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COMISCA
CONSEJO DE MINISTROS DE SALUD DE CENTROAMÉRICA Y REPÚBLICA DOMINICANA

SICA
Sistema de la Integración Centroamericana

ENCUENTRO LECCIONES APRENDIDAS Y EXPERIENCIAS EXITOSAS EN LA

ATENCION *Integral* DE VIH, TUBERCULOSIS Y COINFECCIÓN VIH/TB

San Salvador 19 y 20 de marzo de 2019

HIV transmitted drug resistance surveillance in Guatemala 2010-2013

[1]Centre for Research in Infectious Diseases, National Institute of Respiratory Diseases, Mexico City, Mexico. [2] Infectious Diseases Clinic, Roosevelt Hospital, Guatemala City, Guatemala.

Avila-Ríos S[1], García-Morales C[1], Garrido-Rodríguez D[1], Tapia-Trejo D[1], Girón-Callejas AC[2], Mendizábal-Burastero R[2], Escobar I[2], García L[2], Navas S[2], Pinzón-Meza R[2], Mejía-Villatoro CR[2], Reyes-Terán G[1]

Introducción

La expansión de la terapia ARV ha favorecido el incremento de la resistencia primaria transmitida.

Por lo cual se requieren estudios para valorar la prevalencia de la misma, para establecer las pautas terapéuticas a seguir en los sistemas de salud.

Estudio Mesoamericano

- ✓ Se enrolaron pacientes desde octubre del 2010 a septiembre del 2013 que asistían a la Clínica de Enfermedades Infecciosas en coordinación con el CIENI –INER.
- ✓ Los criterios de resistencia se basaron en los criterios de OMS.
- ✓ Para el análisis de resistencia se utilizo la herramienta de Stanford
- ✓ Las recombinantes y los sub tipos no-B se confirmaron usando la herramienta de la base de datos de VIH de Alamos.

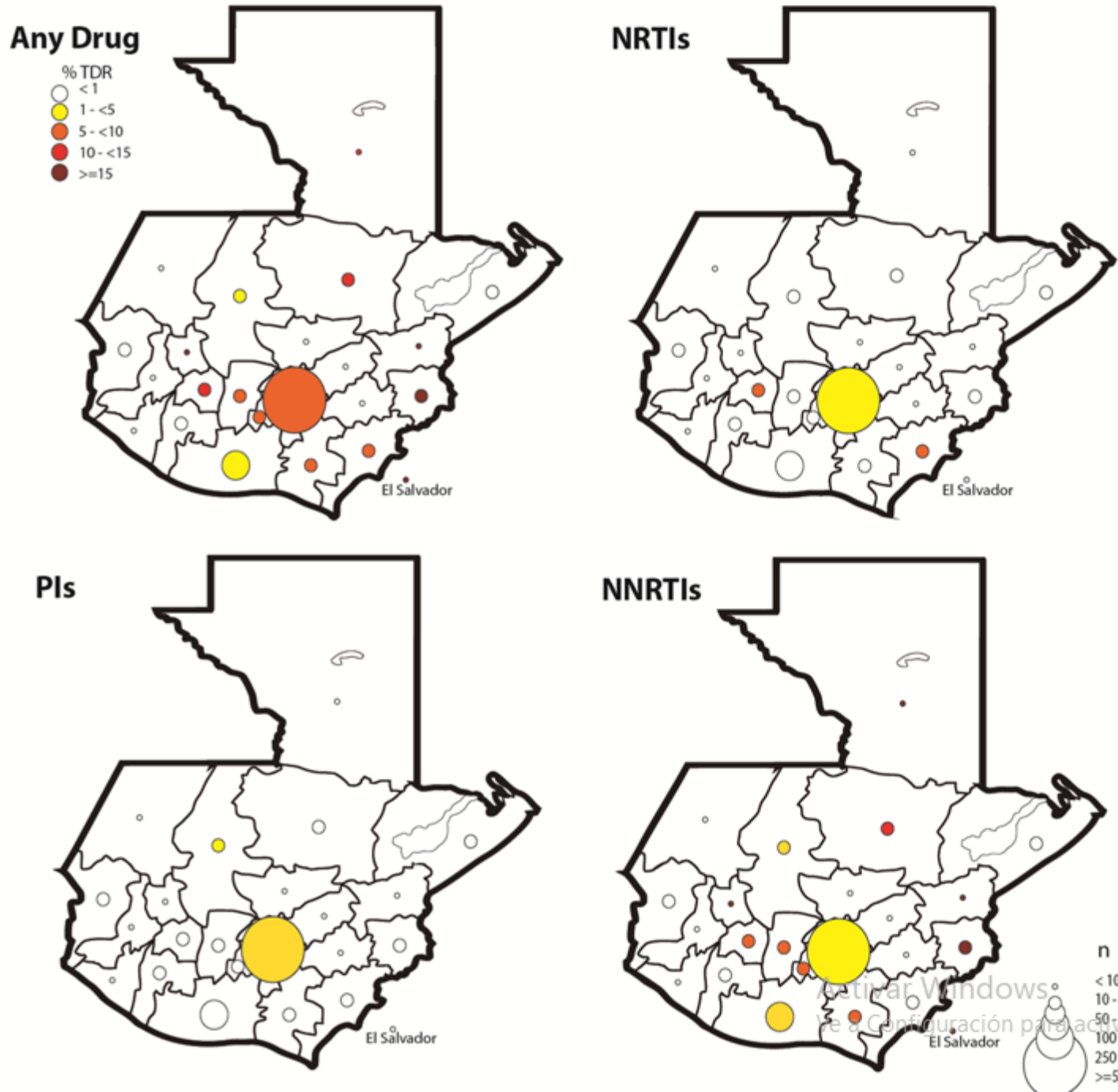


Figure 2. Geographic distribution of HIV TDR in Guatemala 2010-2013. TDR was estimated for 1035 HIV sequences from ART-naïve Guatemalan individuals enrolled at the Roosevelt Hospital in Guatemala City, using WHO mutation list for HIV TDR surveillance. TDR prevalence for each department was estimated. Circle size reflects the number of patients enrolled from each department.

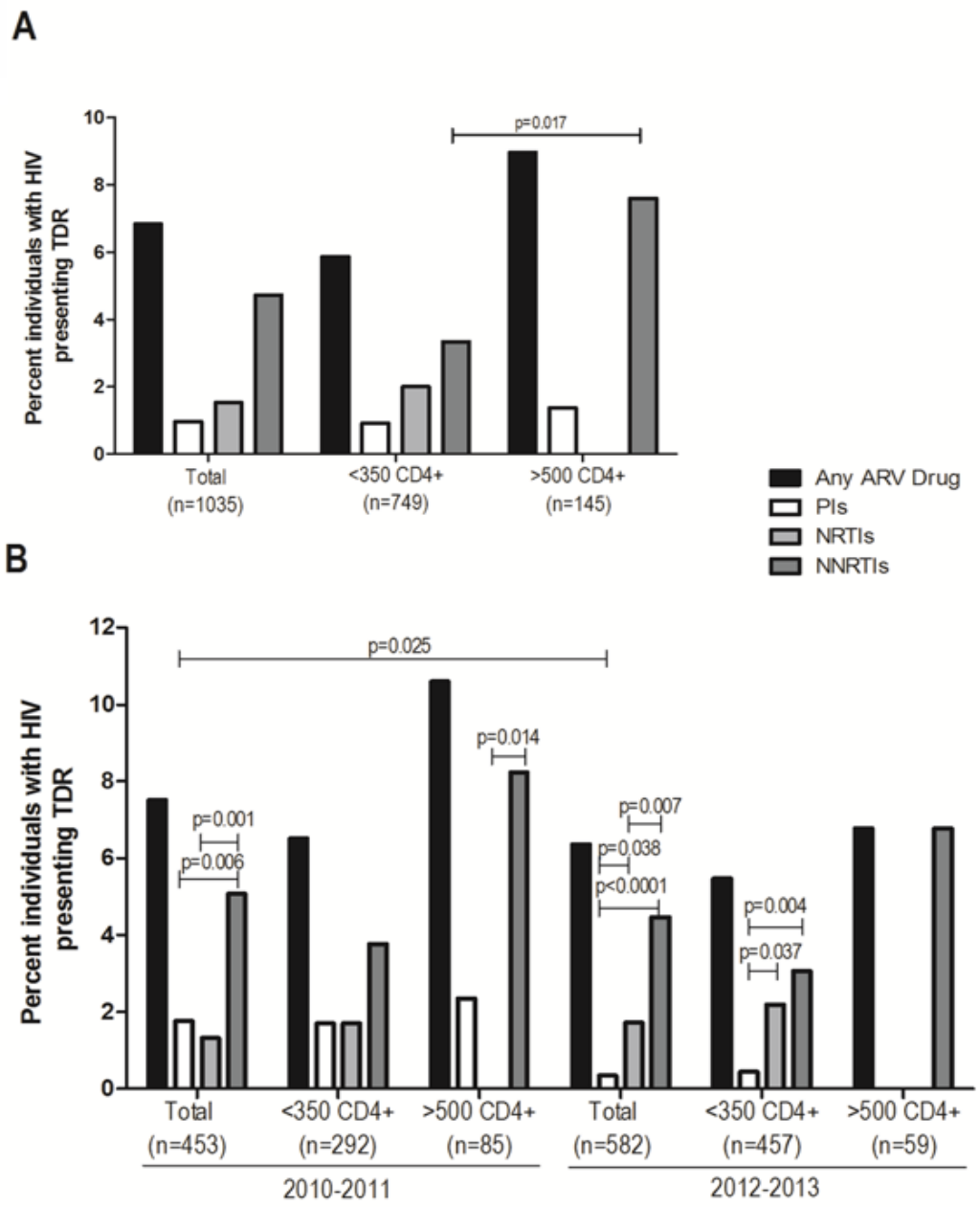


Figure 1. HIV TDR prevalence in Guatemala 2010-2013. TDR was estimated for 1035 HIV sequences from ARVT-naïve Guatemalan individuals enrolled at the Roosevelt Hospital in Guatemala City, using the WHO mutation list for HIV TDR surveillance. A. TDR prevalence for the whole dataset including TDR estimations in recently infected individuals (>500 CD4+ T cells/μL) and individuals starting ART (<350 CD4+ T cells/μL). B. TDR estimations divided by year of enrolment, including prevalence in recently infected and pre-ART individuals. Group comparisons were performed with Chi square test. Significant differences are shown.

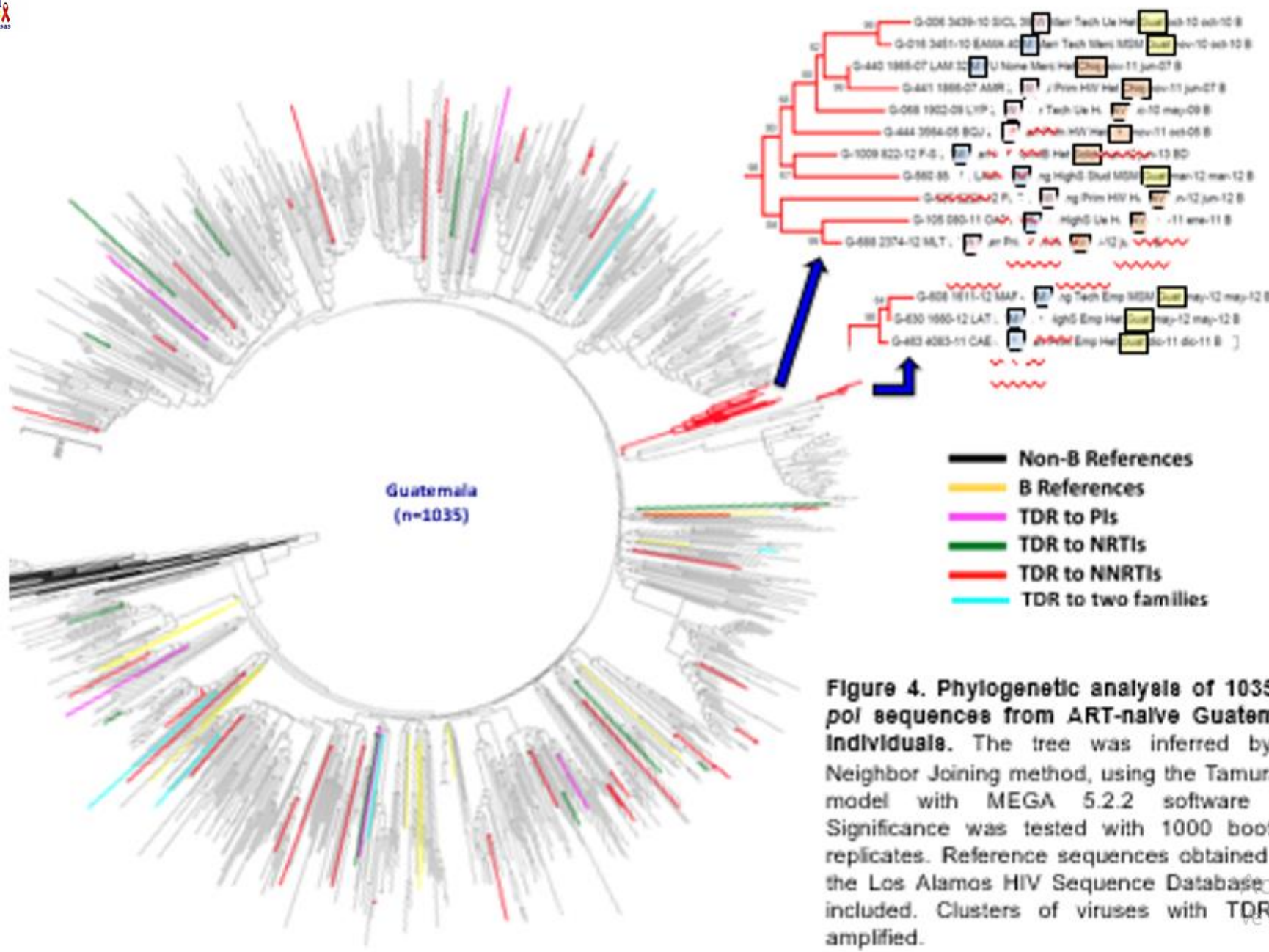


Figure 4. Phylogenetic analysis of 1035 HIV *pol* sequences from ART-naive Guatemalan individuals. The tree was inferred by the Neighbor Joining method, using the Tamura Nei model with MEGA 5.2.2 software [19]. Significance was tested with 1000 bootstrap replicates. Reference sequences obtained from the Los Alamos HIV Sequence Database were included. Clusters of viruses with TDR are amplified.

Table 3. Prevalence of TDR mutations in a cohort of 1035 Guatemalan individuals.

Protease Inhibitors (PIs)			Nucleoside RT Inhibitors (NRTIs)			Non Nucleoside RT Inhibitors (NNRTIs)		
Mutation	Frequency in cohort [n(%)]	Frequency in Individuals with TDR [n(%)][a]	Mutation	Frequency in Cohort [n(%)]	Frequency in individuals with TDR [n(%)][a]	Mutation	Frequency in Cohort [n(%)]	Frequency in individuals with TDR [n(%)][a]
L10IV	135 (13.0)	2 (20.0)	M41L-WHO	4 (0.4)	4 (25.0)	V90I	0 (0.0)	0 (0.0)
L10F	0 (0.0)	0 (0.0)	M41R	0 (0.0)	0 (0.0)	A98G	0 (0.0)	0 (0.0)
V11I	3 (0.3)	0 (0.0)	E44D	3 (0.3)	1 (6.3)	**L100I-WHO**	**0 (0.0)**	**0 (0.0)**
K20I	2 (0.2)	0 (0.0)	A62V	2 (0.2)	0 (0.0)	K101Q	0 (0.0)	0 (0.0)
L23I-WHO	**1 (0.1)**	**1 (10.0)**	**K65R-WHO**	**0 (0.0)**	**0 (0.0)**	K101N	2 (0.2)	2 (4.1)
L24I-WHO	**1 (0.1)**	**1 (10.0)**	D67T	0 (0.0)	0 (0.0)	**K101E-WHO**	**15 (1.5)**	**15 (30.6)**
D30N-WHO	**0 (0.0)**	**0 (0.0)**	D67H	0 (0.0)	0 (0.0)	**K103NS-WHO**	**28 (2.7)**	**28 (57.1)**
V32I-WHO	**0 (0.0)**	**0 (0.0)**	**D67NG-WHO**	**0 (0.0)**	**0 (0.0)**	K103R	0 (0.0)	0 (0.0)
L33F	7 (0.7)	0 (0.0)	**D67E-WHO**	**2 (0.2)**	**2 (12.5)**	**V106A-WHO**	**0 (0.0)**	**0 (0.0)**
E35G	0 (0.0)	0 (0.0)	T69A	1 (0.1)	0 (0.0)	**V106M-WHO**	**2 (0.2)**	**2 (4.1)**
K43T	3 (0.3)	0 (0.0)	**T69D-WHO**	**1 (0.1)**	**1 (6.3)**	V108I	15 (1.5)	1 (2.0)
M46IL-WHO	**4 (0.4)**	**4 (40.0)**	**T69ins-WHO**	**0 (0.0)**	**0 (0.0)**	E138KQ	12 (1.2)	10 (20.4)
I47A-WHO	**0 (0.0)**	**0 (0.0)**	T69N	12 (1.2)	2 (12.5)	E138GAR	12 (1.2)	1 (2.0)
I47V-WHO	**0 (0.0)**	**0 (0.0)**	T69C	0 (0.0)	0 (0.0)	V179AT	30 (2.9)	0 (0.0)
G48VM-WHO	**0 (0.0)**	**0 (0.0)**	T69I	0 (0.0)	0 (0.0)	V179D	39 (3.8)	1 (2.0)
I50L-WHO	**0 (0.0)**	**0 (0.0)**	T69G	0 (0.0)	0 (0.0)	V179E	16 (1.6)	0 (0.0)
I50V-WHO	**0 (0.0)**	**0 (0.0)**	T69S	3 (0.3)	1 (6.3)	**V179F-WHO**	**0 (0.0)**	**0 (0.0)**
F53L-WHO	**0 (0.0)**	**0 (0.0)**	K70G	0 (0.0)	0 (0.0)	**Y181IV-WHO**	**0 (0.0)**	**0 (0.0)**
F53Y-WHO	**1 (0.1)**	**0 (0.0)**	K70N	0 (0.0)	0 (0.0)	**Y181C-WHO**	**0 (0.0)**	**0 (0.0)**
I54VA-WHO	**1 (0.1)**	**1 (10.0)**	**K70R-WHO**	**0 (0.0)**	**0 (0.0)**	**Y188L-WHO**	**0 (0.0)**	**0 (0.0)**
I54L-WHO	**0 (0.0)**	**0 (0.0)**	**K70E-WHO**	**0 (0.0)**	**0 (0.0)**	**Y188H-WHO**	**1 (0.1)**	**1 (2.0)**
I54M-WHO	**0 (0.0)**	**0 (0.0)**	**L74I-WHO**	**0 (0.0)**	**0 (0.0)**	**Y188C-WHO**	**0 (0.0)**	**0 (0.0)**
I54ST-WHO	**0 (0.0)**	**0 (0.0)**	**L74V-WHO**	**2 (0.2)**	**2 (12.5)**	**G190S-WHO**	**0 (0.0)**	**0 (0.0)**
Q58E	9 (0.9)	0 (0.0)	V75L	1 (0.1)	0 (0.0)	**G190A-WHO**	**1 (0.1)**	**1 (2.0)**
A71IVT	168 (16.2)	1 (10.0)	V75I	1 (0.1)	0 (0.0)	**G190E-WHO**	**1 (0.1)**	**1 (2.0)**
G73CSTA-WHO	**0 (0.0)**	**0 (0.0)**	**V75A-WHO**	**0 (0.0)**	**0 (0.0)**	G190C	0 (0.0)	0 (0.0)
T74S	0 (0.0)	0 (0.0)	**V75T-WHO**	**1 (0.1)**	**1 (6.3)**	**P225H-WHO**	**2 (0.2)**	**2 (4.1)**
L76V-WHO	**0 (0.0)**	**0 (0.0)**	**V75S-WHO**	**0 (0.0)**	**0 (0.0)**	F227L	0 (0.0)	0 (0.0)
V82A-WHO	**0 (0.0)**	**0 (0.0)**	**V75M-WHO**	**1 (0.1)**	**1 (6.3)**	M230L-WHO	0 (0.0)	0 (0.0)
V82F-WHO	**0 (0.0)**	**0 (0.0)**	**F77L-WHO**	**1 (0.1)**	**1 (6.3)**	L234I	0 (0.0)	0 (0.0)
V82T-WHO	**0 (0.0)**	**0 (0.0)**	**Y115F-WHO**	**0 (0.0)**	**0 (0.0)**	K238T	1 (0.1)	0 (0.0)
V82S-WHO	**0 (0.0)**	**0 (0.0)**	**F116Y-WHO**	**0 (0.0)**	**0 (0.0)**	Y318F	0 (0.0)	0 (0.0)
V82M-WHO	**1 (0.1)**	**1 (10.0)**	V118I	54 (5.2)	3 (18.8)			
V82C-WHO	**0 (0.0)**	**0 (0.0)**	**Q151M-WHO**	**0 (0.0)**	**0 (0.0)**			
V82L-WHO	**0 (0.0)**	**0 (0.0)**	**M184VI-WHO**	**1 (0.1)**	**1 (6.3)**			
N83D-WHO	**1 (0.1)**	**1 (10.0)**	**L210W-WHO**	**1 (0.1)**	**1 (6.3)**			
I84VAC-WHO	**0 (0.0)**	**0 (0.0)**	**T215Y-WHO**	**0 (0.0)**	**0 (0.0)**			
I85V-WHO	**0 (0.0)**	**0 (0.0)**	T215A	2 (0.2)	0 (0.0)			
N88D-WHO	**0 (0.0)**	**0 (0.0)**	**T215F-WHO**	**0 (0.0)**	**0 (0.0)**			
N88S-WHO	**0 (0.0)**	**0 (0.0)**	**T215CDESIV-WHO**	**2 (0.2)**	**2 (12.5)**			
			K219QEN-WHO	**2 (0.2)**	**2 (12.5)**			
			K219R-WHO	**0 (0.0)**	**0 (0.0)**			
			G333D	0 (0.0)	0 (0.0)			
			G333E	0 (0.0)	0 (0.0)			

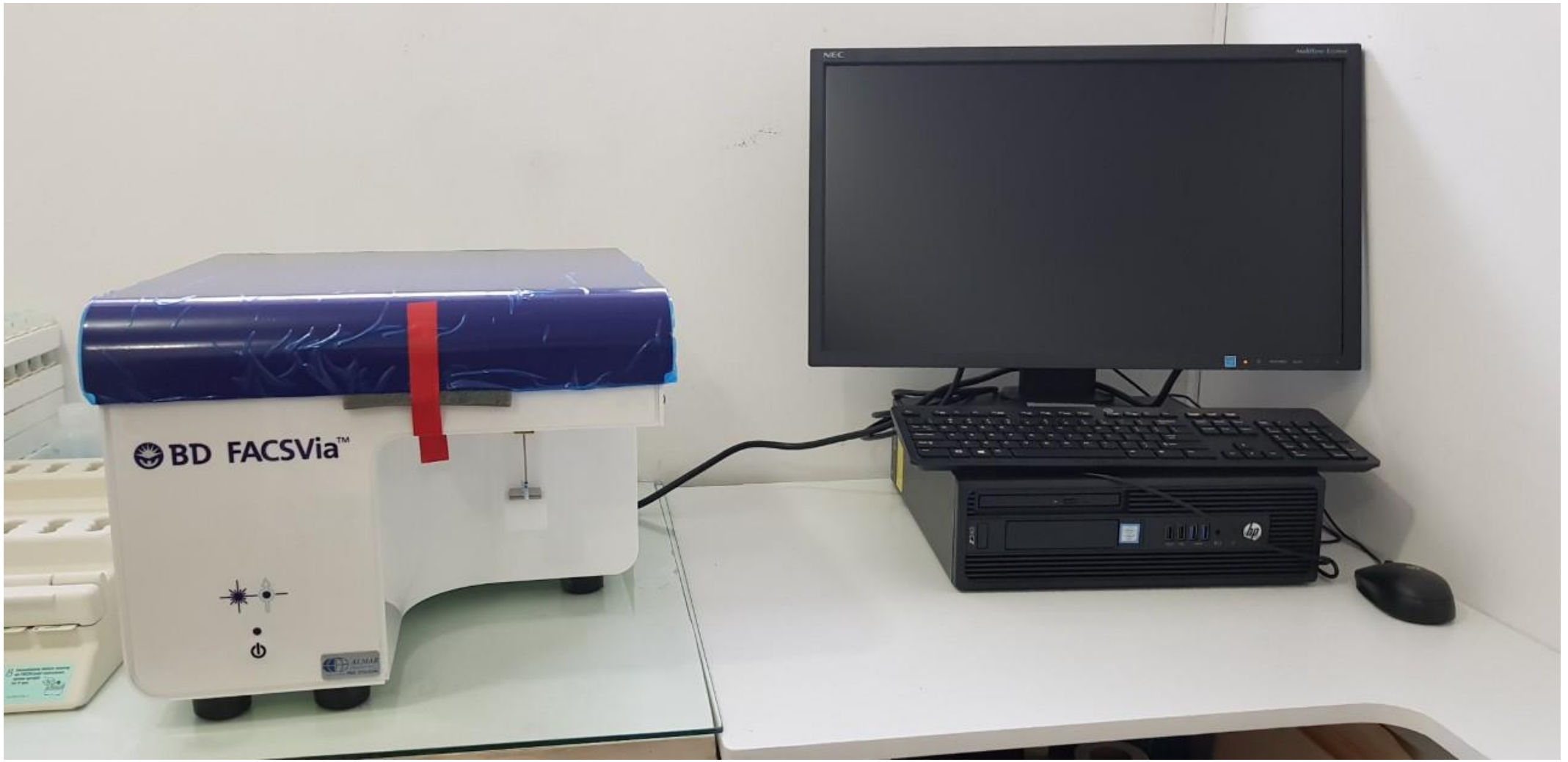
Resultados

- La resistencia primaria transmitida fue del 6.89% , en el total de la cohorte durante el periodo de estudio.

- La resistencia primaria trasmitida a los NNRTI fue el más alto 4.73%, NRTI 1.55% y de los inhibidores de proteasa 0.97%


LABORATORIO
Hospital
Roosevelt


cobas x 480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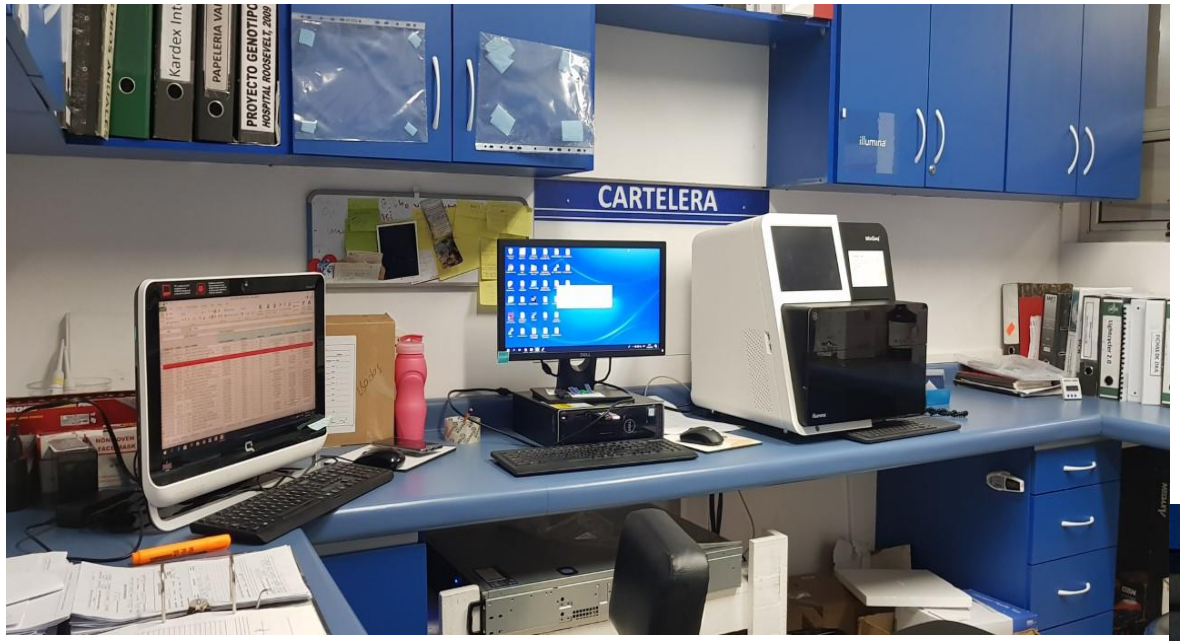
CARTELERA
PROYECTO GENOTIPO HOSPITAL ROOSEVELT 2009
illumina


CARTELERA
illumina
MiniSeq

Datos Recientes

Genotipos procesados localmente de enero 2018 – enero 2019

Genotipos basales procesados	115
Genotipos de resistencia secundaria	201
total	316

Genotipos

Tipo de Resistencia	**Familia**	**Familia**	**Familia**
	NRTI	**NNRTI**	**IP**
Resistencia primaria transmitida (n= 115)	1 (0.9%)	9 (7.8%)	5 (4.35%)
Resistencia secundaria (n = 201)	121 (60%)	138 (68%)	12 (6 %)

Resistencia secundaria N = 201	Masculinos	Femeninos
Adultos	79	85
Pediátricos	24	13
Resistencia primaria N=115		
Adultos	101	14

¡MUCHAS GRACIAS!

Solidaridad entre los pueblos para la integración regional en salud

www.sica.int/comisca info.comisca@sica.int (503) 2248 6901

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