determined using an avidity assay based algorithm or seroconversion within 2 years; incident infection was determined through annual HIV testing for up to 3 years. We estimated prevalence ratios (PRs) and 95% confidence intervals (CIs) for MCP predictors, adjusting for age, gender, and clustering by community.*

***Results**: Data were available for 11,965 (94.9%) of the 12,610 Botswana Combination Prevention Project participants. Among 9,363 sexually active persons in prior 12 months, 2,770 (29.6%) were engaged in MCP. Persons reporting MCP were more likely to be male (aPR=1.6; 95%CI:1.5–1.7), employed (aPR=1.12; 95%CI:1.02–1.2), and to report transactional sex (aPR=1.8; 95%CI:1.5–2.1), intergenerational sex (aPR=1.2; 95%CI:1.1–1.3), high alcohol consumption (aPR=1.5; 95%CI:1.3–1.6), and that their partners had other partners (aPR=2.0; 95%CI:1.8–2.2). Participants who knew their HIV-positive status were less likely to have MCP (aPR=0.8; 95%CI:0.72–0.90). MCP was associated with seroconversion during follow-up (PR=1.5; 95%CI:1.01–2.1) but not with prevalent nor recent HIV infection at baseline.*

***Conclusion**: MCP was common despite efforts to reduce MCP in Botswana and was associated with incident HIV. Knowledge of positive HIV status was associated with ower MCP.*

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Conclusion

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THANKS