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Eastern Europe and central Asia, the Middle East and North Africa and Latin America have all seen increases in annual HIV infections over the past decade.

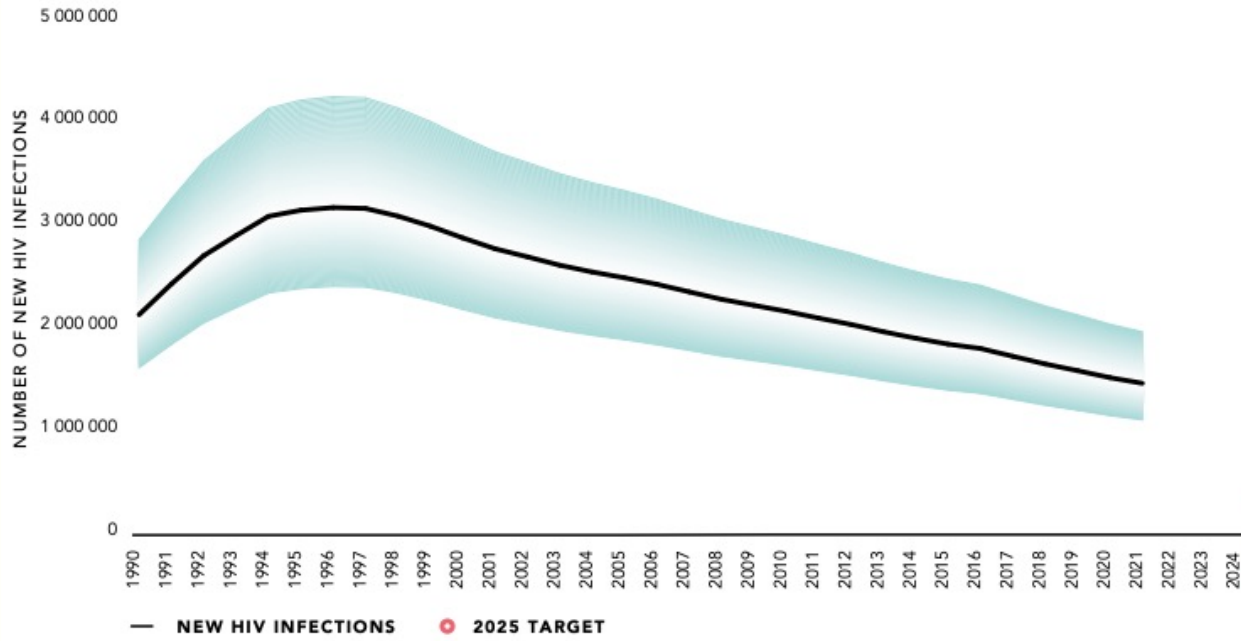
**4000**

PEOPLE BECOME INFECTED  
WITH HIV EVERY DAY

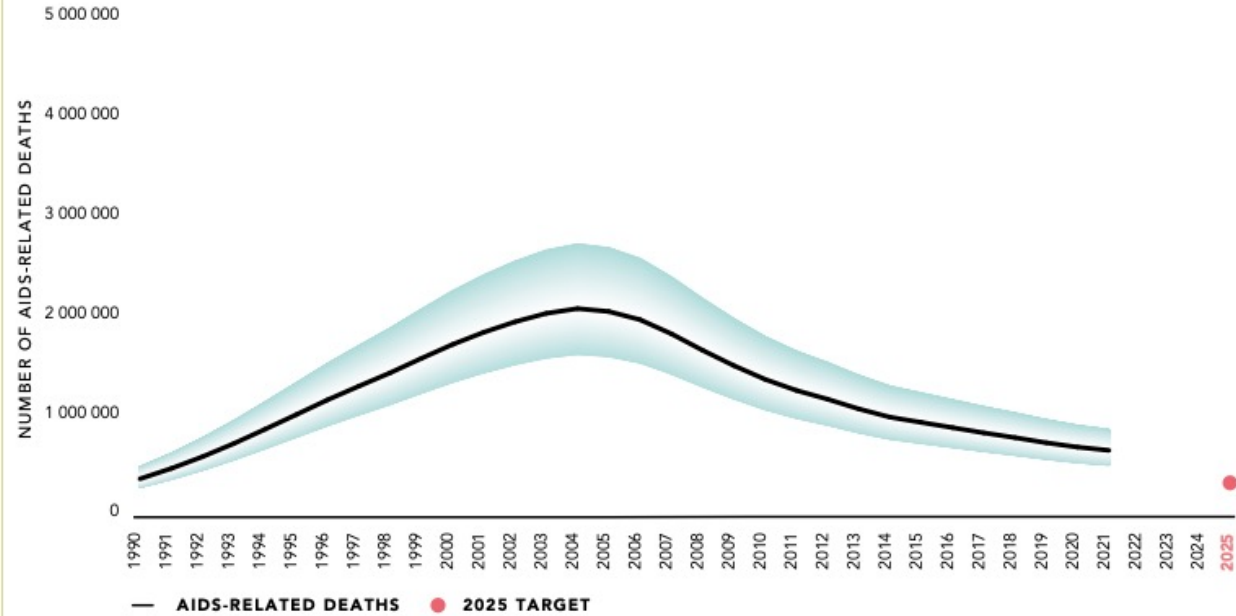
**650 000**

PEOPLE DIED OF AIDS-  
RELATED CAUSES IN 2021

**FIGURE 1.3** Number of new HIV infections, global, 1990–2021, and 2025 target

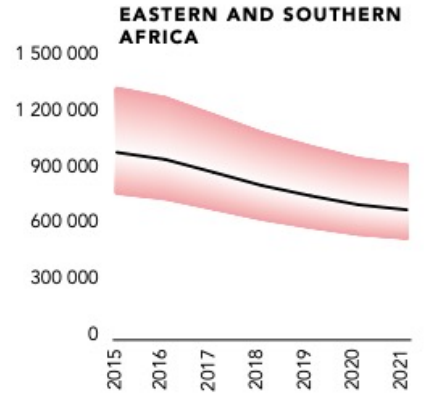
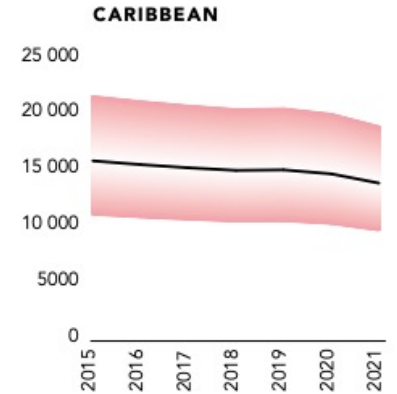
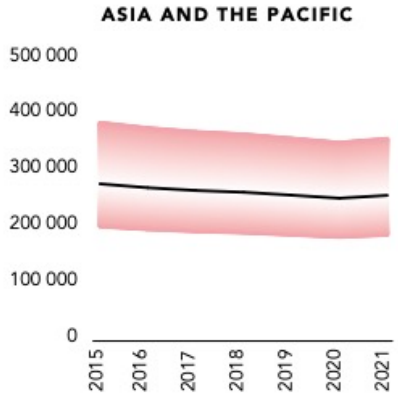


**FIGURE 1.10** Number of AIDS-related deaths, global, 1990–2021, and 2025 target

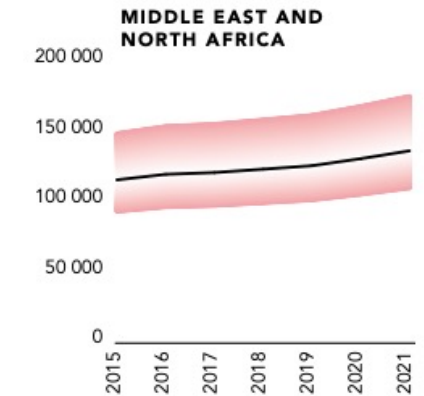
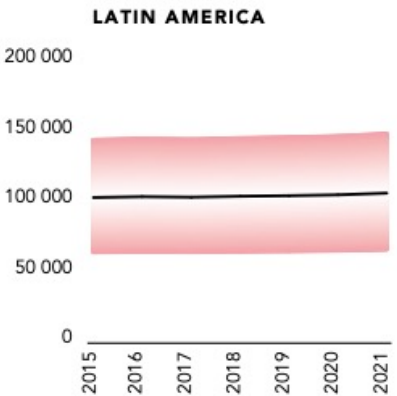
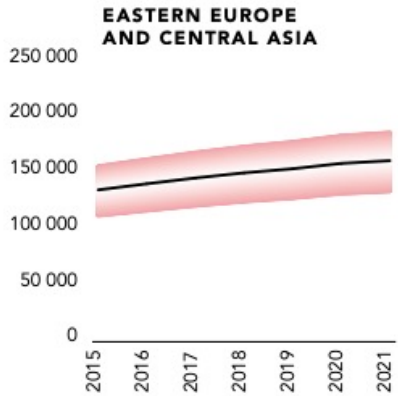


Source: UNAIDS epidemiological estimates, 2022 (<https://aidsinfo.unaids.org/>).

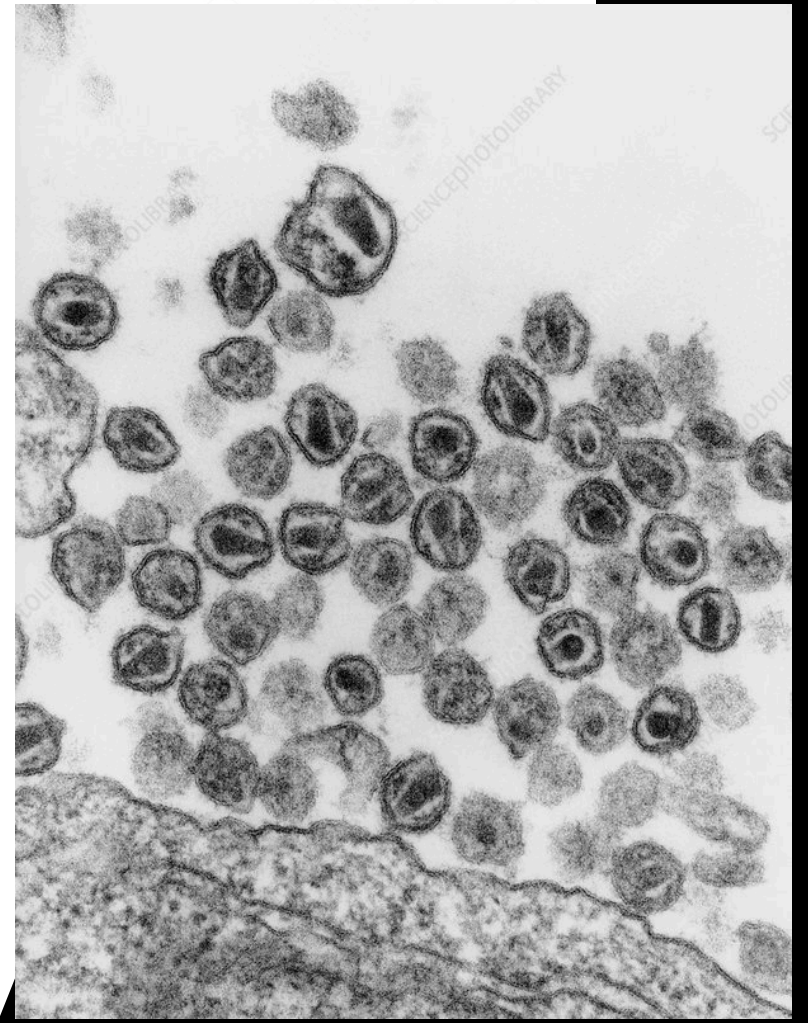
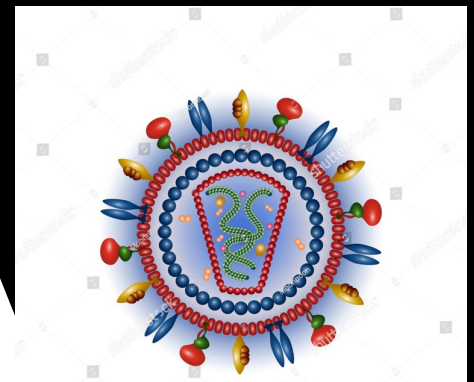
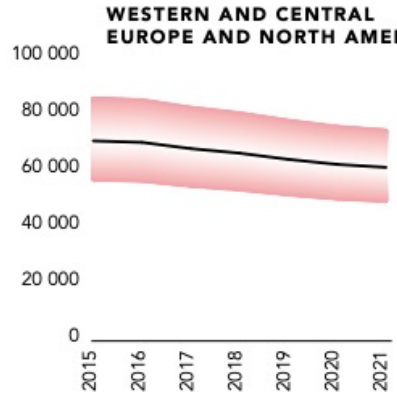
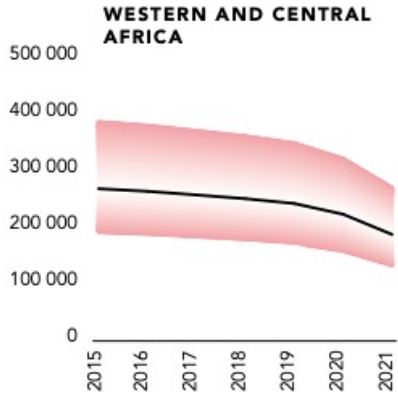
NUMBER OF NEW HIV INFECTIONS



NUMBER OF NEW HIV INFECTIONS

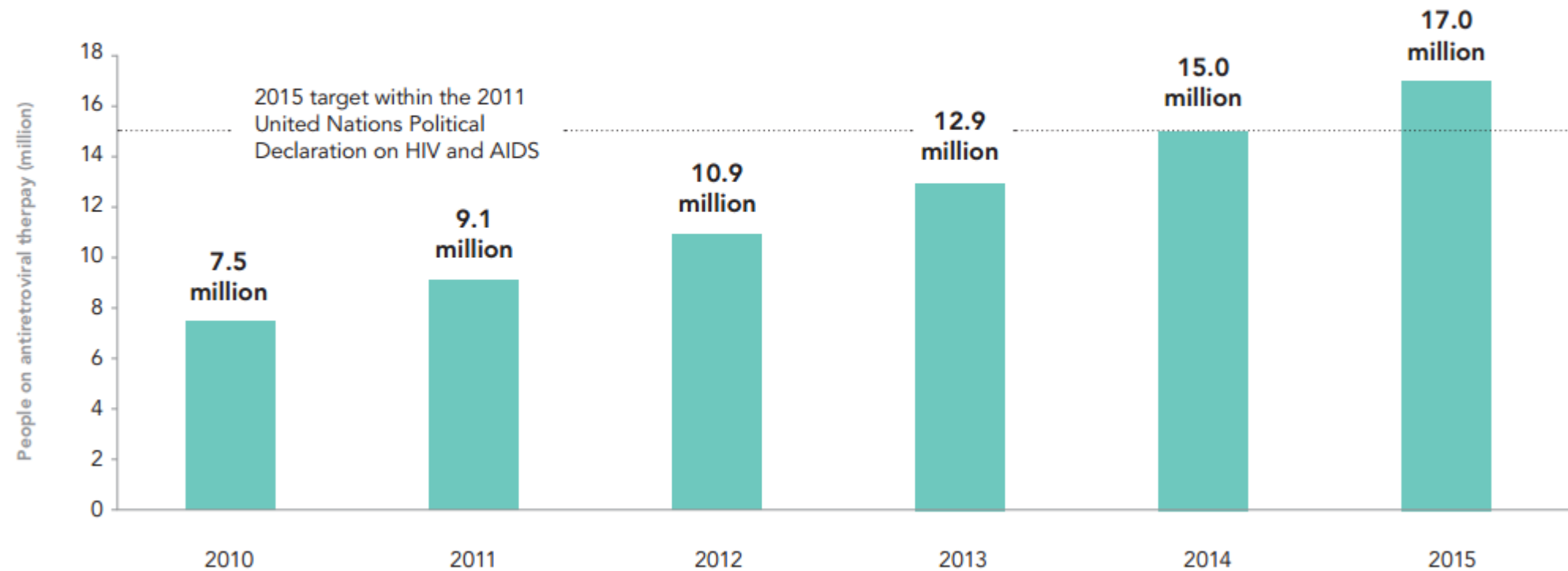


NUMBER OF NEW HIV INFECTIONS



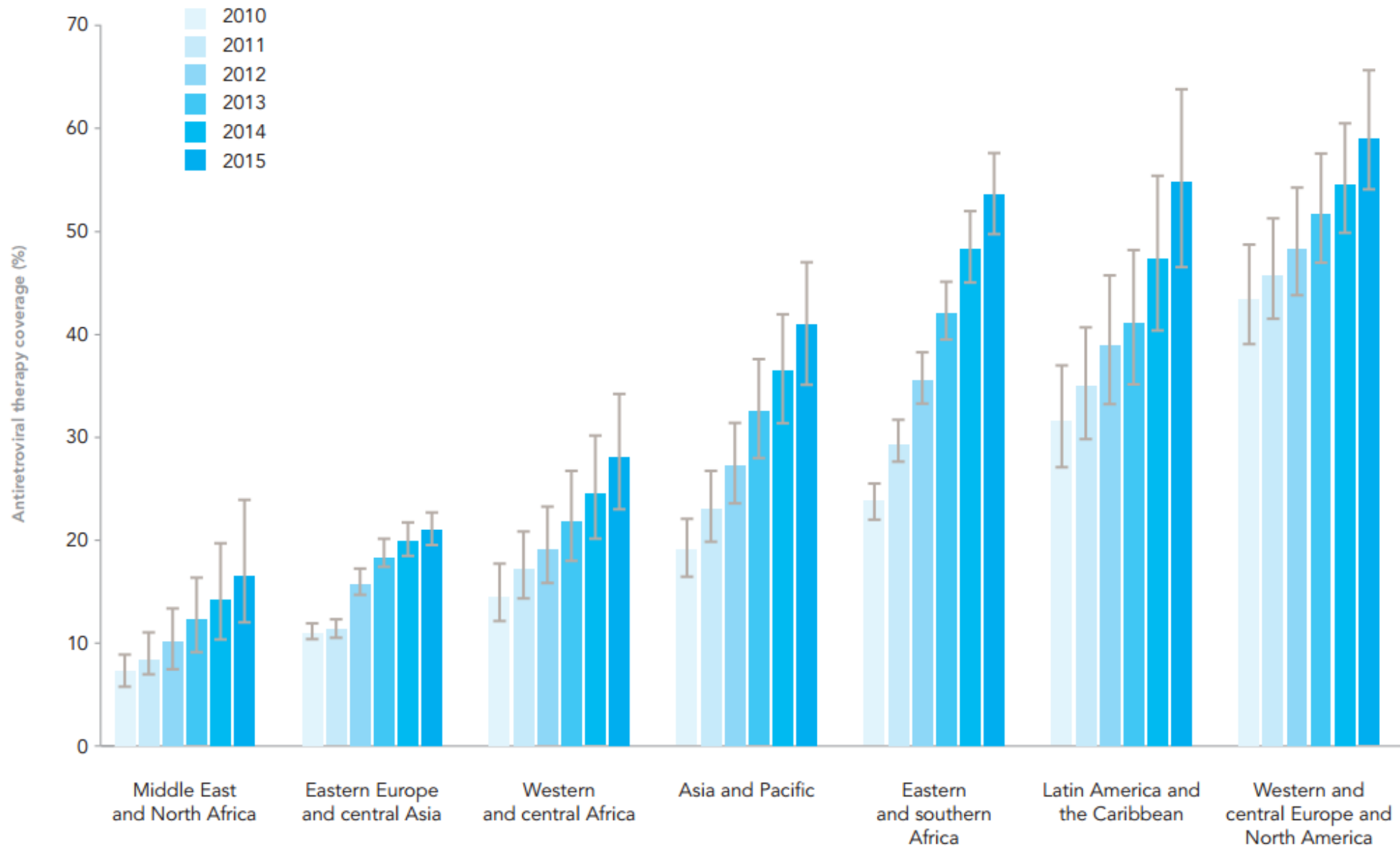
Source: UNAIDS epidemiological estimates, 2022 (<https://aidsinfo.unaids.org/>).

## Number of people living with HIV on antiretroviral therapy, global, 2010–2015

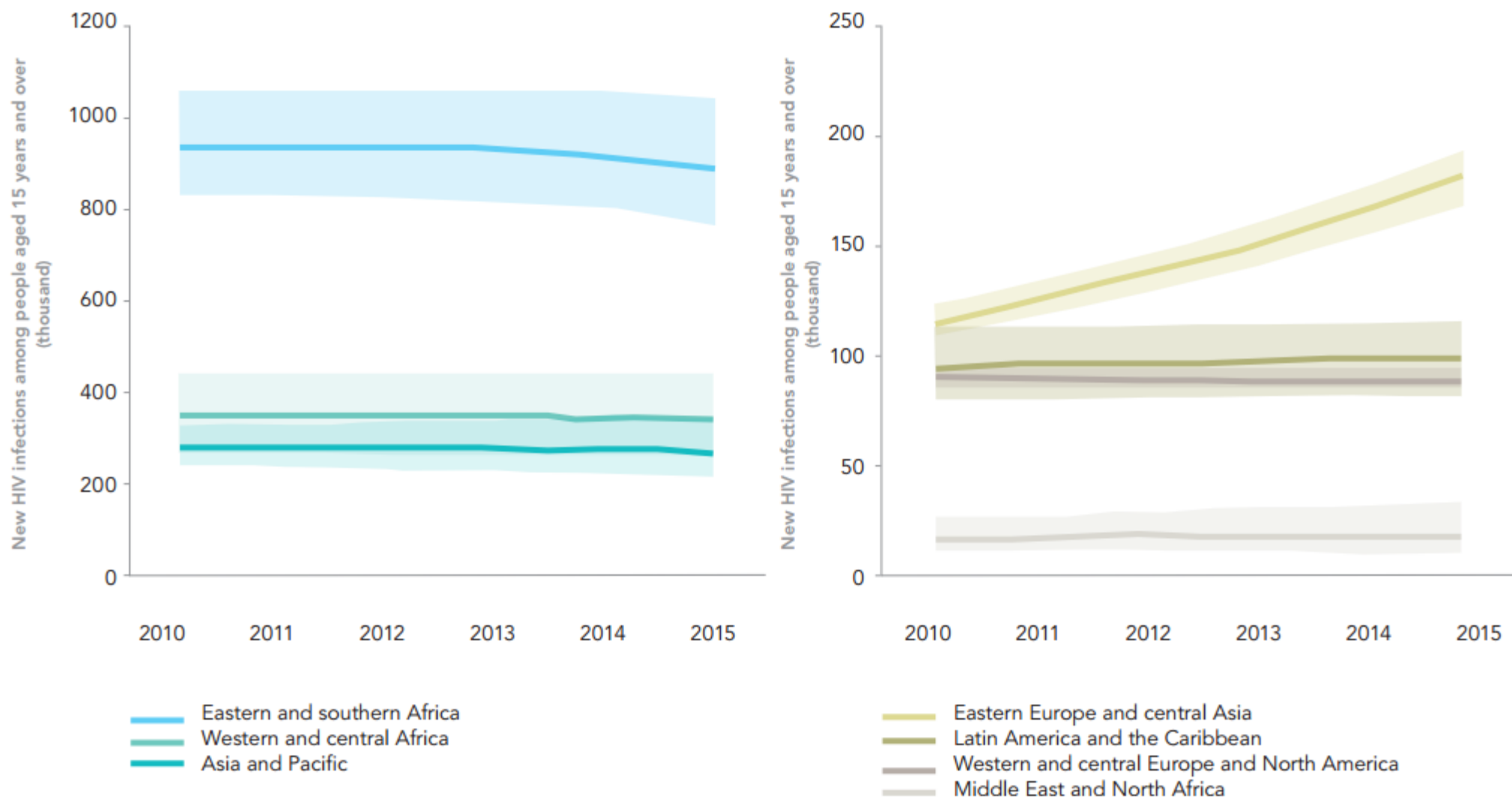


	People living with HIV on antiretroviral treatment (all ages)		AIDS-related deaths (all ages)	
	2010	2015*	2010	2015
<b>Global</b>	7 501 100	17 025 900	1.5 million [1.3 million–1.7 million]	1.1 million [940 000–1.3 million]
<b>Asia and Pacific</b>	907 600	2 071 900	240 000 [200 000–270 000]	180 000 [150 000–220 000]
<b>Eastern and southern Africa</b>	4 087 500	10 252 400	760 000 [670 000–860 000]	470 000 [390 000–560 000]
<b>Eastern Europe and central Asia</b>	112 100	321 800	38 000 [33 000–45 000]	47 000 [39 000–55 000]
<b>Latin America and the Caribbean</b>	568 400	1 091 900	60 000 [51 000–70 000]	50 000 [41 000–59 000]
<b>Middle East and North Africa</b>	13 600	38 200	9500 [7400–12 000]	12 000 [8700–16 000]
<b>Western and central Africa</b>	905 700	1 830 700	370 000 [290 000–470 000]	330 000 [250 000–430 000]
<b>Western and central Europe and North America</b>	906 200	1 418 900	29 000 [27 000–31 000]	22 000 [20 000–24 000]

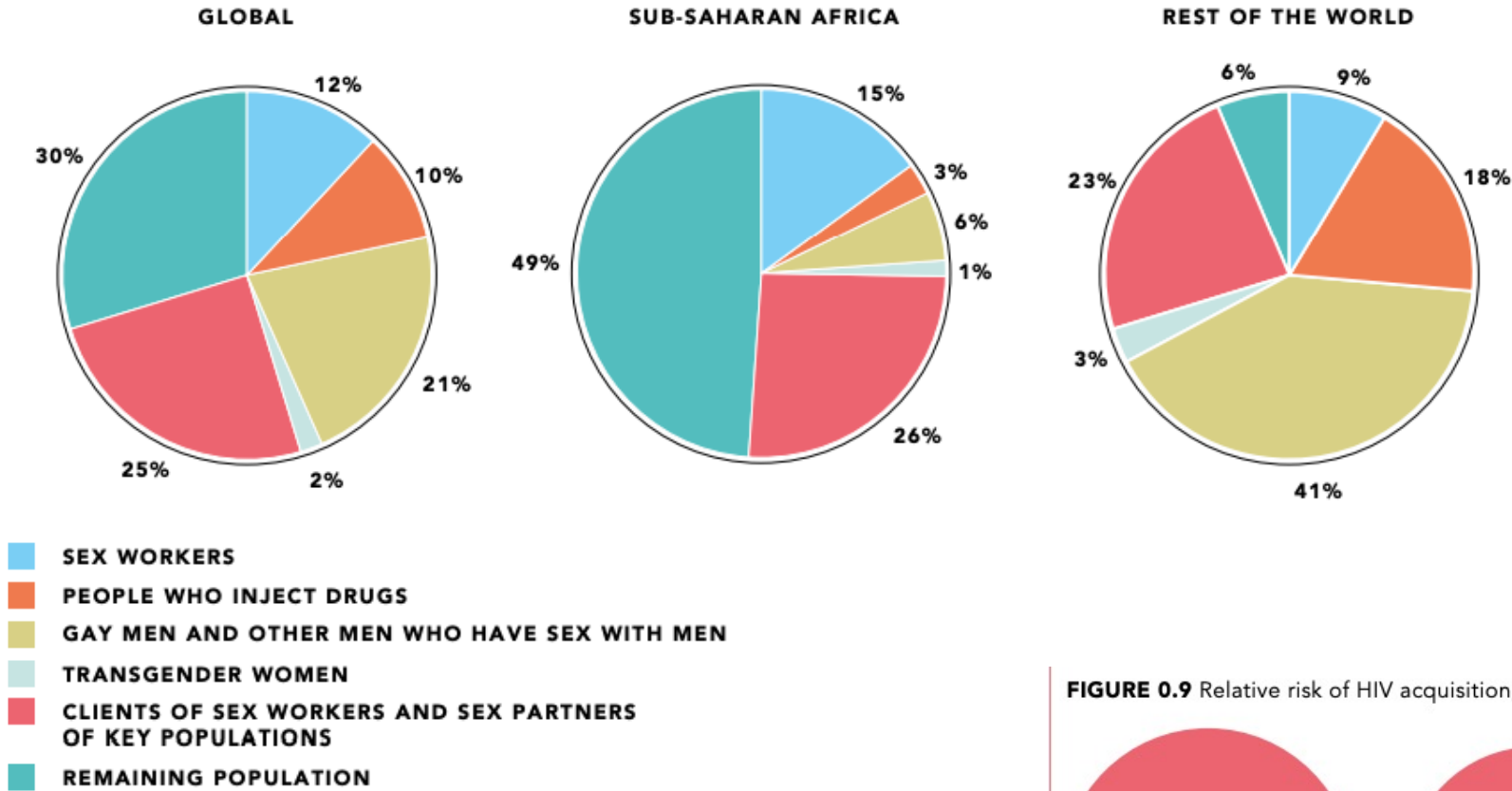
# Antiretroviral therapy coverage among people living with HIV, by region, 2010–2015



## New HIV infections among people aged 15 years and over, by region, 2010–2015



**FIGURE 0.8** Distribution of acquisition of new HIV infections by population, global, sub-Saharan Africa and rest of the world, 2021



Source: UNAIDS special analysis, 2022 (see Annex on Methods).

Note: Due to variations in the availability of data from one year to the next, we do not provide trends in the description of the calculation.

**FIGURE 0.9** Relative risk of HIV acquisition, global, 2021

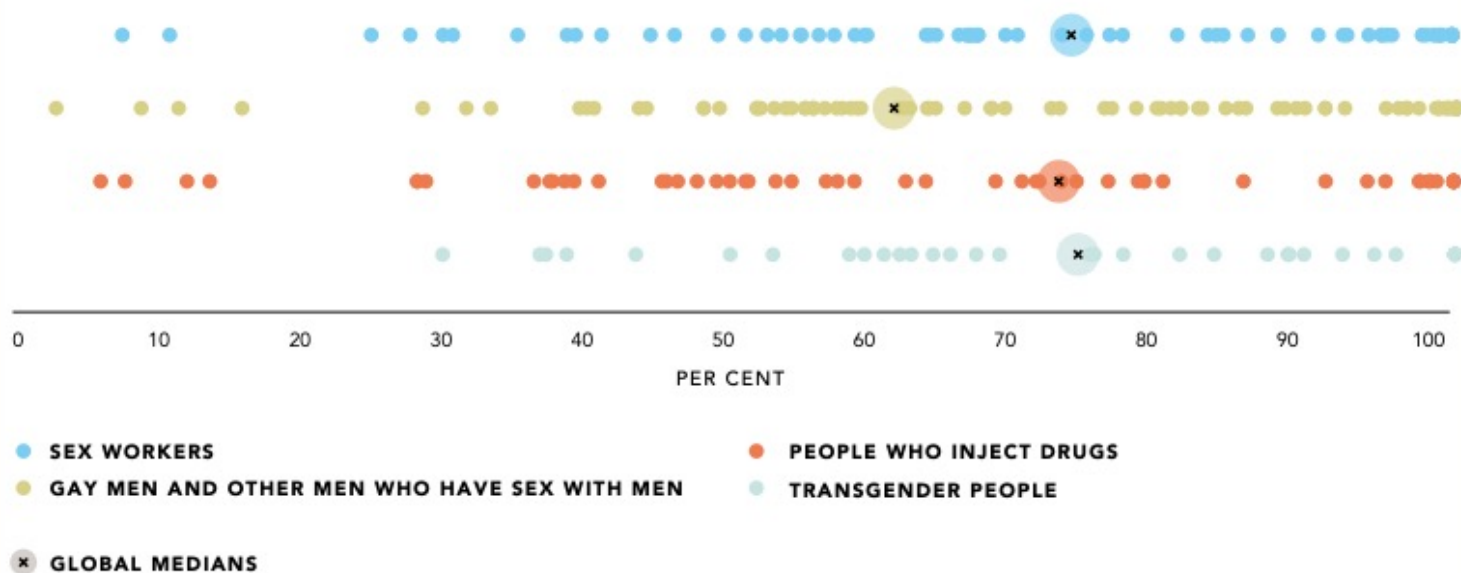


Source: UNAIDS special analysis, 2022 (see Annex on Methods).

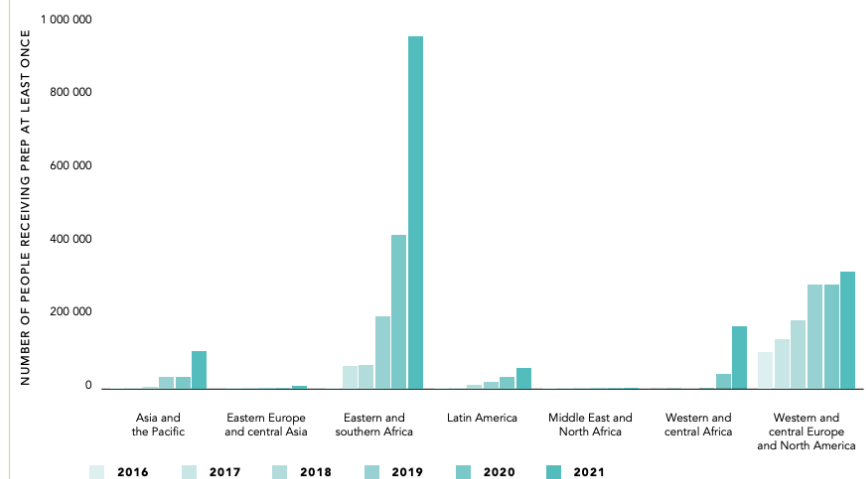


# The percentage of key populations who have been recently tested for HIV and made aware of their status is still low.

**FIGURE 1.14** HIV testing and status awareness among key populations, global, 2017–2021

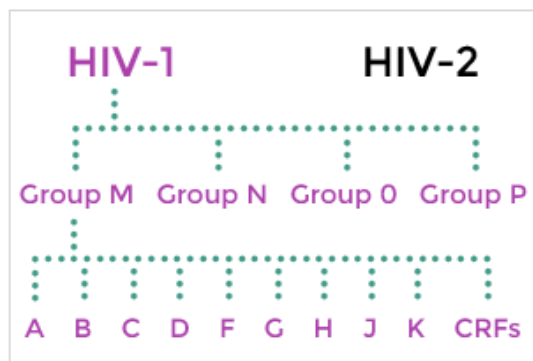


**FIGURE 1.6** Number of people who received pre-exposure prophylaxis (PrEP) at least once during the reporting period, by region, 2017–2021



## Groups within HIV-1

### HIV Types and Strains



**HIV-1 Group M** is the strain of HIV that is responsible for the global HIV epidemic.

The strains of HIV-1 can be classified into four groups.<sup>4</sup> The most important group, M, is the 'major' group and is responsible for the majority of the global HIV epidemic.

The other three groups are N, O and P. They are quite uncommon and only occur in Cameroon, Gabon and Equatorial Guinea.

### Subtypes within HIV-1 group M

Within group M there are known to be at least nine genetically distinct subtypes of HIV-1. These are subtypes A, B, C, D, F, G, H, J and K.

Additionally, different subtypes can combine genetic material to form a hybrid virus, known as a 'circulating recombinant form' (CRFs), of which quite a few have been identified.<sup>5</sup>

The dominant HIV subtype in the [Americas](#), [Western Europe](#) and Australasia is subtype [B](#). As a result, the great majority of HIV clinical research has been conducted in populations where subtype B predominates. However this subtype represents only 12% of global HIV infections.

In contrast, less research is available for subtype C, although just under half of all people living with HIV have subtype C. It is very common in the high prevalence countries of Southern Africa, as well as in the horn of Africa and [India](#).

The greatest diversity of subtypes is found in Cameroon and the Democratic Republic of Congo - the region [where the HIV-1 epidemic originated](#).

However, these geographical patterns in the distribution of subtypes are changing over time, due to migration and the mixing of populations.<sup>6</sup>

# HIV • AIDS

## HIV

De 2007 até junho de 2016, foram notificados no Sinan 136.945 casos de infecção pelo HIV no Brasil, sendo 71.396 no Sudeste (52,1%), 28.879 no Sul (21,1%), 18.840 no Nordeste (13,8%), 9.152 no Centro-Oeste (6,7%) e 6.868 na Região Norte (6,3%). No ano de 2015, foram notificados 32.321 casos de infecção pelo HIV, sendo 2.988 casos na região Norte (9,2%), 6.435 casos na região Nordeste (19,9%), 13.059 na região Sudeste (40,4%), 7.265 na região Sul (22,5%) e 2.574 na região Centro-Oeste (8,0%) (Tabela 1).

Na Tabela 2, são apresentados os casos de HIV notificados no Sinan no período de 2007 a 2016, segundo sexo. Nesse período, foi notificado no Sinan um total de 92.142 casos em homens e 44.766 casos em mulheres. A razão de sexos para o ano de 2015 foi de 2,4.

A Tabela 3 apresenta os casos notificados de infecção pelo HIV no Sinan segundo faixa etária e escolaridade. No período de 2007 a 2016, no que se refere às faixas etárias, observou-se que a maioria dos casos de infecção pelo HIV encontra-se nas faixas de 20 a 34 anos, com percentual de 52,3% dos casos. Com relação à escolaridade, no mesmo período, observou-se um elevado

percentual de casos ignorados (25,0%), o que dificulta uma melhor avaliação dos casos de infecção pelo HIV relativos a esse item.

Com relação à raça/cor da pele autodeclarada, observa-se na Tabela 4 que, entre os casos registrados no Sinan no período de 2007 a 2015, 44,0% são entre brancos e 54,8% em pretos e pardos. No sexo masculino, 46,1% são entre brancos e 52,8% em pretos e pardos; entre as mulheres, 39,2% dos casos são entre brancas e 59,6% em pretas e pardas.

A Tabela 5 apresenta os casos de infecção pelo HIV registrados no Sinan de 2007 a 2015 em indivíduos maiores de 13 anos de idade, segundo a categoria de exposição. Entre os homens, em 2015, verifica-se que 50,4% dos casos tiveram exposição homossexual, 36,8% heterossexual e 9,0% bissexual; entre as mulheres, nessa mesma faixa etária, observa-se que 96,4% dos casos se inserem na categoria de exposição heterossexual.

Por fim, ressalta-se que a notificação compulsória da infecção pelo HIV é muito recente, o que impede uma análise epidemiológica rigorosa com relação às tendências da infecção no Brasil.

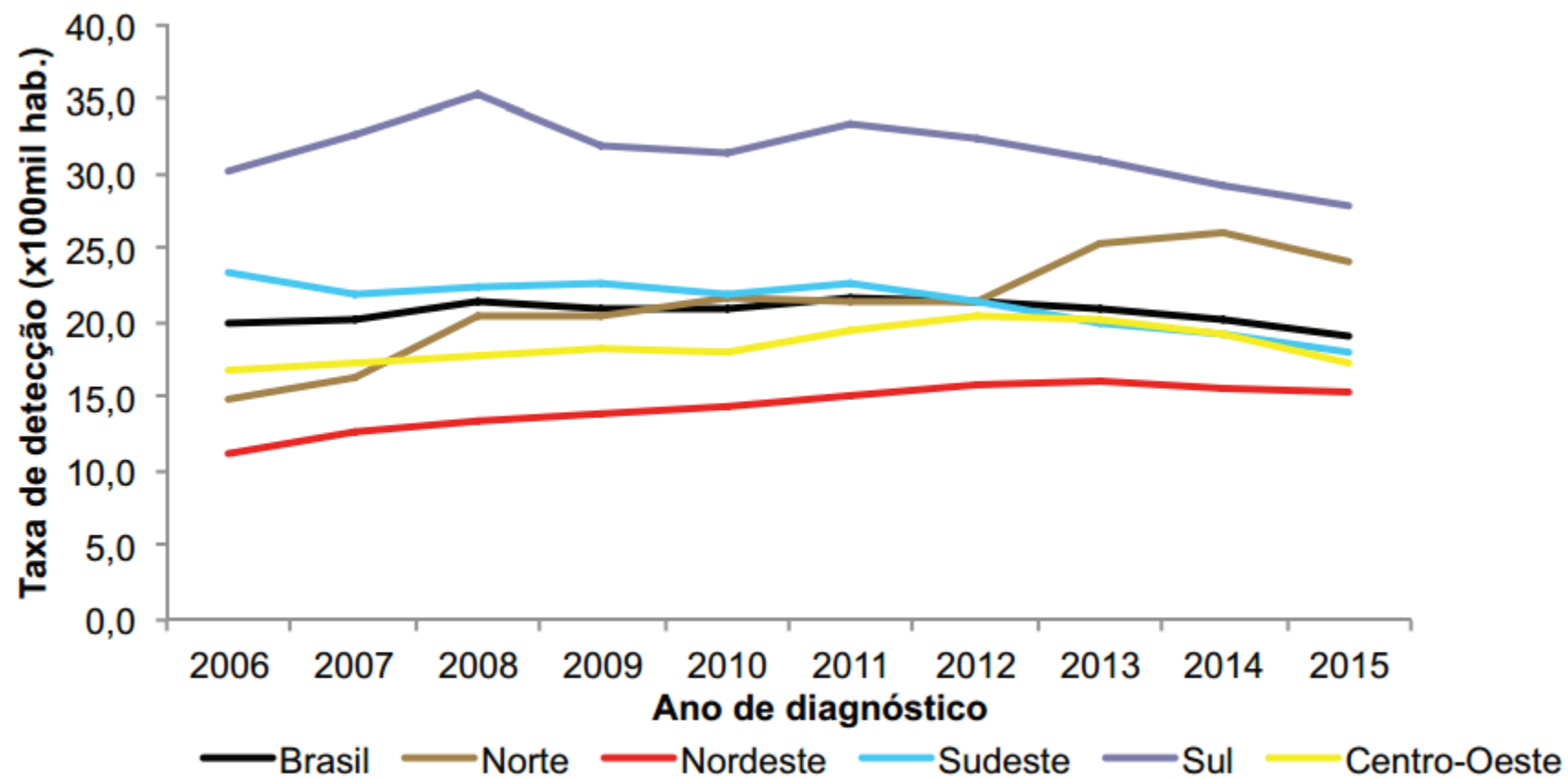


Figura 3. Taxa de detecção de aids (/100.000 mil habitantes) segundo região de residência por ano de diagnóstico. Brasil, 2006 a 2015<sup>(1)</sup>

Fonte: MS/SVS/DIAHV.

Nota: (1) Casos notificados no Sinan e Siscel/Siclom até 30/06/2016 e no SIM de 2000 até 2015.

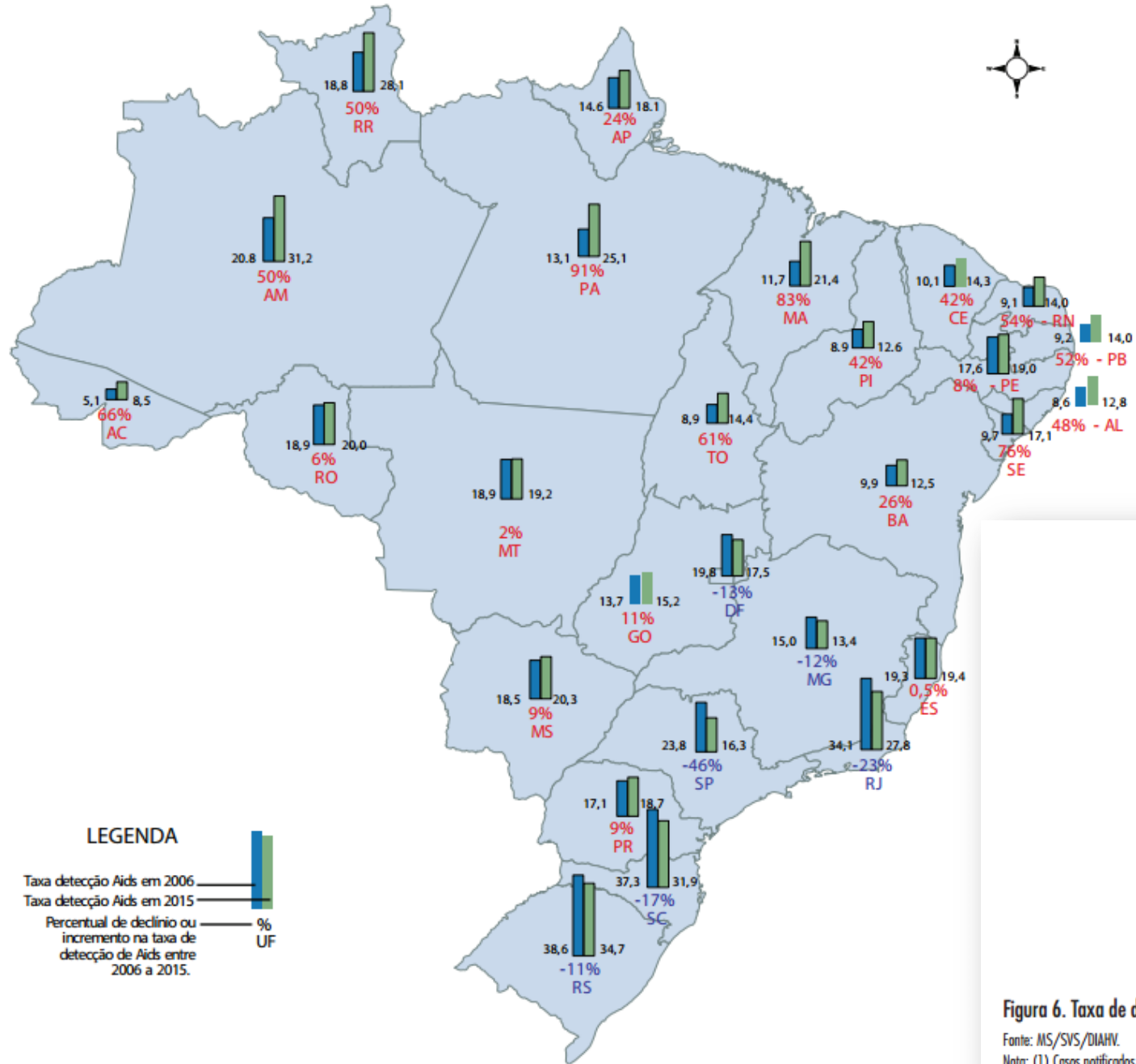


Figura 4. Taxa de detecção de aids (/100 mil habitantes) e percentual de declínio ou incremento segundo UF de residência por ano de diagnóstico. Brasil, 2006 e 2015

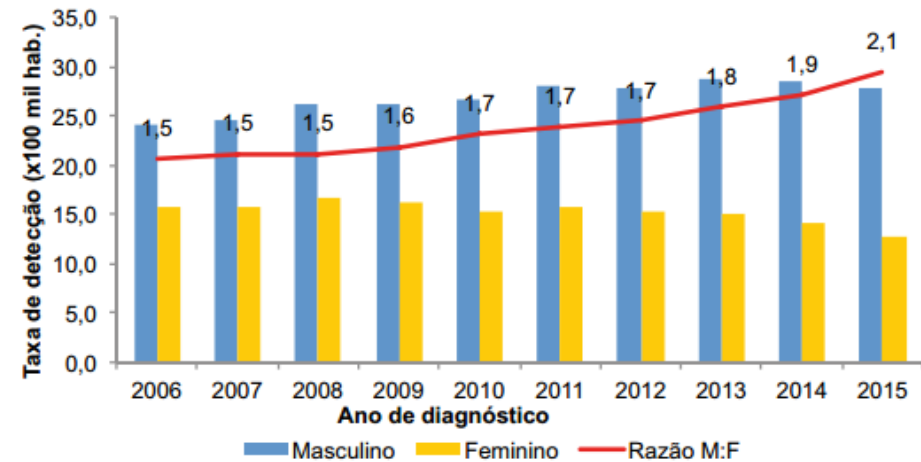


Figura 6. Taxa de detecção de aids (/100 mil habitantes) segundo sexo e razão de sexos por ano de diagnóstico. Brasil, 2006 a 2015<sup>(1)</sup>

Fonte: MS/SVS/DIAHV.

Nota: (1) Casos notificados no Sinan e Siscel/Sicdom até 30/06/2016 e no SIM de 2000 até 2015.

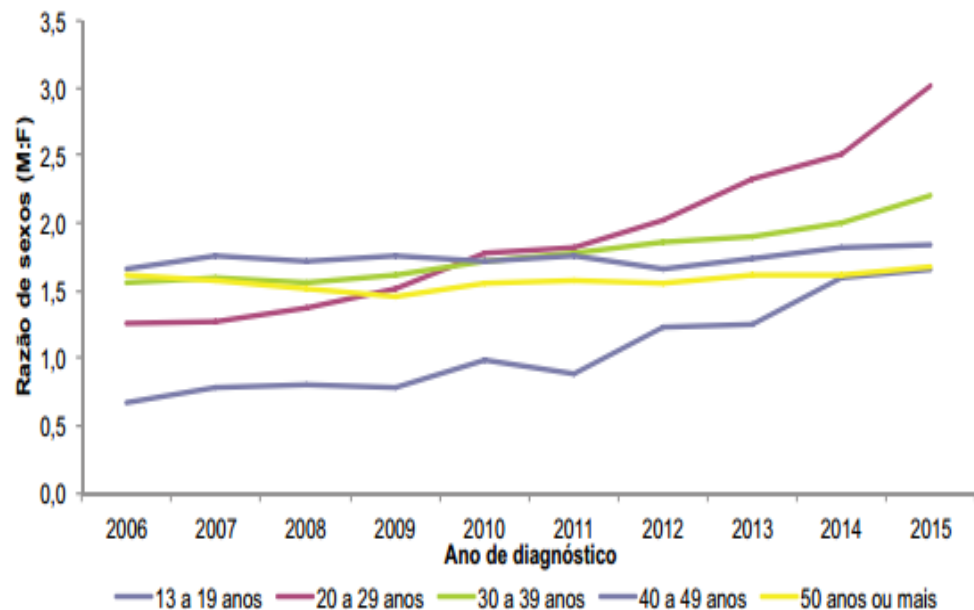


Figura 8. Razão de sexos segundo faixa etária por ano de diagnóstico. Brasil, 2006 a 2015<sup>(1)</sup>

Fonte: MS/SVS/DIAHV.

Nota: (1) Casos notificados no Sinan e Siscel/Sicdom até 30/06/2016 e no SIM de 2000 até 2015.

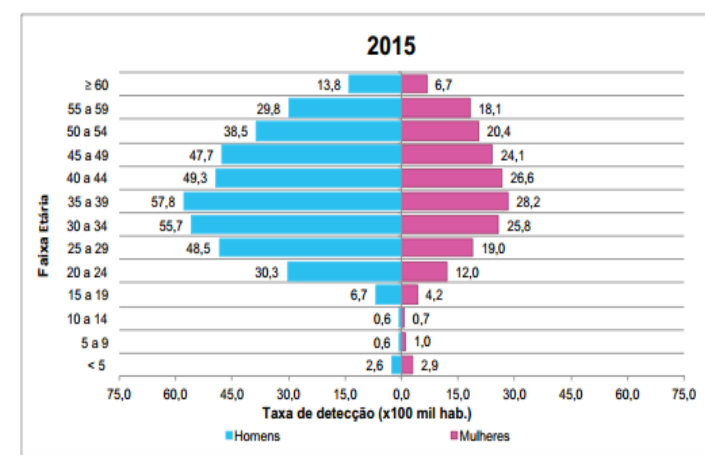
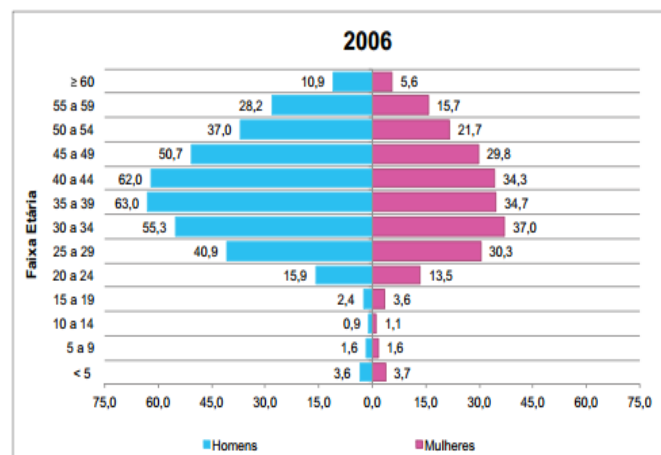


Figura 9. Taxa de detecção de aids (/100 mil habitantes) segundo faixa etária e sexo. Brasil, 2006 e 2015<sup>(1)</sup>

Fonte: MS/SVS/DIAHV.

Nota: (1) Casos notificados no Sinan e Siscel/Sicdom até 30/06/2016 e no SIM de 2000 até 2015.

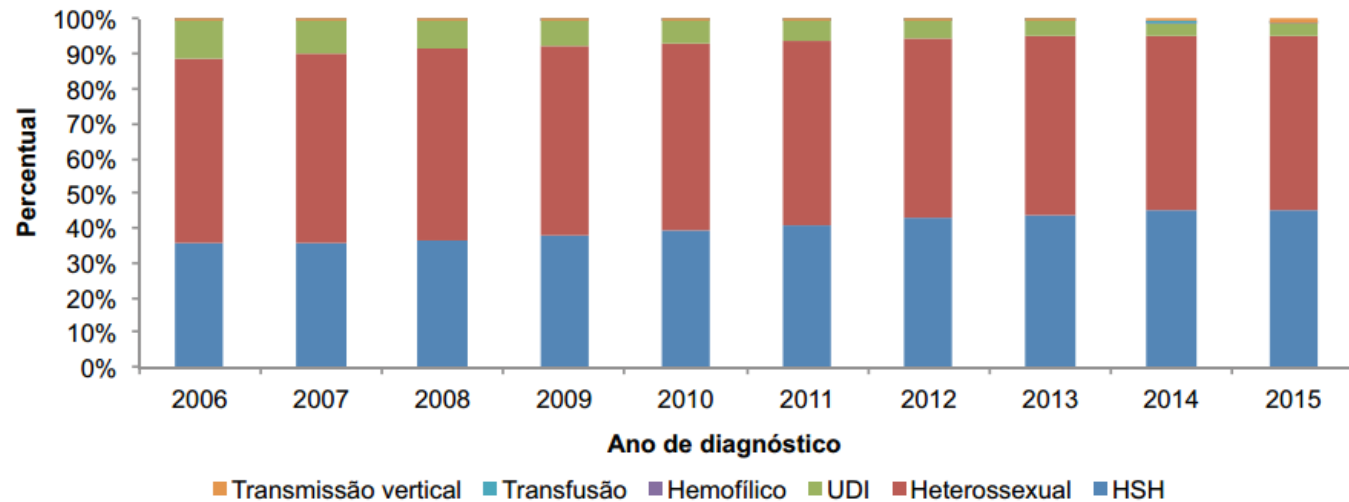


Figura 14. Distribuição percentual dos casos de aids em homens de 13 anos ou mais segundo categoria de exposição por ano de diagnóstico. Brasil, 2006 a 2015.

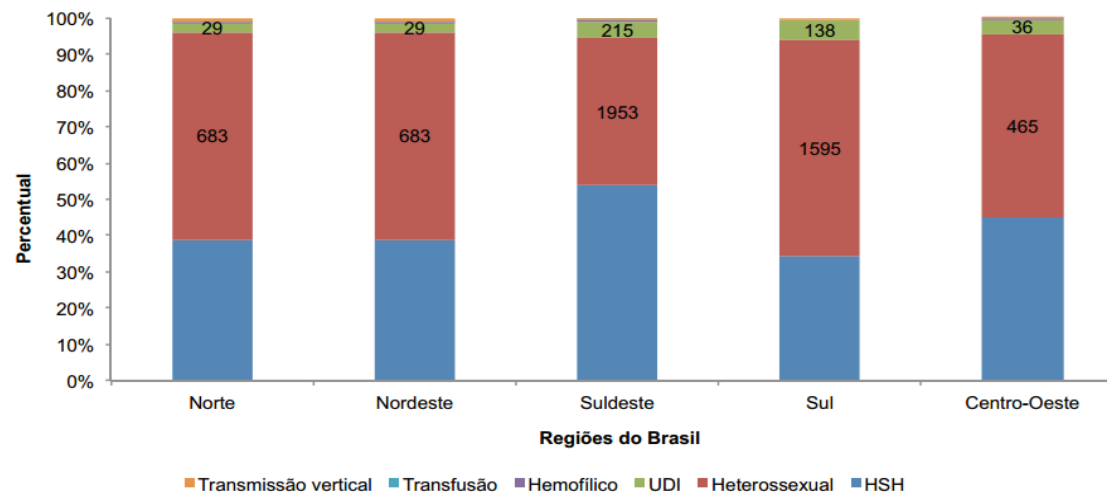
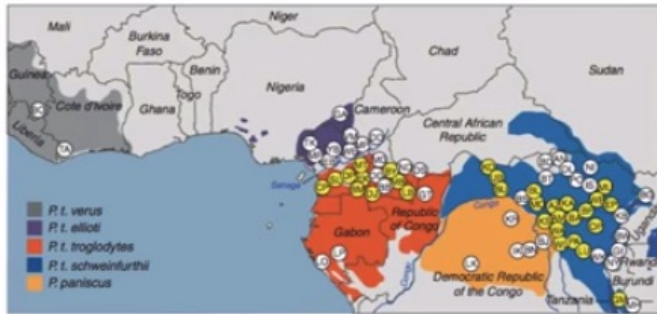


Figura 15. Distribuição percentual dos casos de aids em homens de 13 anos ou mais segundo categoria de exposição por região de residência. Brasil, 2015.

Fonte: MS/SVS/DIAHV.

## What was the source of HIV-1?

- SIV first isolated from chimpanzee in 1989 (SIVcpz)
- Analysis of >7,000 chimpanzee fecal samples from 90 field sites confirmed natural SIVcpz reservoir
- Only *Pan troglodytes troglodytes* and *P. T. schweinfurthii* harbor SIVcpz



Virology Lectures 2017 • Prof. Vincent Racaniello • Columbia University

Cold Spring Harb Perspect Med 2011;1:a006841

### Virology Lectures 2017 #23: HIV and AIDS

10.583 visualizações



100



5



COMPARTILHAR



HIRSCH MS, Olmsted RA, Murphey-Corb M, Purcell RH, Johnson PR. An African primate lentivirus (SIVsm) closely related to HIV-2. *Nature*. 1989; 339: 389-392



# Biologia e Estrutura do HIV

- Attachment – Invasion gp41 e gp-120
- Transcrição Reversa – Transcriptase Reversa
- Também possui domínio Nuclease
- Integrase – Corta 2nt prime ends do dsDNA e integra no Genoma do Hospedeiro.



Photo: Magunia/SCANPIX

Harald zur Hausen



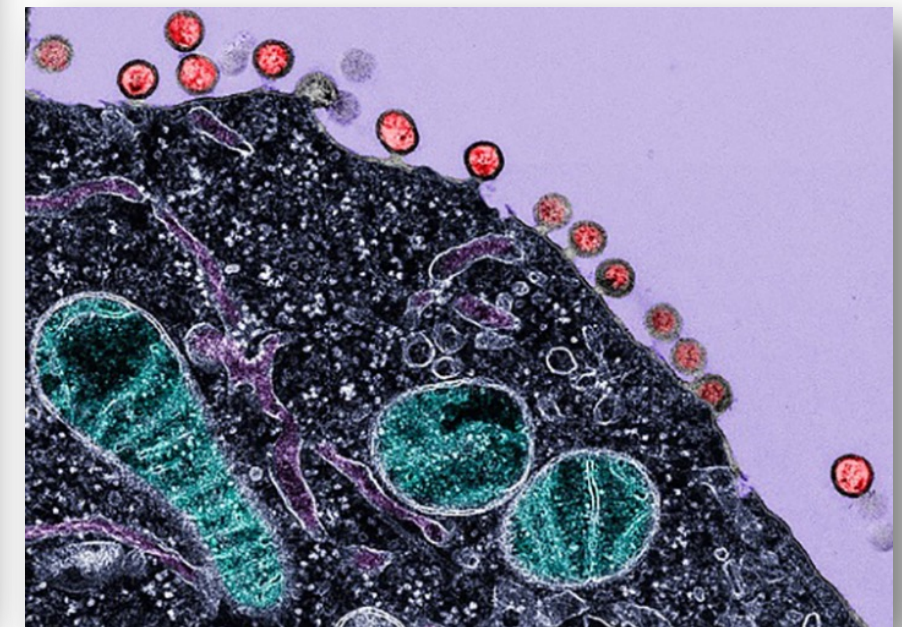
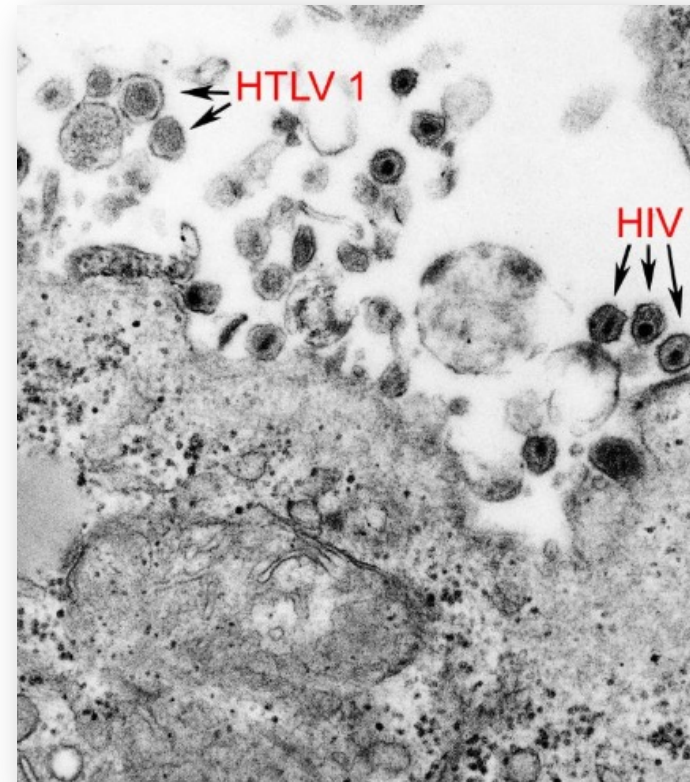
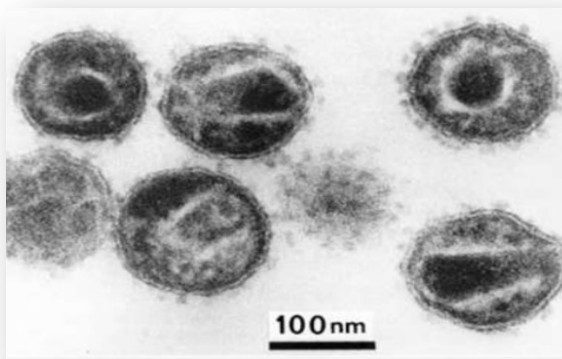
Photo: L. Dolega/SCANPIX

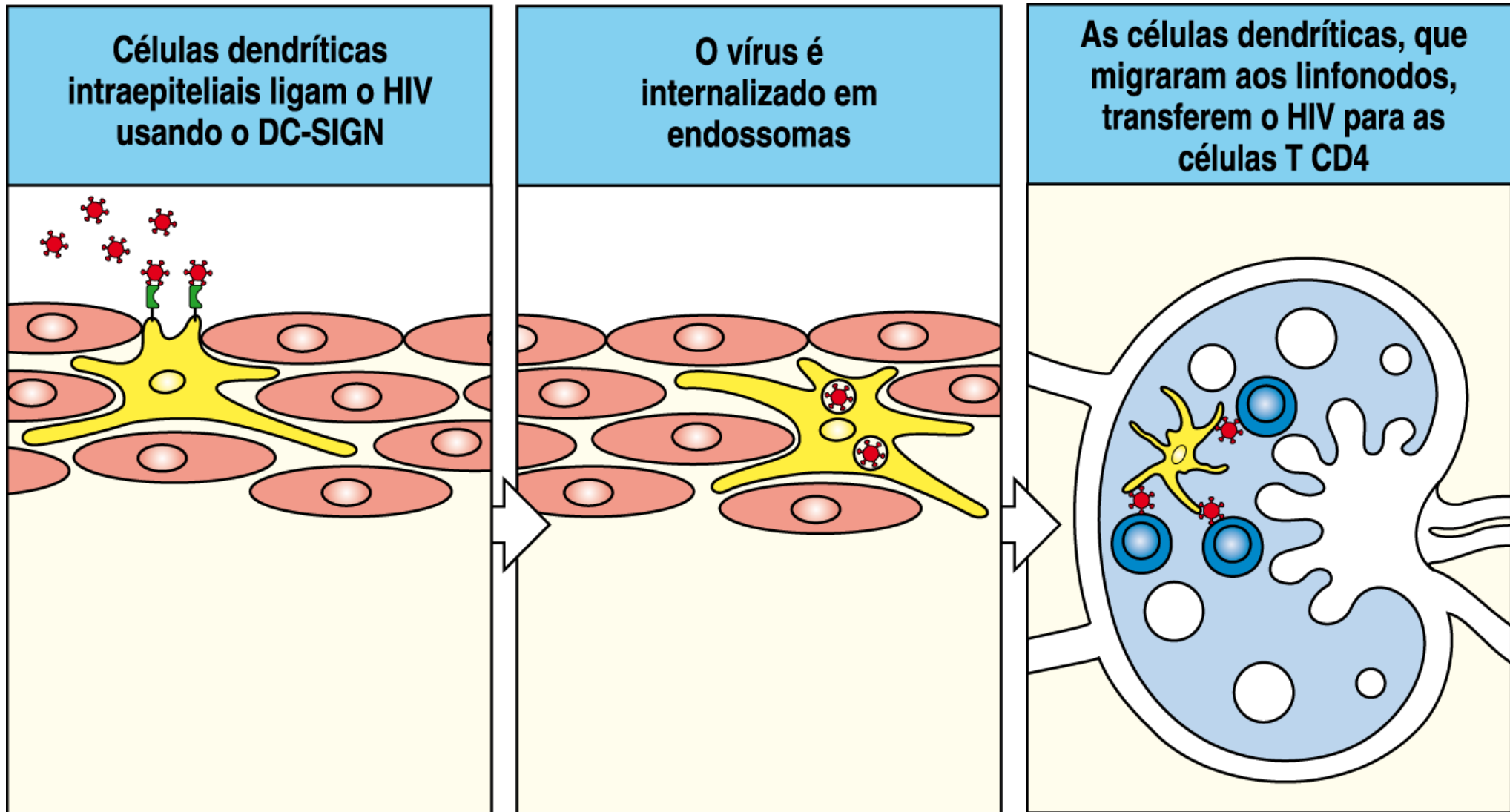
Françoise Barré-Sinoussi



Photo: DKFZ/SCANPIX

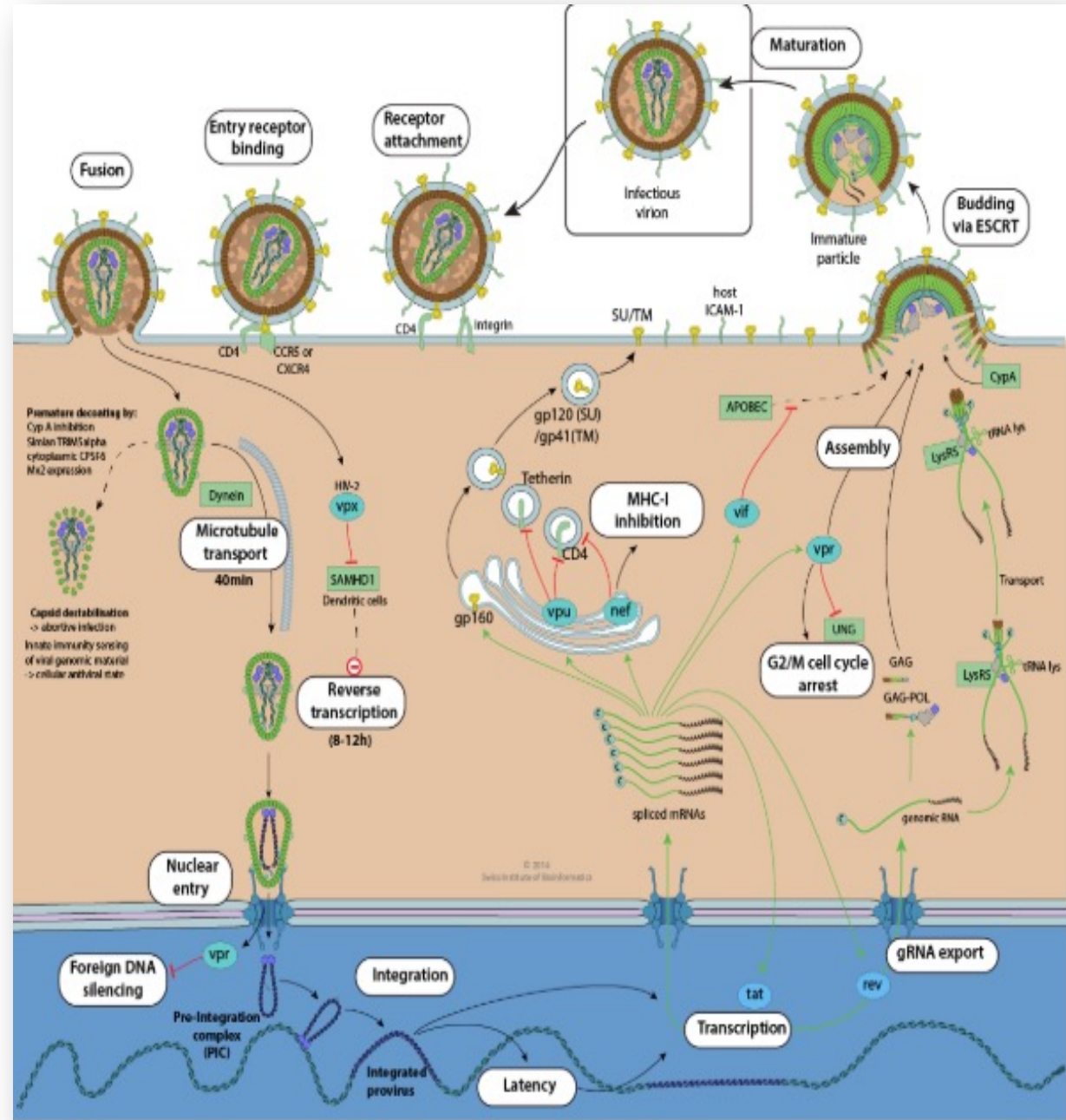
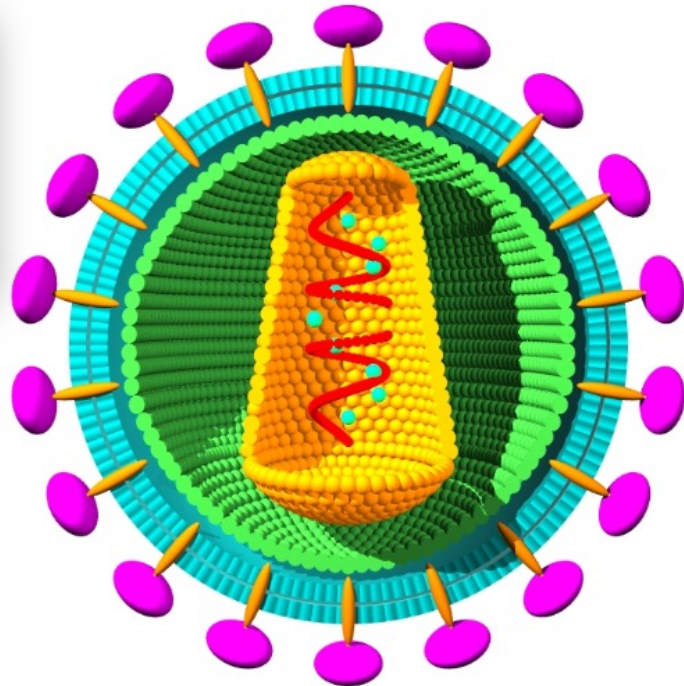
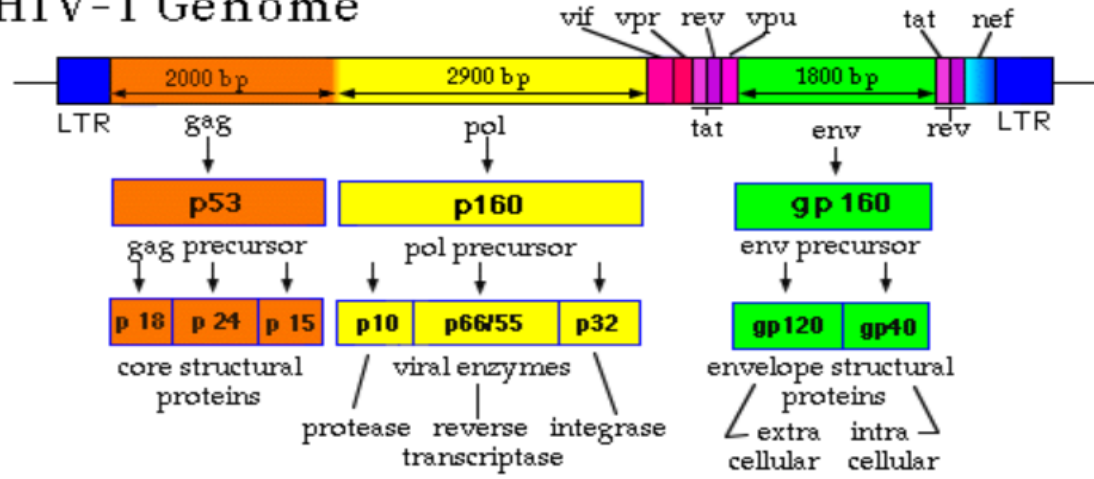
Luc Montagnier

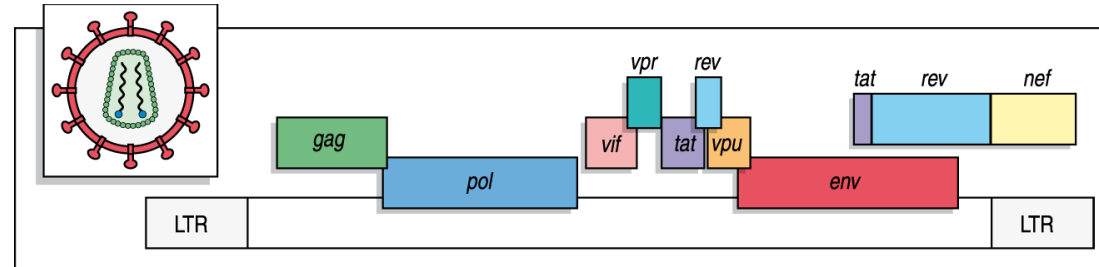




As células dendríticas e Macrófagos iniciam a infecção transportando o vírus HIV das superfícies mucosas para os tecidos linfoides.

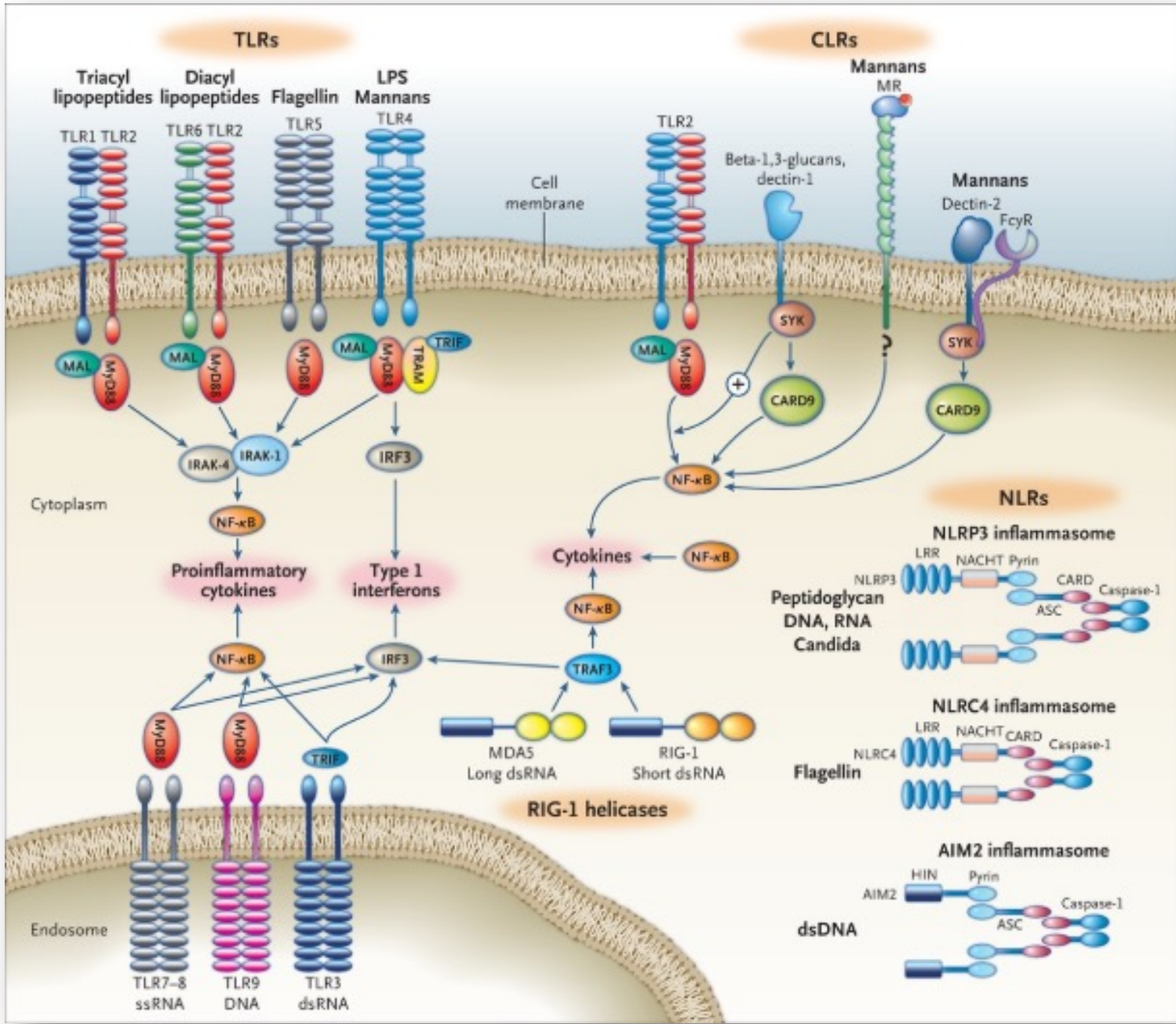
# HIV-1 Genome





Gene		Produto gênico/função
<i>gag</i>	Antígeno grupo-específico	Proteínas do núcleo e da matriz
<i>pol</i>	Polimerase	Enzimas transcriptase reversa, protease e integrase
<i>env</i>	Envelope	Glicoproteínas transmembrana. gp120 liga-se a CD4 e CCR5; gp41 é necessária à fusão e internalização do vírus
<i>tat</i>	Transativador	Regulador positivo da transcrição
<i>rev</i>	Regulador da expressão viral	Permite a exportação de transcritos não-processados e parcialmente processados do núcleo
<i>vif</i>	Infectividade viral	Afeta a infectividade da partícula
<i>vpr</i>	Proteína viral R	Transporte de DNA para o núcleo celular. Aumenta a produção de vírion. Parada do ciclo celular
<i>vpu</i>	Proteína viral U	Promove a degradação intracelular do CD4 e aumenta a liberação de vírus na membrana celular
<i>nef</i>	Fator de regulação negativa	Aumenta a replicação viral <i>in vivo</i> e <i>in vitro</i> . Diminui a expressão de CD4 e do MHC de classes I e II

Figura 12.24 A organização genômica do HIV.



Ativação das Vias de NF-κB ou IFNs Tipo I

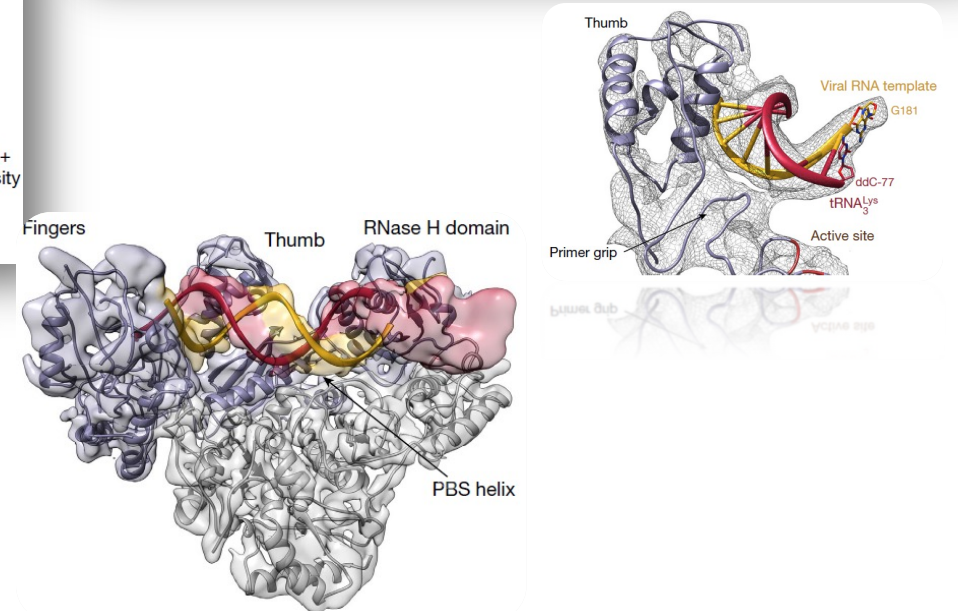
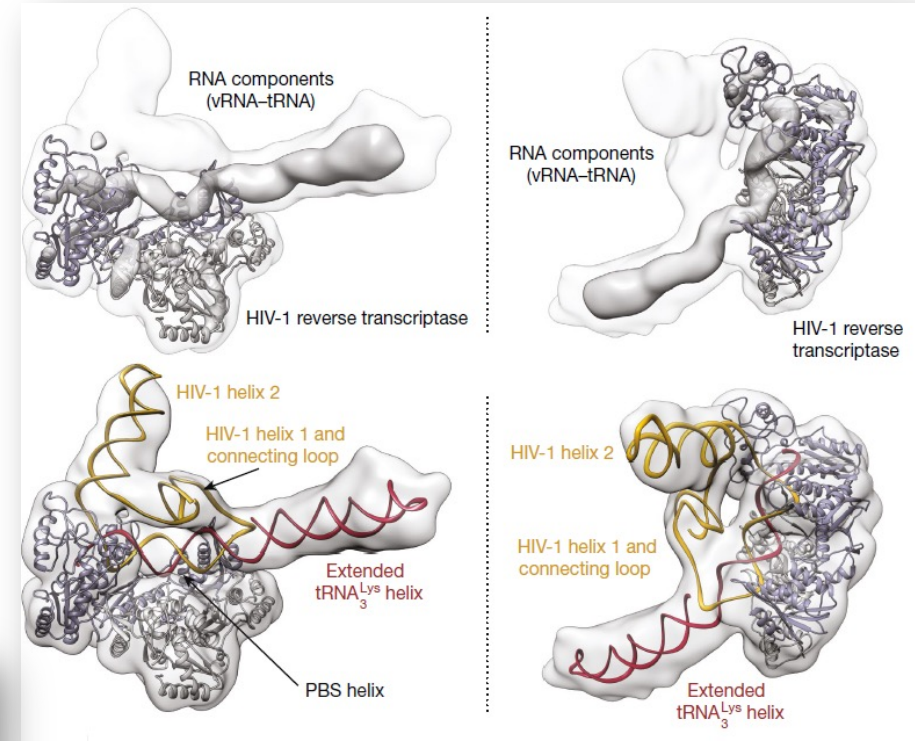
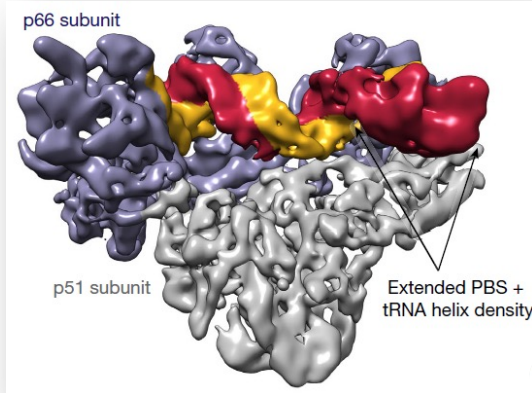
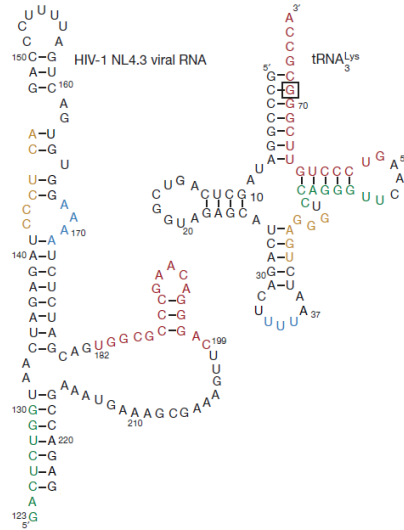
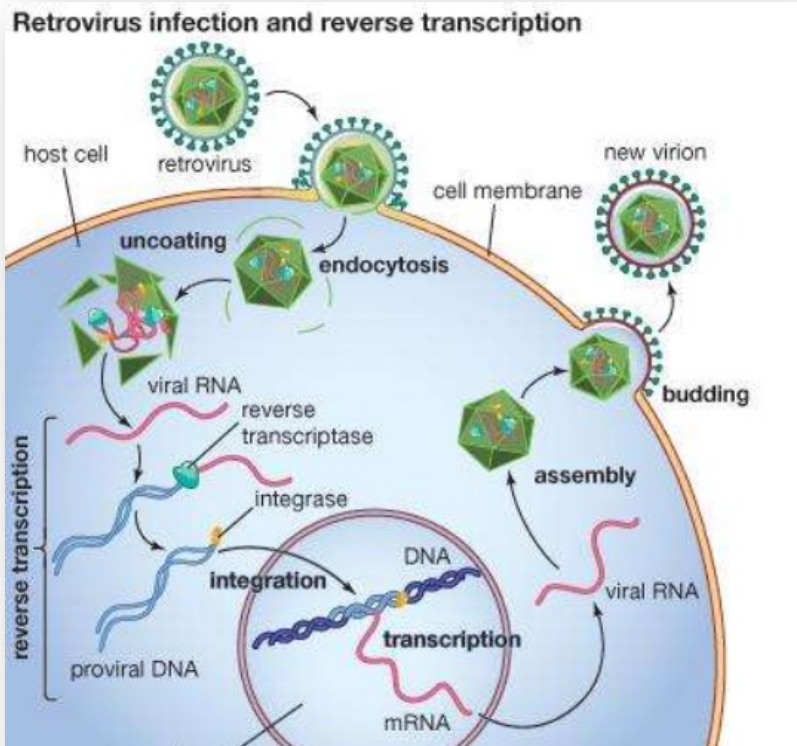
- Maioria experimentos usa recombinantes
  - De proteínas do HIV
    - TLR-3, 7, 8 e 9
      - RIG-I
      - MDA-5
  
- Evidência com vírus ativo ainda faltam

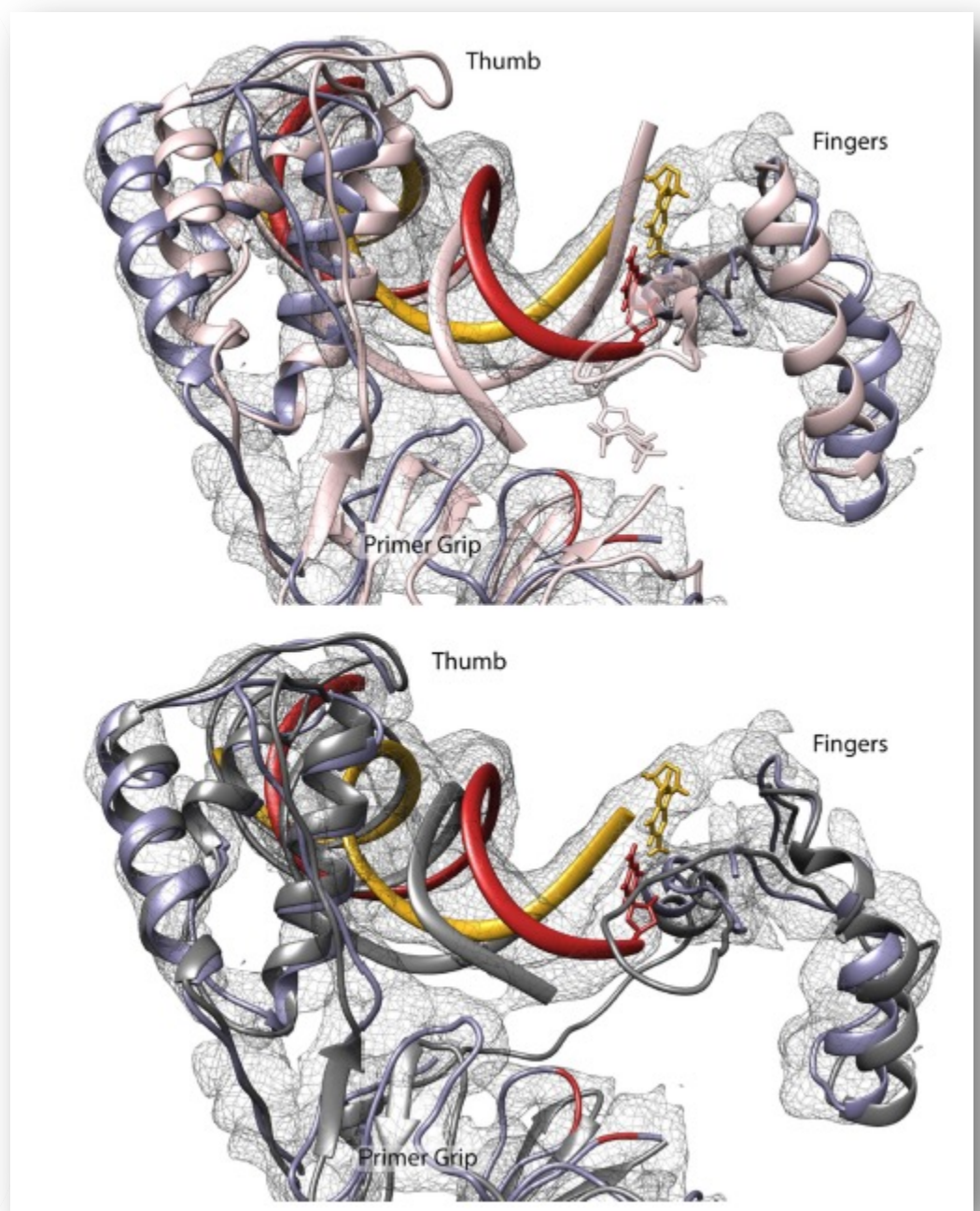
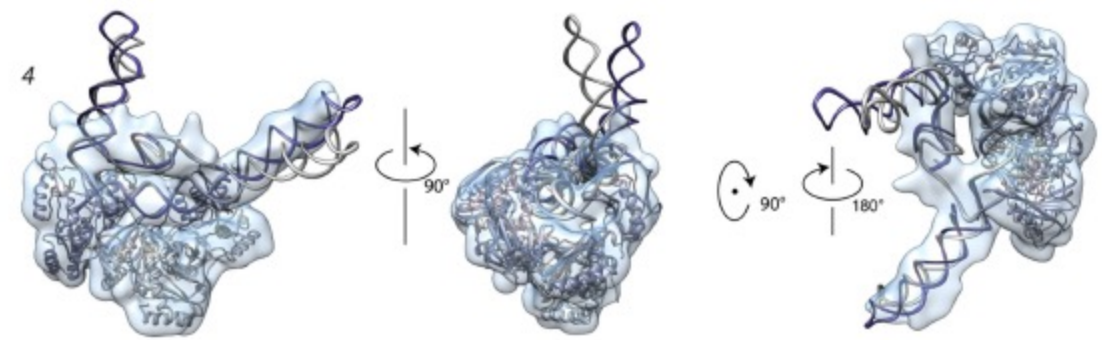
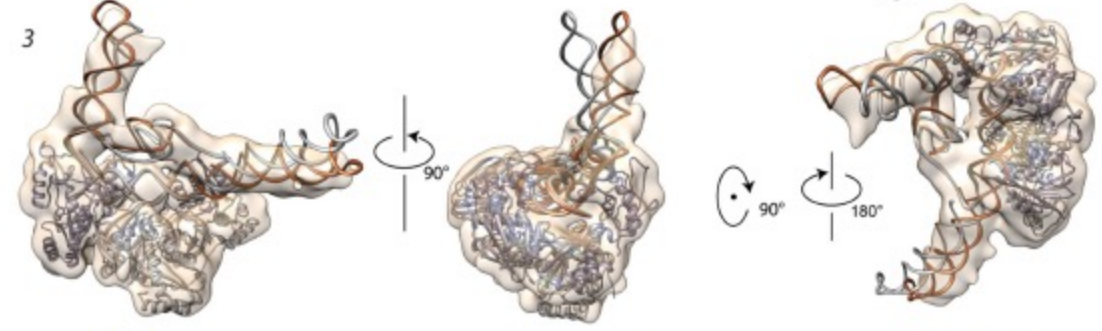
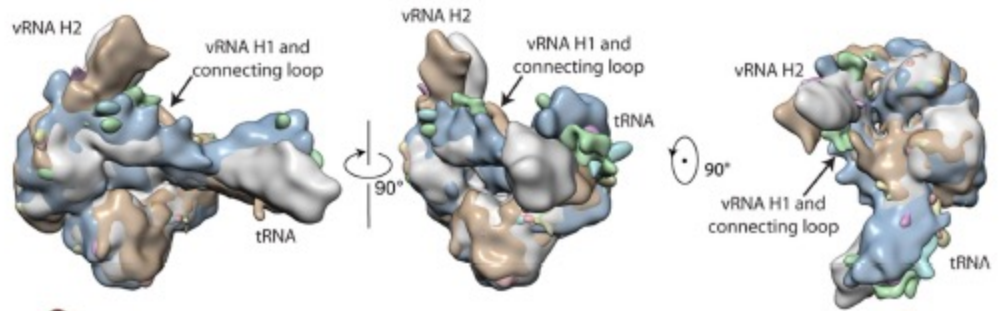
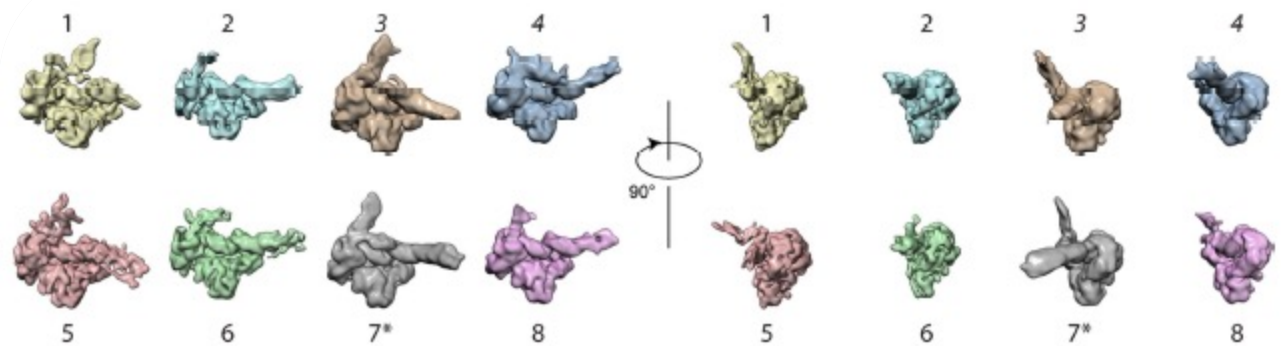
## Architecture of an HIV-1 reverse transcriptase initiation complex

Kevin P. Larsen<sup>1,2,4</sup>, Yamuna Kalyani Mathiharan<sup>3,4</sup>, Kalli Kappel<sup>1</sup>, Aaron T. Coey<sup>1,2</sup>, Dong-Hua Chen<sup>2</sup>, Daniel Barrero<sup>2</sup>, Lauren Madigan<sup>2</sup>, Joseph D. Puglisi<sup>2</sup>, Georgios Skiniotis<sup>2,3</sup> & Elisabetta Viani Puglisi<sup>2\*</sup>

### RTIC – Retro transcription Initiation Complex

RNA carrega finais Lys 3- RNA transportador





## Architecture and Secondary Structure of an Entire HIV-1 RNA Genome

Joseph M. Watts<sup>1</sup>, Kristen K. Dang<sup>2</sup>, Robert J. Gorelick<sup>5</sup>, Christopher W. Leonard<sup>1</sup>, Julian W. Bess Jr.<sup>5</sup>, Ronald Swanstrom<sup>3</sup>, Christina L. Burch<sup>4</sup>, and Kevin M. Weeks<sup>1</sup>

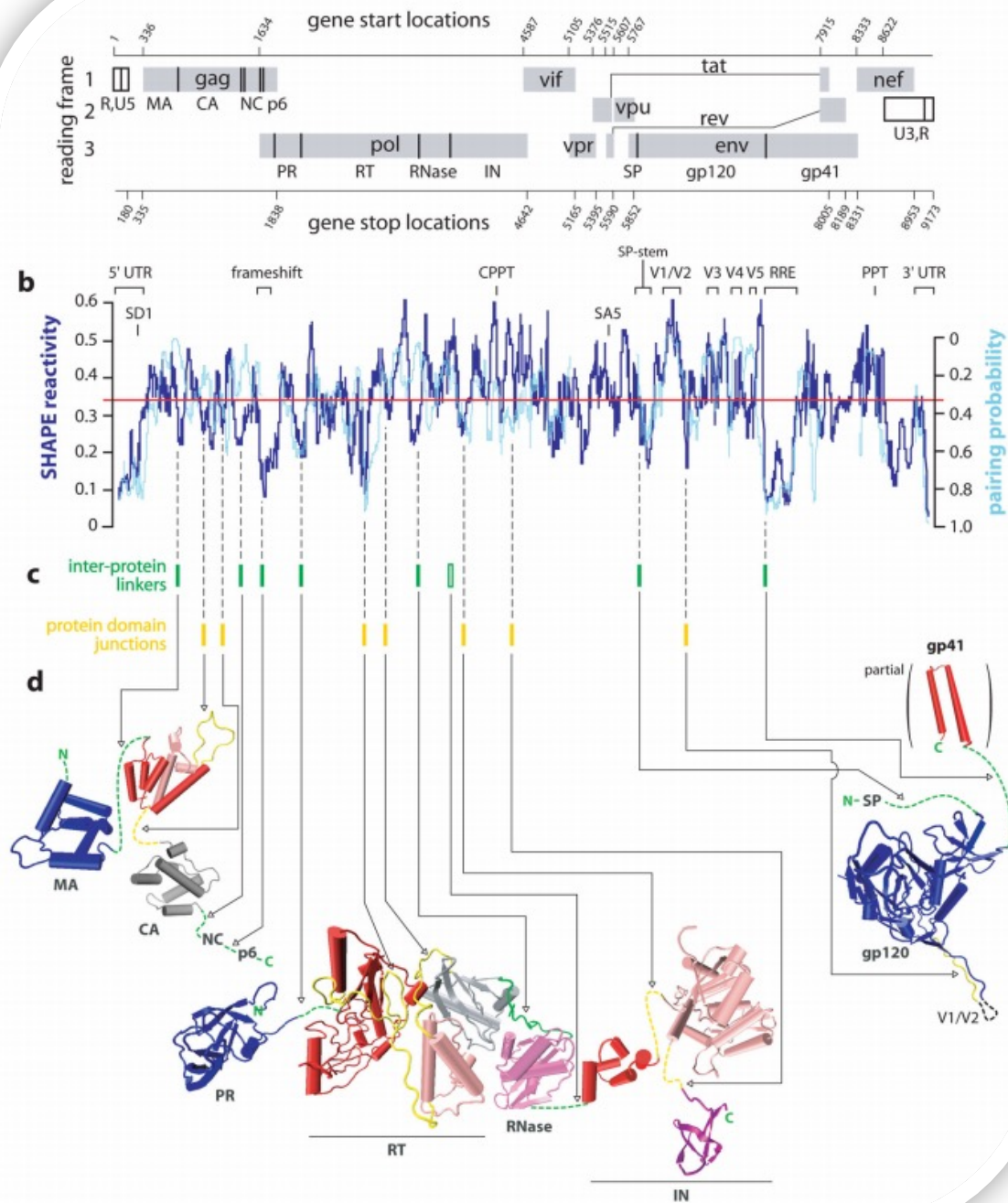
<sup>1</sup> Department of Chemistry, University of North Carolina, Chapel Hill, NC 27599-3290

<sup>2</sup> Department of Biomedical Engineering, University of North Carolina, Chapel Hill, NC 27599-3290

<sup>3</sup> Linenberger Cancer Center, University of North Carolina, Chapel Hill, NC 27599-3290

<sup>4</sup> Department of Biology, University of North Carolina, Chapel Hill, NC 27599-3290

<sup>5</sup> AIDS and Cancer Virus Program, SAIC-Frederick, Inc., NCI-Frederick, Frederick, MD 21702-1201





**TABLE 1** | PRR detection of HIV in HIV target cells.

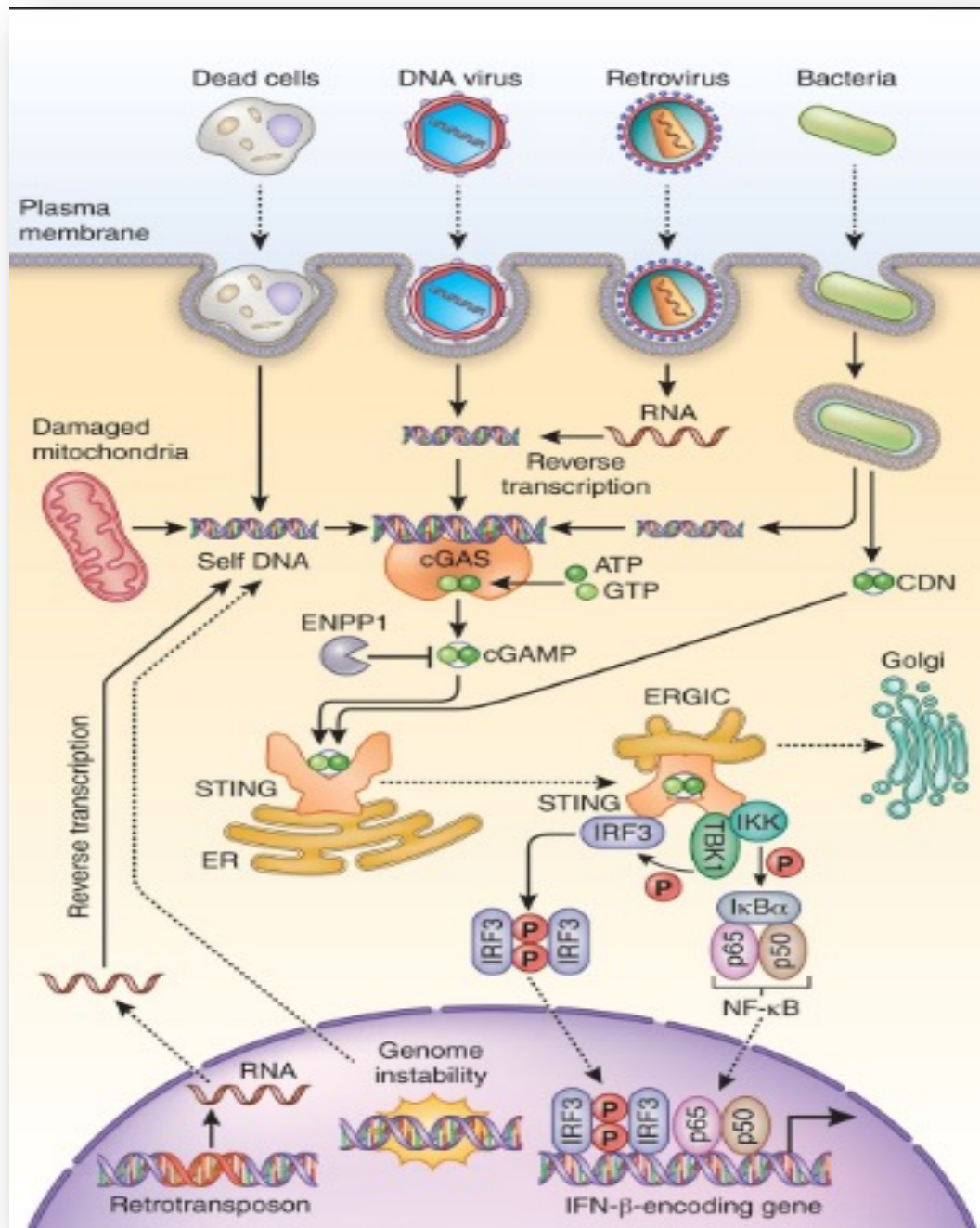
Cell type	PRR	How was the PRR implicated?	PAMP	Consequence	Reference
pDCs	TLR7	TLR7 antagonist	Purified genomic RNA	IFN, pro-inflammatory cytokines	(54)
Immature DCs	TLR8	Depletion by siRNA	ssRNA during infection	NF- $\kappa$ B activation, transcription of the integrated provirus	(55)
MDDC	cGAS	Depletion by shRNA, cGAMP production, and depletion by siRNA	RT products	CD86 expression, IFN and ISG induction	(32, 43, 44)
	PQBP1	Depletion by siRNA	RT products	ISG induction	(44)
	DDX3	Depletion by siRNA	Abortive RNA transcripts	IFN induction	(56)
MDM	cGAS	cGAMP production	RT products	NF- $\kappa$ B and IRF3 activation, IFN and ISG induction	(33, 43)
	IFI16	siRNA	RT products	Reduced replication and ISG induction	(45, 57)
	DDX3	Depletion by siRNA	Abortive RNA transcripts	IFN induction	(56)
Monocytes	NLRP3	Depletion by siRNA	Post-integration step	IL-1 $\beta$ and IL-18 production	(46, 58)
GECs	TLR2 and TLR4	Neutralizing Abs to TLRs	gp120	NF- $\kappa$ B activation and pro-inflammatory cytokine production	(59)
HLACs	IFI16	Depletion by shRNA	Abortive RT products	Pyroptosis	(60)
CD4+ T cells	DNA-PK	Chemical inhibitors	Viral integration	Cell death	(61)
	cGAS	Depletion by shRNA	Post-integration step	IFN and ISG induction	(62)
	cGAS	cGAMP production	Not determined	cGAMP production but no IFN response	(63)
	TLR7	Depletion by shRNA	Viral RNA	Anergy	(64)

IFN, interferon; DC, dendritic cell; pDC, plasmacytoid DC; PRR, pattern recognition receptor; PAMP, pathogen-associated molecular pattern; ISG, IFN-stimulated gene; TLR, toll-like receptor; MDDC, monocyte-derived dendritic cell; MDM, monocyte-derived macrophage; RT, reverse transcription; cGAS, cyclic GMP-AMP synthase; PQBP1, polyglutamine-binding protein 1; HLACs, human lymphoid-aggregated cultures; IFI16, interferon- $\gamma$  inducible protein 16; GECs, Genital epithelial cells.

**TABLE 2** | PRR detection of HIV in other cell types.

Cell type	PRR	How was the PRR implicated?	PAMP	Consequence	Reference
THP-1	cGAS	Depletion by shRNA	RT products	IRF3 activation, IFN and ISG induction	(43, 57)
	IFI16	Depletion by shRNA	RT products	IRF3 activation, IFN and ISG induction	(57)
	PQBP1	siRNA and hypomorphic mutation by CRISPR	RT products	ISG induction	(44)
	NLRP3	Depletion by shRNA	Post-integration step	IL-1 $\beta$ production	(46)
Huh7.5	RIG-I	Cell line is defective for RIG-I	Purified secondary-structured genomic RNA	ISG induction	(65)

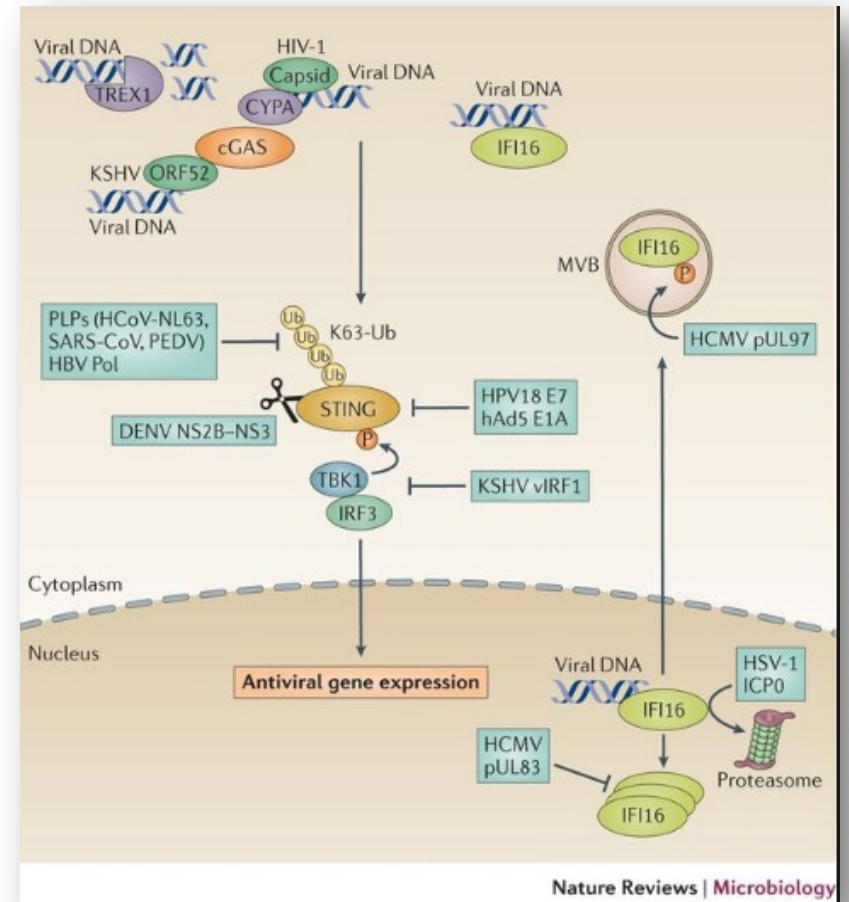
IFN, interferon; PRR, pattern recognition receptor; PAMP, pathogen-associated molecular pattern; ISG, IFN-stimulated gene; RT, reverse transcription; cGAS, cyclic GMP-AMP synthase; PQBP1, polyglutamine-binding protein 1; IFI16, interferon- $\gamma$  inducible protein 16.



## STING

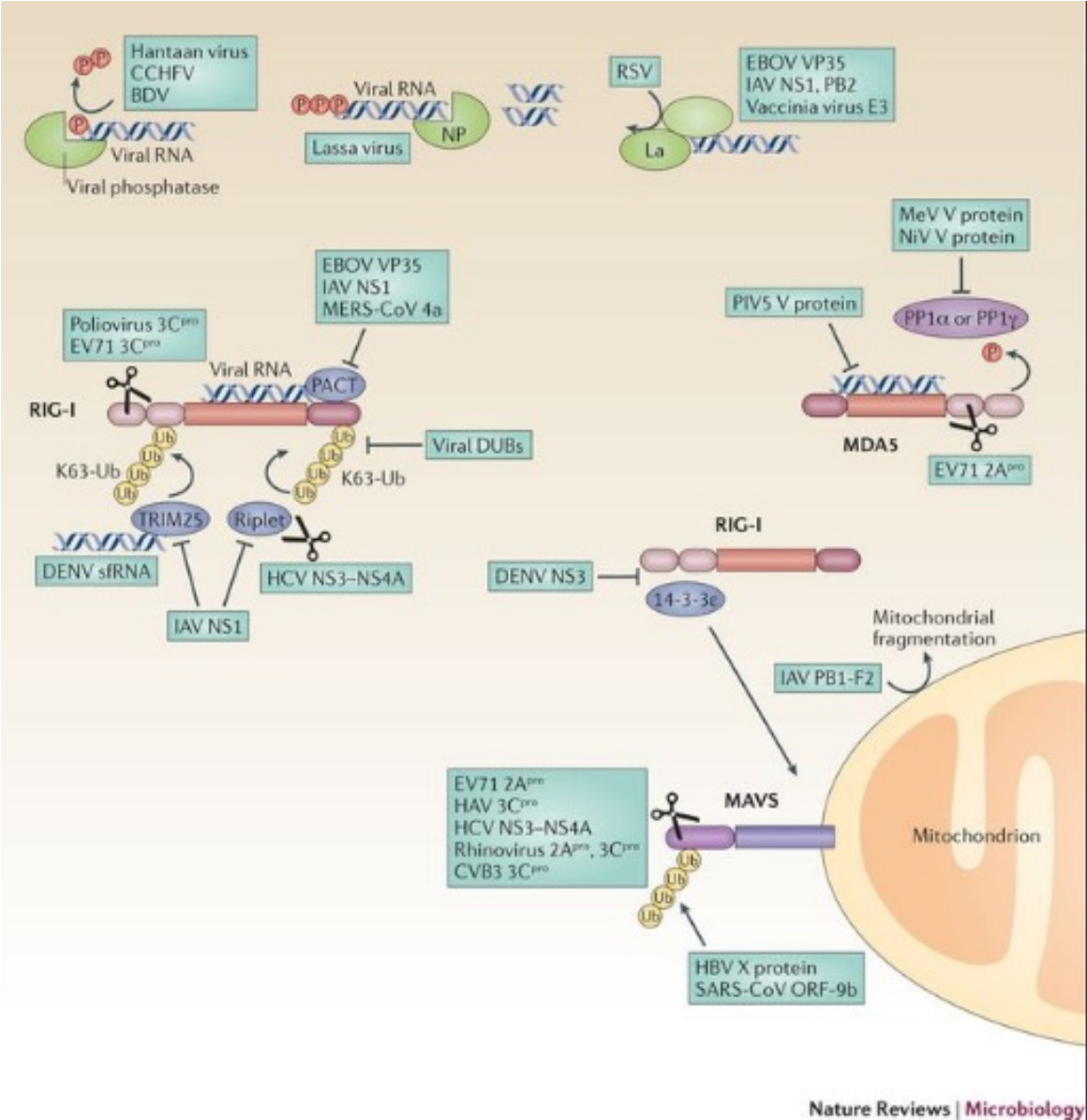
Sensor de DNA pós Transcrição Reversa

Ativação de NF- $\kappa$ B



# Outros Mecanismos

TRIM 25  
Riplet



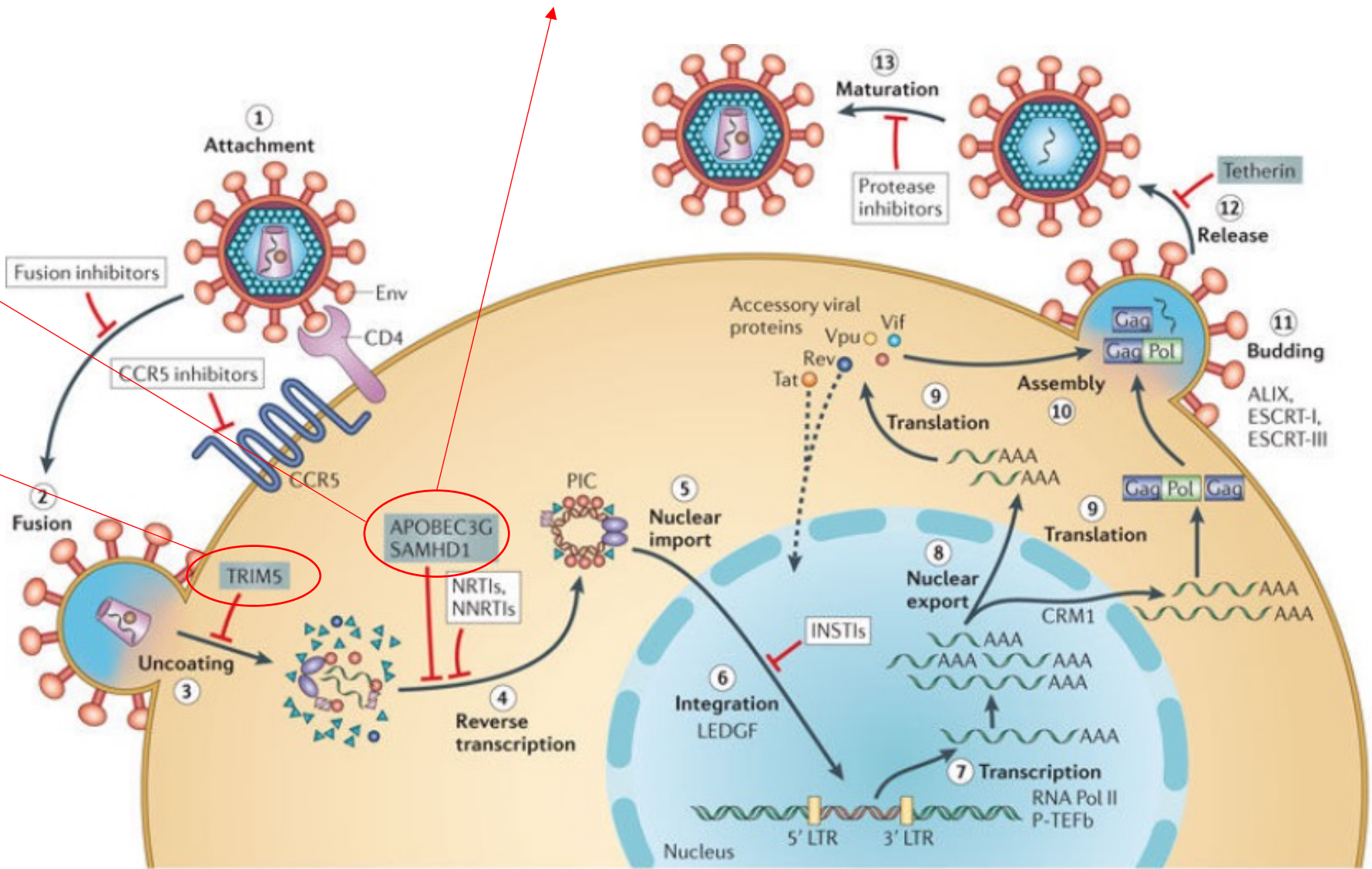
# Mas e nos Linfócitos T ?

**APOBEC**  
DNA deaminases  
Bloqueia síntese de DNA

**SAMHD1**  
dnTPase  
Reduz a concentração  
De dnTPS e restringe  
Replicação

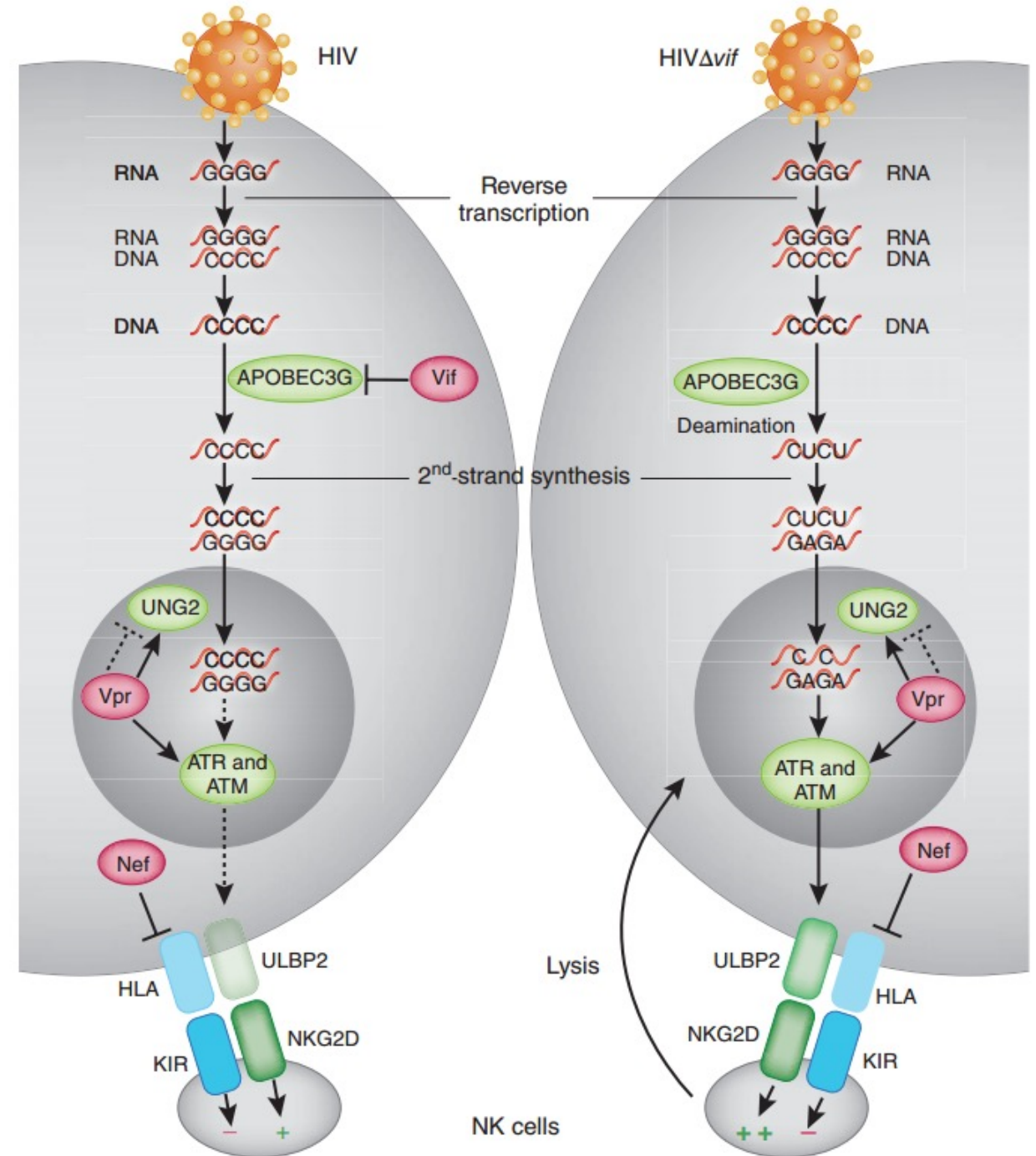
**TRIM5**  
Reconhece Capsídeo

Ativa  
NF-κB



APOBEC – VIF

Ubiquinação



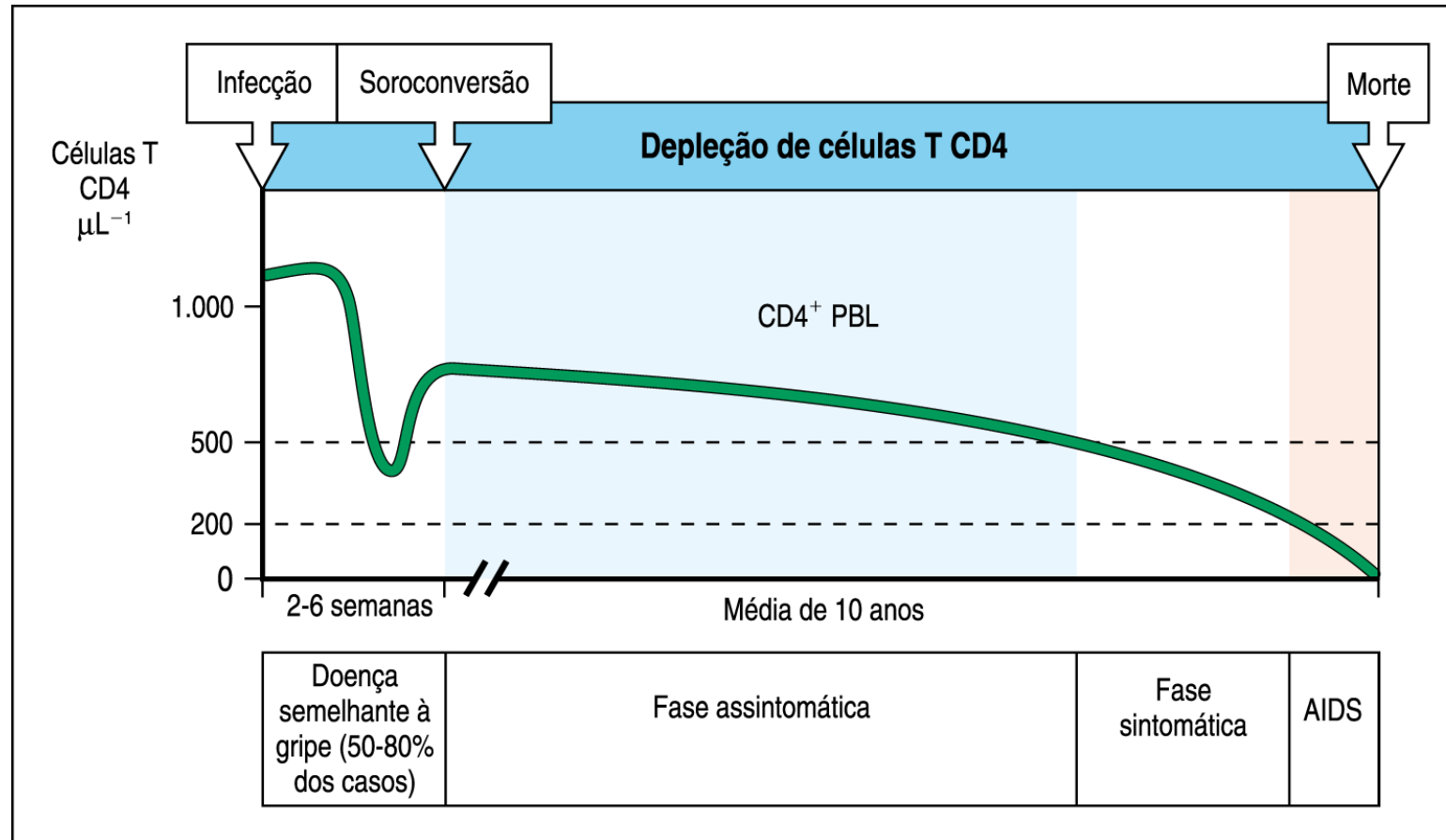


Figura 12.19 O curso típico de infecção pelo HIV.

Genes que influenciam a progressão da AIDS				
Gene	Alelo	Modo	Efeito	Mecanismo de ação
<b>Entrada do HIV</b>				
<i>CCR5</i>	$\Delta 32$	Recessivo	Prevêm a infecção	Nocautem na expressão do CCR5
		Dominante	Prevêm linfoma (L)	Diminui a disponibilidade de CCR5
	P1		Retarda AIDS	
<i>CCR2</i>	I64	Recessivo	Acelera AIDS (E)	Aumenta a expressão de CCR5
<i>CCL5</i>	In1.1c	Dominante	Retarda AIDS	Interage com e reduz CXCR4
<i>CXCL12</i>	3'A	Dominante	Acelera AIDS	Impede a transição CCR5-CXCR4 (?)
<i>CXCR6</i>	E3K	Recessivo	Retarda AIDS	Altera a ativação de células T (?)
<i>CCL2-CCL7-CCL11</i>	H7	Dominante	Intensifica a infecção	Estimula a resposta imune (?)

Figura 12.22 (Parte 1) Genes que influenciam a

~ ~ ~ AIDS ~ ~ ~

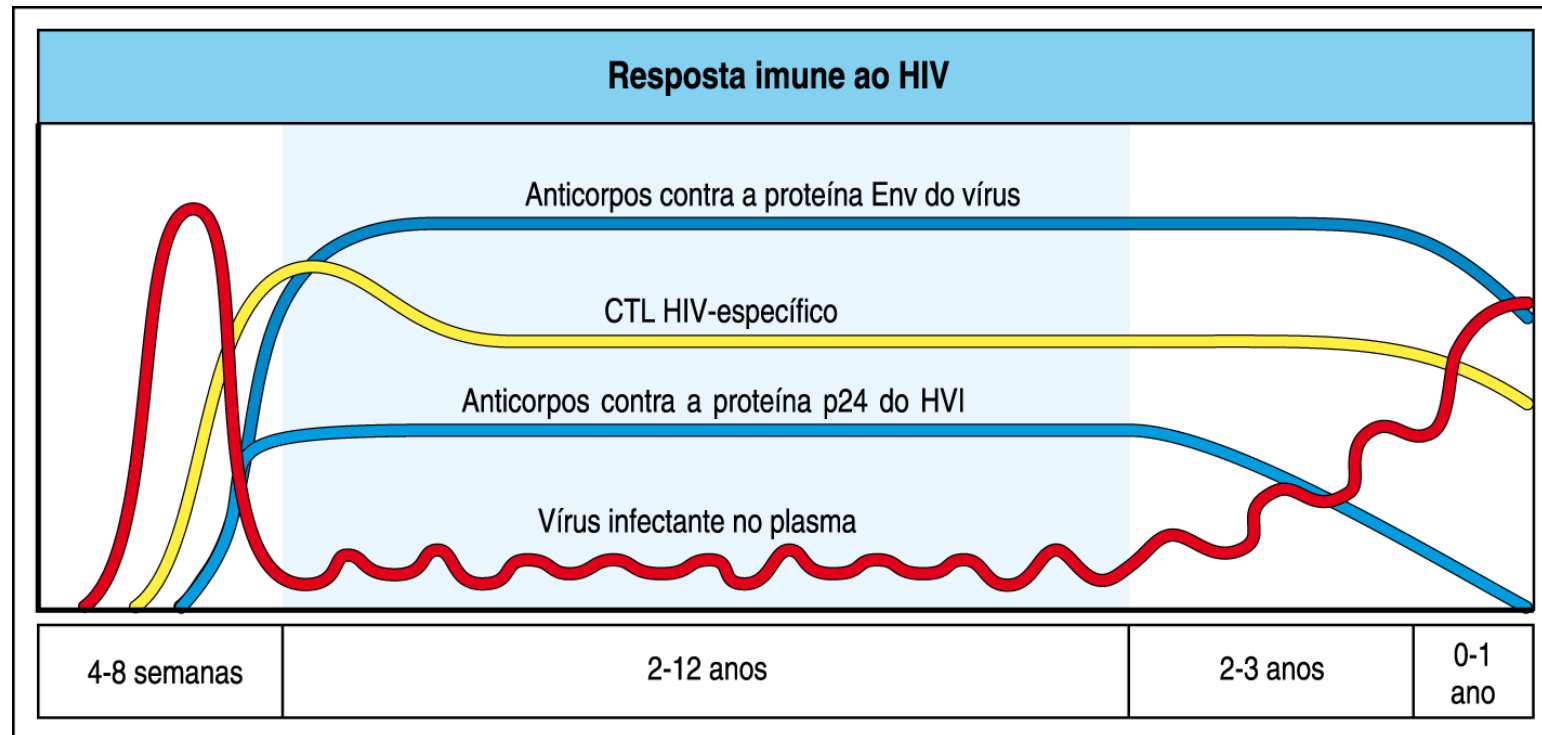


Figura 12.26 A resposta imune ao HIV.



Infecções	
Parasita	<i>Toxoplasma</i> sp. <i>Cryptosporidium</i> sp. <i>Leishmania</i> sp. <i>Microsporidium</i> sp.
Bactérias intracelulares	<i>Mycobacterium tuberculosis</i> <i>Mycobacterium avium intracellulare</i> <i>Salmonella</i> sp.
Fungos	<i>Pneumocystis carinii</i> <i>Cryptococcus neoformans</i> <i>Candida</i> sp. <i>Histoplasma capsulatum</i> <i>Coccidioides immitis</i>
Vírus	Herpes simples Citomegalovírus Varicela zoster
Tumores malignos	
Sarcoma de Kaposi – HHV8 Linfoma não-Hodgkin, incluindo linfoma de Burkitt EBV-positivo Linfoma primário do cérebro	

Figura 12.27 Diversos patógenos oportunistas e cânceres

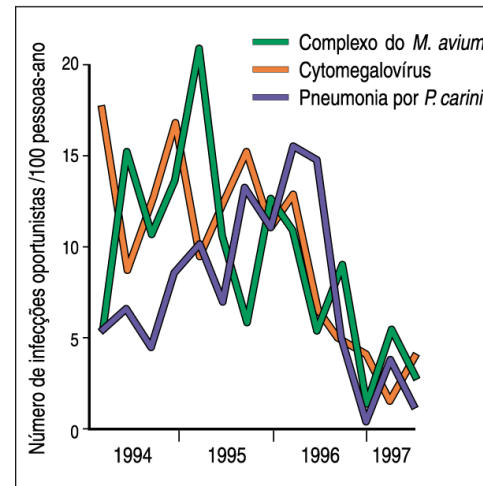
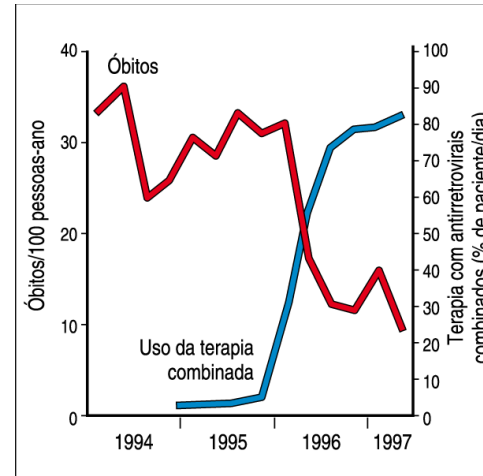


Figura 12.28 A morbidade e a mortalidade da infecção avançada

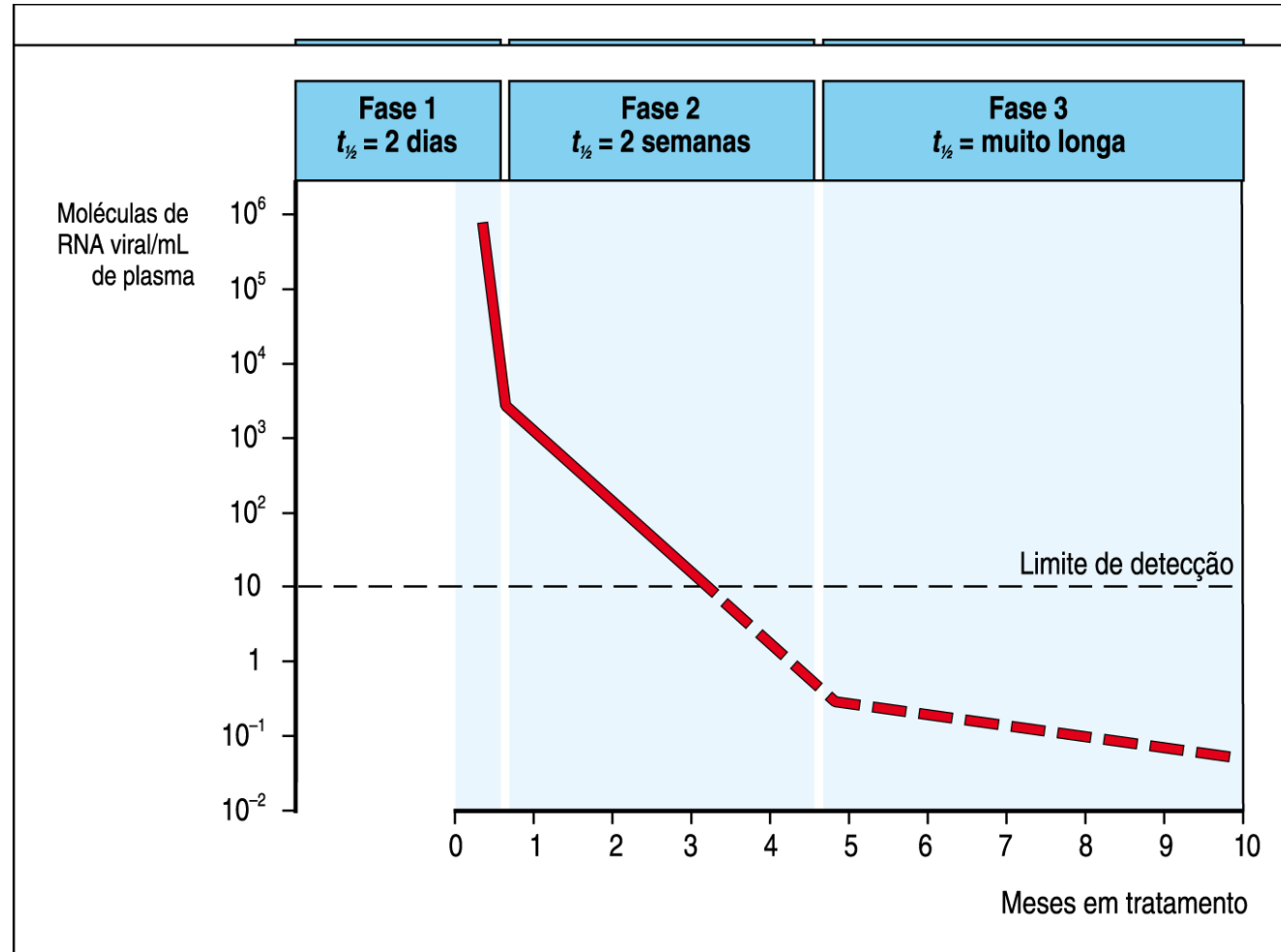


Figura 12.29 Decaimento viral durante a farmacoterapia.

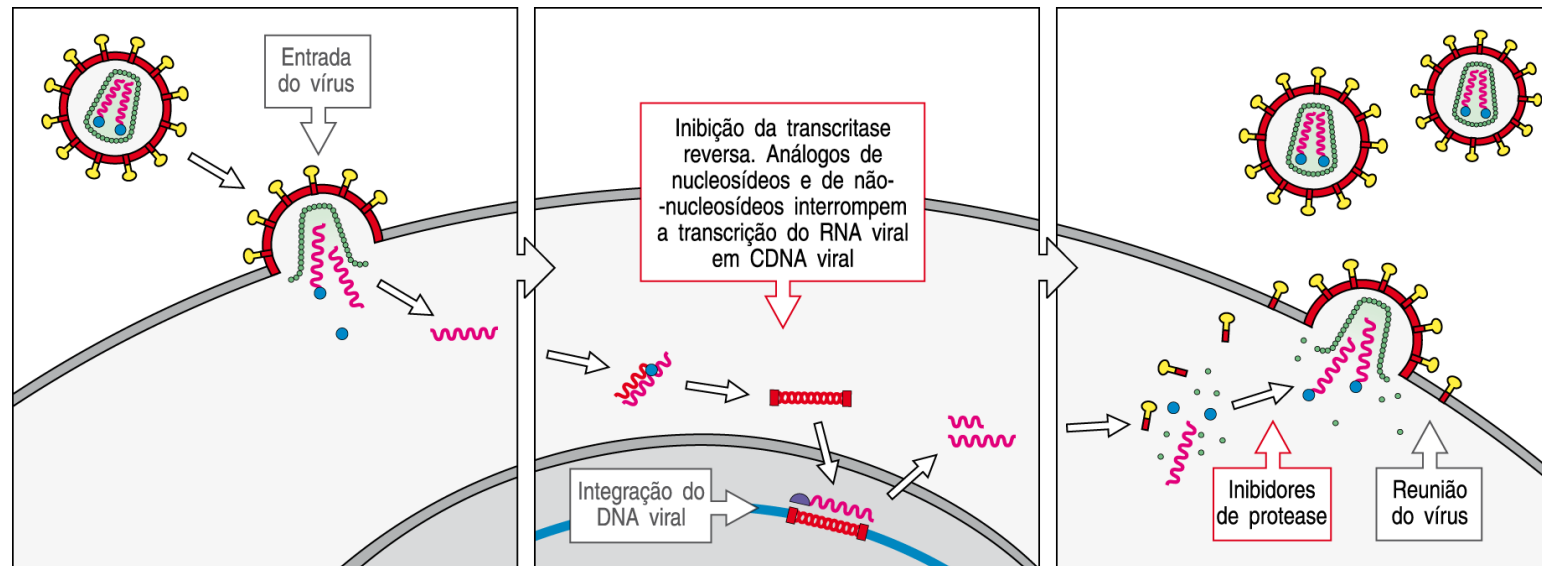


Figura 12.30 Possíveis alvos para a interferência do HIV com o ciclo viral.

# 1. Infections common to HIV/ AIDS

## • Tuberculosis (TB)

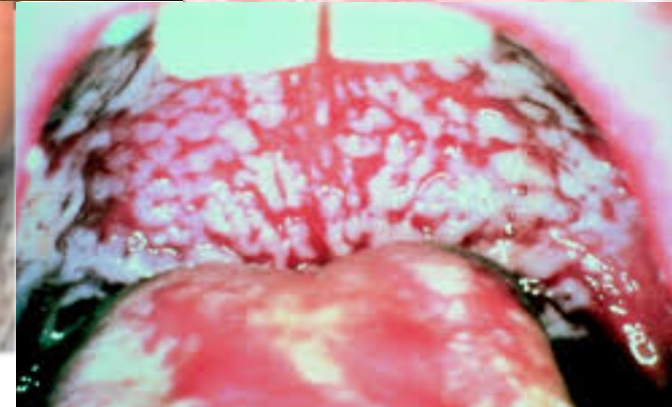
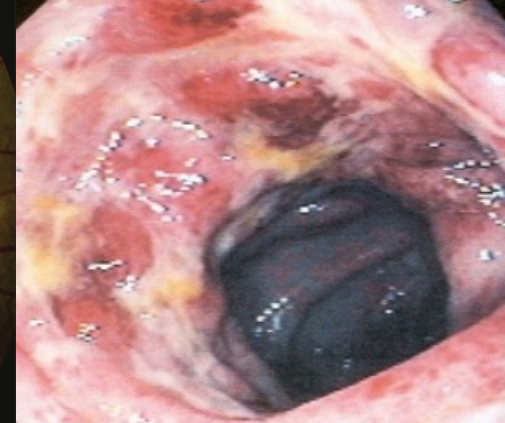
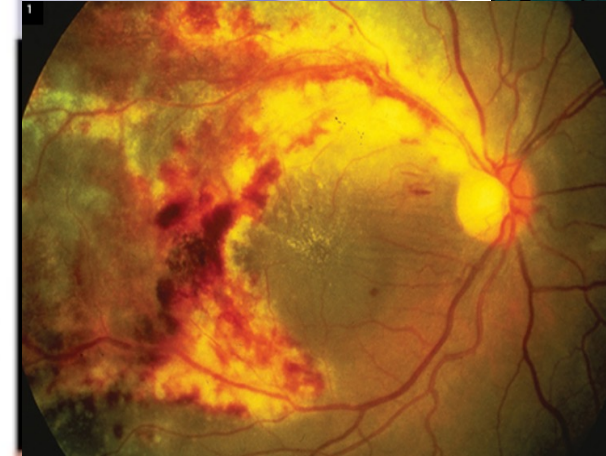
- In resource-poor nations, TB is the most common opportunistic infection associated with HIV and a leading cause of death among people with AIDS.

## • Cytomegalovirus

- This common herpes virus is transmitted in body fluids such as saliva, blood, urine, semen and breast milk.
- A healthy immune system inactivates the virus, and it remains dormant in your body.
- If your immune system weakens, the virus resurfaces — causing damage to your eyes, digestive tract, lungs or other organs.

## • Candidiasis

- Candidiasis is a common HIV-related infection.
- It causes inflammation and a thick, white coating on the mucous membranes of your mouth, tongue, oesophagus or vagina.



### 3. Other

- **Wasting syndrome.**

- Aggressive treatment regimens worsen wasting syndrome, but it still affects many patients.
- It's defined as a loss of at least 10% of body weight, accompanied by diarrhoea, chronic fever, and weight loss.

- **Neurological complications.**

- Although AIDS doesn't appear to cause neurological symptoms such as dementia, anxiety and difficulty walking.
- One of the most common neurological complications is Kaposi's sarcoma, which leads to behavioral changes and impaired functioning.

- **Kidney disease.**

- HIV-associated nephropathy (HIVAN) is a type of kidney disease that prevents your kidneys from removing excess fluid and waste from your blood and passing them to your urine.

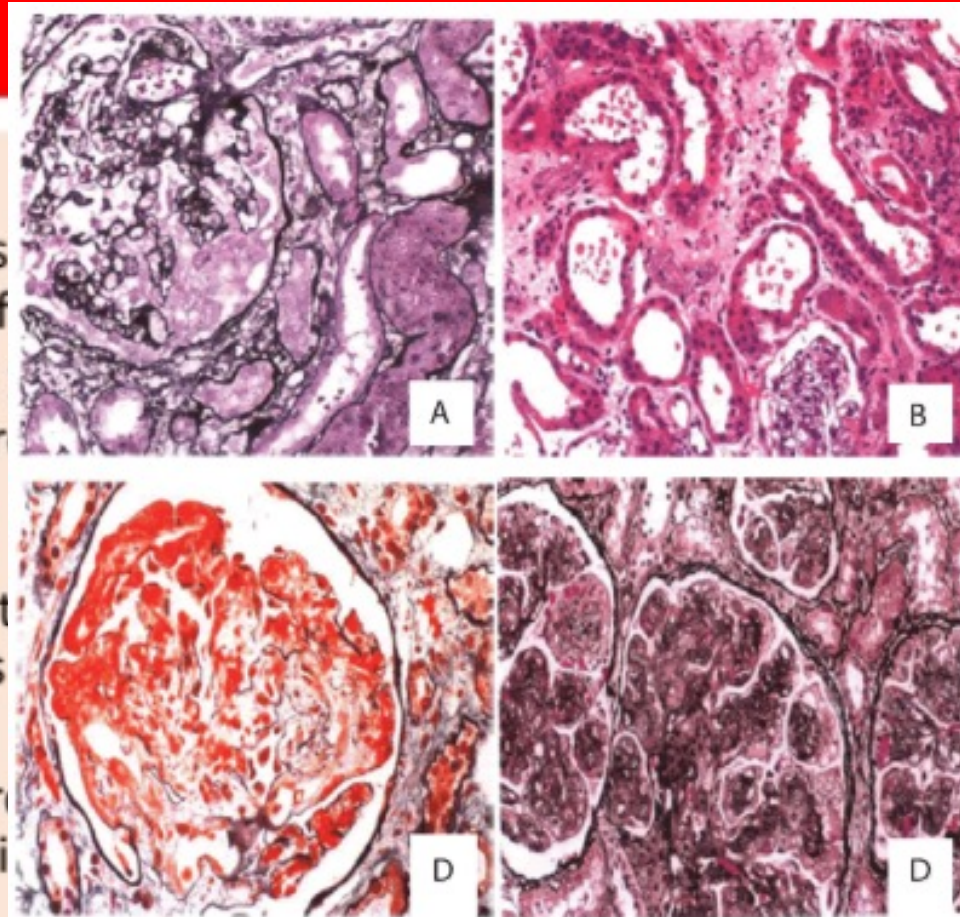
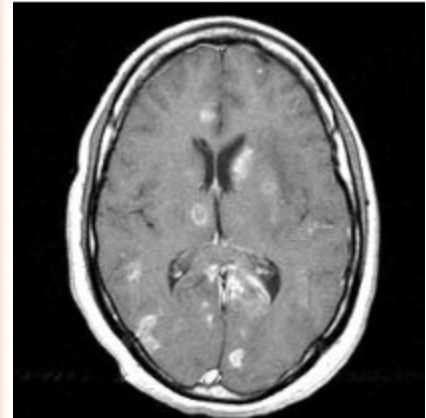


Figure 1: Pathological Characteristics of HIV infection-associated AKI

A) HIV-associated nephropathy (HIVAN): lobular glomerular collapse with overlying epithelial cell crowding and hypertrophy; B) Acute tubular necrosis: Focal tubular necrosis at multiple points along the nephron and rupture of the basement membrane; C) Thrombotic microangiopathies: mesangiolytic, double contour basement membrane; D) membranoproliferative glomerulonephritis (MPGN): intense glomerular hypercellularity, diffuse thickening of the glomerular basement membrane with the appearance of "double contours"[14].



Toxoplasma  
Gondii

# AIDS Related Infections

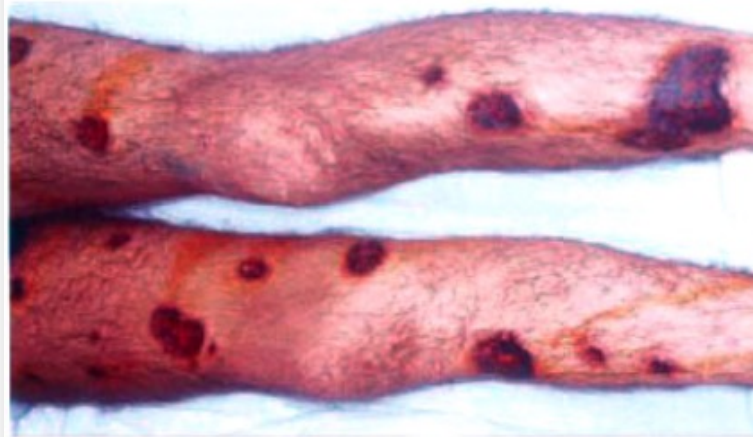


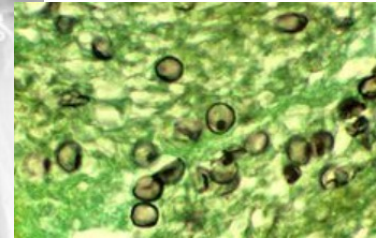
Figure 1 - Disseminated cutaneous sporotrichosis in AIDS patient.



Molusco contagioso



Sarcoma Kaposi

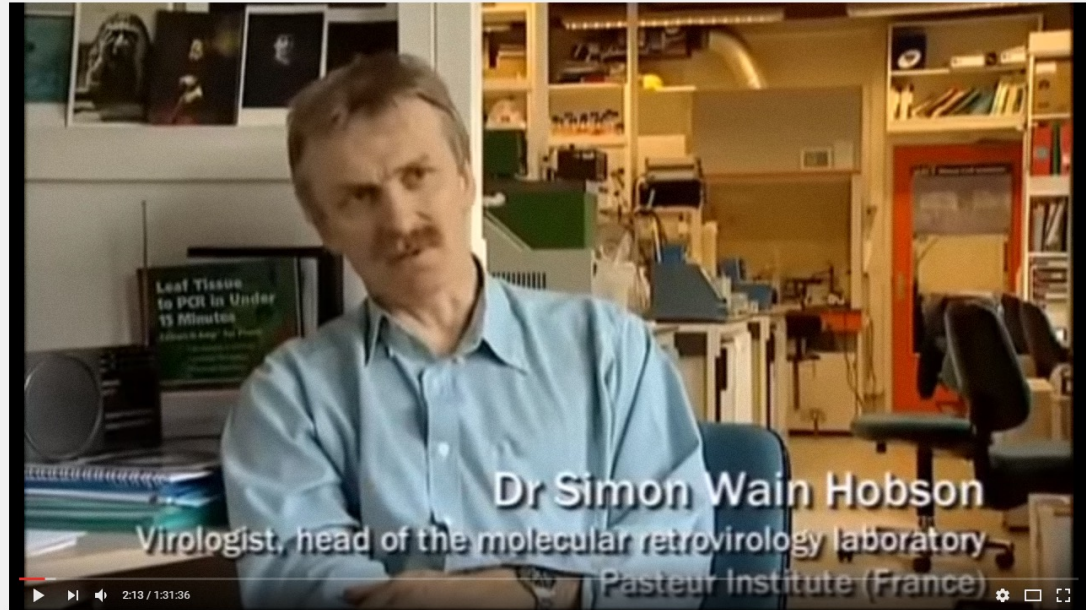


Pneumocistis carinii



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