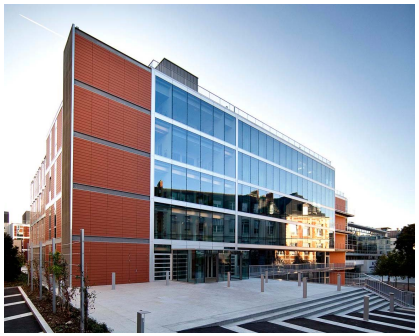
Visioconférence du 19 Décembre 2017
Centre d'Enseignement

« Eradication du VIH/Sida: où en est-on? »

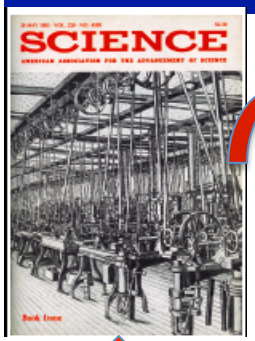
Françoise BARRÉ-SINOUSSI



Agence autonome de l'Inserm

ENSEMBLE CONTRE LE SIDA

35 ans de recherche translationnelle et de mobilisation internationale…



Une nouvelle ère depuis 1996…

Isolation of HIV
HIV sequencing
AZT
AIDS
Viral load PCR
HAART
Generic ARVs
Tetherin
HIV controllers
Circumcision
RV144 Vaccine Trial
Berlin Patient
HPTN 052
SAMHD1
Post-treatment controllers
Temprano/ Start

1981 1983 1985 1987 1989 1991 1993 1995 1997 1999 2001 2003 2005 2007 2009 2011 2013 2015

1982 1984 1986 1988 1990 1992 1994 1996 1998 2000 2002 2004 2006 2008 2010 2012 2014

CD4 receptor
PMTCT (AZT)
Co-receptors
APOBEC3G
TRIM5α
Microbial Translocation -> Immune Activation
CAPRISA 004
iPrEX
PreP

Legend:

HIV biology & pathogenesis
Treatment
Prevention

Depuis 1996: Ere des traitements combinés (cART) et du combat pour l'accès universel!

Infection VIH

↓

Traitement antirétroviral (cARV)

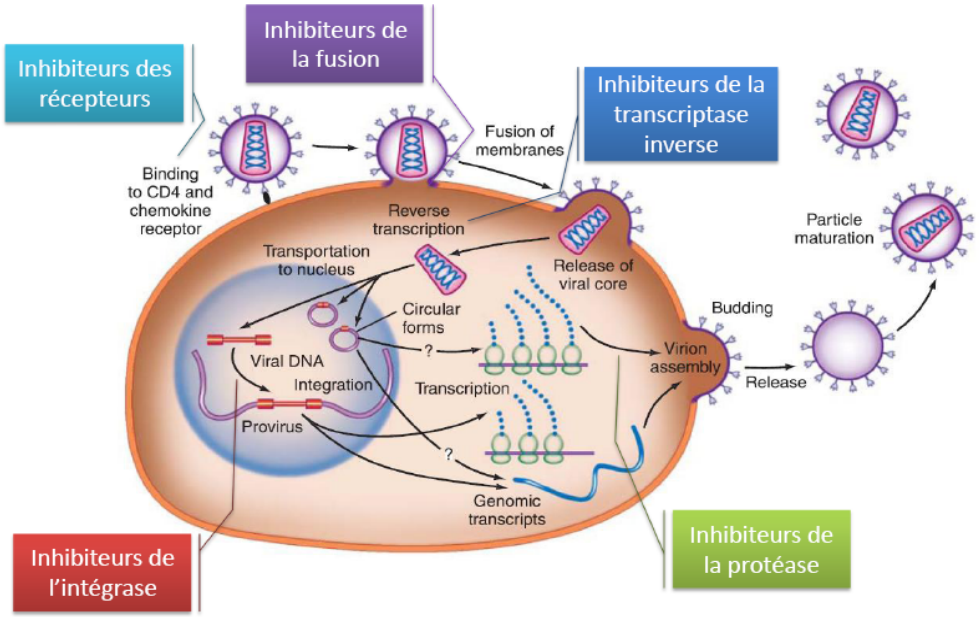
↓

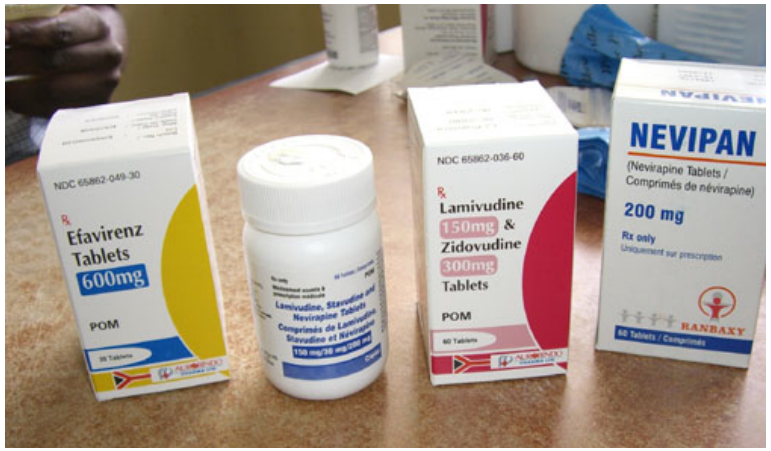
Contrôle de l'infection VIH
Restauration Fonction Immunitaire

↓

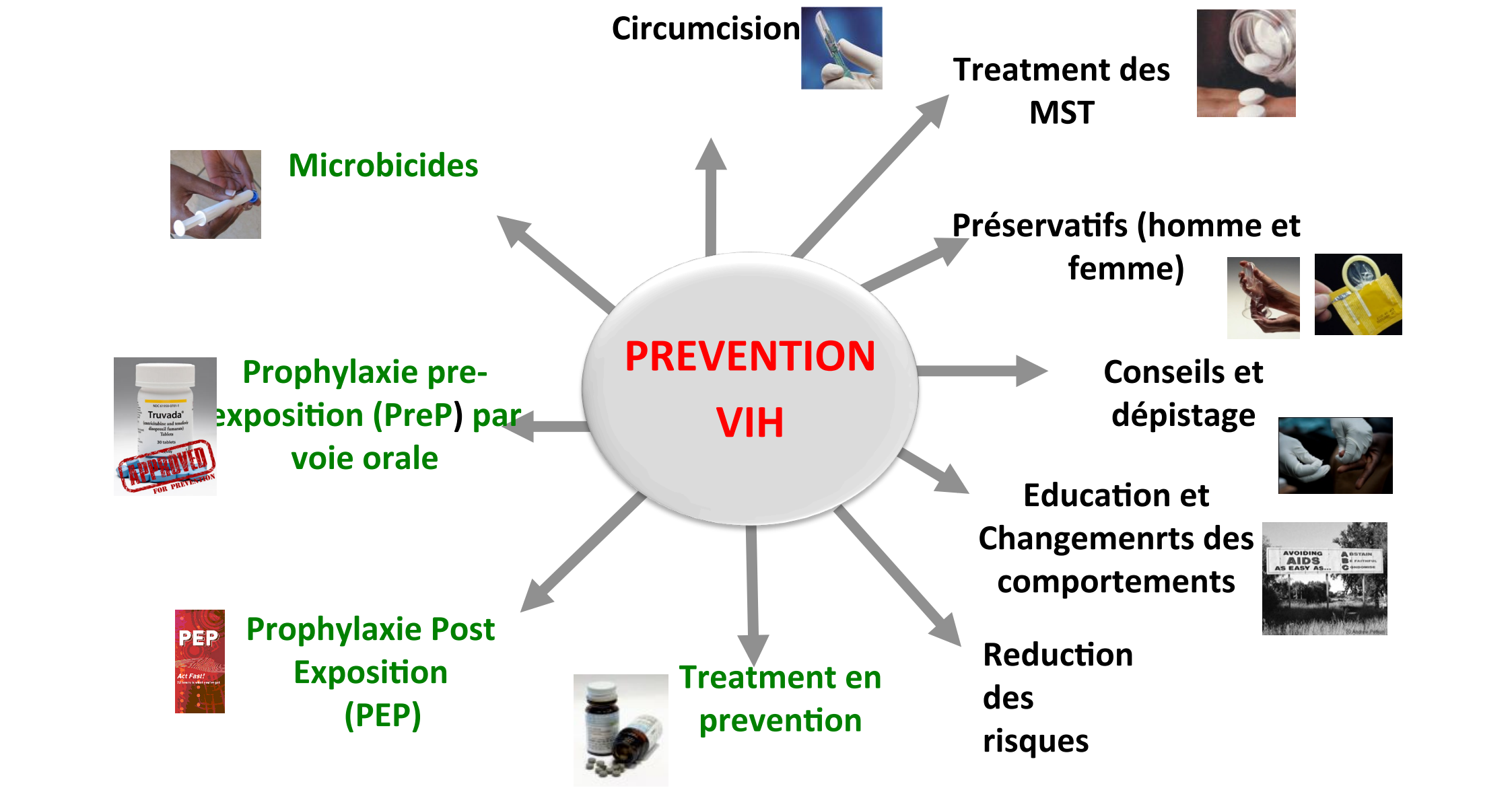
Amélioration de la qualité de vie
Espérance de vie similaire à VIH-
Prévention du Sida

> 30 molécules antirétrovirales et 13 combinaisons approuvées



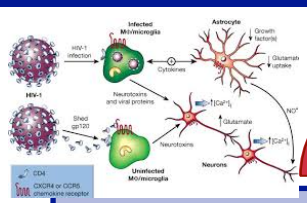


Prevention: une combinaison d'outils scientifiquement validés



ARV prophylaxie (0-96% efficacité, selon adhérence..)

Mobilisation internationale sans précédent basée sur des evidences scientifiques

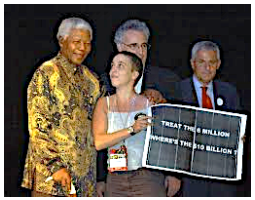


Preuves scientifiques

Activisme

Leaders politiques et autorités de santé

Participation des communautés VIH



Recherche multi-disciplinaire

Formation, renforcement des capacités & structures

Interventions

Organisation des soins et accès aux traitements

Décisions au bénéfice de TOUTES les populations quelqu'elles soient, où qu'elles soient…

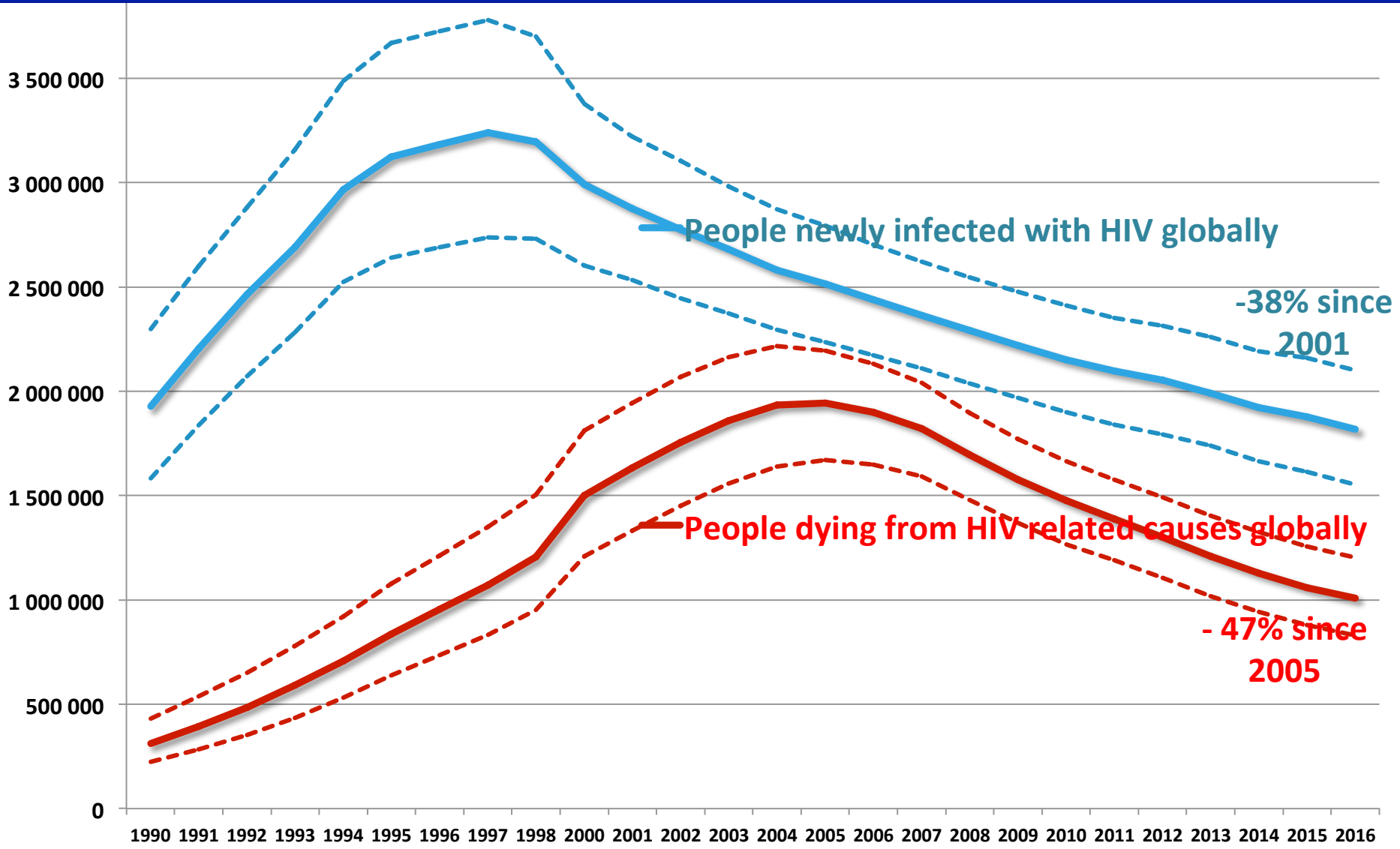
2003-2016: Accès élargi au traitement VIH

- Evidences scientifiques et activisme
- Investissement internationaux depuis 2003 (GF, Pepfar..)
- Reduction des prix et génériques (30000$/year US versus 100$ LMRS)
- Nouveaux services de prises en charge, impliquant les communautés, santé mobile et digitale..

Couverture thérapeutique trop hétérogène avec trop peu de pays ayant >80% PLWH sous ARV

UNAIDS. Ending AIDS. Progress towards the 90-90-90 targets, 2017

Depuis 1996: réduction du nombre de décès et de personnes vivant avec le VIH…



Source: UNAIDS/WHO estimates.

OMS Situation épidémie mondiale 2016

36.7 million

people now estimated to be living with HIV

[30.8–42.9 million]

During 2016…

1.8 million

people newly infected

[1.6–2.1 million]

1.0 million

HIV-related deaths

[830 000–1.2 million]

VIH/Sida: Défis et priorités actuels

Prévenir les nouvelles infections *(éducation, préservatifs, PreP, circoncision, réduction des risques…)*

Tester, traiter et retenir dans les soins *(30-50% des personnes VIH+ ignore leur statut; Continuité des soins chez 40 à 75% des patients)*

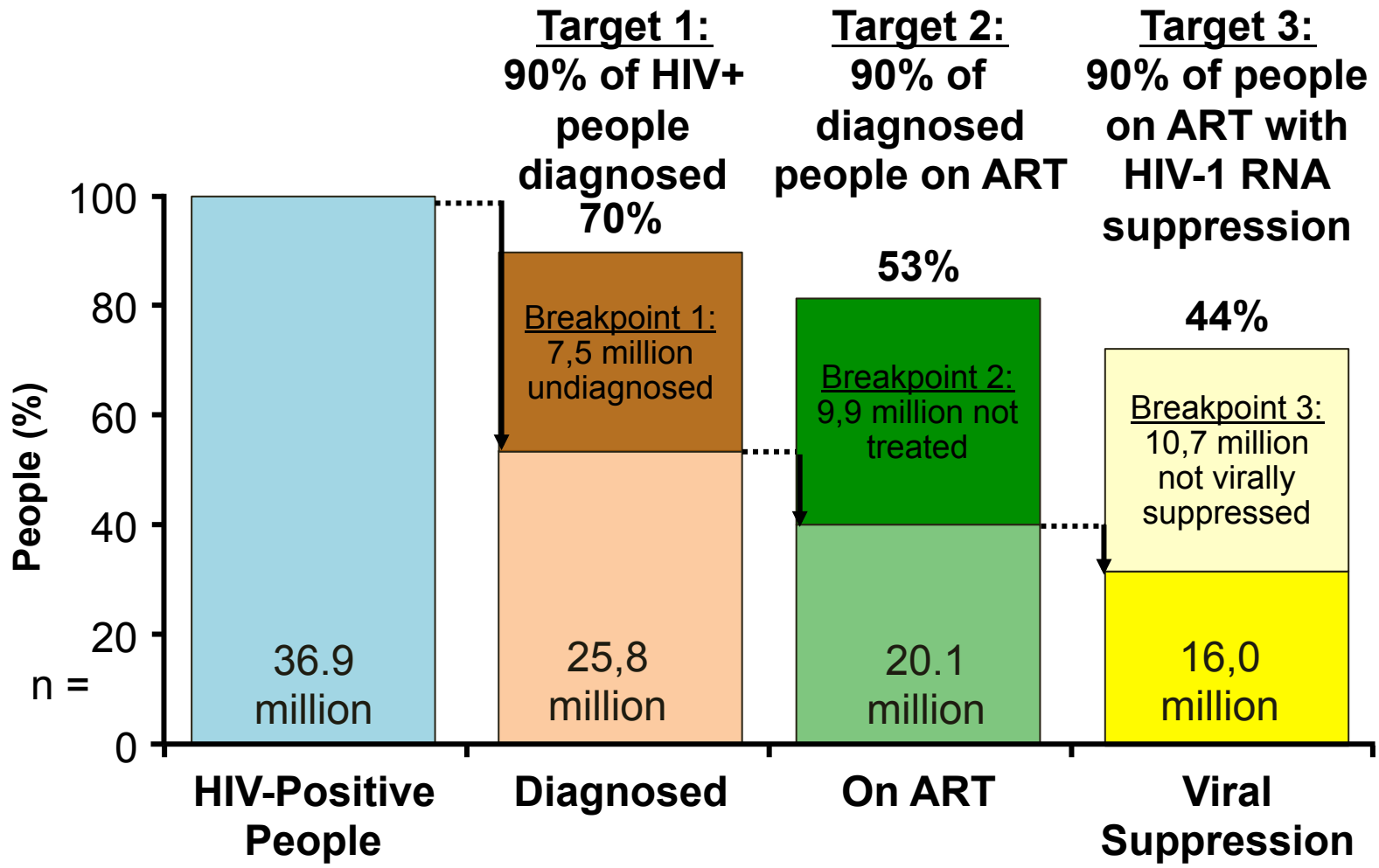
- **Education/Tolérance**
- **Renforcer les systèmes de santé, gouvernance et leaders**
- **Politiques nationales coordonnées et intégrées** *(recherche, intervention, associations..)*
- **Optimiser l'offre de soin** *(dépistage et traitement précoces, approvisionnement et distribution ART, interventions communautaires différenciées, mHealth, cART à action retard…*

RECHERCHE OPERATIONNELLE

PROGRAMME de SANTE PUBLIQUE PERFORMANT…

- Volonté/Décision politique
- Investissement internationaux
- Lutte contre la stigmatisation/discrimination, les législations répressives *(74 pays..)*…

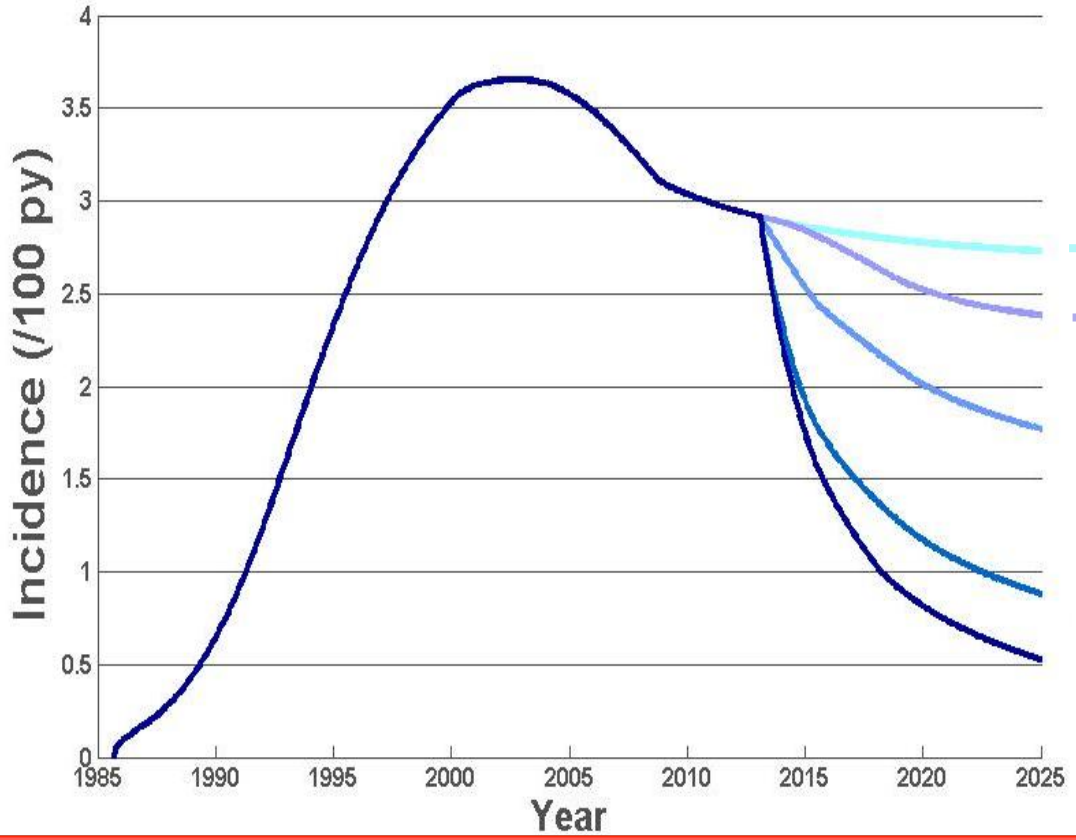
Fin de l'épidémie Sida en 2030: 90-90-90 de nouveaux objectifs ambitieux OMS/ONUSIDA…



UNAIDS/WHO estimates .

Est-ce réaliste et réalisable?

Est-il possible de controler l'épidémie VIH?



Status quo

+ 100% ART at CD4 200

+ Circumcision

+ Early ART

+ PrEP

Oui, mais éradiquer l'épidémie ne sera possible qu'avec un vaccin et un traitement curatif!

Source: Cremin I. et al. AIDS 2013

VIH/Sida: Défis et priorités de la recherche…

Vaccin VIH	Comorbidités sous ART	« HIV Cure »
Toujours pas de vaccin mais Progrès significatifs dans la recherche vaccinale depuis 2009	VIH, une infection chronique sous ART mais Anomalies immunes résiduelles sous ART	Infection VIH controllée sous ART mais persistante…

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Développer nos connaissances fondamentales sur la persistance virale, l'immunologie et la pathogénèse des l'infection VIH.

Nouvelles stratégies vaccinales et thérapeutiques?

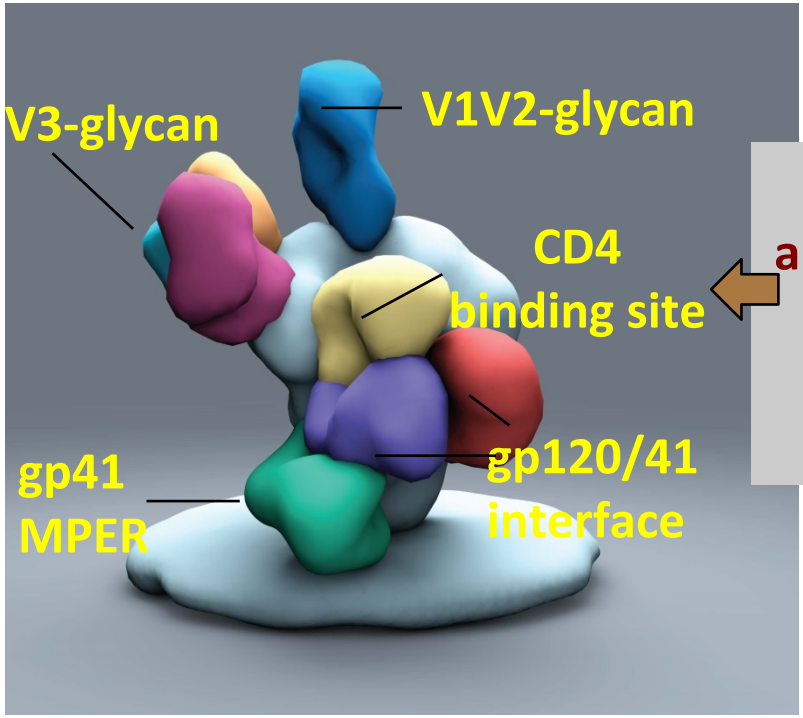
Vaccin prophylactiques anti-VIH: plus de 200 études mais peu d'essais Phase IIb/III

Dates	Clinical efficacy studies	Strategy	Viral targets	Immune response	Efficacy
1999-2003	AidsVax	Protein subunit (AIDSVAX)	monomeric rgp120	Type specific binding Ab	No
2005-2007	Step Phambili	Viral vector (Ad5)	gag/pol/nef	CD8+T (+++)	No
2005-2009	RV144 (Thai trial)	Prime: ALVAC-vCP152 + Boost: AIDSVAX	gag/pol/env + Monomeric rgp120 B/E	Polyfunctional CD4+ T cell (+/-) + Type specific binding V1V2 env Ab	Yes 31% reduction
2009- 2013	HVTN505	Prime: DNA + Boost: Ad5	gag/pol/nef/env		No (around 20 infections in each arm)

Depuis 2009: progrès significatifs
Nouvelles perspectives de la recherche vaccinale….

- **Identification d'anticorps neutralisants à large spectre chez des patients VIH+** ***(blocage infection cellule à cellule, activité liée à fonction effectrice Fc…)***
- **Identification de nouveaux sites de vulnérabilité de Env**
- **Anticorps non neutralisants mais protecteurs (ADCC, autres?)**

Christina Corbaci, Andrew Ward et al.

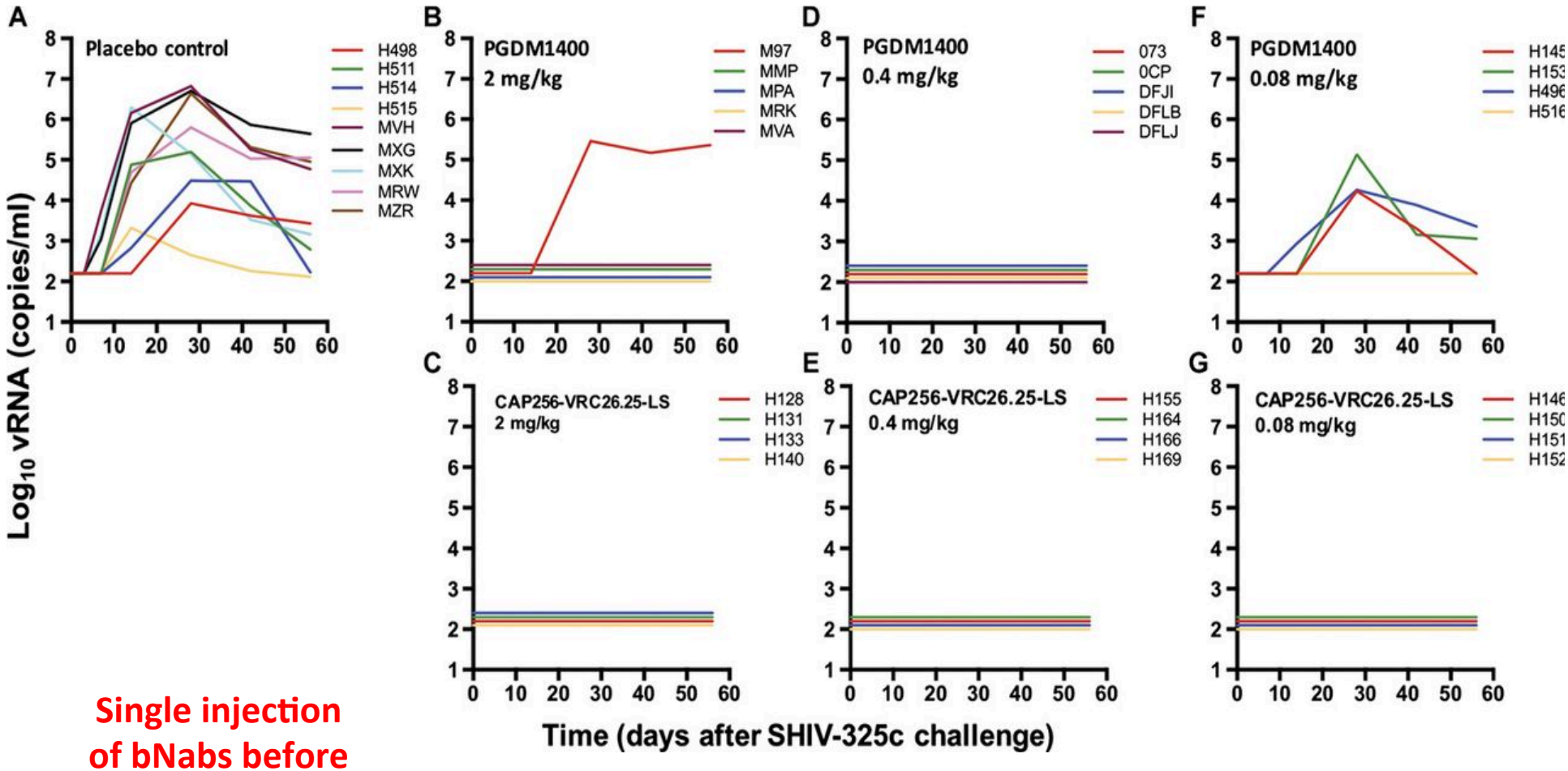


Plusieurs anticorps en clinique (VRC01, 3BNC117)

Conception d'immunogènes basés sur des connaissances structurales

- *Transfert de genes (CAR, TCR…)*
- *DC targeting…*

Immunisation passive efficace par des anticorps V2 env specifiques (PGDM1400 and CAP256-VRC26.25-LS) contre SHIV-325c



Single injection of bNabs before viral challenge

Boris Julg et al.,
Sci Transl Med 2017;9:eaal1321

Science Translational Medicine
AAAS

Depuis 2009: Vaccin anti-VIH, de nouveaux concepts…

- **Energie considerable dans ce domaine avec des études qui seront à l'origine de nouveaux développement dans la prochaine décennie *(prime-boost, bNabs, Réponse protectrice non conventionnelle….)***
- **Pour la premiere fois, la recherche fondamentale est intégrée et coordonnée avec la recherche préclinique et clinique!**

Enfin pas de Dogmes!

- **Strategie intégrée et coordonnée**
- **Nouvelles technologies**
- **Concepts innovants et à risques**

Vaccin? Cure?

Pouquoi un tratiement curatif? Attente des patients…

2011 Workshop on HIV persistence, St Marteen
Fred Verdult and Steve Deeks

- **Quelles sont les priorités et préférences des patients?**
- **Quelles sont les obstacles et les solutions à l'engagement des patients?**
- **Comment préparer au mieux des essais cliniques?**
- **Quelles seront les meilleures options pour assurer un accès equitable au traitement?**

- Enquête: Est-ce important de guérir du VIH?
  - **Oui: >70%**
- Pourquoi?
  - **Raisons médicales mais aussi:**
  - **Peur de transmettre à d'autres,**
  - **Anxiété vis à vis du futur**
  - **Discrimination sous Tx…**

Ethique, Sciences Sociales et Humaines…

« Vers un traitement curatif »?

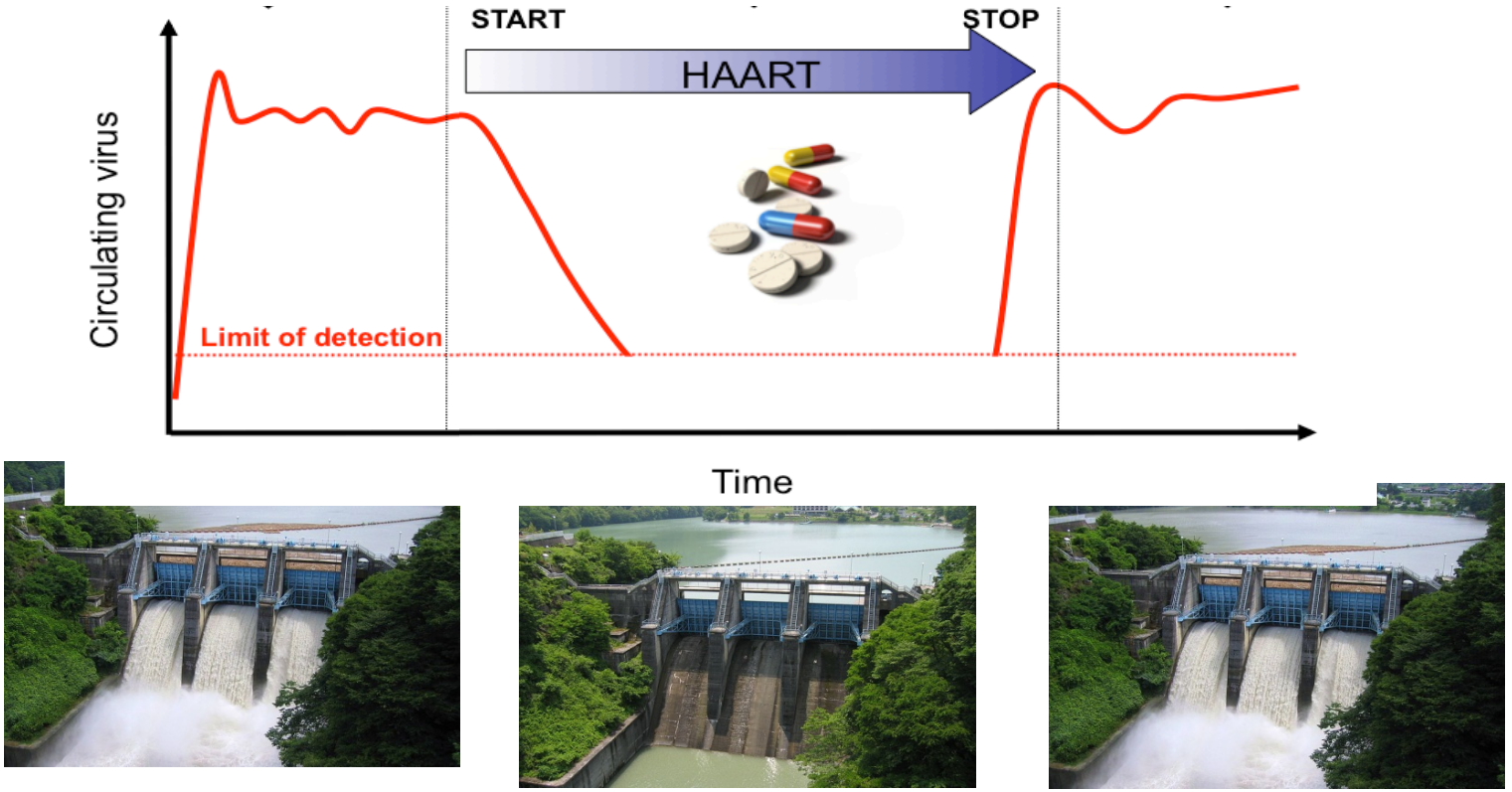
Attente des patients!

- **37 millions PVVIH: 20,9 millions sous cART**
  - **Trop peu de pays avec une couverture >80%**
  - **2 millions environ de nouvelles infections/an**
- **Nombre croissant de patients ayant besoin de traitement coûteux de 2ème ou 3ème ligne et de résistance aux ARV….**
- **Traitement à vie** ***(problème d'adhérence, de Stigmatisation/discrimination, de toxicité, de morbidité non Sida)***
- **Coût à long-terme de cART et investissements internationaux incertains…**
- **Nouveaux Traitements = nouveaux outils de prévention**

Traitement ART à vie pour tous reste un défi majeur…

Défis…..

Pourquoi le traitement doit être pris à vie?



- **Pénétration des ARV dans les tissus**
- **Replication résiduelle liée à l'inflammation/activation immunitaire**
- **Infection latente de cellules T CD4 naïves, quiescentes (TCM et TTM) et prolifération**
- **Réservoirs anatomiques**

Multiples réservoirs VIH cachés et persistants dans les tissus lymphoides…

✓**Major reservoirs are resting central & transitional CD4+ memory T cells** (*Persistent and stable on cART >10 years);*

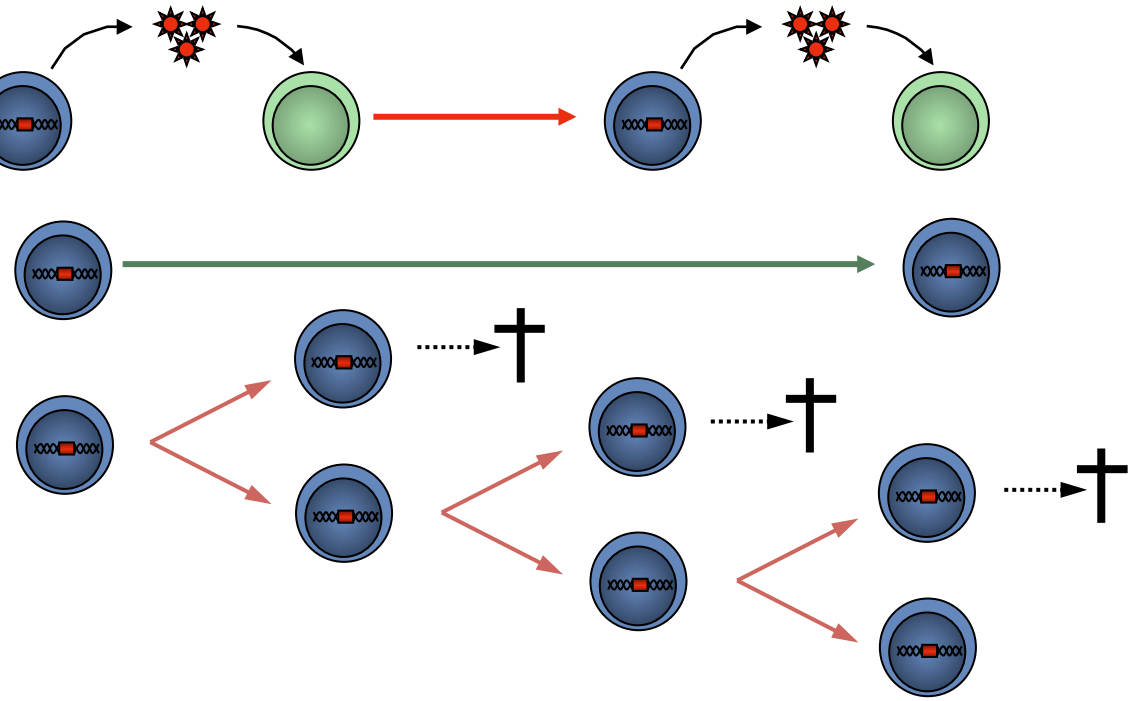
✓**Other reservoir cells**: *naiveT cells, memory stem T cells, T follicular helper cells (EC), meyloid cells, astrocytes, hematopoietic progenitor cells, etc…*

✓**Anatomic reservoirs**: *GI & genital tract, lymphoid tissu, CNS…*

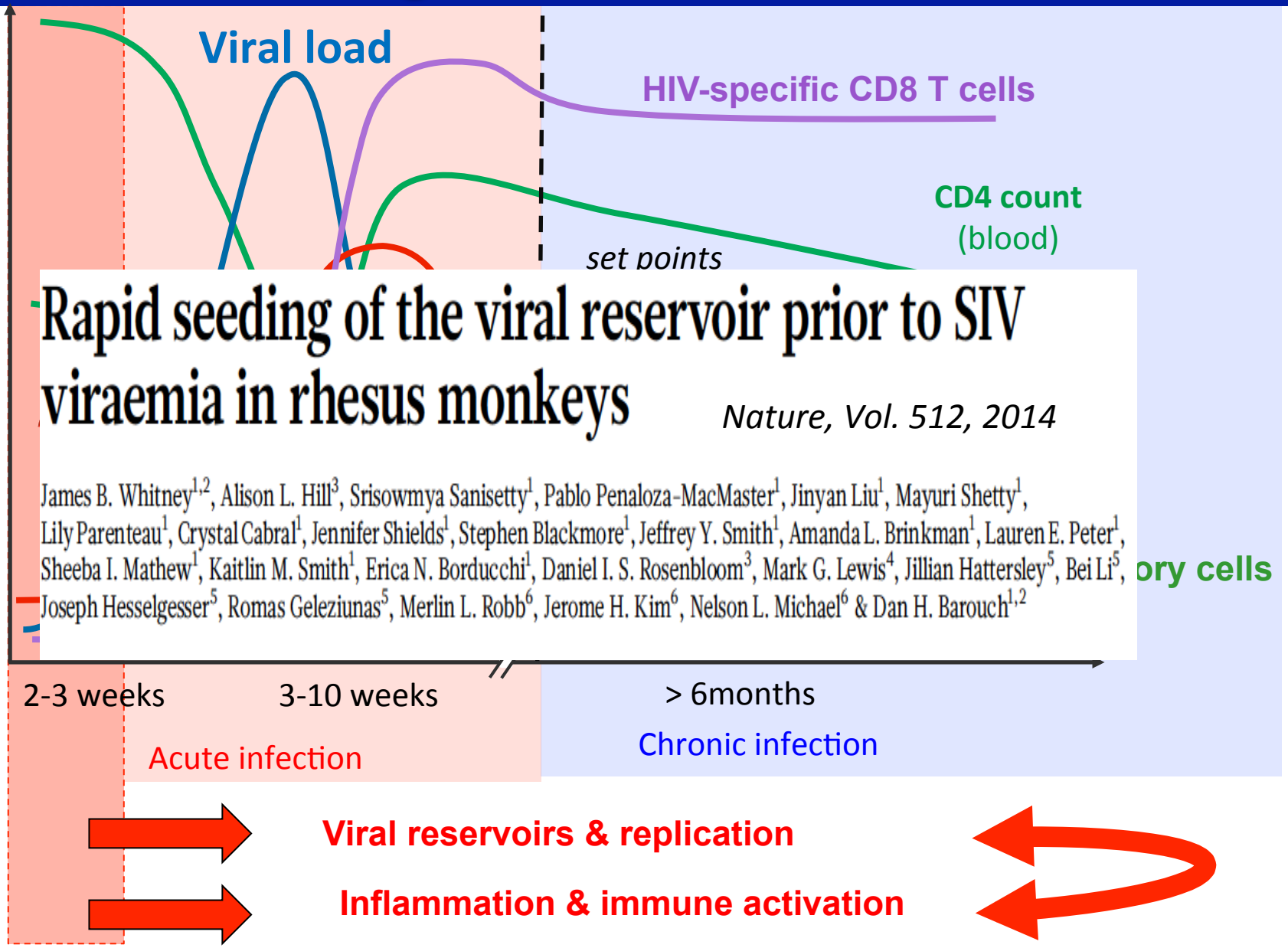
Residual viral replication

T cell survival

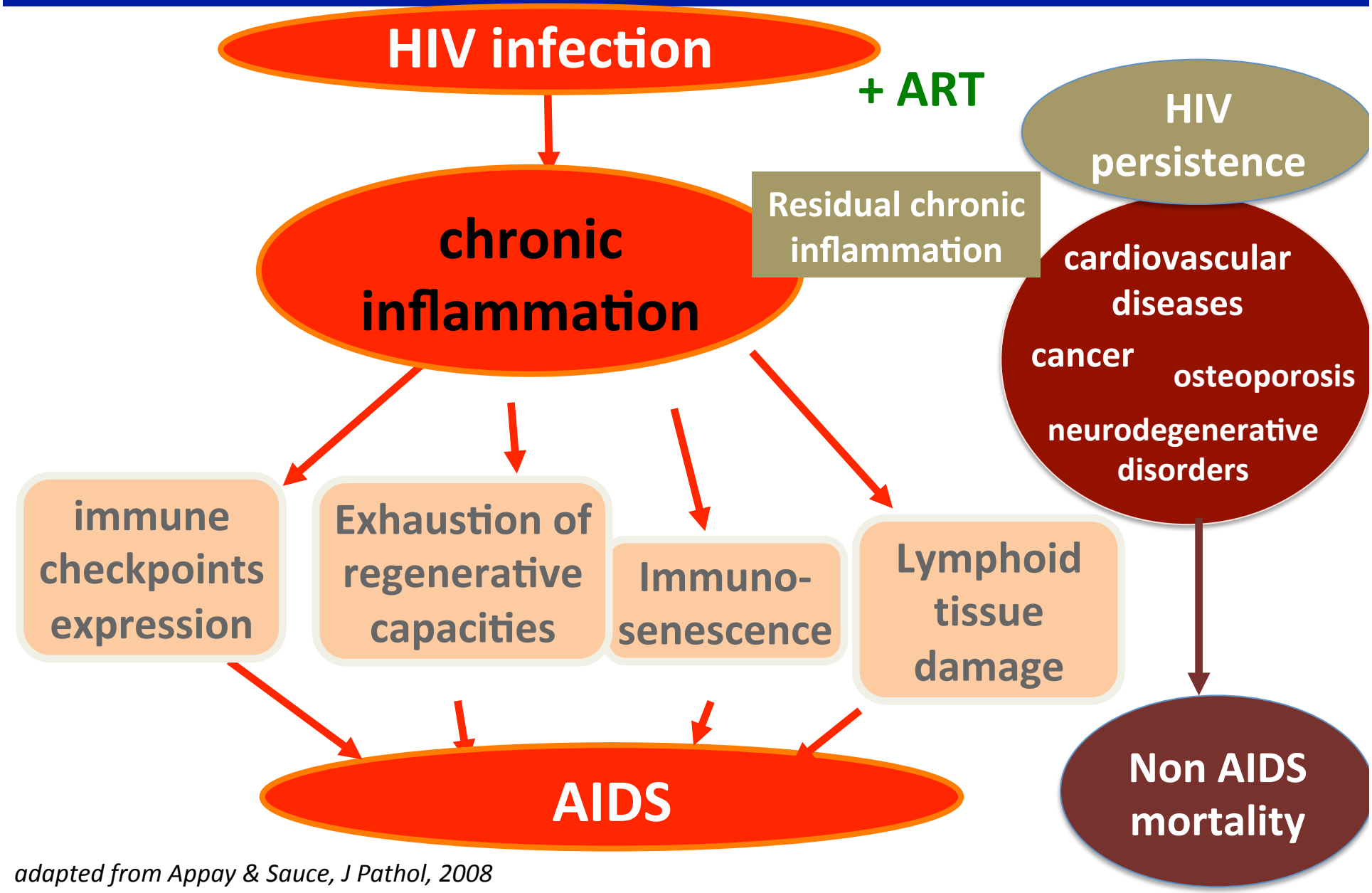
Homeostatic Proliferation
(clonal expansion): *expression of Immune checkpoints molecules (PD-1, LAG-3, TIGIT, CTLA-4)*



Pathogenèse de l'infection VIH



Inflammation chronique , persistance VIH et mortalité non Sida



adapted from Appay & Sauce, J Pathol, 2008

“HIV Cure”: Que cherche-t-on?

Reservoirs VIH sous ART …

Guerison

Elimination de toutes les cellules infectées de façon latente

Berlin Patient?

Remission durable

Contrôle permanent sans Tx➔

Vivre avec le VIH sans traitement et sans transmettre.

Concept réaliste!

Pourquoi pense-t-on qu'a minima une rémission de l'infection est possible?

Un seul cas de guérison…

Transplantation de moelle osseuse: *preuve de concept du* **"Patient de Berlin"** *(donneur CCR5Δ32),*

Cas de rémission sans traitement

Contrôleurs du VIH: patients infectés, naïfs de tout traitement, contrôle naturellement leur infection *(CV non détectable; faible niveau de réservoirs).*

Cas de rémission après un traitement très précoce:

ANRS EP 47 VISCONTI *(Saez-Cirion et al, PloS Pathogens 2013*): **23 patients VIH+ traités 10 semaines PI pendant 3 ans, >12 ans de contrôle sans ARV et un enfant traité à la naissance en remission depuis près de 15 ans…**

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Agenda International de priorités scientifiques….

PERSPECTIVES

OPINION

Towards an HIV cure: a global scientific strategy

The International AIDS Society Scientific Working Group on HIV Cure

Abstract | Given the limitations of antiretroviral therapy and recent advances in our understanding of HIV persistence during effective treatment, there is a growing recognition that a cure for HIV infection is both needed and feasible. The International AIDS Society convened a group of international experts to develop a scientific strategy for research towards an HIV cure. Several priorities for basic, translational and clinical research were identified. This Opinion article summarizes the group's recommended key goals for the international community.

Although in the current decade the HIV epidemic continues unabated, a notable success is that more than 20 different antiretroviral drugs are now available in many countries. When these antiretroviral drugs are used in combination, they improve health and prolong life in HIV-infected individuals and reduce the rates of transmission of the virus. Indeed, HIV-infected individuals who harbour a drug-susceptible viral strain, have access to antiretroviral drugs and are fully compliant with therapy can achieve and maintain complete, or near complete, viral suppression for years to decades. However, despite these successes, standard therapies do not fully restore health or a normal immune status in HIV-infected individuals, and patients still experience co-morbidities, such as increased cardiovascular disease, bone disorders and cognitive impairment[1]. In addition, interruption of antiretroviral therapy almost invariably leads to the re-emergence of detectable viral replication and the progression of AIDS. Perhaps more importantly, only a minority of HIV-infected individuals globally have access to antiretroviral therapy.

The cost of antiretroviral therapy has decreased substantially in recent years, and the availability of these drugs in resource-poor settings has steadily increased. However, the cost associated with delivering antiretroviral drugs to the 35 million people who are now living with HIV is overwhelming many organizations and public health systems. It is estimated that for every HIV-infected person who starts antiretroviral therapy, two individuals are newly infected with HIV; this is clearly unsustainable[2]. The continued presence on a global level of a large number of untreated HIV-infected individuals — who are the main source of ongoing HIV transmission[3] — means that the infected population is likely to grow. Given these well-recognized issues, there is a growing interest in developing curative strategies to tackle HIV[4–7]. Theoretically, a safe, affordable and scalable cure could address both the individual and public health limitations that are associated with lifelong antiretroviral therapy.

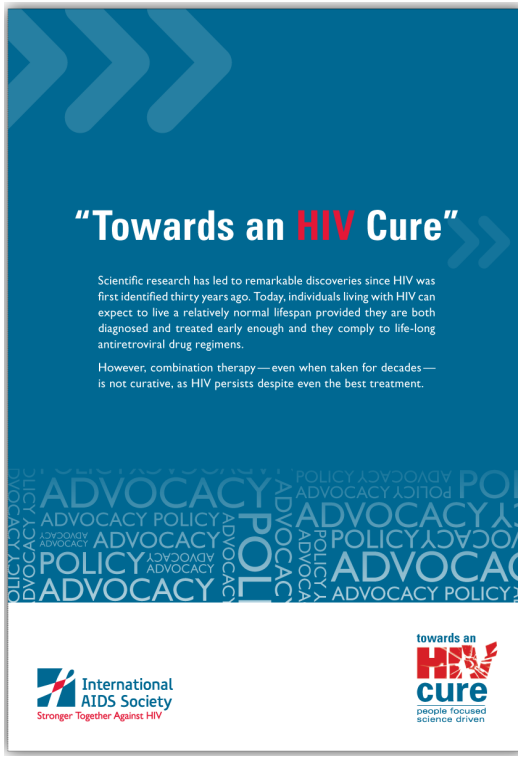
The International AIDS Society (IAS) convened a team of more than 40 scientists who are active in the field of HIV research. We have met frequently over the past 2 years. Throughout this process, the IAS has engaged with a broad range of stakeholders from around the world and has exhaustively solicited advice on the steps that should be taken to develop a cure for HIV infection. These efforts include the creation of a stakeholders' advisory board, and online and in-person discussions with hundreds of community activists, representatives from pharmaceutical and biotechnology industries, funding and regulatory agencies, and key HIV and non-HIV researchers from across the world.

In this Opinion article, we provide a concise, multidisciplinary plan that identifies a set of key scientific priorities that should bring us measurably closer to our vision of developing a cure for HIV infection (BOX 2). These priorities span the areas of basic, translational and clinical investigation. Two broadly defined approaches for curing HIV infection were considered by the group: first, the elimination of all HIV-infected cells (a sterilizing cure); and, second, the generation of effective host immunity to HIV that would result in lifelong control of the virus in the absence of therapy, despite not achieving the complete eradication of HIV (a functional cure). Here, we describe how the priorities identified by the IAS can allow us to achieve a sterilizing or functional cure for HIV.

Basic science aspects of HIV cure research

Multiple mechanisms are likely to contribute to HIV persistence during long-term, otherwise effective, antiretroviral therapy. These include the persistence of pools of latently infected CD4⁺ T cells[8], *de novo* infection of target host cells (ongoing viral replication)[9,10] and the failure of the host immune system to recognize and eliminate infected cells. In the following sections, we discuss the mechanisms that we need to understand in order to identify a strategy that will lead to an HIV cure.

Mechanisms that establish HIV latency. Most CD4⁺ T cells that are productively infected with HIV are likely to die from virus-induced cytopathic effects, but a small subset of long-lived 'resting' memory T cells that harbour integrated HIV DNA persist indefinitely[11] (a phenomenon generally referred to as latent infection) (FIG. 1,2). Although less well characterized, latent HIV infection may also occur in cell populations other than memory CD4⁺ T cells, including naive CD4⁺ T cells, tissue macrophages, astrocytes, thymocytes and perhaps haematopoietic progenitor cells[12–14]. The establishment of latency in resting memory T cells is due either to the infection of resting CD4⁺ T cells (which is difficult to achieve ex vivo)[15,16] or to the infection of highly susceptible activated CD4⁺ T cells followed by their reversion to



IAS working group
Towards an HIV cure: a global scientific strategy.
Nature Rev. Immunol. July 2012,
Update in Nat. Med. August 2016

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Quelles stratégies?

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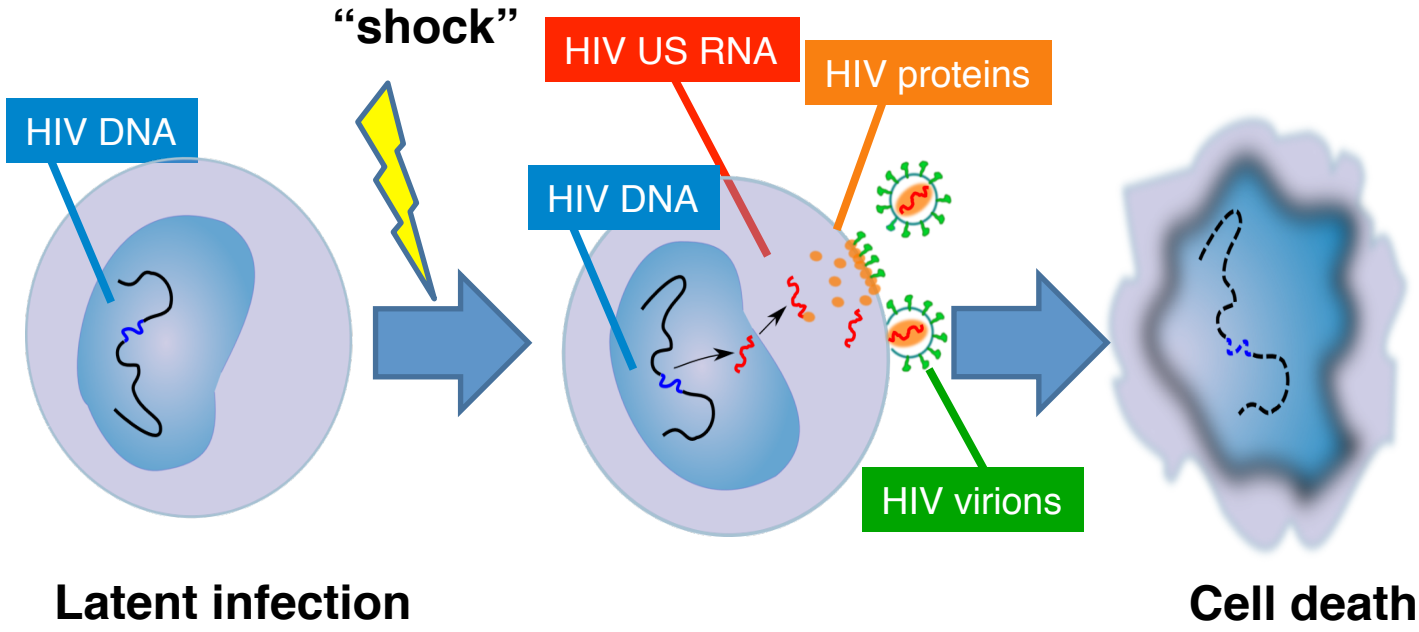
Activation de la latence virale : “shock”



Latency reactivating agents (LRA) eg., modify chromatin

LRA accelerating cell death eg., disulfiram, TLR agonists

Plusieurs molécules activatrices de la latence, mais pas d'elimination des cellules réservoirs.

Latency reversing agent	Site of action	HIV latency	US HIV RNA	Plasma RNA	HIV DNA
Vorinostat	HDACi	Single dose[1] Intermittent[2] Continuous[3]	↑	↔	↔
Panobinostat	HDACi	Intermittent dose[4]	↑	+/-	↔
Romidepsin	HDACi	Weekly dose[5]	↑↑	↑↑	↔
Disulfiram	AKT activation	High dose 2g/ day[6]	↑	↑	↔
Bryostatin	PKC agonist	Low dose 10-20ug/m^2	↔	↔	↔

HDACi = histone deacetylase inhibitor; US HIV RNA = unspliced HIV RNA

[1] Archin et al., *Nature* 2012; [2] Archin et al., *J Infect Dis* 2014; [3]Elliott J et al., *Plos Pathogens* 2014; [4]Rasmussen et al., *Lancet HIV* 2014; [5]Sogaard et al., *Plos Pathogens* 2015; [6]Elliott J et al., *Lancet HIV* 2015; [7]Gutierrez et al., AIDS 2016

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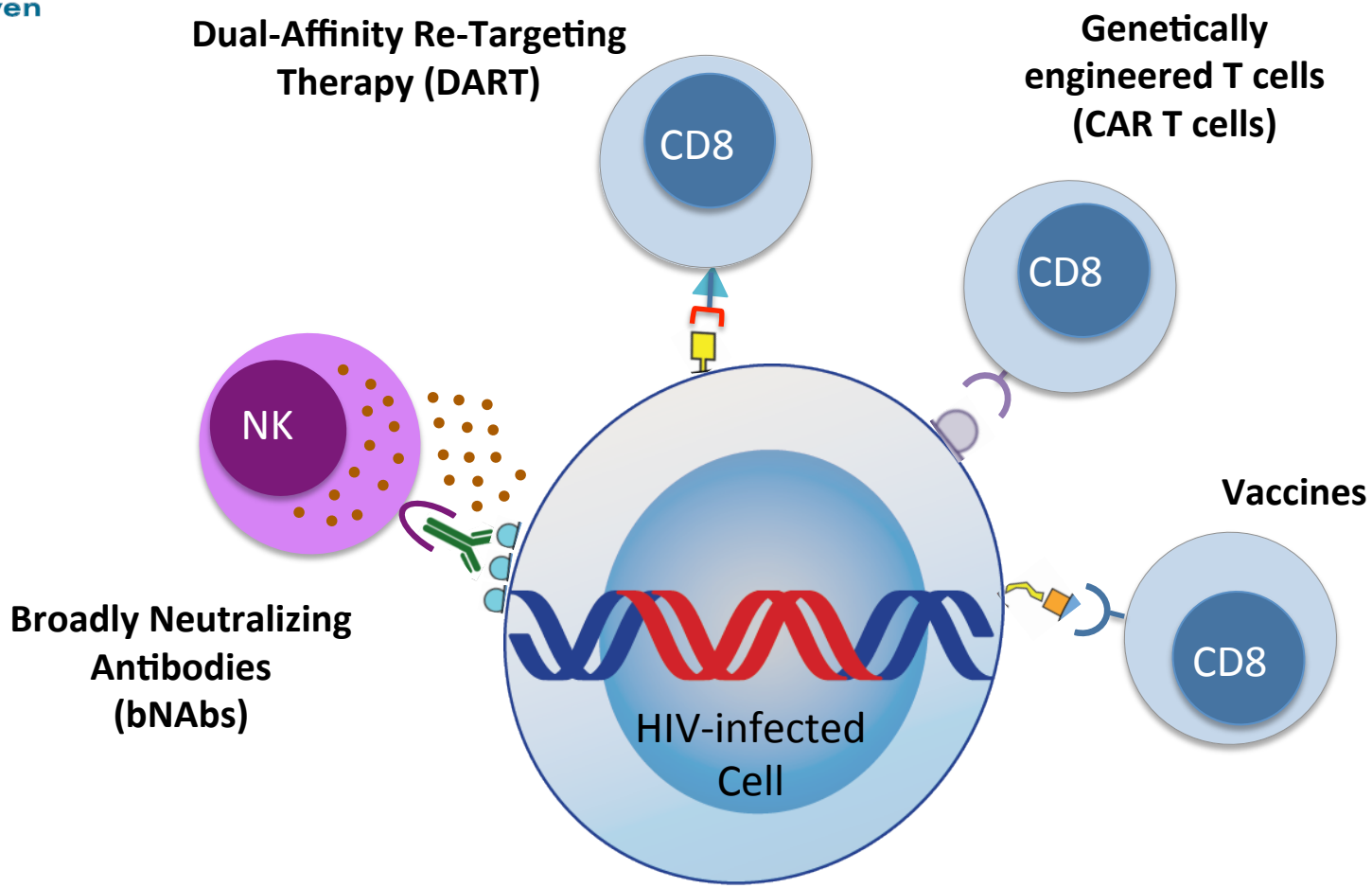
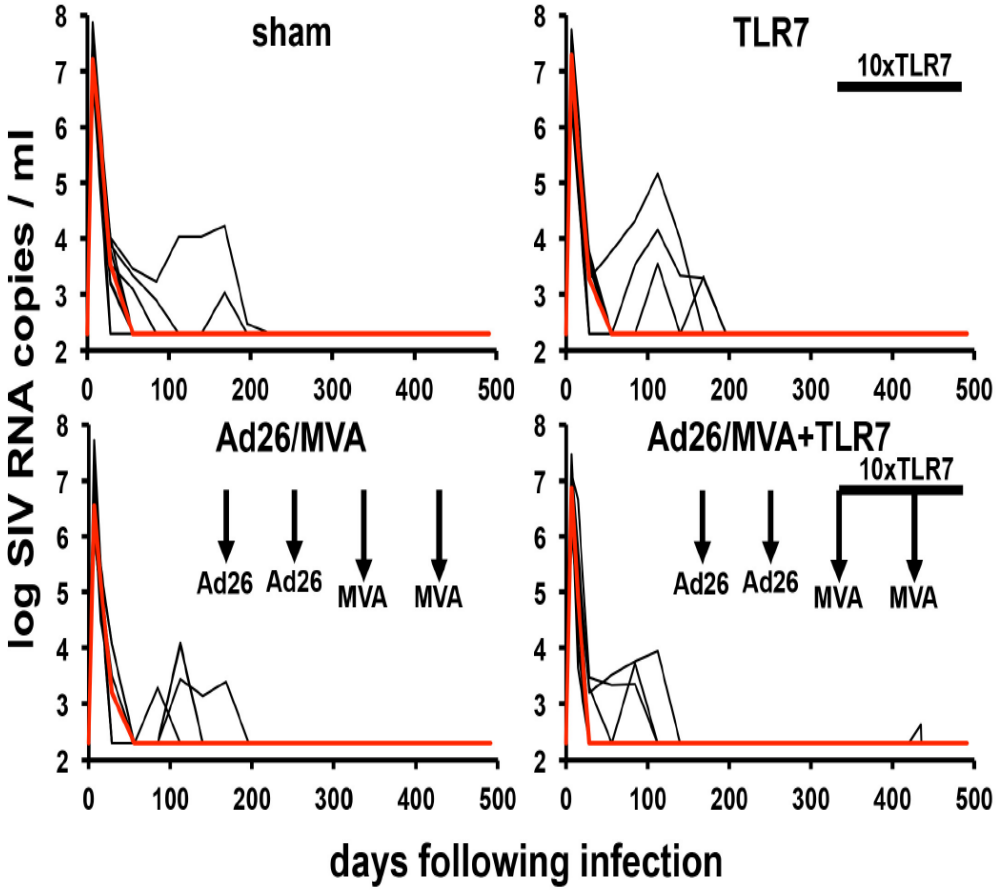
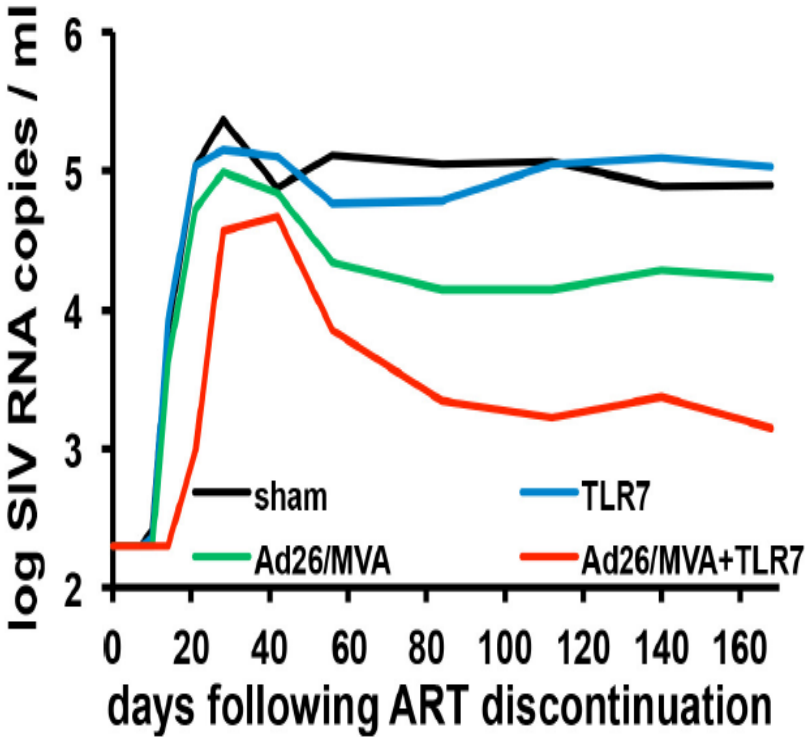
Comment optimiser l'élimination des cellules infectées, réactivées?



Jintanat Ananworanich,Joep Lange Memorial Lecture, IAS 2017, Paris

Vaccins Ad26/MVA + TLR7 (adjuvant ou activateur latence virale): control SIV à arrêt ART

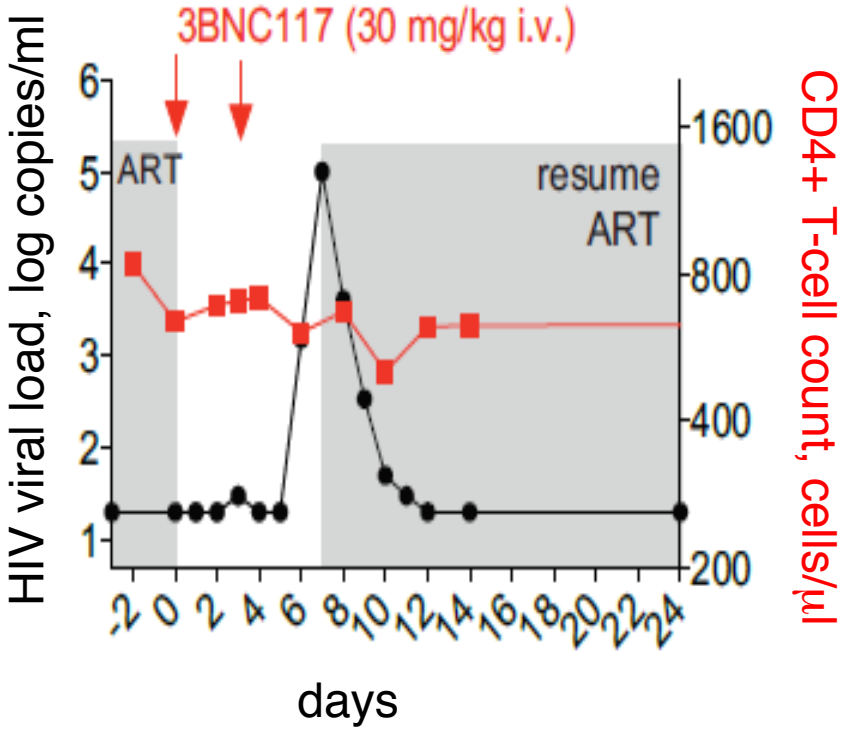




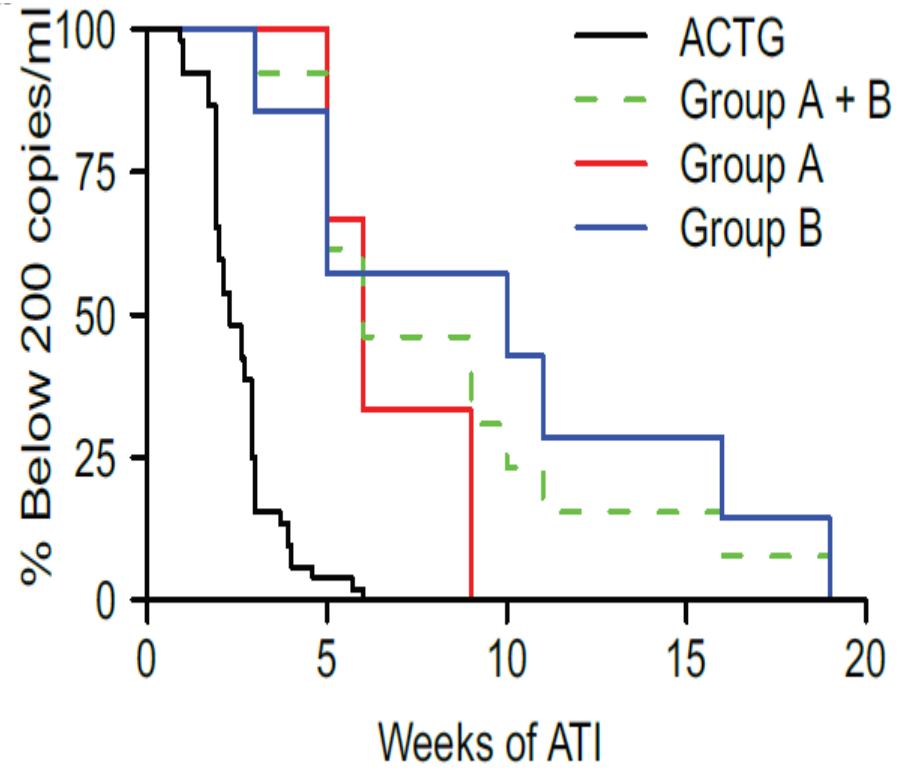
TLR7 agonist (Gilead) and Adenovirus (Ad26) + Modified Vaccine Ankara (MVA, Janssen)

Borducchi et al., Nature 2016

bNabs: elimination des cellules infectées et rebond viral plus tardif après arrêt cART.



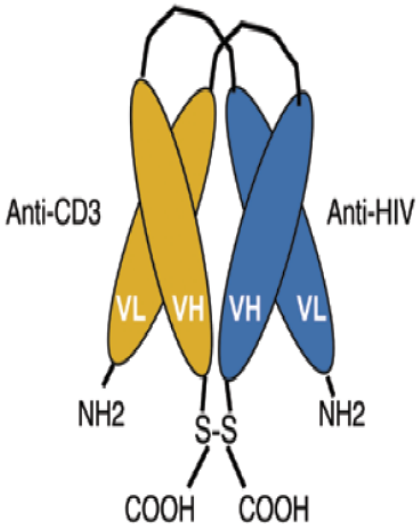
HIV-infected individuals on ART
Antiretroviral treatment interruption 2 days after first infusion

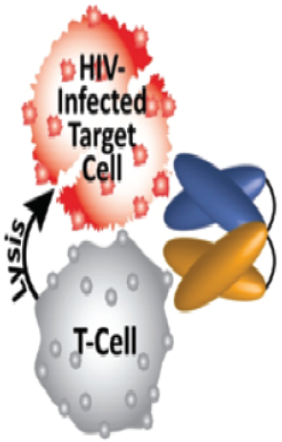


ACTG = historical controls;
Group A = 3BNC1017x2 infusions;
Group B = 3BNC1017 x 4 infusions

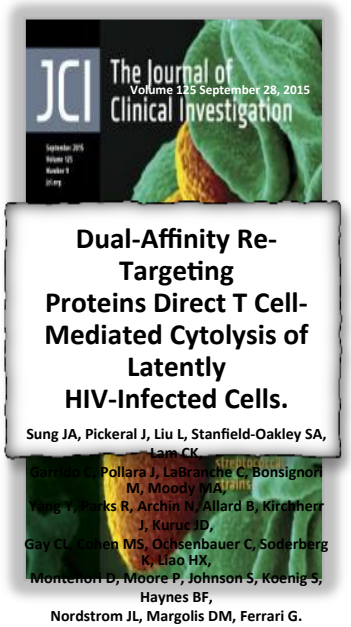
Scheid et al., *Nature* 2016; Bar et al., *N Engl J Med* 2016

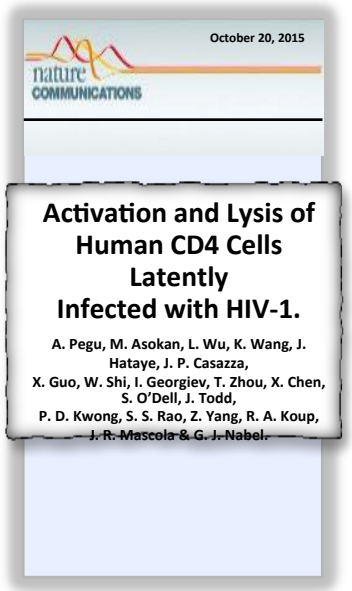
Anticorps bi-specifiques?

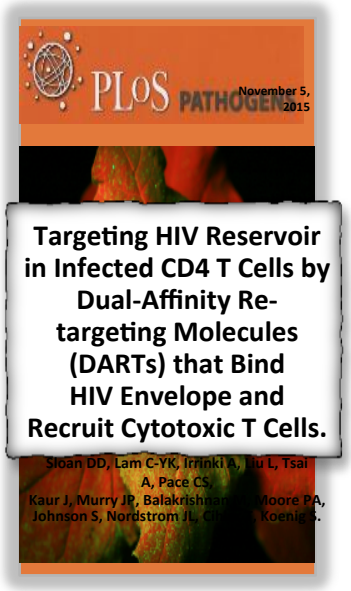




Sung et al., *J Clin Inv* 2015; Pegu et al., *Nat Comms* 2015





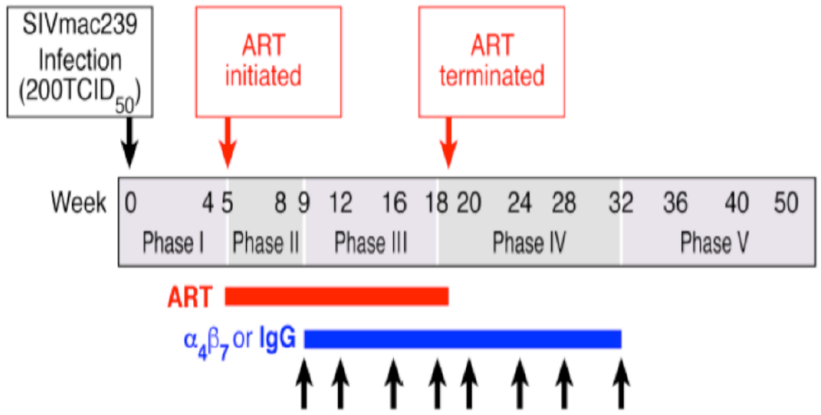


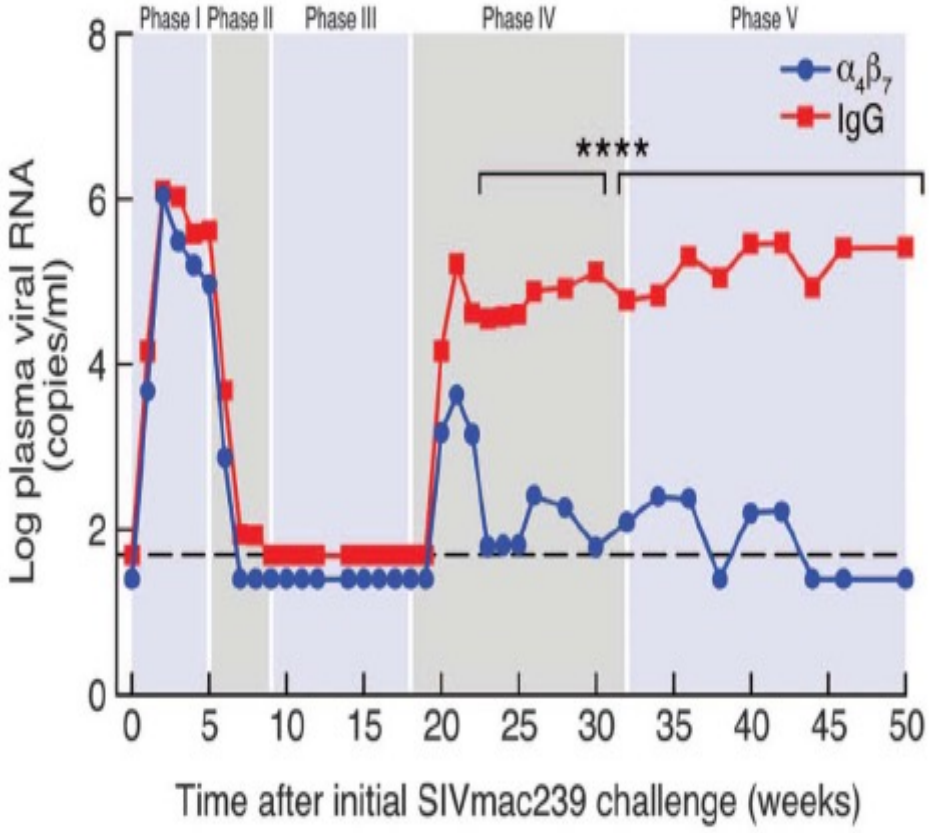
- **BITEs and DARTs mediate killing of HIV-infected cells in vitro**
- **Little in vivo data for treatment of HIV, even in animal models, but ongoing studies**
- **Products exist and have entered clinical trials for cancer**

Cibler le transport des reservoirs vers l'intestin en bloquant a4b7?

- a4b7 is an integrin and enables migration of CD4+ T-cells to GI tract
- a4b7 is a co-receptor for HIV infection
- a4b7 antibody (vedalizumab) is licensed for IBD

Byareddy et al., Science 2016





Vedaluzimab: essais cliniques en cours chez des patients HIV+ sous cART

towards an HIV cure
people focused science driven

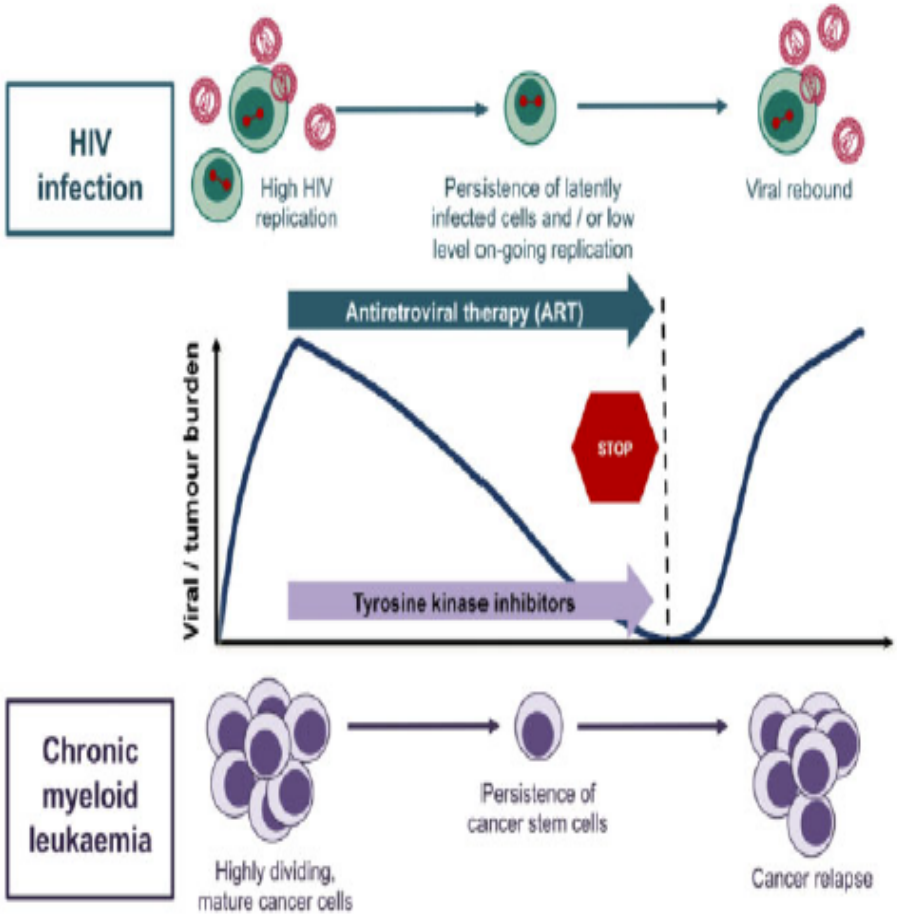
Persistance du VIH et du cancer:

Des défis similaires…

Chronic Inflammation

Immune Suppression

Cancer/ HIV persistence



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Persistance du VIH et du cancer:

Des stratégies thérapeutiques similaires…

Chronic Inflammation

Immune Suppression

Cancer/ HIV persistence

- LRA *(HDAC inhibitors; JAK/STAT inhibitors; PKC agonists)*
- TRL4/7 agonists
-
- Cytokines and/or anti-cytokines *(IL-1, IL-21, IL-15, anti-IFNα,, anti-IL-7…)…*
- **ICB blockers *(anti-PD1, PD1-L or anti CTLA-4…)***

ICB: études cliniques en cours.

Imune checkpoint blocker	Study design	Patient population	Study name (Location)	Outcome
Anti PD1 (Merck)	**Multi-dose phase 1B**	**Malignancy**: AIDS-defining or non-AIDS	CITN; US	Reservoir substudy
Anti PD1 + Anti CTLA4 (BMS)	**Phase 1 Dose escalation**	**Malignancy**: HIV-associated tumors including lung,anal and KS	AMC; US, Sydney	Reservoir substudy
All ICB	**Observational study**	**Malignancy**: melanoma or small cell ca of lung	Australia, US, Denmark, Germany	Ongoing
All ICB	**ANRS OncoVIH cohort**	All cancer patients	France	Ongoing
Nivolumab (anti PD1)	**Phase 2 trials**	NSCLC and Hodgkin	France	Ongoing

ACTG = AIDS Clinical Trials Group; CITN = Cancer Immunotherapy Network; AMC = AIDS Malignancy Consortium; ANRS Clinical trials and cohorts

Adapted from Rasmussen T et al., *Curr Opinion HIV/AIDS* 2016

Réduction importante des réservoirs VIH chez un patient atteint d'un cancer pulmonaire, traité par du nivolumab

Letter to the editor, JP.Spano, B. Autran et al. Annals of Oncology, Dec. 2017.

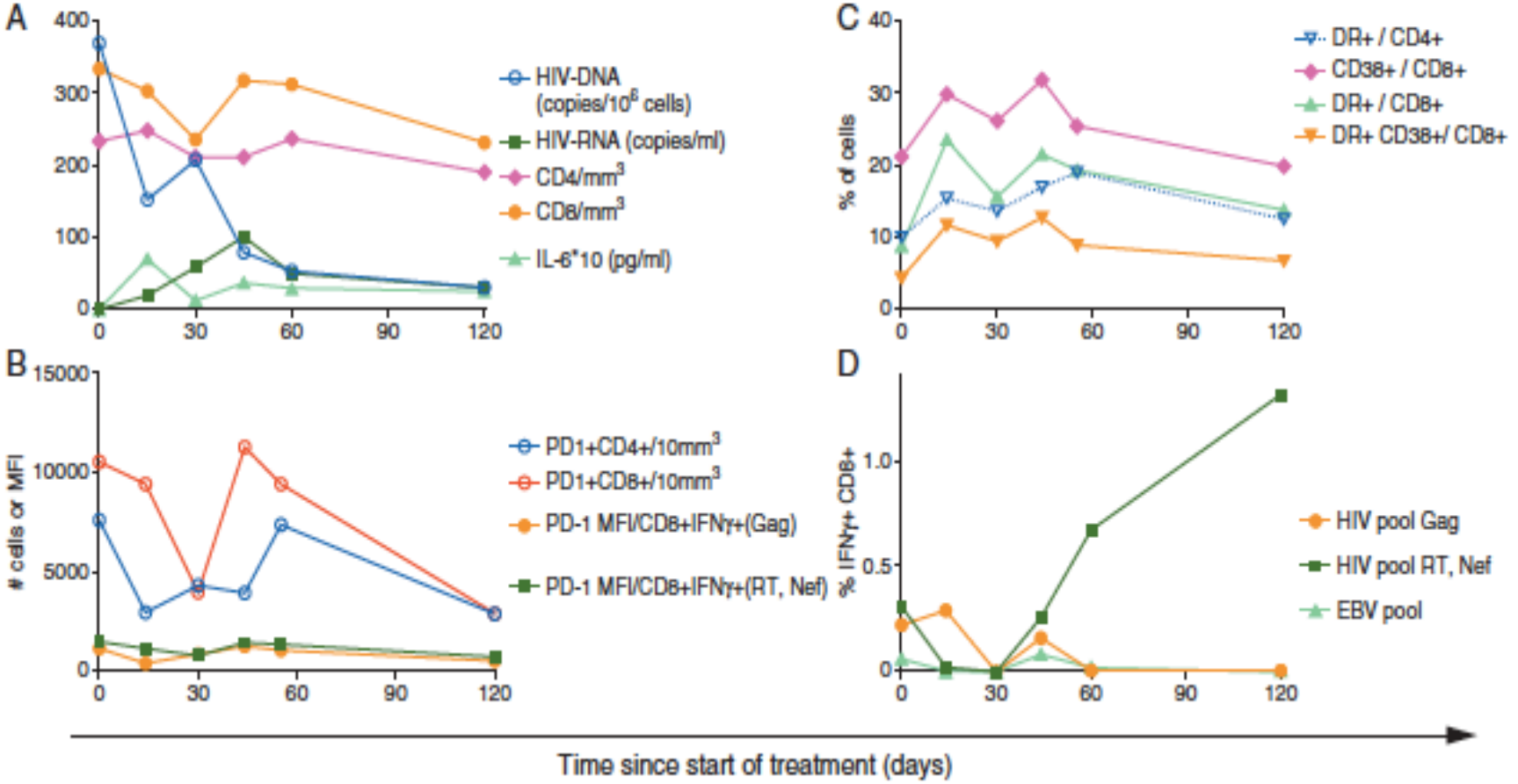
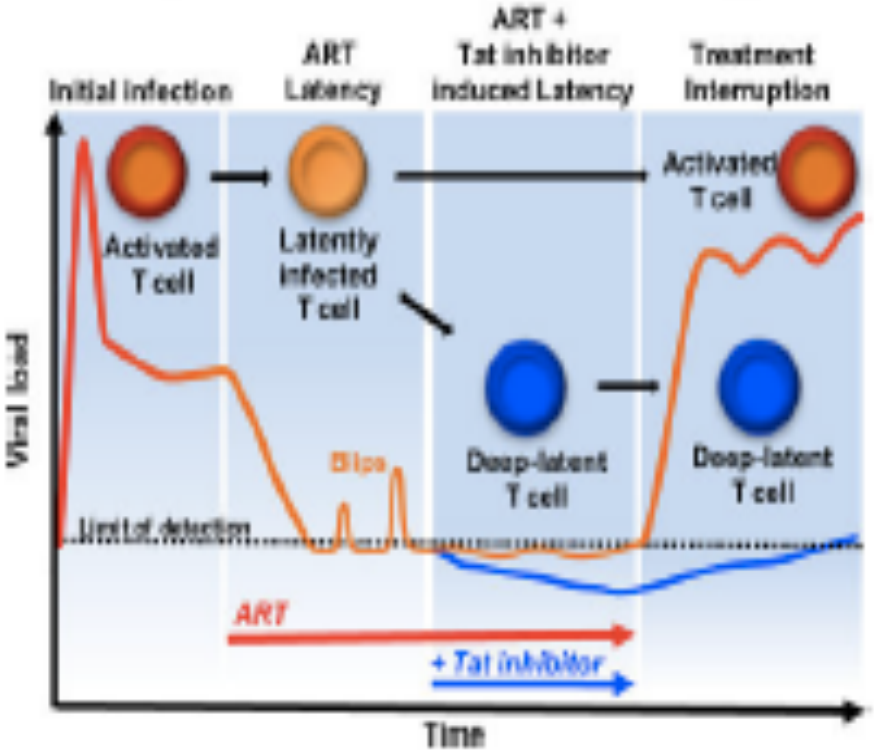
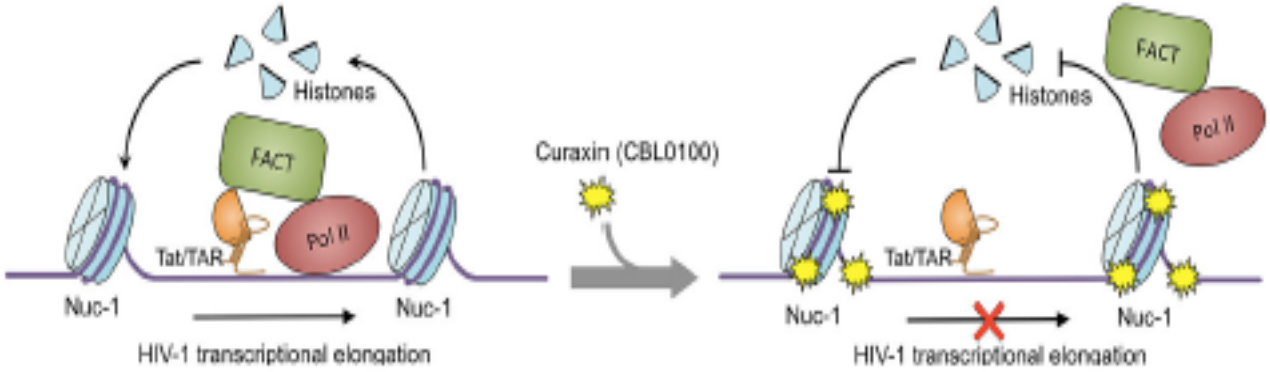


Figure 1. Immunovirological modulations under anti-PD-1 therapy in an HIV-infected patient treated for lung cancer. (A) CD4 cell count, interleukin (IL)-6 plasma levels, HIV-1 plasma viral load measured with ultrasensitive technique (USVL), and total HIV-DNA (DNA copies/million cells) through time. (B) PD-1 expression on total CD4+ and CD8+ T cells, on HIV Gag-specific CD8+ T cells, and on HIV RT/Nef-specific CD8+ T cells. Results are expressed as absolute number of total PD-1+ T cells/mm^3, or as mean fluorescence intensity (MFI) for HIV-specific T cells. (C) HLA-DR and CD38 activation markers expression on total CD4 and CD8 peripheral T cells. (D) Frequencies of HIV Gag, RT/Nef, and Epstein Barr Virus (EBV)-specific IFNγ+CD8+ T cells (stimulation with optimal CD8 peptides).

Stratégie “Block and Lock”



Tat inhibitor like dCA (didehydrocorstatin)



Epigenetic silencing by Curaxin 100 (CBLO100), an inhibitor of transcriptional elongation

Thérapie génique et cellulaire?

Re-infusion of ZFN-CCR5 modified T cells in few patients: sustained decline in HIV DNA and CD4+T cell increase over one year *(Sangamo study by Dale Ando et al.)*

The NEW ENGLAND JOURNAL of MEDICINE

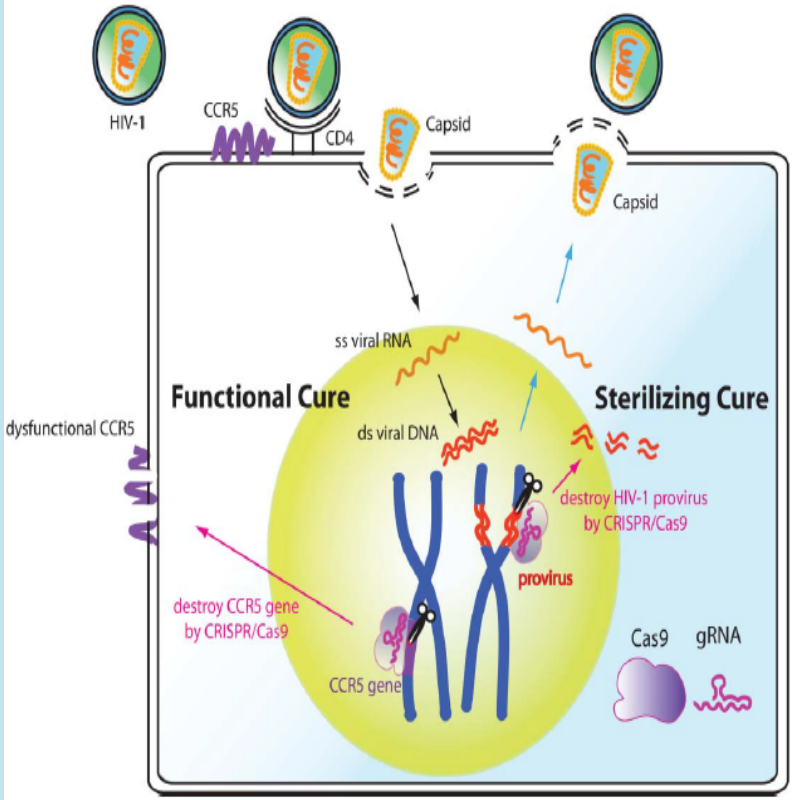
ESTABLISHED IN 1812 MARCH 6, 2014 VOL. 370 NO. 10

Gene Editing of *CCR5* in Autologous CD4 T Cells of Persons Infected with HIV

Pablo Tebas, M.D., David Stein, M.D., Winson W. Tang, M.D., Ian Frank, M.D., Shelley Q. Wang, M.D., Gary Lee, Ph.D., S. Kaye Spratt, Ph.D., Richard T. Surosky, Ph.D., Martin A. Giedlin, Ph.D., Geoff Nichol, M.D., Michael C. Holmes, Ph.D., Philip D. Gregory, Ph.D., Dale G. Ando, M.D., Michael Kalos, Ph.D., Ronald G. Collman, M.D., Gwendolyn Binder-Scholl, Ph.D., Gabriela Plesa, M.D., Ph.D., Wei-Ting Hwang, Ph.D., Bruce L. Levine, Ph.D., and Carl H. June, M.D.

What are the best strategies?

- **Gene editing using CRISPR-Cas9 (modified CCR5, siRNA, CARs, TCRs…)?**
- **Which Cell (T cells or human stem cells, autologous vs. allogeneic) to engineer?**
- **HSC engraftment concerns?**
- **Animal models and best patients for clinical studies?**
- **Safe, effective, affordable and scalable approach?**



Hua et al. PNAS 2015; Liao et al. Cell Cycle 2015

Vers où allons nous?

Stratégies à l'étude chez l'homme: vers une combinaison?

towards an HIV cure
people focused science driven

MINIMIZE RESERVOIR

Limit reservoir with early treatment

Antiretroviral therapy

Broadly neutralizing antibodies

Combination

SHOCK

Reactivation of latently infected cells by transcription activators

Inhibit histone deacetylase

Inhibit bromodomain extraterminal

Activate toll-like receptors

Activate PKC, JAK/STAT

BLOCK and LOCK

Latency Promoting Agent (LPA)

Curaxin100 (CBLO100): inhibitor of transcriptional elongation.

dCA (tat inhibitor didehydroxycorsatin)

KILL

Viral clearance by the immune system

Therapeutic vaccines

bNAbs, anti-a4b7

Bispecific abs

anti-PD1, PD1-L, anti-CTLA-4

HIV RESISTANT CELLS or DESTROY HIV

Transfusing cells with modified CCR5 gene

Gene-editing therapy using CRISPR-Cas9

Bone marrow or cord blood transplantation

BESOIN URGENT de nouveaux biomarqueurs prédictifs d'efficacité!

CD32a? Autres?

Traitement personnalisé?

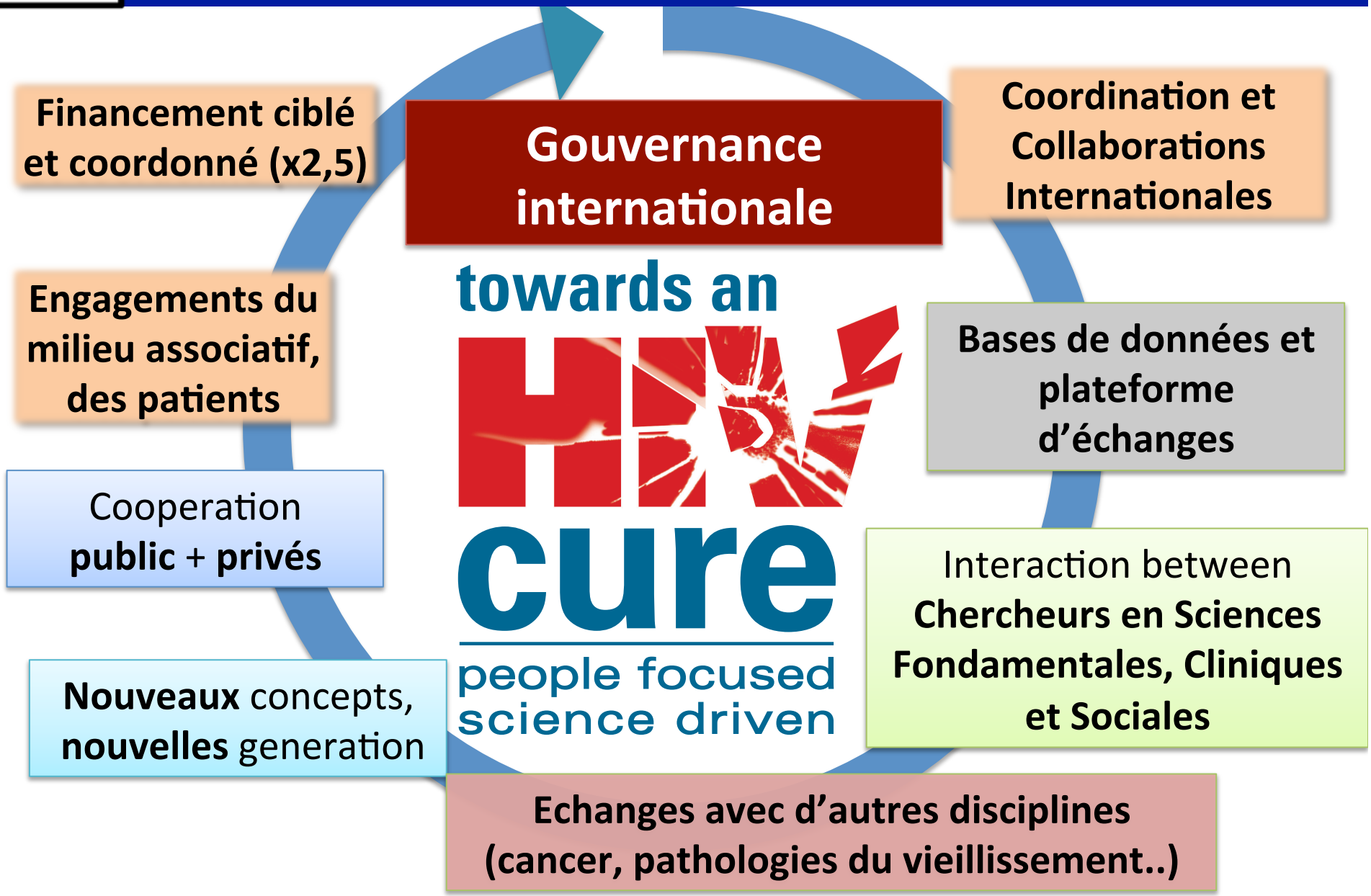
Accessible à tous! (<$1400)…

Eradication du VIH/Sida?

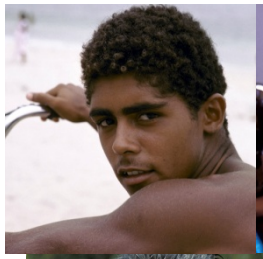
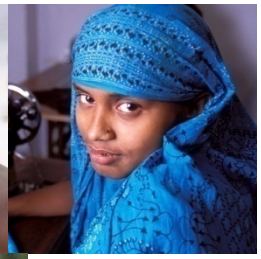
Répondre aux grands défis sociétaux, interventionnelles et scientifiques

Pas pour demain….

Stratégie Intégrée et Mobilisation internationale…



Gardons en mémoire la vision de Louis Pasteur…


L'action sans vision ne fait que passer le temps, la vision sans action n'est que rêverie, mais vision et action ensemble peuvent changer le monde…. N. Mendela





IAS

Plus fort tous ensemble!