HIV Drug Resistance Overview and Application at Primary Health Care

Prof. Tulio de Oliveira
Africa Centre for Health and Population Studies, University of KwaZulu-Natal, South Africa
Research Department of Infection, University College of London (UCL), U.K.
Southern African Treatment Resistance Network (SATuRN)

http://www.bioafrica.net/saturn/
Drug Resistance Mutations

Presence of drug resistance mutations give rise to resistance viruses.

E.g. Mutation M -> V at RT position 184 (M184V).
Clinical effect of drug resistance

No decrease of viral load (> 50 copies/ml) - green line
No increase of CD4 - blue line
Drug resistance accumulation


<table>
<thead>
<tr>
<th>NRTI:</th>
<th>31/03/2004</th>
<th>27/04/2004</th>
<th>22/03/2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>AZT</td>
<td>S</td>
<td>R (215Y 41L)</td>
<td>R (215N 103N)</td>
</tr>
<tr>
<td>DDC</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>DDI</td>
<td>S (184V)</td>
<td>S (215Y 184V 41L)</td>
<td>S (215Y 184V 41L)</td>
</tr>
<tr>
<td>3TC</td>
<td>R (184V)</td>
<td>R (184V)</td>
<td>R (184V)</td>
</tr>
<tr>
<td>D4T</td>
<td>S</td>
<td>R (215Y 41L)</td>
<td>R (215N 103N)</td>
</tr>
<tr>
<td>ABC</td>
<td>S (184V)</td>
<td>S (215Y 184V 41L)</td>
<td>S (215Y 184V)</td>
</tr>
<tr>
<td>FTC</td>
<td>R (184V)</td>
<td>R (184V)</td>
<td>R (184V)</td>
</tr>
<tr>
<td>TDF</td>
<td>S</td>
<td>S (215Y 41L)</td>
<td>S (215Y)</td>
</tr>
<tr>
<td>NNRTI:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NVP</td>
<td>R (103N)</td>
<td>R (103N)</td>
<td>R (103N)</td>
</tr>
<tr>
<td>DLV</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>EFV</td>
<td>R (225Y 103N)</td>
<td>R (225Y 103N)</td>
<td>R (225Y 103N)</td>
</tr>
</tbody>
</table>
Gene sequencing in the clinic: HIV resistance testing

Many ‘resistance mutations’ have been defined for HIV - these are changes in viral protein sequence that are associated with decreased susceptibility to specific drugs

<table>
<thead>
<tr>
<th>Drug</th>
<th>Position</th>
<th>Abacavir</th>
<th>Didanosine</th>
<th>Emtricitabine</th>
<th>Lamivudine</th>
<th>Stavudine</th>
<th>Tenofovir</th>
<th>Zidovudine</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>65</td>
<td>74</td>
<td>115</td>
<td>184</td>
<td>65</td>
<td>65</td>
<td>41</td>
</tr>
<tr>
<td></td>
<td></td>
<td>R</td>
<td>V</td>
<td>V</td>
<td></td>
<td>R</td>
<td>K</td>
<td>N</td>
</tr>
<tr>
<td></td>
<td></td>
<td>65</td>
<td>L</td>
<td></td>
<td></td>
<td>65</td>
<td>M</td>
<td>67</td>
</tr>
<tr>
<td></td>
<td></td>
<td>74</td>
<td>F</td>
<td></td>
<td></td>
<td>41</td>
<td>K</td>
<td>70</td>
</tr>
<tr>
<td></td>
<td></td>
<td>115</td>
<td></td>
<td></td>
<td></td>
<td>70</td>
<td>R</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>184</td>
<td></td>
<td></td>
<td></td>
<td>184</td>
<td>V</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>210 215 219</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>210 215 219</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

IAS-USA 2008
Matrix “score” for mutations

<table>
<thead>
<tr>
<th>Mutation Scoring</th>
<th>AZT</th>
<th>D4T</th>
<th>DDI</th>
<th>DDC</th>
<th>ABC</th>
<th>3TC</th>
<th>NVP</th>
<th>EFV</th>
<th>DLV</th>
</tr>
</thead>
<tbody>
<tr>
<td>M41L</td>
<td>10</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M184V</td>
<td>-10</td>
<td>-5</td>
<td>20</td>
<td>20</td>
<td>20</td>
<td>100</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L210LW</td>
<td>10</td>
<td>5</td>
<td></td>
<td></td>
<td>5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T215Y</td>
<td>30</td>
<td>15</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total:</td>
<td>40</td>
<td>20</td>
<td>30</td>
<td>30</td>
<td>35</td>
<td>100</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Changing Regimens Due to Resistance Profile:

### RT Resistance Mutations:
- M41L, M184V, L210W, T215Y

### RT Other Mutations:
- K10Q, K122E, I178L, R211K

<table>
<thead>
<tr>
<th>Nucleoside RT Inhibitors</th>
<th>Non-Nucleoside RT Inhibitors</th>
</tr>
</thead>
<tbody>
<tr>
<td>AZT</td>
<td>EFV</td>
</tr>
<tr>
<td>DDI</td>
<td>DLV</td>
</tr>
<tr>
<td>DDC</td>
<td>NVP</td>
</tr>
<tr>
<td>D4T</td>
<td></td>
</tr>
<tr>
<td>ABC</td>
<td></td>
</tr>
<tr>
<td>3TC</td>
<td></td>
</tr>
</tbody>
</table>

**Intermediate resistance**

**Low-level resistance**

**High-level resistance**

**Susceptible**

### RT Comments
- M41L increases AZT resistance when present with T215Y/F.
- M184V/I cause resistance to 3TC and low level resistance to ddi, ddc, and ABC.
- L210W increases AZT resistance when present with T215Y or T215F.
- T215Y/F AZT resistance. T215S/C/D represent transitions between T and Y or F.
- The presence of multiple AZT resistance mutations at codons 41, 67, 70, 210, 215, and 219 is associated with low-level d4T and ABC resistance and a decreased virologic response to d4T and ABC-containing regimens.
- M184V partially reverses AZT and possibly d4T resistance caused by other mutations. AZT mutations in this isolate include: M41L, L210LW, T215Y.
SURVEILLANCE OF HIV DRUG RESISTANCE
The threat from transmitted HIV-1 drug resistance

Sub-optimal treatment can lead to the emergence of resistant virus strains.

Transmission of drug resistance strains.

Widespread transmission of resistance could undermine treatment efforts.

- Once transmitted, resistant strains can be ‘archived’ in resting T-cells, and may not be detectable.
- However, they will quickly remerge if regimens to which they are resistant are restarted.

- The WHO has therefore developed a standard protocol for surveillance of transmitted resistance.

Likely reasons for sub-optimal treatment are poor adherence and interruptions in supply.

Drug susceptible infection

Drug resistant infection
Primary (i.e. Transmitted) resistance timeline

Trend in the prevalence of TDR in South Africa, 2000-2010

SATuRN - Surveillance of transmitted resistance

![Graph showing the frequency of mutations over different years.

- **Year**:
  - before-2000
  - 2001-2002
  - 2003-2004
  - 2005-2006
  - 2007-2009

- **Frequency of mutations**:
  - 0.00%
  - 2.50%
  - 5.00%
  - 7.50%
  - 10.00%

- **Data**:
  - n=99
  - n=160
  - n=489
  - n=488
  - n=214

- **Legend**:
  - Any DR mutation

- **Note**:
  - The graph illustrates the trend of mutations over time, with a peak around 2001-2002 and a gradual decrease thereafter.
## Primary & Acquired Drug Resistance

<table>
<thead>
<tr>
<th>Results</th>
<th>TDR 2010 (n=72)</th>
<th>TDR 2011 (n=83)</th>
<th>PDR 2011 (n=320)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Estimates of HIVDR prevalence</td>
<td>0%</td>
<td>7%</td>
<td>5%</td>
</tr>
<tr>
<td>Estimates of HIVDR to &gt;= 2 drugs</td>
<td>0%</td>
<td>2%</td>
<td>2%</td>
</tr>
</tbody>
</table>

*TDR - transmitted drug resistance in sero-converters  
PDR - primary drug resistance in patients before ART

Manasa et al. AHRH 2012, Manasa et al. in preparation
## Primary & Acquired Drug Resistance

<table>
<thead>
<tr>
<th>Results</th>
<th>Adult* (n=240)</th>
<th>Children* (n=88)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Estimates of HIVDR prevalence</td>
<td>87%</td>
<td>83%</td>
</tr>
<tr>
<td>Estimates of HIVDR to &gt;= 2 drugs</td>
<td>84%</td>
<td>71.8%</td>
</tr>
<tr>
<td>GSS for the standard second-line regimen was &lt;2, suggesting a significantly compromised standard regimen</td>
<td>17%</td>
<td>7%</td>
</tr>
</tbody>
</table>

- Average time on therapy: 47 months (Adults) vs. 39 months (Children)
- Average time on failing regimen: 27 months (Adults) vs. 20 months (Children)

*Adults and children with viral load > 1,000 on failing regimen.*

Viral load monitoring and recommended responses

- **Viral load measurement**
  - **VL <400 copies/ml**
    - Routine monitoring as per schedule
    - Adherence support
  - **VL 400-1000 copies/ml**
    - Adherence assessment (and intervention)
    - Repeat VL 6 months
  - **VL >1000 copies/ml**
    - Intense adherence assessment (and intervention)
    - Repeat VL 3 months

  - **VL ≤1000 copies/ml**
    - Return to monitoring as per schedule
  - **VL >1000 copies/ml**
    - If adherence issues addressed, switch to second-line regimen
First - Free State Cohort

Prevalence of primary resistance 2008: 10/425 (2.3%)

Prevalence of treated resistance:
1st line Adult: 138/154 (89.54%)
2nd line Adult: 16/45 (11.1% PI)

Abstract IAS and SA AIDS 2011:
- Van Vurren et al. HIV Drug resistance in adult patients failing first-line antiretroviral therapy (ART) in the Free State Province of South Africa.
SATuRN’s APPROACH TO DRUG RESISTANCE
SATuRN Vision

Develop advanced yet affordable HIV & TB drug resistance diagnostics, implement it at primary health care clinics in resource limited settings and create a collaborative system for surveillance, research and capacity building.
What is the SATuRN?

a network consisting of biomedical scientists, clinicians, epidemiologists and public health experts

SATuRN managed at the Africa Centre and the SA-MRC

CURRENT PARTNERS includes 24 partners in southern Africa

FUNDERS AND COLLABORATORS believe that the system has the potential to be implemented nationally, that is the reason for the meeting with the DoH and funders.

Collaborators & implementation sites info at www.bioafrica.net/saturn/
Genotyping system is the most accurate diagnostic test for HIV drug resistance. It was prohibited for national implementation due to its cost and complexity.

AFFORDABLE & OPEN ACCESS GENOTYPING

Genotyping price reduced from US$ 250 to Approx. US$ 50 per sample (Sanger ABI).

Following an open letter submitted to Life Technologies on the need for cheap resistance testing in Africa, a discount of nearly 50% on all sequencing reagents has been agreed.

We have now started working with Sanger, which should reduce the cost to approximately US$ 20 per genotype.
Research & Development: Bioinformatics databases and tools

Bioinformatics system for HIV and other pathogens

PUBLIC & FREE OPEN SOURCE SOFTWARE

Use of gold, standard, free open source software databases and tools.

RegaDB for clinical management and Stanford HIVDB for genotypes after publication.

We aim to enhance the capacity of physicians and scientists to produce research results and of policy makers to use research evidence.

Can our clinical management model replicated nationally?

Suggested by the South African HIV Clinician Society as the system to be used for national implementation

South Africa Department of Health (SADoH) official newsletter: Impilo dialogues.

Suggested for national implementation by the NDoH Drug Resistance Workgroup.

Operation and inclusion in the functioning of the provincial ART program

Databases and resistance genotyping implemented officially as part of National programme
17 PHC Clinics in rural KwaZulu-Natal

Medical officer

HIV specialist

Africa Centre Lab (UKZN)

RegaDB MRC

Transport by road
Internet access
Email (computer/cell)
Anonymized study name
No clinical and laboratory information - no genotype.

Objective is to provide enough information to be used for clinical decision.

Laboratory tests: VL, CD4, HBV, Creatinine clearance

Treatment: current and previous regimens

Demographic: age, gender

Other OIs: TB and other Ois

Adherence: questions, social worker interview
Dear Clinician,

I enclose the report of the genotyping that you requested.

Patient ID on the SATuRN Rega database: RES007 /
*Please notice that no patient personal identification information should be stored in this database, please use an sequential study number as patientID.

Sample ID / Sample Date: ACRES007 - 19/01/2011
Antiretroviral experience: [D4T, 3TC, TDF, EFV, LPV/r]
Subtype: HIV-1 Subtype C
Resistance interpretations: HIVDB 6.0.5

<table>
<thead>
<tr>
<th>Drug</th>
<th>Mutations</th>
<th>Description</th>
<th>Level</th>
<th>GSS</th>
</tr>
</thead>
<tbody>
<tr>
<td>zidovudine</td>
<td>184V</td>
<td>Susceptible</td>
<td>1</td>
<td>1.0</td>
</tr>
<tr>
<td>zalcitabine</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>didanosine</td>
<td>184V</td>
<td>Susceptible</td>
<td>1</td>
<td>1.0</td>
</tr>
<tr>
<td>lamivudine</td>
<td>184V</td>
<td>High-level resistance</td>
<td>5</td>
<td>0.0</td>
</tr>
<tr>
<td>stavudine</td>
<td>184V</td>
<td>Susceptible</td>
<td>1</td>
<td>1.0</td>
</tr>
<tr>
<td>abacavir</td>
<td>184V</td>
<td>Potential low-level resistance</td>
<td>2</td>
<td>1.0</td>
</tr>
<tr>
<td>emtricitabine</td>
<td>184V</td>
<td>High-level resistance</td>
<td>5</td>
<td>0.0</td>
</tr>
<tr>
<td>tenofovir</td>
<td>184V</td>
<td>Susceptible</td>
<td>1</td>
<td>1.0</td>
</tr>
<tr>
<td>nevirapine</td>
<td>106M 190A</td>
<td>High-level resistance</td>
<td>5</td>
<td>0.0</td>
</tr>
<tr>
<td>delavirdine</td>
<td>106M</td>
<td>High-level resistance</td>
<td>5</td>
<td>0.0</td>
</tr>
<tr>
<td>efavirenz</td>
<td>106M 190A</td>
<td>High-level resistance</td>
<td>5</td>
<td>0.0</td>
</tr>
<tr>
<td>etravirine</td>
<td>106M 190A</td>
<td>Low-level resistance</td>
<td>3</td>
<td>0.5</td>
</tr>
<tr>
<td>saquinavir</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>saquinavir/rt</td>
<td>N/A</td>
<td>Susceptible</td>
<td>1</td>
<td>1.0</td>
</tr>
<tr>
<td>ritonavir</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>indinavir</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>indinavir/rt</td>
<td>N/A</td>
<td>Susceptible</td>
<td>1</td>
<td>1.0</td>
</tr>
<tr>
<td>nelfinavir</td>
<td>N/A</td>
<td>Susceptible</td>
<td>1</td>
<td>1.0</td>
</tr>
<tr>
<td>fosamprenavir</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>fosamprenavir/rt</td>
<td>N/A</td>
<td>Susceptible</td>
<td>1</td>
<td>1.0</td>
</tr>
<tr>
<td>lopinavir</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>atazanavir</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>atazanavir/rt</td>
<td>N/A</td>
<td>Susceptible</td>
<td>1</td>
<td>1.0</td>
</tr>
<tr>
<td>tipranavir</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>darunavir</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
</tr>
</tbody>
</table>

Additional:
- Antiretrovirals for which the virus showed a reduced sensitivity, may still be partially active in a combination therapy. Antiretrovirals agents against resistant virus are not recommended but may still exhibit a temporary activity when on HAART (> 3 Antiretrovirals).
- The interpretations of the genotypic and phenotype (PI) tests are based on limited clinical information. These interpretations should be taken with care.

List of all amino acid mutations observed in:

Clinical chart and resistance interpretation:
This individual has resistance to two of the three ARVs that she currently on. She has High-level resistance to the NNRTI, EFavirenz (EFV) and the NRTI, Lamivudine (3TC). Her HIV population has the NNRTI mutation K103N and V106M. For resistance to NRTIs there is the 3TC specific mutation M184V. The currently circulating viral population is still susceptible to Tenofovir (TDF).

This patient’s viral load has never fully suppressed in had a very good immunological response after the initiation of therapy. However this lasted for less than a year only and the CD4 count started on a downward trend. Her last three viral loads done in a space of fifteen months have all been above 2000 RNA copies/ml.

I.D. treatment switch suggestion: !
Interpretation of genotype: This patient has not accumulated any TAMs or TDF resistance, despite failing for quite some time.

Adherence: Intensive adherence support is needed and the use of alternative remedies and social deterrents to adherence should be thoroughly explored.

Treatment recommendation: Since the virus is still susceptible to TDF, the patient should do well on a standard second line consisting of TDF, 3TC and LPV/r.

General comments: The renal function should be monitored before initiation and again at three months. If the patient has a high risk if renal disease, pre-existing renal compromise (especially HT and DM patients)
Clinical chart and resistance interpretation:
This individual has resistance to two of the three ARVs that she currently on. She has High-level resistance to the NNRTI, EFavirenz (EFV) and the NRTI, Lamivudine (3TC). Her HIV population has the NNRTI mutation K103N and V106M. For resistance to NRTIs there is the 3TC specific mutation M184V. The currently circulating viral population is still susceptible to Tenofovir (TDF).
I.D. treatment switch suggestion: !

Interpretation of genotype: This patient has not accumulated any TAMs or TDF resistance, despite failing for quite some time.

Adherence: Intensive adherence support is needed and the use of alternative remedies and social deterrents to adherence should be thoroughly explored.

Treatment recommendation: Since the virus is still susceptible to TDF, the patient should do well on a standard second line consisting of TDF, 3TC and LPV/r.

General comments: The renal function should be monitored before initiation and again at three months. If the patient has a high risk if renal disease, pre-existing renal compromise (especially HT and DM patients)
This patient has high-level resistance to two of the three antiretroviral drug that she is currently taking. She has high-level resistance to the NRTI Lamivudine associated with the M184V mutation. For the NNRTI (Efavirenz), the high level resistance is associated with the K103S and V106M mutations. Her viral isolate is still susceptible to Tenofovir.

The first two years of this patient’s antiretroviral therapy were associated with complete viral suppression. However, she stopped therapy after the second quarter of 2010 and her subsequent viral load was 8,200 RNA Copies/ml. Even after resume her ART she never managed to re-suppress her viral, her last three viral loads were greater than 10,000 RNA Copies/ml.
Interpretation of clinical chart & genotype: This patient had a very good virological and immunological response to treatment for the first two years. Unfortunately she then had an unscheduled treatment interruption which led to a rapid decline in CD4+ cell count back to pre-treatment levels...

Adherence: It seems that initially adherence was very good but that the treatment interruption itself might have led to the emergence of resistance. It is unfortunate that the patient stopped treatment without seeking care and advice at the clinic and this suggests suboptimal treatment literacy or incomplete trust in the clinic staff. The patient should be strongly encouraged to identify a reliable treatment supporter (ideally the mother rather than the six-year old child!) and to bring that supporter to adherence counselling before starting second-line therapy...

Treatment recommendation: I recommend that the patient is switched to the second-line regimen of TDF/3TC/LPVr. Avoidance of TDF might have been preferred given the likely concurrent use of kanamycin for MDR-TB and the combined risk of nephrotoxicity. However, I note the Hb is 7.7 g/dL and therefore AZT would be inadvisable. If the clinical condition is stable, I would recommend delaying the treatment switch until after at least two weeks of MDR-TB treatment, by which time rifampicin will have been eliminated and normal dose of LPVr can be used.

General comments: U&Es should be monitored at least monthly whilst on the intensive phase of MDR-TB treatment. There should also be close clinical monitoring for GI toxicity. The viral load should be checked in six months....
Learn how to diagnose, manage and prevent drug resistance in HIV and TB

Drug resistance is one of the main challenges confronting HIV and TB programmes in Africa. Facing these challenges requires understanding of how drug resistance develops as well as up-to-date knowledge of how to diagnose and manage drug-resistant HIV and TB disease. This book uses a case-based learning approach to present to health care workers the most important information needed to offer their patients the best possible care, and to improve their programmes to prevent the emergence and spread of drug resistance.

- Learn how drug resistance develops in HIV and Mycobacterium tuberculosis
- Understand when to suspect drug resistance and which diagnostic tests can be used to diagnose resistance
- Get up-to-date knowledge on how to interpret genotypic resistance tests for HIV and new diagnostic tests for TB, including Xpert® MTB/RIF
- Acquire information on how to select second-line regimens for HIV and TB and how to offer comprehensive care for patients with drug-resistant HIV and TB

Some comments from reviewers of the HIV & TB Drug Resistance & Clinical Management Case Book

“This book is an excellent practical compendium of knowledge in the field of HIV and TB drug resistance that will be of immense use to clinicians, nurses, and other caregivers. It provides expert guidance as to what to do in difficult clinical situations and what steps should be taken to prevent the emergence and transmission of drug resistance.”

Professor Mark A. Wainberg, McGill University, Canada, former president of the International AIDS Society (IAS)

“This is indeed a wonderful book that will be very handy for clinicians, virologists and bacteriologists. It presents a simple but expert introduction to the field of HIV & TB drug resistance, including a brief review of technical details that are needed to understand the cases. The cases are presented in a very structured way, and I specifically like the key learning points. I have no doubt that this will be a book on everybody’s desk, either in the office or in the clinic.”

Professor Anne-Mieke Vandamme, Rega Institute, AIDS Reference Laboratory, Belgium
SATuRN HIV & TB Drug Resistance Case Book

Roland van Geer
Ambassador of the EU to South Africa, authors and Ronnie Anderson of UP.

8,000 copies being distributed to health care workers, book used by many teaching medical.

Book open accessible at Google Book and SATuRN website: www.bioafrica.net/saturn
CAPACITY BUILDING AND COMMUNITY ENGAGEMENT
SATuRN education and capacity building

In our previous 6 editions we have trained > 2,500 participants (mostly physicians, health care workers and researchers trained).


Open access books and reports produced by SATuRN:

HIV/TB Drug Resistance & Clinical Management Case Book
SATuRN Life Technologies Discounted genotyping manual
SATuRN 2013 Annual Report

Website: www.bioafrica.net/saturn

Popular website with 200 unique visits a day. Interaction with the community by blogs, news, twitter.
8th Southern African HIV & TB Drug Resistance & Treatment Monitoring Workshop

The workshop includes theoretical lectures and practical sessions on the usage and interpretation of HIV-1 and TB drug resistance genotyping in the management of HIV patients on anti-retroviral (ARV) treatment.

Who should apply?
The workshop is targeted at clinicians, clinical virologists, nurses, medical students and researchers working in the public and private sector who are currently involved in the treatment of patients with HIV & TB.

Invited Presenters:

Deadline for application: 10 October 2013.
Venue: Free State School of Medicine, Bloemfontein.
Date of the workshop: 20 to 22 November 2013.

Registration is free of charge
workshop includes CPD points, lectures and refreshments.

Organizers: Tulio de Oliveira, Tobias Rinke de Wit, Chris Seebregts, Dewald Steyn, Cloete van Vuuren, Dominique Goehals.

Contact: Xolile Kineri, email: xkineri@afriacentre.ac.za,
Tel: +27 35 550 7500, Fax: +27 35 550 7565

Application forms & information at http://www.blicafrica.net
More info: www.bioafrica.net/saturn