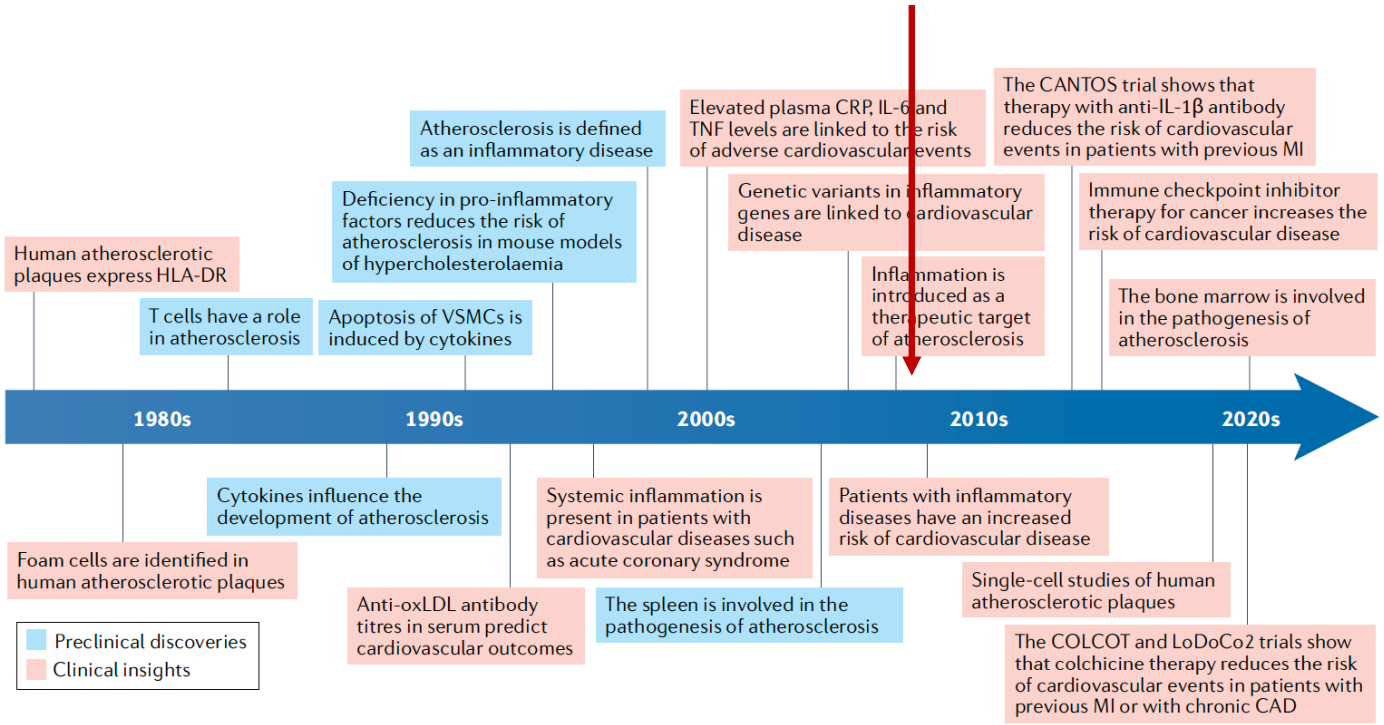
Targeting Inflammation and Cardiovascular Disease in People with HIV: 2024 and onward

CROI March 2024

Priscilla Hsue, MD FACC FAHA
Professor of Medicine
University of California, San Francisco
Maurice Eliaser, Jr., MD, Distinguished Professorship in CVD
Chief of Cardiology, UCSF at Zuckerberg San Francisco General

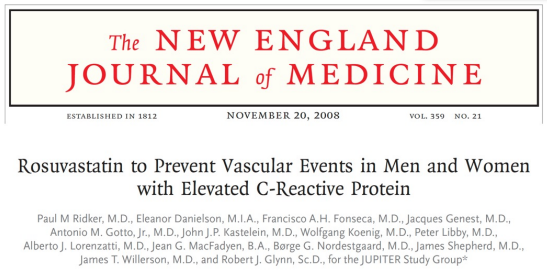
Role of inflammation in CVD: 40 years of work, role in PWH emerging

Role of inflammation in HIV



2024: No clinical endpoint studies using immunotherapy in HIV

Engelen SE Nature Reviews Cardiology 2022



The NEW ENGLAND JOURNAL of MEDICINE

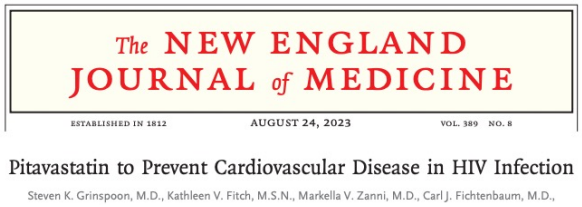
ESTABLISHED IN 1812 NOVEMBER 20, 2008 VOL. 359 NO. 21

Rosuvastatin to Prevent Vascular Events in Men and Women with Elevated C-Reactive Protein

Paul M Ridker, M.D., Eleanor Danielson, M.I.A., Francisco A.H. Fonseca, M.D., Jacques Genest, M.D., Antonio M. Gotto, Jr., M.D., John J.P. Kastelein, M.D., Wolfgang Koenig, M.D., Peter Libby, M.D., Alberto J. Lorenzatti, M.D., Jean G. MacFadyen, B.A., Børge G. Nordestgaard, M.D., James Shepherd, M.D., James T. Willerson, M.D., and Robert J. Glynn, Sc.D., for the JUPITER Study Group*

JUPITER: LDL reduced by 50%, hsCRP by 37%; 44% reduction in primary endpoint

While some believed that HIV should be considered a risk equivalent with respect to statin use like DM, we lacked evidence to change guidelines, hence the need for a clinical trial.



The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 AUGUST 24, 2023 VOL. 389 NO. 8

Pitavastatin to Prevent Cardiovascular Disease in HIV Infection

Steven K. Grinspoon, M.D., Kathleen V. Fitch, M.S.N., Markella V. Zanni, M.D., Carl J. Fichtenbaum, M.D.,

REPRIEVE: LDL reduced by 29%, MACE reduced by 35%
Statins now indicated for nearly all PWH > 40 years

HIV inflammation hypothesis was not fully tested by REPRIEVE

- N=804, treated for 2 years
- Pitavastatin had a significant reduction in LDL-C while having nonsignificant reductions in hsCRP
- Pitavastatin did not significantly lower IL-6, sCD163, sCD14

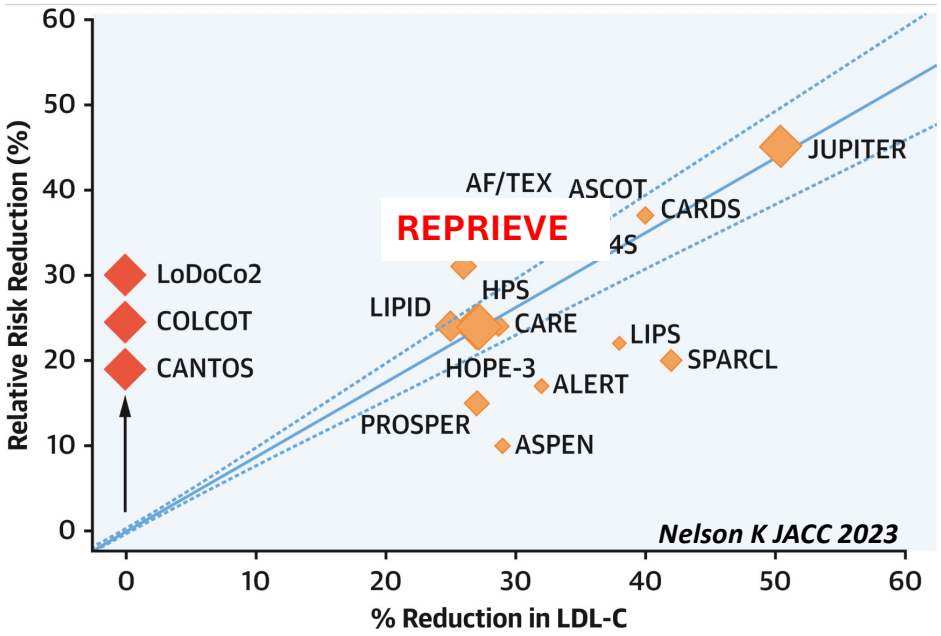
Lu M et al JAMA Cardiology Feb 2024

Abs 151 Kollossvary – Pitavastatin does not impact inflammatory pathways using a proteomics approach

Are direct anti-inflammatory strategies needed to prevent CVD in the general population?

Will people with HIV have unique needs for anti-inflammatory strategies?

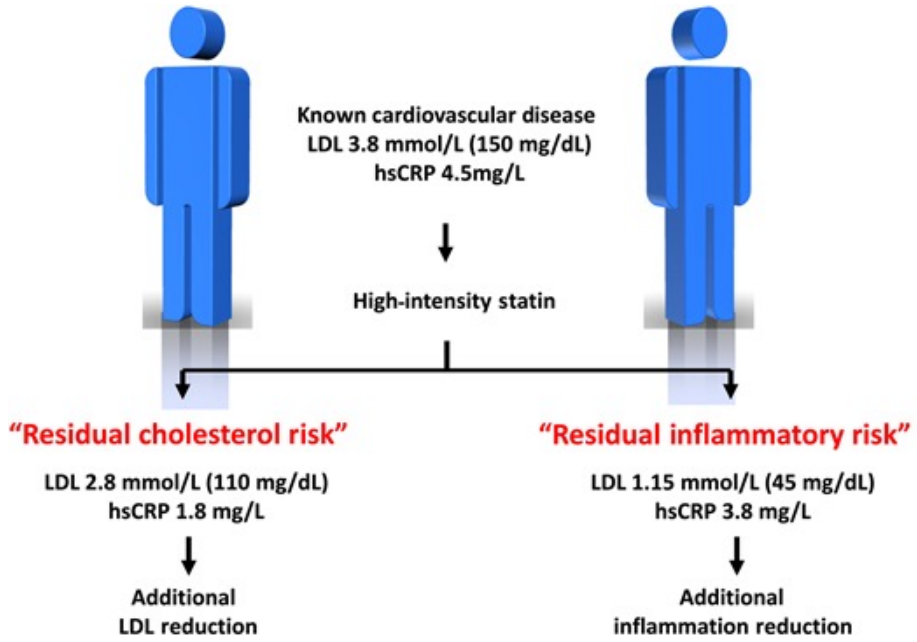
Residual inflammatory risk in the general population



Colchicine and canakinumab reduce CV risk without reducing LDL-C, while statins reduce both

"We believe that combined use of aggressive lipid-lowering and anti-inflammatory therapies might become standard of care for atherosclerotic disease in the future" (Ridker PM Lancet 2023).

Residual inflammatory risk in the general population
Best outcomes achieved when both LDL and CRP are low



Ridker PM European Heart Journal 2016

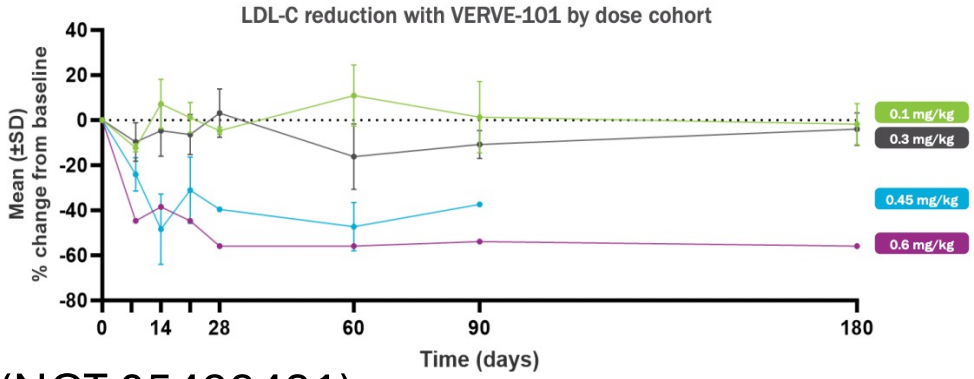
Optimizing CVD prevention in PWH: Future directions (very low, earlier lowering and new agents)

PCSK9 inhibitors: Very low LDL-C on top of statin, lowering earlier

- Lowers LDL safely in PWH (Boccara F JACC 2020)
- EPIC-HIV trial at UCSF (NCT03207945)

CRISPR-gene editing

- Single infusion (non HIV)

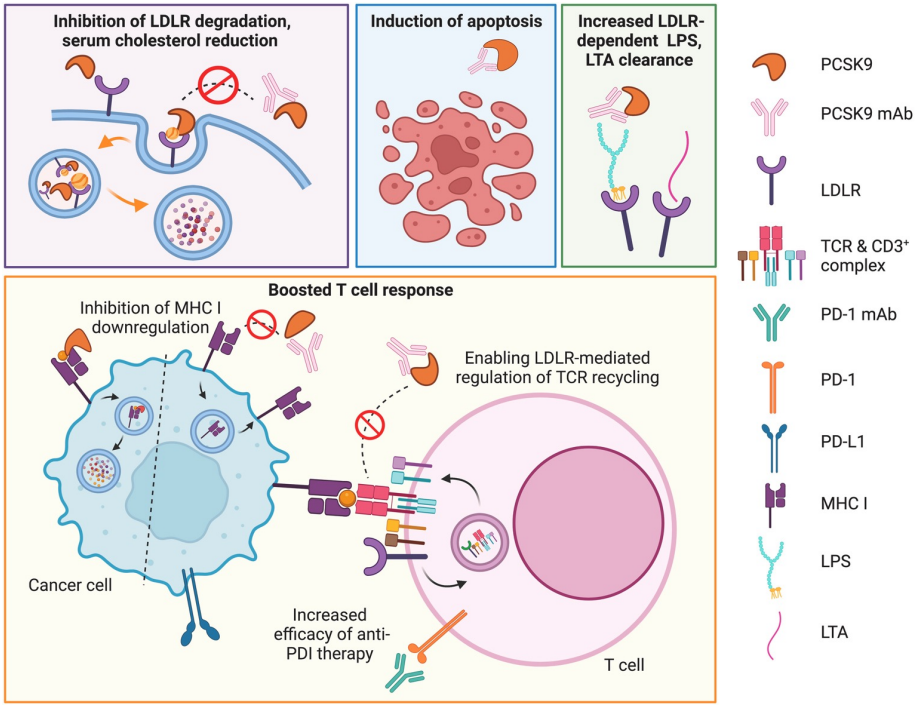


Bempedoic acid

- CLEAR HIV trial at UCSF and UCLA (NCT 05488431)

Lp(a) – clinical event trials ongoing

PCSK9 – not just lipids, implicated in cancer



- Cancer cells rely on cholesterol for growth
- PCK9 inhibition potentiates immune checkpoint inhibitor therapy for cancer

Palak O Euro Journal of Pharmacology 2023; Liu X Nature 2020

Inflammation agenda in PWH extends beyond CVD:

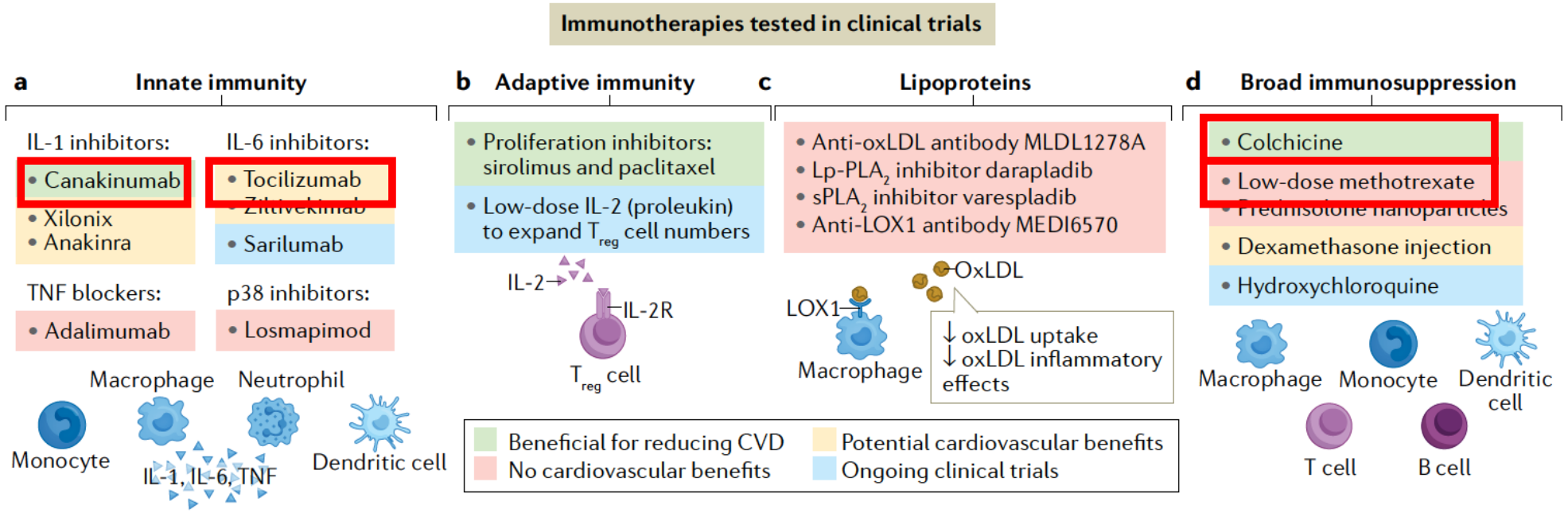
- Mortality (Kuller, PLoS Med, 2008; Tien, JAIDS, 2010; Tenorio, JID, 2014; Hunt, JID, 2014)
- Cancer (Breen, Cancer Epi Bio Prev, 2010; Borges, AIDS, 2013)
- Venous Thromboembolism (Musselwhite, AIDS, 2011)
- COPD (Attia, Chest, 2014; Kirkegaard-Klitbo, AIDS, 2017)
- Renal Disease (Gupta, HIV Med, 2015; Kirkegaard-Klitbo, AIDS, 2017)
- Bacterial Pneumonia (Bjerk, PLoS One, 2014)
- Cognitive Dysfunction (Burdo, AIDS, 2013; Sattler, JAIDS 2015)
- Depression (Martinez, JAIDS, 2014)
- Frailty (Erlandson, JID, 2013)
- Type 2 DM / insulin resistance (Brown, Diabetes Care, 2010; Reid, AIDS, 2017)
- Cure

Will we ever be able to reduce inflammation safely?

Will reduction in inflammation prevent cardiovascular disease in HIV? Will it prevent other co-morbidities?

Will we ever be able to conduct another clinical endpoint study in the post-REPRIEVE era?

Multiple anti-inflammatory drugs/strategies have advanced to clinical testing in the cardiology space



Engelen SE Nature Reviews Cardiology 2022

Low dose methotrexate did not lower inflammation or prevent CVD events in the general population

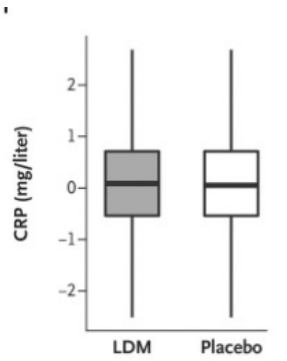
Nov. 2018

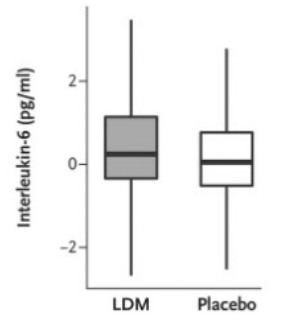
The NEW ENGLAND JOURNAL of MEDICINE

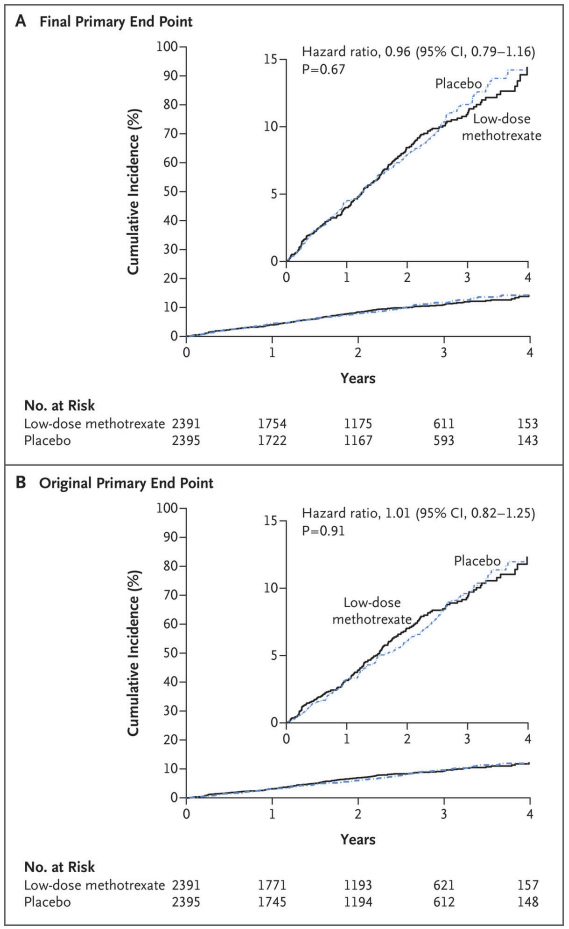
ORIGINAL ARTICLE

Low-Dose Methotrexate for the Prevention of Atherosclerotic Events

Paul M Ridker, M.D., Brendan M. Everett, M.D., Aruna Pradhan, M.D., Jean G. MacFadyen, B.A., Daniel H. Solomon, M.D., Elaine Zaharris, B.A., Virak Mam, B.S., Ahmed Hasan, M.D., Yves Rosenberg, M.D., Erin Iturriaga, M.S.N., Milan Gupta, M.D., Michelle Tsigoulis, Subodh Verma, M.D., Michael Clearfield, D.O., Peter Libby, M.D., Samuel Z. Goldhaber, M.D., Roger Seagle, M.D., Cyril Ofori, M.D., Mohammad Saklayen, M.D., Samuel Butman, M.D., Narendra Singh, M.D., Michel Le May, M.D., Olivier Bertrand, M.D., James Johnston, M.D., Nina P. Paynter, Ph.D., and Robert J. Glynn, Sc.D., for the CIRT Investigators*







Low dose methotrexate did not lower inflammation in PWH, reduced CD8 T-cell activation and T cell proliferation

Clinical Infectious Diseases

MAJOR ARTICLE

Safety and Impact of Low-dose Methotrexate on Endothelial Function and Inflammation in Individuals With Treated Human Immunodeficiency Virus: AIDS Clinical Trials Group Study A5314

Priscilla Y. Hsue,[1] Heather J. Ribaudo,[2] Steven G. Deeks,[1] Tanvir Bell,[3] Paul M. Ridker,[4] Carl Fichtenbaum,[5] Eric S. Daar,[6] Diane Havlir,[1] Eunice Yeh,[2] Ahmed Tawakol,[7] Michael Lederman,[8] Judith S. Currier,[6] and James H. Stein[9]

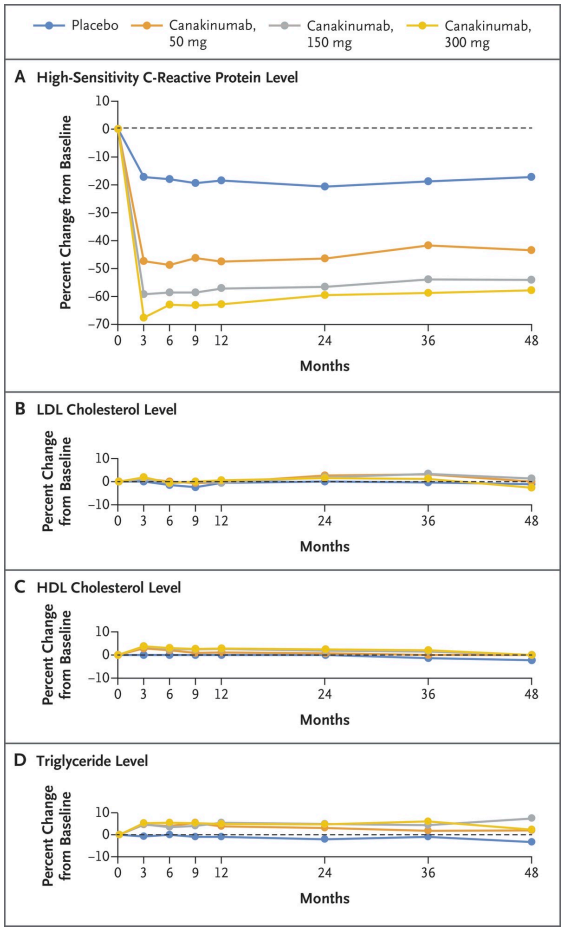
[1]Department of Medicine, University of California, San Francisco School of Medicine; [2]Center for Biostatistics in AIDS Research, Harvard T. H. Chan School of Public Health, Boston, Massachusetts; [3]McGovern Medical School, University of Texas Health Science Center at Houston; [4]Cardiology Division, Brigham and Women's Hospital and Harvard Medical School, Boston, Massachusetts; [5]University of Cincinnati College of Medicine, Ohio; [6]David Geffen School of Medicine, University of California, Los Angeles; [7]Cardiology Division, Massachusetts General Hospital and Harvard Medical School, Boston; [8]Case Western Reserve University School of Medicine, Cleveland, Ohio; and [9]University of Wisconsin School of Medicine and Public Health, Madison

Methotrexate Inhibits T Cell Proliferation but Not Inflammatory Cytokine Expression to Modulate Immunity in People Living With HIV

Michael L. Freeman[1], Brian M. Clagett[1], Daniela Moisi[1], Eunice Yeh[2], Charles D. Morris[1], Angela Ryu[1], Benigno Rodriguez[1†], James H. Stein[3], Steven G. Deeks[4], Judith S. Currier[5], Priscilla Y. Hsue[6], Donald D. Anthony[1,7,8], Leonard H. Calabrese[9], Heather J. Ribaudo[2] and Michael M. Lederman[1*]*

Frontiers in Immunology 2022

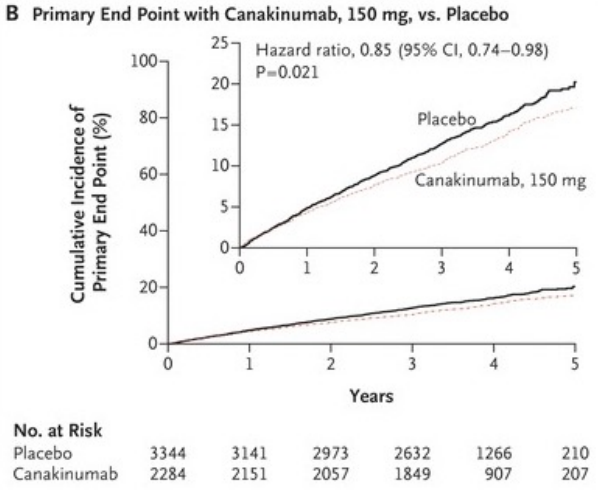
Targeting IL-1β using canakinumab lower inflammation, reduces CV events, but failed to achieve regulatory approval



The NEW ENGLAND JOURNAL of MEDICINE
ESTABLISHED IN 1812 SEPTEMBER 21, 2017 VOL. 377 NO. 12

Antiinflammatory Therapy with Canakinumab for Atherosclerotic Disease

Ridker PM et al

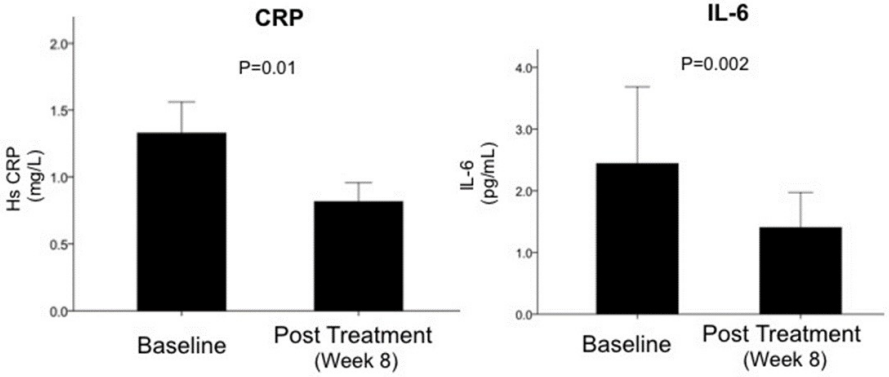


Hopes Fade for a CV Indication for Canakinumab: What's Next for the Inflammatory Hypothesis?

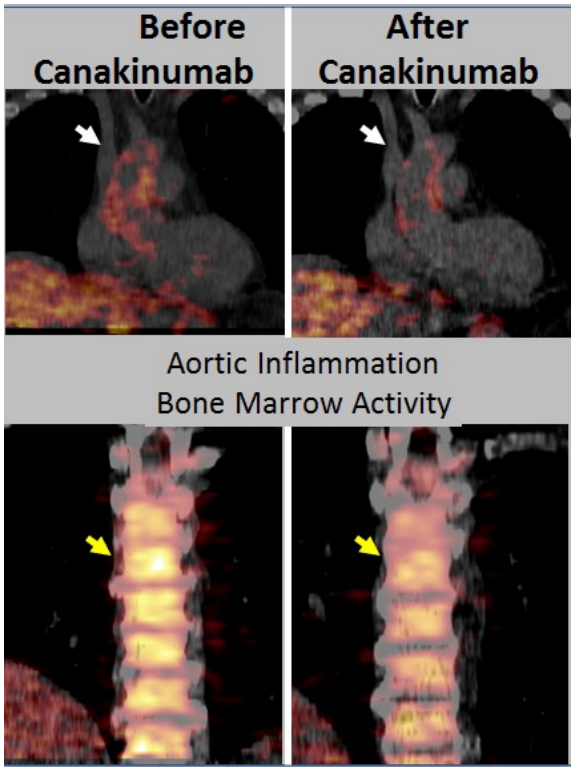
The CANTOS sponsor has given up on a CV indication for its monoclonal antibody, leaving some to ask if marketing trumped medicine.

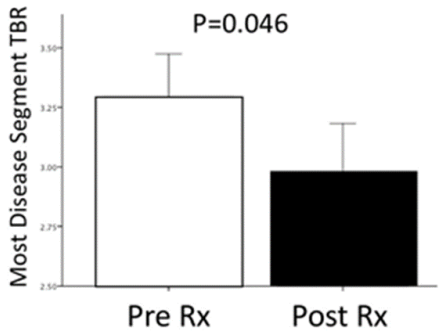
by Michael O'Riordan | FEBRUARY 01, 2019

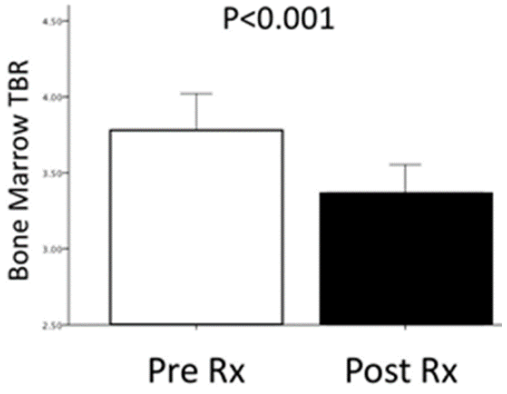
IL-1β inhibition with canakinumab reduces inflammatory markers and tissue inflammation in PWH



hsCRP reduced by 41%
IL-6 reduced by 30%







Hsue PY and Tawakol A JACC 2018

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 DECEMBER 26, 2019 VOL. 381 NO. 26

Efficacy and Safety of Low-Dose Colchicine after Myocardial Infarction

Jean-Claude Tardif, M.D., Simon Kouz, M.D., David D. Waters, M.D., Olivier F. Bertrand, M.D., Ph.D., Rafael Diaz, M.D., Aldo P. Maggioni, M.D., Fausto J. Pinto, M.D., Ph.D., Reda Ibrahim, M.D., Habib Gamra, M.D., Ghassan S. Kiwan, M.D., Colin Berry, M.D., Ph.D., José López-Sendón, M.D., Petr Ostadal, M.D., Ph.D., Wolfgang Koenig, M.D., Denis Angoulvant, M.D., Jean C. Grégoire, M.D., Marc-André Lavoie, M.D., Marie-Pierre Dubé, Ph.D., David Rhainds, Ph.D., Mylène Provencher, Ph.D., Lucie Blondeau, M.Sc., Andreas Orfanos, M.B., B.Ch., Philippe L. L'Allier, M.D., Marie-Claude Guertin, Ph.D., and François Roubille, M.D., Ph.D.

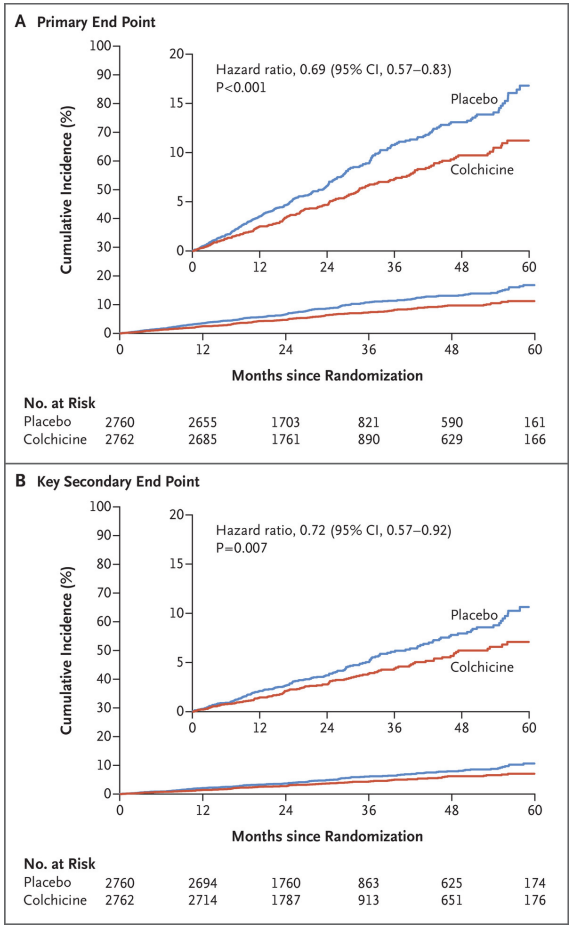
The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

2020

Colchicine in Patients with Chronic Coronary Disease

S.M. Nidorf, A.T.L. Fiolet, A. Mosterd, J.W. Eikelboom, A. Schut, T.S.J. Opstal, S.H.K. The, X.-F. Xu, M.A. Ireland, T. Lenderink, D. Latchem, P. Hoogslag, A. Jerzewski, P. Nierop, A. Whelan, R. Hendriks, H. Swart, J. Schaap, A.F.M. Kuijper, M.W.J. van Hessen, P. Saklani, I. Tan, A.G. Thompson, A. Morton, C. Judkins, W.A. Bax, M. Dirksen, M. Alings, G.J. Hankey, C.A. Budgeon, J.G.P. Tijssen, J.H. Cornel, and P.L. Thompson, for the LoDoCo2 Trial Investigators*

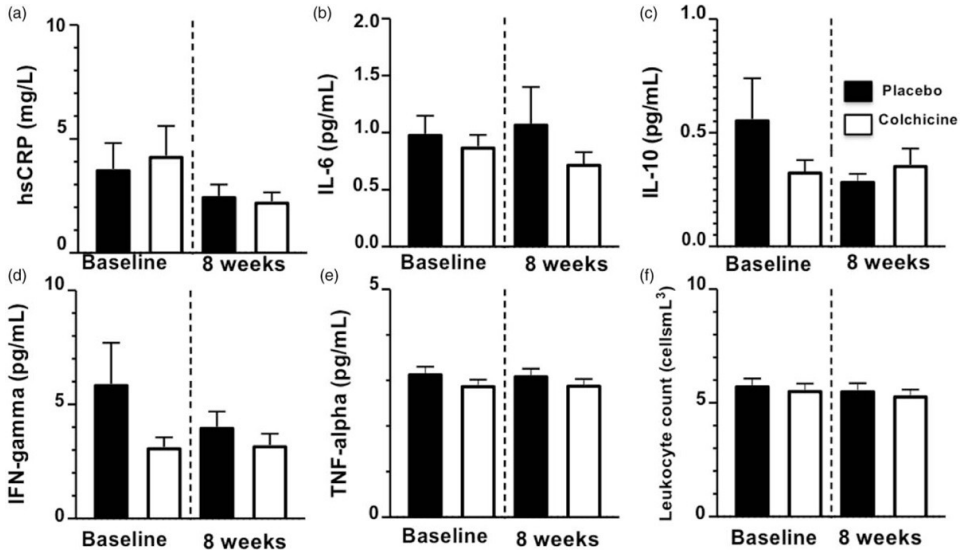


COLCOT: 30 days after MI, colchicine reduced risk of ischemic CV events 23% vs. placebo

LoDoCo2: 31% reduction in MACE when colchicine added to standard prevention

June 20, 2023: FDA approves colchicine as first anti-inflammatory drug to reduce risk for MI, CVA, revascularization and CV death in people with established ASCVD or risk factors for CVD

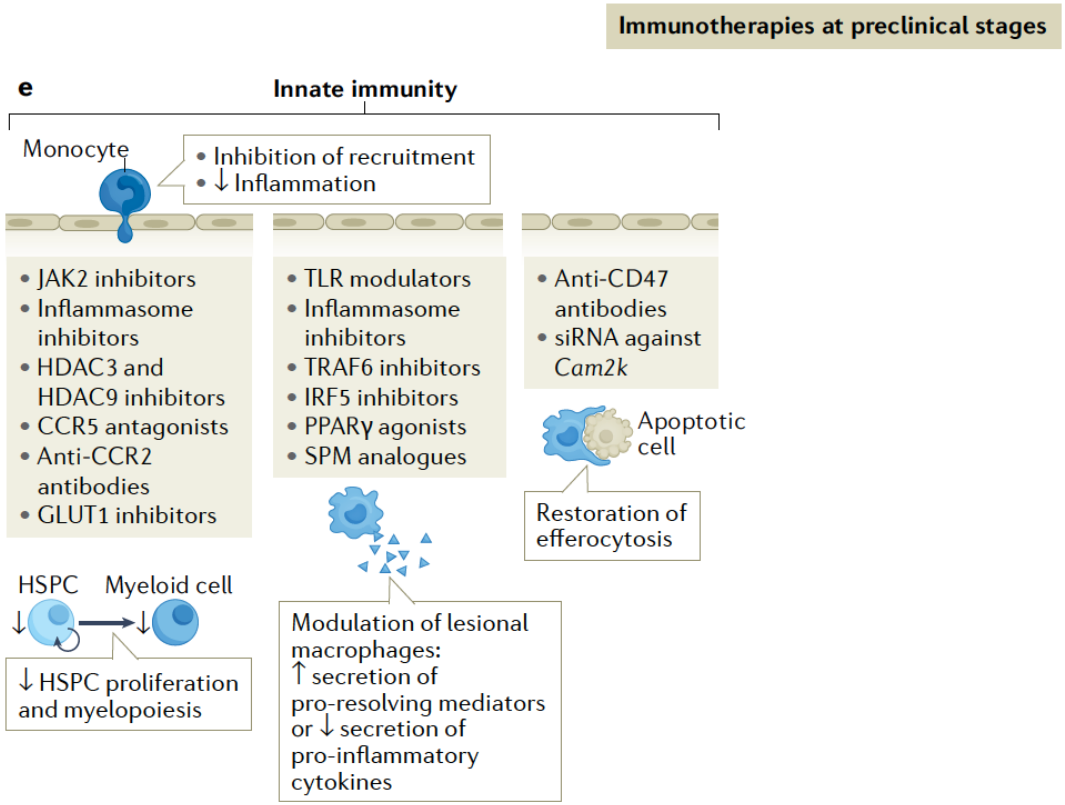
Low dose colchicine is safe in PWH, although a small RCT (n=81) failed to showed an impact on inflammation



- RCT of colchicine (0.6 mg/day) in 81 PWH
- No impact on inflammatory markers or coronary or systemic endothelial function
- Larger studies in PWH will be needed to define the potential role of this safe, scalable approach

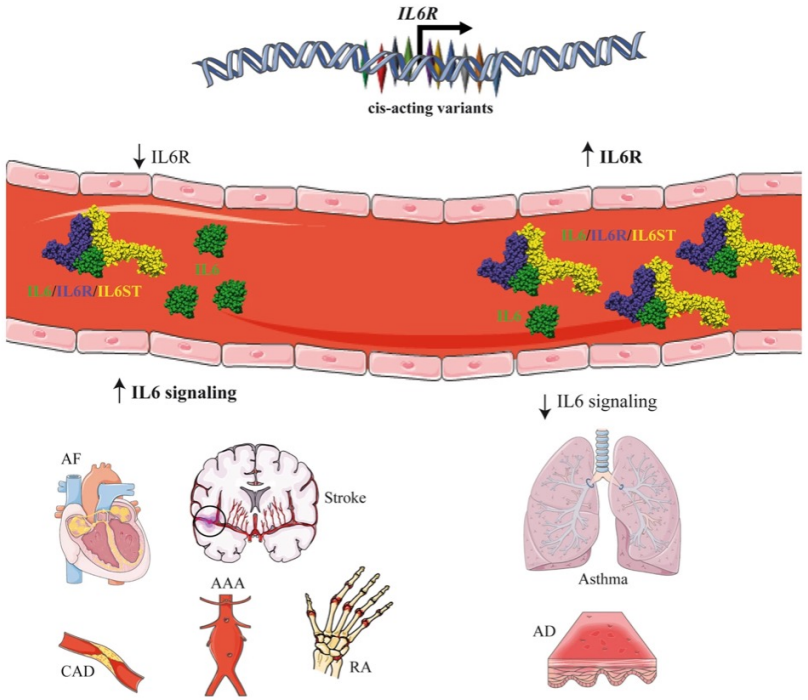
Hays AG, et al AIDS 2021

Immunotherapies in development for atherosclerosis in general population



Engelen SE Nature Reviews Cardiology 2022

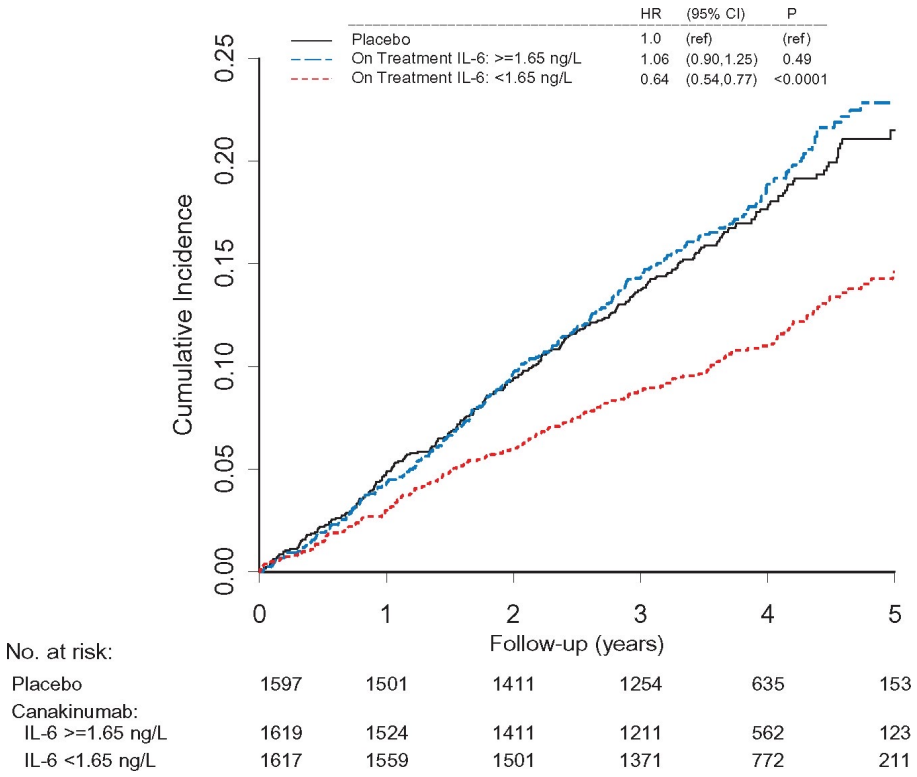
IL-6 signaling as a cause rather than correlate of disease
Mendelian randomization improves causal inference



- <u>Genetic determinants</u> of protein level predict <u>both</u>:
  - Measured protein levels *AND*
  - Clinical endpoints
- Provides <u>strong causal inference</u>
  - Host genetic determinants cannot be "caused" by confounding risk factors (e.g., smoking, diet, etc).
- In CVD field, MR established that:
  - IL-6 signaling <u>causes</u> disease
  - CRP <u>does not</u>

Rosa et al, NPJ Genomic Medicine, 2019

IL-6 is predictive of CVD and the magnitude of IL-6 reduction with immunomodulation associates with clinical benefit in CANTOS

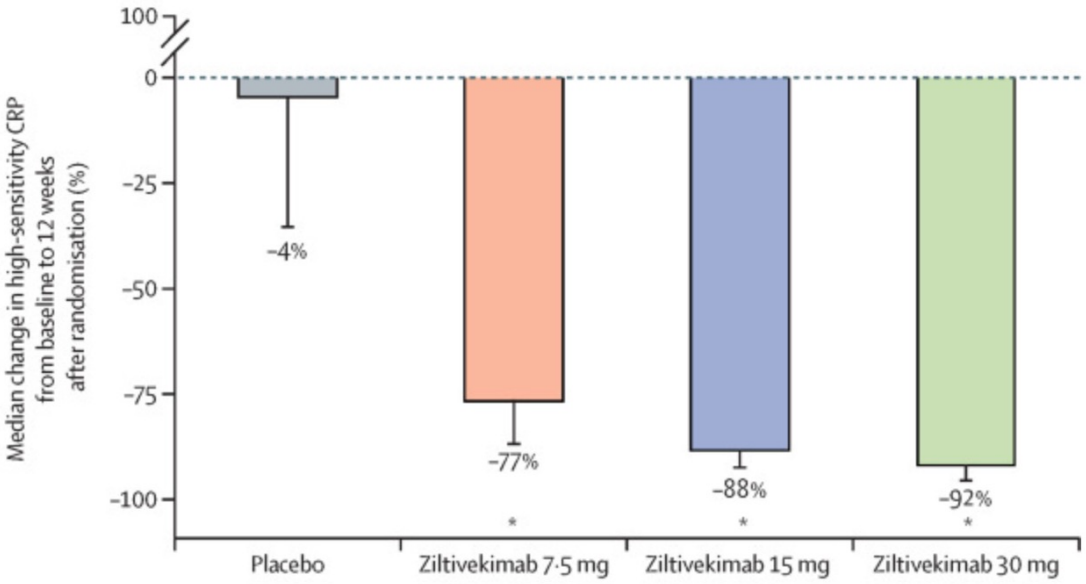


- After a single dose, pts achieving IL-6 <1.65ng/L had 32% reduction in MACE, 52% reduction in CV mortality, 48% reduction in all cause mortality, independent of lipid lowering

Ridker PM EHJ 2018

IL-6 inhibition with ziltivekimab in patients at high atherosclerotic risk (RESCUE): a double-blind, randomised, placebo-controlled, phase 2 trial

Ridker PM Lancet 2021 et al



- Ziltivekimab targets IL-6 ligand (as opposed to receptor)
- Dosed every 4 weeks x 24 weeks
- 77% to 92% reduction in hsCRP
- Limited effect on lipids
- ZEUS: Phase 3 RCT (n=6200, NCT 05021835), estimated completion 10/2025

IL-6 inhibition in PWH is safe and reduces inflammation

Clinical Infectious Diseases

MAJOR ARTICLE

Interleukin 6 Blockade With Tocilizumab Diminishes Indices of Inflammation That Are Linked to Mortality in Treated Human Immunodeficiency Virus Infection

Nicholas T. Funderburg,[1,a] Carey L. Shive,[2,3,a] Zhengyi Chen,[4] Curtis Tatsuoka,[5] Emily R. Bowman,[1] Chris T. Longenecker,[6] Grace A. McComsey,[2,7] Brian M. Clagett,[2] Dominic Dorazio,[2] Michael L. Freeman,[2] Scott F. Sieg,[2] Daniela Moisi,[2] Donald D. Anthony,[2,3,8] Jeffrey M. Jacobson,[2] Sharon L. Stein,[9] Leonard H. Calabrese,[10] Alan Landay,[11] Charles Flexner,[12,13,14] Keith W. Crawford,[15] Edmund V. Capparelli,[16] Benigno Rodriguez,[2,b] and Michael M. Lederman[2]

- N=30, cross over trial, 10 week treatment, 12 week washout
- Tocilizumab associated with significant reduction in hsCRP, sCD14, D-Dimer, increase in IL-6
- Tocilizumab associated with decreased T cell cycling in naïve CD4+ T-cells
- Tocilizumab also associated with lipid abnormalities: Increased TC, HDL-C, LDL-C, oxidized LDL, and $LpPLA_2$

Other anti-inflammatory strategies have been studied in PWH

JAK1/2 inhibition in PWH is safe, did not reduce IL-6 but did lower T cell activation, cellular lifespan and translocation markers

Clinical Infectious Diseases

MAJOR ARTICLE

Randomized Trial of Ruxolitinib in Antiretroviral-Treated Adults With Human Immunodeficiency Virus

Vincent C. Marconi,[1,2,3,4] Carlee Moser,[5] Christina Gavegnano,[1] Steven G. Deeks,[6] Michael M. Lederman,[7] Edgar T. Overton,[8] Athe Tsibris,[9] Peter W. Hunt,[6] Amy Kantor,[5] Rafick-Pierre Sekaly,[7] Randall Tressler,[10] Charles Flexner,[11] Selwyn J. Hurwitz,[1] Daniela Moisi,[7] Brian Clagett,[7] William R. Hardin,[12] Carlos del Rio,[1,2] Raymond F. Schinazi,[1] and Jeffrey J. Lennox[1]

- Ruxolitinib: JAK ½ Inhibitor
- ACTG 5336, N=60 treated/suppressed PWH randomized 2:1 x 5 weeks
- No significant impact on IL-6
- Ruxolitinib was associated with significant decreased markers of immune activation and cell survival

A5337 mTOR inhibition in PWH

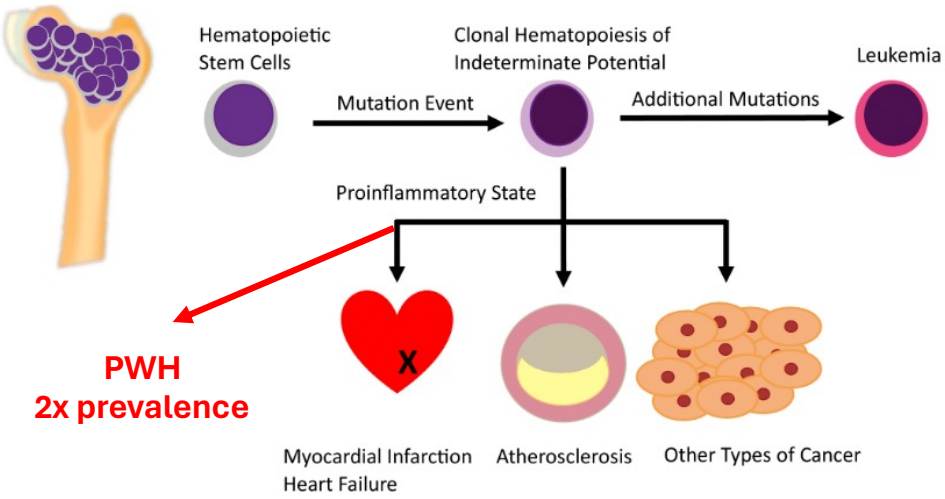
Summary

- Sirolimus was associated with a relatively high rate of treatment discontinuation
  - strict protocol-defined stopping criteria
- 20 weeks of sirolimus use associated with a **significant decrease in peripheral CD4+ T cell-associated HIV-1 DNA**
- Sirolimus use associated with significant reductions in the percentages of T cells expressing cell cycling markers (CD4 & CD8), immune exhaustion and CCR5 (CD8)
- Complex impact on markers of immune activation and inflammation

ACTG
AIDS CLINICAL TRIALS GROUP

Can we target the cause of inflammation to reduce cardiovascular disease?

Emerging role of mutations in hematopoietic stem cells as an important cause of inflammation and cardiovascular disease

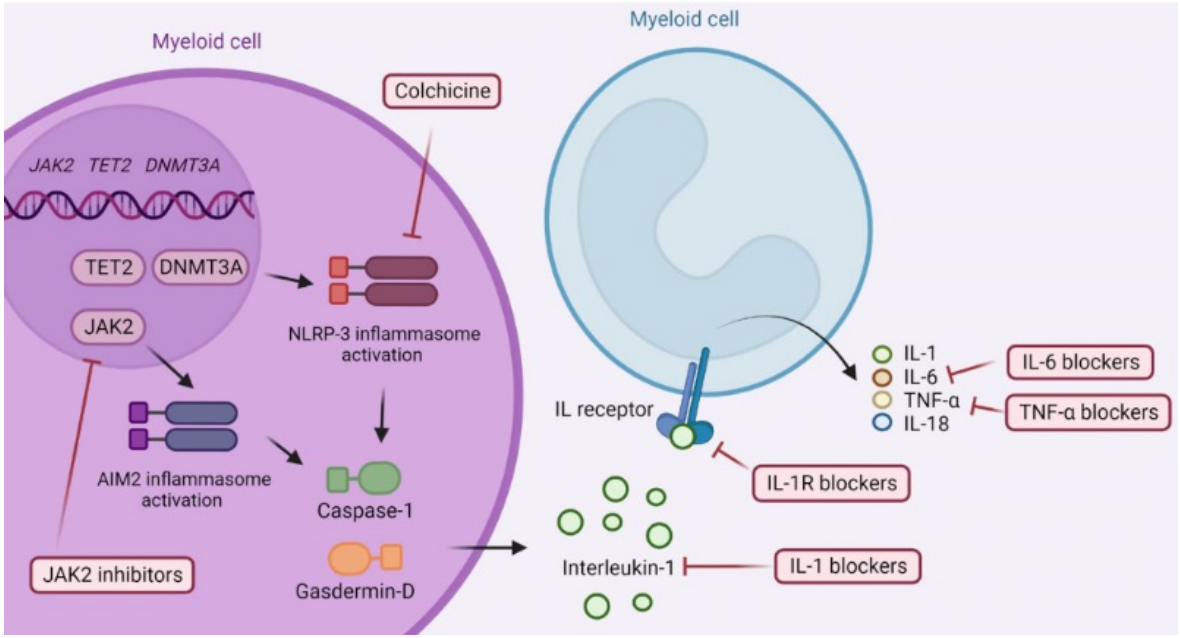


- Clonal hematopoiesis of indeterminate potential (CHIP) is a new risk factor for CVD and cancer
- Associated with proinflammatory state
- 2x prevalence CHIP in PWH
  - Associated with markers of HIV reservoir
  - Bone marrow activity and CHIP:Durstenfeld M Poster 769

40% risk for CVD **Hematologic malignancies < 1% per year**

Libby P Circulation 2018
Libby P JACC 2020; Aboumsallem JP JAHA 2020;
Bick A Sci Reports 2022, Van der Heijden JID 2022

Potential therapies for clonal hematopoiesis of indeterminate potential (CHIP)-related inflammation

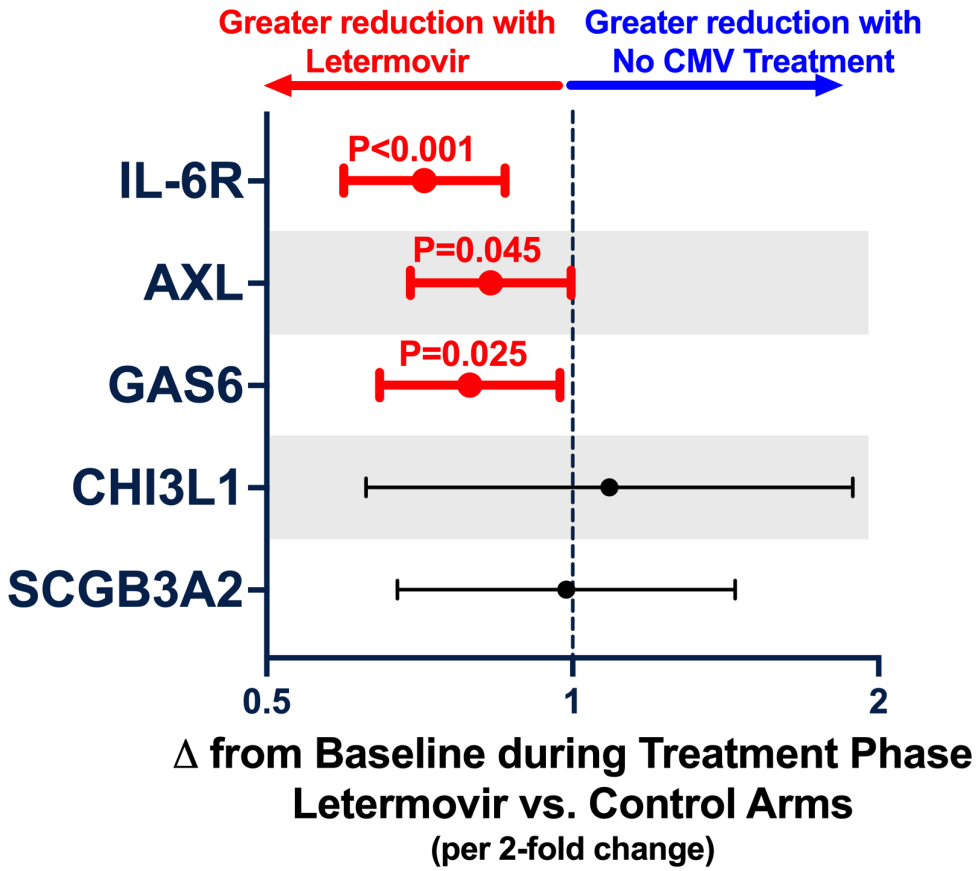


Upstream targets of inflammatory cytokines (JAK inhibitors, colchicine)

Downstream targets (IL-6, TNF-alpha, IL-1R,IL-1 blocker)

Sikking MA JAHA 2023

Emerging role of CMV as a cause of inflammation and cardiovascular disease in PWH



- ACTG 5383: RCT of letermovir vs placebo in PWH (n=40)
- Letermovir decreased 3/5 of the plasma proteins causally linked to CVD events in treated HIV (IL-6R, AXL, GAS6).
  (Reilly et al, JID, 2023)
- AXL and GAS6 are unique causal predictors of CVD in HIV

Gianella et al, Poster 354 LB, CROI 2024

Inflammation as a cause of immunosuppression and disease

Cancer and co-morbidities

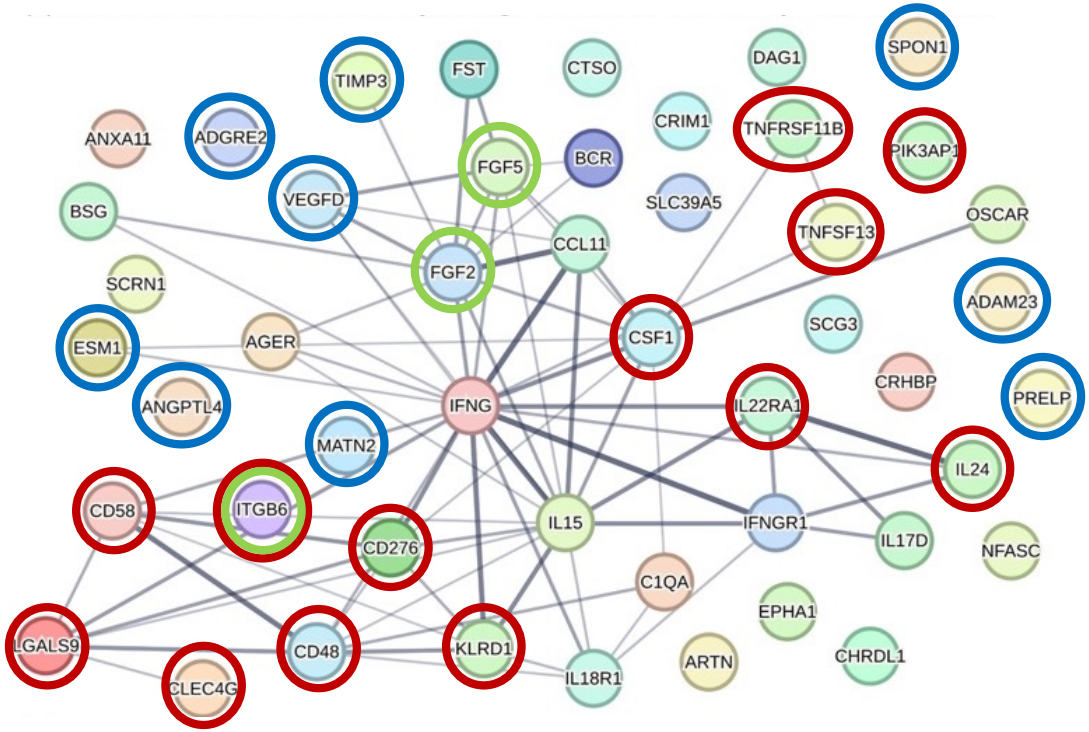
HIV cure

Immunology 101: Most of the host inflammatory response is immunoregulatory in nature

Schnittman et al, Tuesday, Oral #143, CROI 2024

- Inflammation is harmful
- Inflammation stimulates a potent and sustained immunosuppressive response
  - T regulatory cells (TGF-β, IL-10)
  - Immune checkpoint receptors
  - Myeloid-derived suppressor cells
- These immunosuppressive responses can blunt immune responses to infection (HIV) and cancer

Immunoregulatory pathways preferentially predict mortality in people with treated HIV
(CNICS vs. UK Biobank)

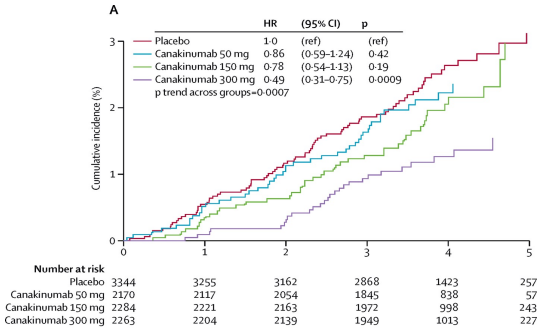


The largest clusters comprised immunoregulatory proteins linked to suppressing T, NK, and myeloid cell activation or pro-fibrotic and endothelial cell/ECM regulatory processes

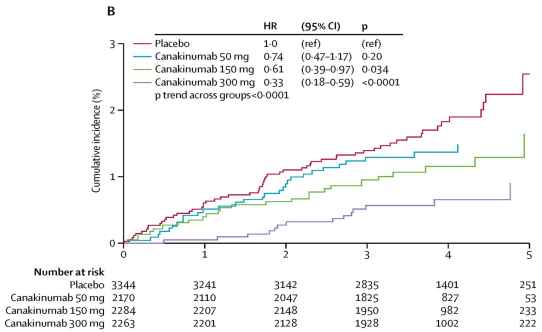
These markers predicted mortality either uniquely in PWH or to at least a 50% greater degree than in the general population

Schnittman et al, Tuesday, Oral #143, CROI 2024

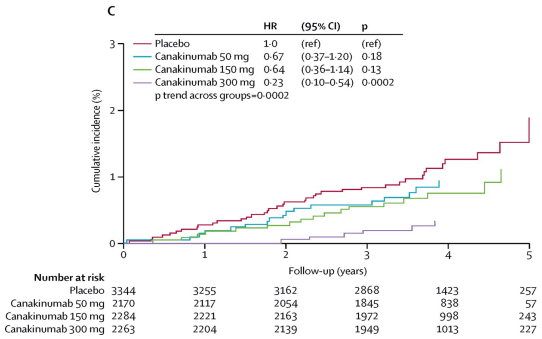
IL-1β inhibition: Canakinumab as a case study of how confusing immunology can be, particularly to a cardiologist



All fatal cancer



Lung cancer



Fatal lung cancer

Effect of interleukin-1β inhibition with canakinumab on incident lung cancer in patients with atherosclerosis: exploratory results from a randomised, double-blind, placebo-controlled trial

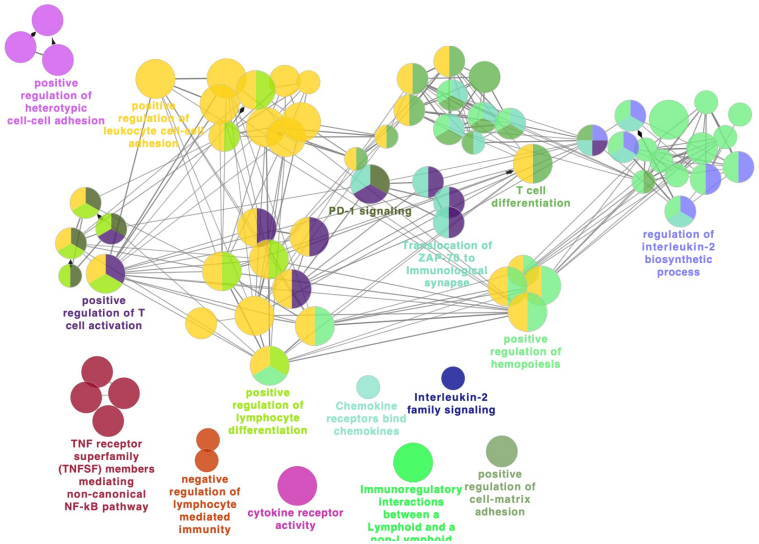
Paul M Ridker, Jean G MacFadyen, Tom Thuren, Brendan M Everett, Peter Libby, Robert J Glynn*, on behalf of the CANTOS Trial Group†*

Lancet 2017

IL-1β inhibition using canakinumab reduced incident lung cancer and lung cancer mortality

Why? Could reducing inflammation reduce counter-regulatory immunosuppression, leading to control of cancer?

Inhibition of IL-1β : Good for the heart, prevents cancer, predictive of CAD in HIV, and might even address important barriers to an HIV cure



- Lee (Abs 768) IL-1β predicts incident CAD in treated HIV
- Canakinumab (UCSF): RCT of canakinumab in PWH
- Hypothesis: Chronic inflammation leads to poor T cell function
- Tomalka (Emory): Inhibiting IL-1β enhances T cell function which in turn was associated with lower reservoir

EMORY UNIVERSITY SCHOOL OF MEDICINE | Pathology Advanced Translational Research Unit, Department of Pathology and Laboratory Medicine
EMORY VACCINE CENTER

IL-1β blockade enhances antiviral immunity and cytotoxic effector functions in ART suppressed PWHIV

00507

Ashish A Sharma[1,2], Naseem Sadek[1,2], AshokKumar Dwivedi[3], David Siegel[3], Hilmi Al-Shakshir[1], Aarthi Talla[4], CANTOS Trial Group, Rachel Rutishauser[3], Rafick Sekaly[1,2], Priscilla Hsue[3], Sulggi Lee[3], and **Jeffrey A Tomalka[1,2]**
[1]Emory University, Atlanta, GA, USA, [2]Emory Vaccine Center, Atlanta, GA, USA, [3]University of California San Francisco, San Francisco, CA, USA, [4]Allen Institute for Immunology, Seattle, WA, USA

Immunoregulation and the evolving HIV cure agenda

Interleukin-10 contributes to reservoir establishment and persistence in SIV-infected macaques treated with antiretroviral therapy

Justin Harper, … , Rafick-Pierre Sekaly, Mirko Paiardini

Article https://doi.org/10.1038/s41467-024-49555-x

TGF-β blockade drives a transitional effector phenotype in T cells reversing SIV latency and decreasing SIV reservoirs in vivo

Jinhee Kim[1], Deepanwita Bose[2,11], Mariluz Araínga[2,11], Muhammad R. Haque[3], Christine M. Fennessey[4], Rachel A. Caddell[5], Yanique Thomas[3], Douglas E. Ferrell[2], Syed Ali[2], Emanuelle Grody[3,6], Yogesh Goyal[3,6,7], Claudia Cicala[8], James Arthos[8], Brandon F. Keele[4], Monica Vaccari[5,9], Ramon Lorenzo-Redondo[1,10], Thomas J. Hope[1], Francois Villinger[2] & Elena Martinelli[1]

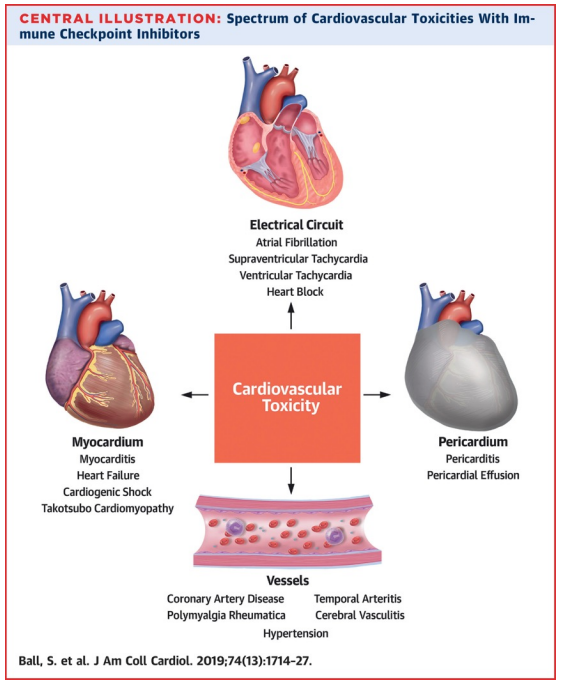
- Immunoregulatory environment contributes to SIV/HIV persistence
  - Reduced T cell turnover
  - Failure to clear
  - Failure to control
- Targeting IL-10 and TGF-β being explored in NHP model

Will emerging strategies aimed at the immunoregulatory response be good for CVD?
What is good for HIV or cancer might be bad for the heart

- **IL-10**
  - Implicated in plaque stabilization
  - Lower levels associated with unstable angina
  - Inversely related to atherosclerosis in PWH
- **TGF- β**
  - Atheroprotective
- **JAK STAT inhibitors**
  - Decrease inflammation
  - Increase thrombotic complications

Pinderski Oslund LJ ATVB 1999; Smith D Circulation 2001; Fourman L JID 2020; Grainger D ATVB 2004; Baldini C EHJ 2021

Checkpoint inhibitors



What is needed to move the field forward?

- Clinical impact of therapies to lower inflammation among PWH
  - Extends beyond CVD (other comorbidities, cancer, cure)
- Which agents to study and how to study them?
  - General anti-inflammatory approaches
  - Focus on immune pathways specific to HIV
  - Cost, safety
- Which individuals will be most likely to benefit from anti-inflammatory interventions?
  - Consider risk stratification at baseline?
  - Can we ascertain who is most likely to benefit based on specific pathway being intervened upon (ie CANTOS)?
- Need to break down silos isolating those working on CVD, cancer, HIV cure
- Everything we do should be focused on developing a safe, scalable anti-inflammatory approach that can be tested in REPRIEVE 2.0

Acknowledgements

Hsue Research Team
Clinical:
Matt Durstenfeld
Saate Shakil
Danny Li
Marta Levkova
Veronica Schaffer
Megan Mclaughlin
Yifei Ma
John Kornak
Anjali Thakkar
Diane Jeon
Sophia Xiao
Colette DeJong
Shannon Walker

Vascular/US Tech:
Yuaner Wu
Claire Mills

ZSFG HIV, ID, Global
SCOPE/LIINC: Steve Deeks, Michael Peluso, Sulggi Lee
Becky Hoh, Meghann Williams
Laurence Huang
Diane Havlir
Annie Luetkemeyer

UCSF Cardiology
Peter Ganz
David Waters
Zian Tseng
Sithu Win

Subjects at ZSFG

DEM
Tim Henrich
Peter Hunt

UCLA-Judith Currier, Rushi Parikh
MGH- Ahmed Tawakol, Michael Lu, Pradeep Natarajan
BWH – Paul Ridker, Peter Libby
UCSD- Neil Chi, Sara Gianella
University of Wisconsin – James Stein
Vanderbilt University – Matt Freiberg, Alex Bick, Wes Ely
Northwestern University–Matt Feinstein
Emory University – Rafick Sekaly, Vince Marconi, Jeff Tomalka
NIAID – Irini Sereti

Grant Support:
R01AI152932-01A1 (PI: Henrich, Hsue, VanBrocklin)
1R01HL152957-01A1 (PI: Hunt, Hsue, Tawakol)
1R01HL158315-01A1 (PI: Chi, Hsue)
1R01HL170600 (PI: Hsue, Natarajan, Freiberg, M, Chi N)
K12HL143961-01 (PI: Huang, Hsue)
K24AI112393 (PI: Hsue)
R01AG085873 (PI: Ely, Marconi, Hsue)
R01HL164337 (PI: Hsue, Tawakol)

Email for questions:
Priscilla.hsue@ucsf.edu