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Comunicaciones Orales

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Comunicaciones Orales 1
24 de noviembre - 10:45-12:45 h

CO-01. RESULTADOS A LARGO PLAZO DEL ESTUDIO PASO-DOBLE (GESIDA 11720): NO INFERIORIDAD VIROLÓGICA Y MENOR GANANCIA DE PESO CON DTG/3TC FRENTE A BIC/FTC/TAF A 96 SEMANAS

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Introducción y objetivos: El estudio PASO-DOBLE (GeSIDA 11720, NCT04884139) compara la eficacia virológica y el impacto metabólico de simplificar el tratamiento antirretroviral a dolutegravir/lamivudina (DTG/3TC) o a bictegravir/emtricitabina/tenofovir alafenamida (BIC/FTC/TAF) en adultos con VIH. Los resultados a 48 semanas se publicaron en *Lancet HIV* (2025); aquí presentamos los finales a 96 semanas.

Métodos: Ensayo clínico aleatorizado, multicéntrico, abierto y de no inferioridad. Se incluyeron adultos con VIH con RNA-VIH-1 < 50 copias/mL, en tratamiento oral estable con ≥ 1 comprimido/día, potenciadores o fármacos con toxicidad acumulativa, y sin exposición previa a DTG o BIC. Los participantes fueron asignados 1:1 a cambiar su terapia a DTG/3TC o BIC/FTC/TAF. El criterio principal fue la proporción con RNA-VIH-1 ≥ 50 copias/mL (FDA Snapshot; margen de no inferioridad 4%). El fracaso virológico según protocolo se definió como RNA-VIH-1 ≥ 50 copias/mL seguido de ≥ 200 copias/mL. Entre los criterios secundarios se incluyeron el cambio de peso y los factores asociados a una ganancia > 5% en la semana 96.

Resultados: Entre el 14 de julio de 2021 y el 24 de marzo de 2023 se aleatorizaron 553 participantes (DTG/3TC n = 277; BIC/FTC/TAF n = 276). En la semana 96, DTG/3TC fue no inferior a BIC/FTC/TAF [diferencia de riesgo: DTG/3TC (n = 1, 0,4%) menos BIC/FTC/TAF (n = 3, 1,1%) = -0,7%; IC95% -2,1-0,7]. Tres participantes (todos del grupo BIC/FTC/TAF) cumplieron criterios de fracaso virológico. Los eventos adversos relacionados con el tratamiento fueron menos frecuentes con DTG/3TC (7,6%) que con BIC/FTC/TAF (13,4%) (p = 0,025). Los eventos adversos que motivaron retirada ocurrieron en 2 participantes (0,7%) con DTG/3TC y en 4 (1,4%) con BIC/FTC/TAF (p = 0,409), principalmente neuropsiquiátricos. El incremento medio de peso fue mayor con BIC/FTC/TAF (2,4 kg; IC95% 1,6-3,1) que con DTG/3TC (0,8 kg; IC95% 0,1-1,6), con una diferencia de 1,5 kg (IC95% 0,7-2,3); más participantes experimentaron un aumento > 5% del peso con BIC/FTC/TAF (n = 96, 34,8%) que con DTG/3TC (n = 55, 20,1%) (OR ajustada 1,9; IC95% 1,3-2,9) (fig.). Además de BIC/FTC/TAF (OR 1,9; IC95% 1,3-2,9), la ganancia de peso > 5% se asoció independientemente con la exposición basal a TDF sin EFV (aOR 1,9; IC95% 1,04-3,4) o con EFV (OR 1,8; IC95% 1,1-2,9), mientras que seguir una dieta saludable resultó protector (OR 0,4; IC95% 0,2-0,9). El aumento de peso > 5% se asoció a un peor perfil lipídico.

Conclusiones: DTG/3TC demuestra a semana 96 no inferioridad virológica y un perfil metabólico más favorable que BIC/FTC/TAF, lo que respalda su uso como opción de simplificación terapéutica.

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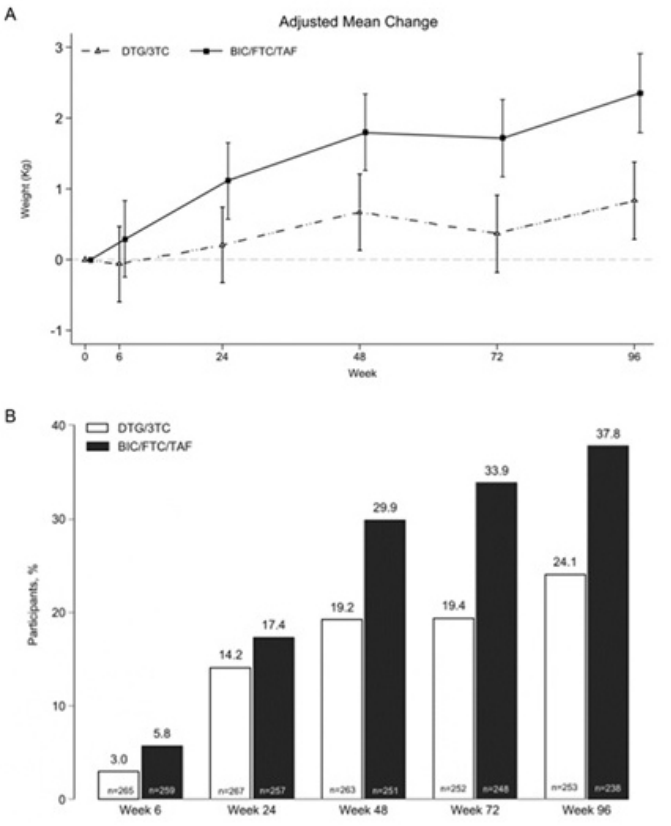
Figura. Cambio medio de peso desde el inicio (ajustado por sexo, TAF, edad, raza, dieta e interacción EFV*TDF) (A) y proporción de participantes que experimentaron una ganancia de peso >5% durante el seguimiento (B).



CO-02. COMPARABLE INFLAMMATORY AND METABOLIC OUTCOMES AFTER SWITCHING TO BICTEGRAVIR/EMTRICITABINE/TENOFOVIR ALAFENAMIDE VERSUS CONTINUING DOLUTEGRAVIR/LAMIVUDINE IN VIROLOGICALLY SUPPRESSED ADULTS WITH HIV

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Introduction and objectives: Dual antiretroviral therapy (ART) with dolutegravir/lamivudine (DTG/3TC) is widely used in virologically suppressed individuals. However, data remain limited on potential differential effects of ART regimens on mid-term systemic inflammation and metabolic health. We evaluated whether switching from DTG/3TC to bictegravir/emtricitabine/tenofovir alafenamide (BIC/FTC/TAF) modifies systemic inflammation or metabolic parameters.
Methods: INSTINCT was a phase IV, multicenter, open-label, randomized trial enrolling adults with HIV-1 on stable DTG/3TC and sustained viral suppression. Participants were randomized (1:1) to continue DTG/3TC or switch to BIC/FTC/TAF and followed for 96 weeks. Plasma biomarkers (sCD14, IL-6, sCD163, hsCRP, D-dimer, and kynurenine/tryptophan ratio) were measured at baseline, week 48, and week 96. Secondary outcomes included $CD4^+$ and CD4/CD8 ratio, virological suppression, weight, lipid profile, and renal function. Longitudinal changes were analyzed using linear mixed-effects models.
Results: A total of 141 participants were randomized. Over 96 weeks, no significant between-group differences were observed in inflammatory biomarkers (fig.). $CD4^+$ T-cell counts and CD4/CD8 ratio remained stable and comparable across arms. Weight changes were modest and similar; the proportion with ≥ 5% weight gain did not differ. No relevant differences were found in lipids, glucose, or eGFR. Virological suppression was maintained in > 95% of participants. Adverse events were mild and balanced between groups.

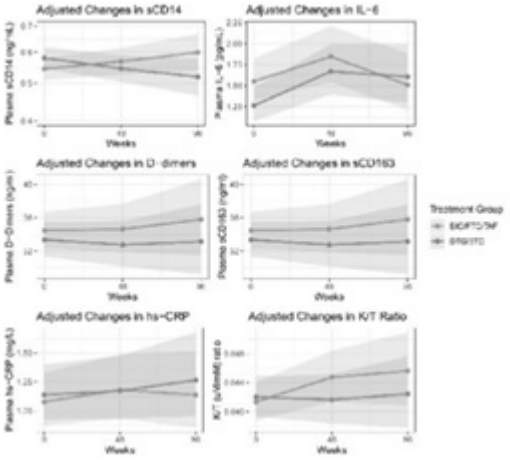

Figure 1: Adjusted trajectories of plasma inflammatory biomarkers over 96 weeks in participants receiving DTG/3TC or switched to BIC/FTC/TAF. Linear mixed-effects models with random intercepts and slopes were used to estimate longitudinal changes in sCD14, IL-6, D-dimer, sCD163, hsCRP, and the kynurenine/tryptophan ratio. Models included fixed effects for treatment group, time (weeks 0, 48, and 96), and their interaction, adjusted for baseline biomarker levels, batch, age, and sex. Marginal predictions are displayed with 95% confidence bands to reflect estimation uncertainty.

Conclusions: Switching from dolutegravir/lamivudine to bictegravir/emtricitabine/tenofovir alafenamide in virologically suppressed adults with HIV did not reduce systemic inflammation or alter metabolic parameters over 96 weeks. Both regimens showed comparable effects on inflammatory biomarkers, immunological markers, and virological efficacy.

CO-03. CAR-T CELLS TARGETING CD4 EFFICIENTLY ELIMINATE HIV-1 INFECTED T CELLS

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Introduction: Chimeric Antigen Receptor (CAR) technology shows significant potential in the pursuit of an HIV-1 cure. However, success in eliminating all latently infected cells by targeting gp120 on the cell surface depends on 100% effective latency reversal, which remains a challenge. In a new approach to bypass this limitation, we have engineered CAR-T cells targeting CD4, the primary HIV receptor, with the aim of eliminating any cells harbouring an HIV-1 viral reservoir.
Methods: We designed two second-generation CAR constructs containing different single-chain variable fragments (scFv) sequences against CD4. The first scFv (CAR-4) was produced *de novo* by hybridoma technology, while the second scFv (CAR-Hu5a8) was derived from the αCD4 antibody ibalizumab. We also produced a mock construct containing the CAR backbone without scFv. $CD8^+$ T-cells were transduced with these constructs and co-cultured with healthy or HIV-1 infected autologous $CD4^+$ T-cells at different effector:target (E:T) ratios. We evaluated cytotoxicity produced by CAR-T cells at 24/48/72h of co-culture by flow cytometry and measured cytokine production using MesoScaleDiscovery.
Results: Both αCD4 CAR-T cells produced a significant cytotoxic effect towards uninfected CD4+ T-cells compared to mock-transduced CAR-T cells. At 24h, CAR-4 T cells killed CD4+ T-cells more efficiently than CAR-Hu5a8 T cells (83.3 ± 11.7% vs. 57.5 ± 21.7% respectively, at 1:1 E:T ratio). At 48h CAR-4 T cells reached complete elimination of CD4+ T-cells, while CAR-Hu5a8 T cells reached a maximum of 95% of CD4+ T-cells elimination at a 4:1 ratio and 72h of co-culture. In HIV-1 infected co-cultures, CAR-4 T cells produced a more potent cytotoxic effect against CD4+ T-cells than CAR-Hu5a8 T cells (85.9 ± 6.3% vs. 54.4 ± 0.8% respectively at 1:1 E:T and 24h). Moreover, while CAR-4 T cells completely eliminated CD4+ T-cells from HIV-1 infected cultures at 48h and 2:1 and higher E:T ratios, Hu5a8-CAR T cells reached a maximum killing of only 73 ± 5.3% in HIV-1 infected co-cultures, which didn't increase regardless of time and ratio. Finally, we observed higher production of IFNγ, GM-CSF and Granzyme-B in supernatants from CAR-4 T cells co-cultures compared to CAR-Hu5a8 T cells, suggesting greater cytotoxic potential.
Conclusions: CAR-4 T cells efficiently eliminate both healthy and HIV-1 infected autologous $CD4^+$ T-cells *in vitro*. CAR-4 T cells, produced with our newly generated scFv, are more efficient in killing target cells than CAR-Hu5a8 T cells, produced with ibalizumab's scFv.

CO-04. ENSAYO CLÍNICO ALEATORIZADO DE NO-INFERIORIDAD COMPARANDO SEGUIMIENTO SEMESTRAL VS. ANUAL EN PERSONAS CON VIH CON BAJA COMPLEJIDAD CONFORME EL MODELO DE ATENCIÓN ESTRATIFICADO POR RIESGO

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Introducción: La implementación en la asistencia de los modelos de atención estratificados por riesgo basados en la complejidad clínica y la vulnerabilidad social son necesarios para personalizar el seguimiento en las personas con VIH y reducir costes. El presente estudio se centra en la optimización del seguimiento de los pacientes estables menos complejos.
Métodos: Ensayo clínico prospectivo, aleatorizado, de no inferioridad, entre mayo'22 y febrero'25, comparando dos estrategias de seguimiento en la atención durante 24 meses. De las 6,250 personas seguidas en la Unidad de VIH del Hospital Clínic de Barcelona, se ofreció participar en el estudio a aquellas que cumplieron el criterio de baja complejidad tras utilizar el **"**Sistema de Estratificación de Pacientes con VIH de GESIDA". Las personas finalmente incluidas se aleatorizaron a seguimiento semestral (SoC) o visitas anuales (intervención). El objetivo primario evaluó la no inferioridad en el control virológico (carga viral- (CV) < 50 cp/mL, *snapshot*-FDA) en población ITT y OT; los secundarios, la calidad de vida/satisfacción del paciente y los costes sanitarios.
Resultados: De 394 personas candidatas, 321 fueron incluidas en el ensayo: 162 en el brazo de SoC y 159 en el de Intervención (71 rechazaron participar, 48% prefirieron mantener el SoC). El 89% hombres, 78% HSH; 63% nacidos en España, 45 años de mediana de edad (RIC 38-54) y 13 años de seguimiento del VIH (RIC 7-20), sin diferencias entre los grupos. Doscientas setenta y siete (86%) personas completaron el seguimiento, sin diferencias entre brazos (p = 0,560). El cambio a terapia *long-acting* en 25 personas (57%) y la pérdida de seguimiento/transferencia en 14 (32%) fueron los motivos de discontinuación más frecuentes. A los 24 meses el control viral fue de 83 vs. 84% (ITT) y 97 vs. 96% (OT) de la rama SoC vs. intervención, cumpliéndose la no inferioridad. La satisfacción de los pacientes, según el porcentaje de promotores en el Net Promoter Score al final del estudio, fue significativamente mayor en la rama de intervención (p = 0,022). La intervención redujo significativamente el coste asociado a visitas farmacéuticas, médicas y analíticas (p < 0,001); no obstante, los costes sanitarios directos fueron comparables entre grupos (p = 0,277).
Conclusiones: La estratificación basada en el riesgo es un modelo factible y eficaz para optimizar la atención a las personas con VIH, con beneficios en eficiencia, asignación de recursos y satisfacción de los pacientes; sin comprometer el control virológico.

CO-05. RANDOMIZED PLACEBO-CONTROLLED TRIAL EVALUATING THE IMPACT OF LAURIC ACID SUPPLEMENTATION ON IMMUNE MODULATION AND HIV RESERVOIR MARKERS IN ART-TREATED INDIVIDUALS

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Introduction: Persistent immune activation and inflammation are common in people living with HIV (PLWH) despite suppressive antiretroviral therapy (ART). Lauric acid (LA), a medium-chain fatty acid, has emerged as a potential immunomodulatory agent capable of enhancing immune recovery and reducing inflammation.
Methods: In a randomized, placebo-controlled clinical trial, 45 PLWH on stable ART received daily oral 3 g LA (n = 15), 1.5 g LA (n = 15) or placebo (n = 15) for 24 weeks, followed by 24 weeks of follow-up. Primary endpoints included tolerance, quantification of the HIV reservoir (intact HIV DNA and intracellular HIV RNA), lymphocyte counts and subsets, innate immune activation, systemic inflammation indices (NLR, PLR, SII), and plasma cytokine profiling at 4, 12, 24, 36 and 48 weeks.
Results: 45 patients were included, with 42 completing the study. Of these, 34 were male, with a median age of 49 years, and a median $CD4^+$ T cells count of 815. There were 5 cases of adverse events not related to the medication, and 7 cases related (mild gastrointestinal effects, which did not require discontinuation: 3 in the 1.5 g group, 3 in the 3 g group, and 1 in the control group). The treatment was safe, with no severe adverse effects or laboratory abnormalities. At week 24, LA led to a significant expansion of $CD4^+$ (median 110 cells, p = 0.0004) and $CD8^+$ T cells (median 100 cells, p = 0.00002), a reduction in neutrophils (median -0.46 cells, p = 0.02), granulocytes (median -0.01 cells, p = 0.07), and significant decreases in systemic inflammation indices (SII p = 0.03; PLR p = 0.04; NLR p = 0.03). Cytokine profil-

ing demonstrated a shift from pro-inflammatory (CXCL9 ↓ p = 0.09; OSM ↓ p = 0.05) to regenerative (EGF ↑ p = 0.03) signaling, with pro-regenerative changes persisting after treatment cessation. At week 48, increases in TGFA (p = 0.03), CXCL10 (p = 0.01), IL-18 (p = 0.05), and CCL19 (p = 0.01) indicated a mixed response, with sustained signals of tissue repair and immune regulation alongside partial rebound of inflammatory activity. In the 3g LA group, trends toward transient reductions in intact HIV DNA (p = 0.06) and intracellular HIV RNA (p = 0.1) at week 12 suggested a potential impact on viral persistence.

Conclusions: LA supplementation was safe, without severe adverse effects, and enhanced T cell recovery, reduced innate immune activation, and promoted regenerative immune signaling. These immunologic effects may foster HIV reservoir reduction and support further investigation of LA as an adjunctive strategy to target persistent inflammation and HIV persistence.

CO-06. RESISTENCIA TRANSMITIDA EN NUEVOS DIAGNÓSTICOS DE VIH EN ESPAÑA: EVIDENCIAS DE 17 AÑOS DE MONITORIZACIÓN EN CORIS (2007-2024)

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Introducción: La evaluación de la resistencia en los nuevos diagnósticos de VIH en España se realiza en CoRIS desde 2007, con actualizaciones anuales hasta 2023. En este estudio, presentamos la actualización de 2024 así como la tendencia interanual 2007-2024 de la transmisión de mutaciones de resistencia en nuevos diagnósticos de VIH y las resistencias a los fármacos de primera línea.

Métodos: Se incluyeron nuevos diagnósticos de los centros CoRIS que accedieron a participar. Tras el control de calidad de las secuencias, se investigaron las mutaciones en RT/Pro/INI asociadas a resistencia (TDR). Además, evaluamos las resistencias clínicamente relevantes a los fármacos de primera línea en las guías de GESIDA en los diferentes periodos analizados. Para el subtipado, se utilizó la herramienta de Stanford y análisis filogenético con Mega.

Resultados: En el periodo 2007-2024, se analizaron 9.636 nuevos diagnósticos de VIH. La TDR mostró una tendencia estable en este periodo, con máximos niveles para los NNRTI. La resistencia a los ARV de primera línea presenta un notable descenso en 2014, cuando los NNRTI dejan de ser recomendados como pautas preferentes de inicio. En 2024, se analizaron 609 nuevos diagnósticos, con datos disponibles en RT, Pro e INI de 602, 597 y 456, respectivamente. La prevalencia de TDR por clases fue del 5% para NRTI (M184V, 1,3%), 11,1% para NNRTI (K103N 7,1%), 2,2% para IP y 0% para INI. Se encontraron RAMS a rilpivirina en el 10,8% de los pacientes, además solo 1 de las 8 personas con la mutación M184V hacía o había tomado PrEP. Para los fármacos de primera línea, las resistencias fueron: 0,2% tenofovir, 0,5% abacavir, 1,7% lamivudina/emtricitabina, 10,5% efavirenz, 2,5% rilpivirina, 1,5% doravirina. La mayoría de las PVIH presentaron subtipos B (75,9%); entre los subtipos no-B, los CRF fueron los más prevalentes (5,6%), con una representación del 1,5% de A6 y 0,5% de A1.

Conclusiones: Nuestros resultados reflejan la situación epidemiológica actual de las TDR y el perfil de resistencias primarias a los antirretrovirales de primera línea en España. No se ha detectado ninguna resistencia completa frente a los inhibidores de la integrasa de segunda generación, y la resistencia a 3TC/FTC y TDF/TAF se mantiene en niveles bajos. Se confirma que los NNRTI continúan siendo la familia con mayor prevalencia de resistencias transmitidas, observándose un incremento de los casos asociados a la mutación K103N.

CO-07. SAFETY, IMMUNOGENICITY, AND EFFECT ON VIRAL REBOUND COMBINING T- AND B-CELL THERAPEUTIC VACCINES IN ART-SUPPRESSED INDIVIDUALS TREATED IN CHRONIC HIV INFECTION: THE BCN03 TRIAL

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Introduction: In two independent, randomized, placebo-controlled clinical trials -AELIX-002 and AELIX-003-, combinations of DNA, ChAdOx1, and MVA vectors expressing the HTI immunogen, with or without the TLR7 agonist vesatolimod, demonstrated that HTI vaccines were safe and highly immunogenic, and that higher levels of vaccine-induced HTI-specific T-cell responses were associated with extended time off ART. However, in both studies, viral rebound occurred universally within 3-4 weeks of analytical treatment interruption (ATI), and all participants remained viremic at ATI completion, highlighting the need for improved intervention strategies. We hypothesized that efficacy of T-cell therapeutic vaccines could be improved with the support of a robust antibody response to synergize with the T cell immunity.

Methods: BCN03 (NCT05208125) was a single-center, randomized, placebo-controlled clinical trial testing as primary objective the safety of a combined regimen of ChAdOx1.HTI (C) and MVA.HTI (M), with the B-cell ConM.SOSIP.v7 (S) vaccine in 30 cis-gender men treated during chronic phase of HIV at HUGTIP, Badalona. Secondary objectives included immunogenicity, and viral rebound kinetics during a 24-week ATI. HTI- and S-specific T cells were evaluated by IFNγ-ELISpot, S-specific IgG was quantified by ELISA, and neutralization of 7 Tier1/2 pseudoviruses (one expressing S) was tested by TZM-bl assay.

ART was resumed if pVL > 100,000 copies/mL, > 10,000 for 8 weeks, and/or < 350 CD4/μL.

Results: Related adverse events were mostly mild and transient. Vaccinees significantly increased HTI-specific T cell magnitude (range) from 113 (50-1,080) to 355 SFC/10^6 PMBC (50-2,265) between baseline to ATI start ($p = 0.0082$). At ATI start, all 20 vaccinees vs. 3 placebos had detectable anti-S IgG ($p < 0.0001$), with levels correlating to neutralization (ID_{50}) of the S-matched strain ($r = 0.69$, $p = 0.0010$). The magnitude of S-specific T cells positively correlated with levels of anti-S IgG. Neither positive nor negative correlations were observed between S- and HTI-specific T cell responses. Viral rebound kinetics were similar between groups.

Conclusions: In BCN03, the CSSMS sequential vaccination translated into a lower magnitude of induced HTI T-cell responses compared to previous studies in etPWH receiving more complex T-cell vaccine regimens, and did not provide improved ATI outcomes. CSSMS was able to induce anti-ConM.SOSIP.v7 IgG antibodies in all vaccinees, with levels correlating to the magnitude of S-specific T cell responses and to neutralization of the vaccine-matched strain. We did not observe any signature of competition between T-/B-cell vaccine immunogens. The combined regimen was safe and provided important insights into T-/B-cell collaboration on which further refined vaccine regimens can be built.

CO-08. HIV INFECTION IN PREP USERS: INCIDENCE, EPIDEMIOLOGICAL, CLINICAL AND VIROLOGICAL CHARACTERISTICS. A MULTICENTER AMBISPECTIVE COHORT IN SPAIN (SCOPE STUDY)

María del Mar Vera[1], Patricia Álvarez-López[2], Haridian Tato[3], David Rial[4], Naya Faro[5], María del Mar Arcos[6], Marta Rosas Cancio-Suárez[7], Alexandre Pérez[8], Hadrián Pernas[9], Santiago Fernández[1], Claudia Broto[2], Eva Ariza-Vioque[3], Nicolás de Loredo[10], Ana González-Cordón[11], Elisa de Lazzari[12], Ángel Rivero[13], Josep M Llibre[14], Juan Ambrosioni[11] and Scope Study Group[10]

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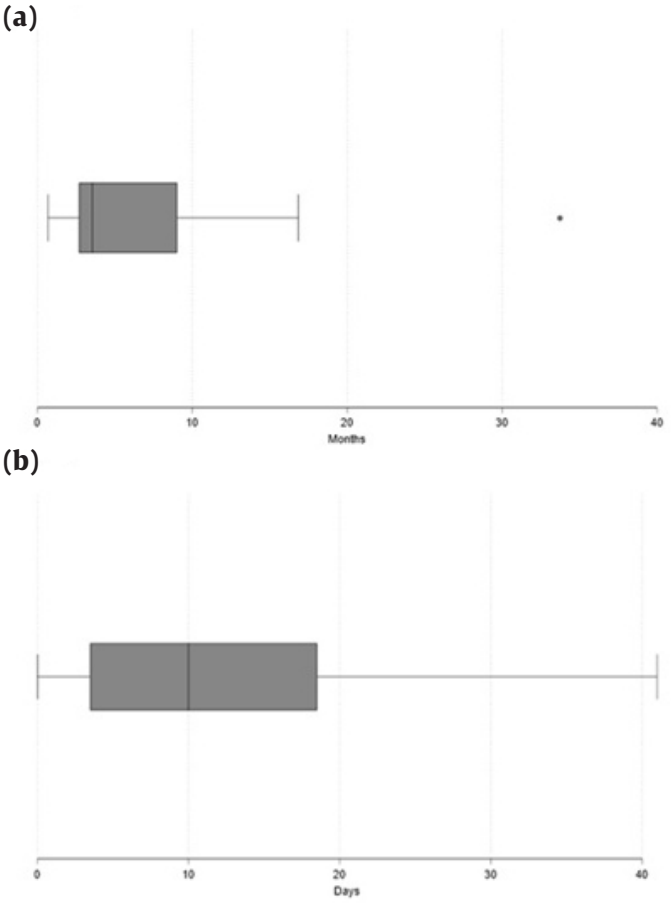
Objectives: PrEP is a highly effective HIV-1 prevention strategy implemented in Spain in 2019. According to official sources, there were an estimated 28800 users in 2024. We aim to address the incidence, clinical characteristics, resistance development, and ART outcomes of HIV infection in this setting in Spain.

Methods: Ambispective observational cohort study reporting cumulative HIV incidence among PrEP users (receiving oral PrEP within six months prior to seroconversion) across 11 Spanish centers, until 26 March 2025. Baseline epidemiological, clinical and laboratory data were analyzed for all cases; follow-up data were available for individuals reaching 48 weeks after HIV diagnosis (retrospective cases).

Results: Out of 18361 PrEP users, 54 seroconversions (Retrospective = 41; Ambispective = 2; Prospective = 11) were identified (cumulative incidence 0.2%, 95%CI: 0.1-0.3). Median age was 32.5 years [IQR: 27-38]; 93% were cisgender men and 94% reported sex with men. Thirty-two (60%) were Latin-American and 17 (32%) Spanish; half (23/46) had completed university education. *Chemsex* use was reported by 44% (24/54), and 48% had a concomitant STI. Daily FTC/TDF was the most frequent regimen (37/51; 73%), while 27% used on-demand. PrEP interruptions were reported by 33 (72%) and 15 (45%) had suboptimal adherence. Time from last PrEP dose to first HIV positive test was 2 days [IQR:0-90]. Baseline median CD4 count was 616.5 cells/mm^3 [IQR: 438-760] and VL 19,608.5 copies/mL [IQR:825-221000]. Five (10%) were already undetectable at diagnosis and 19 (37%) at Fiebig Stage VI. Time from last negative to first positive HIV test was 3.5 months [IQR: 2.7-9.0] (fig. 1a). A baseline genotypic resistance test was available in 93% (50/54) and successfully amplified in 90% (45/50). Substitutions to NRTIs were detected in 51% (M184V/I, K65R), NNRTIs 20% (K103N, E138A, V106A/M), protease 16% (M36I, L63P), and integrase 2% (G163K). ART was started 10 days [IQR:4-18] after HIV diagnosis (fig. 1b). Initial ART regimens included BIC/FTC/TAF (57%), BIC or DTG+DRV/c+TAF/FTC (15%), DTG/3TC (7%), BIC/FTC/TAF+RPV or DOR (4%), and others (17%). Among retrospective cases, 5 (12%) reported ART-simplifications during follow-up and 4 (10%) ART-discontinuation; 36 (88%) individuals completed a 52 weeks follow-up [IQR:48-55], with undetectability rates at M1 = 46%, M3 = 80%, M6 = 88%, and M12 = 88%. No serious adverse events were reported to either PrEP or ART.



Conclusions: Cumulative HIV incidence among oral PrEP users was very low. A significant proportion of seroconverters reported PrEP interruptions and/or poor adherence. Relevant seroconversion delays

were not observed. A significant proportion of PrEP-related and non-PrEP-related resistance substitutions were detected, but ART-suppression rates were high.

Comunicaciones Orales 2
25 de noviembre - 10:45-12:45 h

CO-09. SPECIFIC SPATIAL IMMUNE LANDSCAPES OF LYMPH NODES WITH UNDETECTABLE VERSUS ACTIVE HIV REPLICATION

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Introduction: Despite efficiently suppressing HIV replication, antiretroviral therapy (ART) does not eliminate persistent HIV-1 infected reservoir cells. One of the key anatomical sites where infected $CD4^+$ T cells persist even in the presence of ART are lymphoid tissues, but the complex interplay between immune cells in these organs and how these tissue cell networks reshape in the presence of viral replication remain incompletely understood. Advances in tissue imaging offer a unique opportunity to characterize the specific cell circuits in tissue. Here, we used a spatial proteomic method to map immune microenvironments in HIV infected tissue.
Methods: Lymph node (LN) tissue from 6 people with HIV (PWH; n = 3 undetectable, n = 3 viremic) were obtained from the Pathology Anatomy Unit from Hospital Universitario La Princesa. LNs from people without HIV (PWOH) were included as negative controls (n = 5). We applied MIBI-TOF (Multiplexed Ion Beam Imaging by Time of Flight), to simultaneously image 42 proteins in these tissues. We profiled over 1.8 million cells and characterized the spatial relationships between 20 cell types in their native tissue context. The inclusion of the viral protein p24 allowed us to identify microenvironments with differential active viral replication and their functional signatures.
Results: As expected, LNs from PWH contained fewer $CD4^+$ T cells compared to PWOH ($p = 0.0006$), while there were higher numbers of $CD8^+$ T cells in both the whole tissue ($p = 0.024$) and specifically within follicle areas ($p = 0.021$). These $CD8^+$ T cells expressed more Granzyme B and were enriched in p24+ LN ($p = 0.011$). Further characterization of p24+ regions revealed higher levels of markers associated with activation and exhaustion of memory and effector $CD8^+$ T cells (CD45RO, ICOS, TIGIT, Galectin9, TIM3), as well as Caspase1 ($p = 0.03$) and NLRP3 ($p = 0.03$, FC = 1.90) inflammasome markers. Using a spatial niche detection method, we found that LN from PWH were enriched for $CD8^+$ T cells near antigen-presenting cells SMA+ fibroblasts, and CD31+ endothelial cells. Notably, viremic donors, unregardless of presence of p24+ cells, selectively shaped the microenvironment increasing the presence and induction of inflammasome on monocytes within the myeloid compartment.
Conclusions: Using spatial proteomics, we identified novel markers in tissue PWH with different levels of viremia, implying that active viral replication confers long-term changes on the immune landscape in LNs. Collectively, these data provide new insights into complex cell networks associated with viral replication at an important tissue reservoir site, which could be relevant for future HIV cure strategies.

CO-10. ENFERMEDADES DEFINITORIAS DE SIDA E IMPACTO DIFERENCIAL EN LA MORTALIDAD EN LA CORIS

Enrique Bernal Morell[1], Carlos Báguena[1], Jose Miguel Gómez Verdú[1], Eva García Villalba[1], Cristina Tomás Jiménez[1], Salvador Valero Cifuentes[1], Antonia Alcaraz García[1], Ángeles Muñoz Pérez[1], Santiago Moreno[2], Teresa Gómez García[3], Jose Luis Blanco Arevalo[4], Helena Albendín-Iglesias[5], Pepa Galindo[6], Nuria Espinosa-Aguilera[7] y Jose Ramón Blanco[8]

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Introducción: Las enfermedades definitorias de sida (EDS) se asocian a mayor mortalidad en personas con VIH, aunque su impacto no es uniforme. El objetivo de este estudio fue evaluar la influencia relativa de las distintas EDS en la mortalidad por cualquier causa.
Métodos: Estudio de cohortes retrospectivo en la cohorte multicéntrica española CoRIS. Se incluyeron pacientes con diagnóstico de al menos una EDS, seguidos desde la entrada hasta muerte o fin de seguimiento. El desenlace principal fue la mortalidad global. Se aplicaron modelos de Cox multivariable ajustados por edad, sexo, vía de transmisión, tipo y año de inicio de TAR, país de origen, nivel educativo, coinfecciones por VHB/VHC, carga viral y CD4 al ingreso. Las infecciones bacterianas se usaron como referencia y se compararon el resto de subgrupos de EDS (neoplasias, *Pneumocystis*, infecciones fúngicas, micobacterias, virales, encefalopatía, sarcoma de Kaposi, *wasting syndrome* y otras).
Resultados: Se analizaron 2.913 pacientes (17,5% mujeres), con edad media de 41,3 años y seguimiento medio de 7,9 años. El 72,2% presentó una EDS infecciosa, el 19,4% una neoplasia y el 8,5% otras. El 33,2% inició TAR con un inhibidor de la integrasa, el 31,1% con un inhibidor de la proteasa y el 28,4% con un NNRTI. Durante el seguimiento se registraron 450 muertes. En el análisis ajustado, las neoplasias presentaron mayor riesgo de mortalidad (HR 1,91; IC95% 1,5-2,44; $p < 0,001$), destacando linfoma no Hodgkin (HR 2,66; IC95% 1,6-4,4; $p < 0,001$) y cáncer de cuello uterino (HR 2,7; IC95% 0,91-8,2; $p = 0,07$). Entre las infecciones, *Pneumocystis* mostró menor riesgo frente a infecciones bacterianas (HR 0,53; IC95% 0,32-0,88; $p = 0,014$). El resto de subgrupos no alcanzaron significación estadística. Otros factores asociados a mayor mortalidad fueron: no recibir TAR (HR 7,14), edad ≥ 50 años (HR 1,84), coinfección por VHC (HR 1,45) y VHB (HR 1,52). Un recuento de CD4 ≥ 200 células/µL se asoció con menor riesgo (HR 0,69).
Conclusiones: El impacto de las EDS sobre la mortalidad no es homogéneo. Las neoplasias, en especial linfoma no Hodgkin y cáncer de cuello uterino, se asociaron a un riesgo marcadamente superior al de las infecciones oportunistas. Por el contrario, la infección por *Pneumocystis jirovecii* mostró un riesgo relativo menor que las infecciones bacterianas. Estos hallazgos sugieren que la categoría de "sida" no debería considerarse de forma uniforme, sino estratificada según la EDS presentada.

CO-11. HIV INFECTION IS STRONGLY ASSOCIATED WITH ACUTE MYOCARDIAL INFARCTION, INDEPENDENTLY OF AGE, GENDER, AND COMORBIDITIES: A SPANISH POPULATION-BASED COHORT STUDY

Sergi Rodríguez-Mercader[1], Juan Du[1], Esperanza Cañas[1], Itziar Arrieta[1], Jade Soldado[1], Irene Carbonell[1], Cecilia Canepa[1], Maria Jose Fernández[1], Marta Trenchs-Rodríguez[2], Alicia González-Mena[1], Natalia García-Giralt[1] and Roberto Güerri-Fernández[1]

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Introduction: HIV infection and antiretroviral therapy (ART) are linked to negative impacts on the metabolic profile, potentially elevating cardiovascular risk. Nonetheless, the evidence regarding the specific influence of HIV on acute myocardial infarction (AMI) remains controversial. This study aims to assess the correlation between clinical HIV diagnosis and the risk of AMI in a population-based study.
Methods: We performed a retrospective cohort analysis utilizing the SIDIAP database, including clinical data from more than 8 million persons in Catalonia, Spain. For every HIV-positive case identified (PWH) from 2007 to 2019, three HIV-negative controls were selected, matched by age and gender. AMI diagnoses were identified in both cohorts using predefined clinical criteria. Inverse probability of treatment weighting (IPTW) based on propensity scores was subsequently employed to balance baseline variables across the groups. Adjustment variables were chosen based on clinical significance and data integrity, encompassing both exposure- and outcome-related aspects. The variables analyzed before to the event included: Age, sex, nationality, LDL cholesterol, HDL cholesterol, triglycerides, body mass index,smoking status,renal clearance, alcohol intake, statin use, diabetes, hypertension, and chronic kidney disease. Cox proportional hazards models were employed to assess the association between HIV and the risk of AMI.
Results: A total of 10,326 PWH were identified and matched by sex and age with 30,978 controls. The average age of participants was 43 years, with 74.1% being male. Notable baseline disparities between HIV-positive patients and controls were detected. Individuals with HIV had increased rates of active smoking (71.7% compared to 50.6%),a greater incidence of low HDL cholesterol (< 40 mg/dL: 44.2% versus 17.4%), and raised triglycerides (≥ 150 mg/dL: 37.6% versus 31.6%).PWH had a reduced mean body mass index (24.4 vs. 27.9 kg/m^2) and exhibited moderate to high Charlson comorbidity scores in comparison to the control group. The incidence of AMI was 34.4 per 1,000 person-years in PLWH and 17.6 per 1,000 person years in controls. Unadjusted analysis revealed a significantly higher AMI risk in PLWH (HR: 1.897; 95%CI 1.615-2.228). After IPTW adjustment, the increased risk persisted (HR:1.838; 95%CI: 1.480-2.282). The unadjusted analysis indicated a markedly elevated risk of acute myocardial infarction in HIV-positive persons (Hazard Ratio [HR]:1.897; 95% Confidence Interval [CI]:1.615-2.228). Upon controlling for specific covariates, the risk persisted at an increased level (HR:1.838; 95%CI:1.480-2.282).
Conclusions: HIV infection correlates with a heightened risk of acute myocardial infarction, even when controlling for clinically relevant risk variables. The identified disparities in the metabolic profile and comorbidities underscore the necessity of tailored cardiovascular risk management for patients with HIV.

CO-12. SELECTIVE MODULATION OF NKP44 AND NKP30 LIGANDS ON GASTROINTESTINAL CD4$^+$ T CELLS DURING HIV INFECTION

Alba González Robles[1], Irene Mota Gómez[1], Judith Grau Expósito[1], Eloy Espín Basany[2], Stefania Landolfi[2], Meritxell Genescà Ferrer[1] and María José Buzón Gómez[1]

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Introduction: Despite the success of antiretroviral therapy, HIV persists in tissue reservoirs that remain resistant to immune elimination. Tissue-resident NK (NKTR) cells play a pivotal role in local viral control. The gastrointestinal track, a major site of HIV persistence, represents a critical compartment where NKTR-CD4$^+$ T-cell interactions could influence the balance between viral suppression and immune evasion. Identifying novel ligands for NK natural cytotoxicity receptors within this tissue is key to defining therapeutic targets for the elimination of HIV reservoirs.
Methods: Gastrointestinal resections obtained from routine surgeries were infected ex vivo with HIV and cultured for 7 days using a gelatin-sponge model. NK-ligand detection on intestinal CD4$^+$ T cells (n = 7) was performed with chimeric Fc-fused receptors (NKp30, NKp44, NKp46, CD161, DNAM, NKG2D) and analyzed by flow cytometry. For single-cell RNA sequencing (scRNA-seq) analyses (n = 2), tissue resections were enzymatically digested, and CD45+CD3-CD56+ NK cells together with CD45+CD3+CD8-CD4$^+$ T cells were isolated by cell sorting and processed for scRNA-seq. Populations were subsequently profiled with a focus on tissue-resident NK cell receptors (NCRs: NKp30, NKp44, NKp46) and other activating receptors (CD161, DNAM, NKG2D), as well as their corresponding ligands on CD4$^+$ T cells.
Results: HIV infection induced a significant upregulation of ligands for NKp30 and NKp44 on intestinal CD4$^+$ T cells ($p = 0.031$ and $p = 0.015$, respectively). NKp44 ligands were significantly enriched in p24+ CD4$^+$ T cells ($p = 0.022$), while NKp30 ligands showed a similar trend. No significant induction of other ligands was detected. Single-cell transcriptomics revealed increased expression of Bag3, an activating NKp30 ligand, and the proliferating cell nuclear antigen (PCNA), a known inhibitory ligand of NKp44, in CD4low T cells from infected tissue. Strikingly, these CD4low cells also harbored the highest levels of HIV transcripts.
Conclusions: Our data suggest that HIV infection might modulate the expression of NCR ligands on CD4$^+$ T cells in gut tissue, with evidence of increased NKp44 and NKp30 interactions. The observed upregulation of PCNA in transcriptionally active CD4low T cells points to a potential inhibitory mechanism that could contribute to NKTR evasion. These results highlight candidate mechanisms of NKTR dysfunction and warrant further investigation to inform strategies for the elimination of persistent tissue HIV reservoirs.

CO-13. PROGNOSTIC VALUE OF BLOOD EPIGENETIC BIOMARKERS OF AGING IN PERSONS WITH WELL CONTROLLED HIV INFECTION

Patricia Martínez-Martín[1], Andrés Esteban-Cantos[1], Francisco Jurado[1], Rocío Montejano[1], Lucía Gutiérrez-García[1], Alejandro de Gea Grela[1], Juan Carlos González[1], Cristina Marcelo[1], Alejandro Díez-Vidal[1], María del Mar Arcos-Rueda[1], Rosa de Miguel Buckley[1], Jose I. Bernardino[1], María de Lagarde[2], José Sanz Moreno[3], Pablo Ryan[4], Vicente Estrada Pérez[5], Leire Pérez-Latorre[6], Juan Emilio Losa-García[7], Berta Rodés[1] and José R. Arribas[1]

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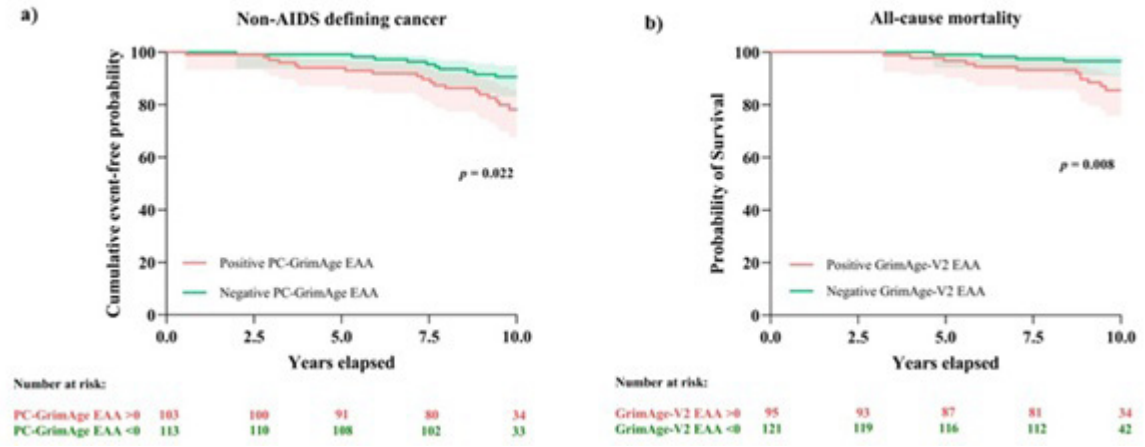
Introduction: Epigenetic age acceleration (EAA) predicts morbidity and mortality in the general population, but its prognostic utility in people with HIV (PWH) on suppressive antiretroviral therapy remains unclear.
Methods: Longitudinal observational cohort study of 216 PWH with sustained virological suppression (median 6.5 years), recruited from the DUAL-GESIDA trial and the La Paz Aging Cohort (Spain). Baseline blood DNA methylation was assessed to evaluate eight epigenetic clocks (Horvath's, Hannum's, Skin&Blood, PhenoAge, GrimAge V1 and V2, DNAmFitAge, and DunedinPACE), their principal-component (PC) derivatives, and the DNA methylation-based estimator of telomere length (DNAmTL). For each biomarker, EAA was calculated as the residual of epigenetic age regressed on chronological age. Over 10 years, we recorded AIDS, serious non-AIDS (SNAEs), and aging-related events. To evaluate the prognostic value of epigenetic aging biomarkers, we stratified participants according to whether they had



positive (EAA > 0) or negative (EAA < 0) epigenetic age acceleration for first and second-generation clocks and their PC-derived counterparts. For DunedinPACE, participants were classified as having a faster (DunedinPACE > 1) or slower (DunedinPACE < 1) pace of biological aging. Associations between EAA measures and outcomes were evaluated by multivariate Cox proportional hazards models adjusted for demographic and HIV-related covariates.

Results: A total of 162 clinical events were observed in 105 participants (1 AIDS event, 65 SNAEs and 96 aging-related conditions). Positive EAA as measured by Horvath's clock predicted aging-related events (HR 1.55, 95%CI 1.02-2.36; p = 0.040), whereas positive EAA according to PC-GrimAge, GrimAge V1 and V2, and Hannum's clock was associated with more than a twofold increased risk of SNAEs (HR 2.50, 95%CI 1.16-5.39; p = 0.019 for PC-GrimAge). Specifically, PC-GrimAge and PhenoAge predicted non-AIDS cancers with HRs of 3.52 (95%CI 1.30-9.54; p = 0.013) and 2.81 (95%CI 1.32-5.96; p = 0.007), respectively (fig. 1A). GrimAge V1 and V2 were strongly associated with all-cause mortality (HR 4.32, 95%CI 1.04-17.90; p = 0.044 and HR 4.69, 95%CI 1.15-19.13; p = 0.031, respectively) (fig. 1B). All deaths (n = 17) occurred among participants with DunedinPACE > 1, whose median baseline pace of aging was significantly higher than survivors (1.19 vs. 1.11; p = 0.002). No associations were found for cardiovascular events or the composite endpoint.

Conclusions: In this preliminary study, GrimAge and PC-GrimAge were associated with an increased risk of adverse outcomes in people with well-controlled HIV infection. These findings suggest their potential relevance as long-term health-risk biomarkers, warranting validation in larger cohorts.

CO-14. IMMUNOLOGIC AND VIROLOGIC PARAMETERS IN A COHORT OF PWH DIAGNOSED WITH CANCER: THE VNEO STUDY

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Introduction: Since the introduction of antiretroviral therapy, the number of non-AIDS defining cancers (NADCs) have increased in people with HIV (PWH). Incidence of tumour development and cancer mortality is higher in PWH compared to general population probably related with impaired immune system functions due to accelerated aging process, leading to diminished tumour surveillance. To have a better insight of the characteristics of PWH diagnosed with cancer in this work we compare viral reservoirs, the degree of chronic-inflammation and immune checkpoint levels (ICPs) between PWH with and without tumour diagnosis.

Methods: We conducted a multicentric case-control study among PWH who received follow-up care between 2017 and 2023 at HIV Unit of Clinic Hospital (Barcelona, Spain) and Hospital La Paz (Madrid, Spain): 55 PWH with cancer diagnoses (VNEO) were identified and matched by age, sex and characteristics of HIV infection with 55 controls. Samples 12-months before cancer diagnose were available in 15 VNEO participants. Levels of 6 ICPs and 21 chronic-inflammation markers (IMs) were analysed from serum samples using Luminex technology. Total and proviral HIV-1 DNA was quantified from $CD4^+$Tcell lysates by qPCR. Mixed linear regression models were conducted for each parameter comparing cases and controls at baseline, and within cases comparing baseline and samples obtained 12-months prior to diagnosis, with random-effects at match and patient level, respectively. For all analyses, only matched pairs with complete data on the parameter of interest for both case and control were included. All parameters were log-10 transformed for analyses.

Results: Integrated HIV-1 DNA was significantly higher (1.93-fold, p = 0.034) at baseline in VNEO compared to controls (median of 555.10 and 281.96 Cp/$10^6$$CD4^+$Tcells, respectively). We observed an increase in most ICPs when comparing baseline samples of VNEO versus controls: 1.41-fold in LAG-3 (p < 0.001), 1.37-fold in PD-1 (p = 0.003), 1.35-fold in TIM-3 (p = 0.003), and 1.29-fold in CTLA-4 (p = 0.038) in VNEO compared to Controls. Regarding the IMs, we found an increase of 1.79-fold in CD30 (p < 0.001), 1.28-fold in CD30L (p = 0.024), 1.33-fold in GRO alpha (p < 0.001), and 1.25-fold in TNF-RII (p = 0.018) in VNEO compared to controls. In n = 15 VNEO patients ICPs and IMs were already increased 12-months prior to cancer diagnosis.

Conclusions: Integrated reservoir, ICPs and IMs were higher in PWH developing cancer at time of diagnosis in comparison with non-cancer PWH. Our results suggest that ICPs and IMs are already altered one year before cancer diagnosis and could be considered in the follow-up of PWH for early diagnosis of NADCs.

CO-15. A FOCUS ON TRANSGENDER WOMEN WITH HIV: CLINICAL OUTCOMES AND QUALITY OF CARE IN THE CORIS COHORT

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Baseline characteristics and clinical outcomes in TWH and other groups (CoRIS 2004-2023)				
	TWH (N 196)	CWH (N 2,515)	MSM (N 10,748)	CHM (N 3,954)
Baseline characteristics				
Age-Median (IQR)-yr.	32.4 (26.2-38.3)	35.2 (28.3-44.2)	32.6 (27.1-39.8)	38.6 (30.5-46.8)
Born in Spain-N (%)	25 (12.8)	1,244 (49.5)	6,823 (63.5)	2,695 (68.2)
HIV acquired by sex-N (%)	98.9 (96.9)	2,153 (85.6)	10,748 (100)	2,523 (63.8)
AIDS at diagnosis-N (%)	2 (13.8)	371 (14.8)	784 (7.3)	826 (20.9)
CD4+ count at diagnosis-Median (IQR)	368 (213-589)	335 (155-543)	435 (273-618)	283 (111-495)
IRR of events (95%CI)				
LTFU	1.55 (1.16-2.08)	0.64 (0.57-0.71)	Ref	0.55 (0.50-0.61)
VF	2.32 (1.68-3.22)	1.57 (1.42-1.73)	Ref	1.57 (1.44-1.71)
New-ADE	2.22 (1.28-3.86)	1.16 (0.97-1.40)	Ref	1.82 (1.58-2.09)
Mortality	1.20 (0.45-3.22)	2.18 (1.78-2.66)	Ref	4.27 (3.66-4.99)

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Introduction and objectives: This study assesses transgender women with HIV(TWH) included in the Spanish CoRIS cohort, comparing their clinical outcomes and quality of care (QoC) with other people with HIV(PWH), including cisgender women (CWH), men who have sex with men (MSM), and cisgender heterosexual men (CHM).
Methods: A longitudinal analysis was conducted using CoRIS data from 2004 to 2023. Gender identity was self-reported. Incidence rates (per 1,000 person-years) were estimated for loss to follow-up (LTFU), virological failure (VF), AIDS-defining events (ADE), and mortality. Poisson regression was used to calculate incidence rate ratios (IRRs), with MSM as the reference. From 2014 to 2023, two QoC metrics were also assessed: linkage to care within one month of diagnosis and viral suppression (< 50 copies/mL) within three months. Adjusted odds ratios (aORs) were estimated using logistic regression, adjusting for sociodemographic and clinical factors.
Results: Of 20,336 individuals enrolled, 17,413 were included: 10,748 MSM, 3,954 CHM, 2,515 CWH, and 196 TWH. Baseline characteristics and IRRs (95%CI) for clinical events over the full study period (2004-2023), with MSM as the reference group, are presented in the Table. Compared to MSM, TWH consistently exhibited worse clinical outcomes, with higher rates of VF, LTFU, and ADE, while no significant differences were observed in mortality. Notably, TWH had the highest IRRs for both VF and LTFU. Regarding QoC indicators, both TWH and CWH had significantly higher odds of linkage to care within one month of diagnosis compared to MSM (aORs: 0.67 and 0.82, respectively), while no significant difference was observed for CHM. Viral suppression within three months of diagnosis did not differ significantly across groups.
Conclusions: This study comprehensively assessed the clinical trajectories of TWH in the CoRIS cohort. TWH in CoRIS face substantially worse outcomes in terms of VF, LTFU, and ADE, despite demonstrating similar or better performance in early care engagement. These findings highlight the need for focused strategies to improve long-term treatment outcomes in TWH.

CO-16. PERSISTENCE OF LATE HIV DIAGNOSIS AND POORER RESPONSE TO ANTIRETROVIRAL THERAPY AMONG MIGRANTS WITH HIV COMPARED WITH THE NATIVE-BORN POPULATION IN SPAIN

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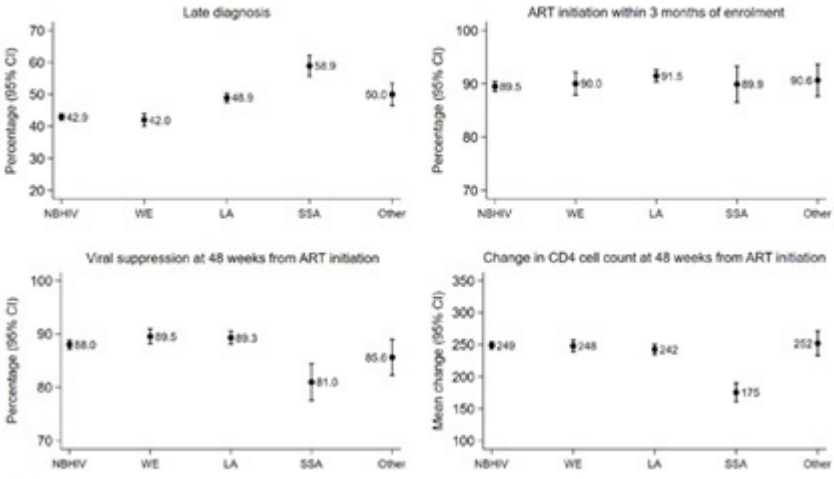
Figure. Late diagnosis, ART initiation within 3 months of enrolment, viral suppression and change in CD4 cell counts at 48 weeks from ART initiation, in NBHIV and MHIV



NBHIV: Native-born people with HIV; WE: Western Europeans; LA: Latin Americans; SSA: Sub-Saharan Africans

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Objectives: Migrants with HIV (MHIV) often face multiple barriers to healthcare access and retention, which may lead to poorer health outcomes. We aimed to compare key outcomes between MHIV and native-born people with HIV (NBHIV), overall and by sex, and to assess whether these differences have changed over time concerning legislative changes affecting migrants' healthcare access (2004-2012, 2013-2018, 2019-2023).

Methods: We included 20,215 adults with HIV enrolled in the CoRIS cohort between 2004 and 2023 (10,928 NBHIV and 9,287 MHIV). Outcomes included late diagnosis (LD; CD4 < 350 cells/µL or AIDS at enrolment), ART initiation within 3 months of enrolment, viral suppression (VS;viral load < 50 copies/ml), and change in CD4 cell counts at 48 weeks after ART initiation.

Results: From 2004 to 2023, the proportion of migrants increased from 37.1% to 59.3%, mainly due to Latin Americans (LA), who rose from 14.8% to 47.8%. Figure shows key outcomes for NBHIV and MHIV. In multivariable analyses, LD was more frequent in LA (adjusted OR [aOR]: 1.58, 95%CI: 1.44;1.73) and Sub-Saharan Africans (SSA) (1.43, 1.24;1.64) compared to NBHIV. This pattern was consistent in men, whereas among women, SSA showed a similar LD prevalence to Spanish women (1.18, 0.96;1.46). Differences in LD between MHIV and NBHIV persisted across all periods. ART was initiated within 3 months of enrolment in 90.2% of participants with no significant differences between MHIV and NBHIV, except for a lower probability observed among LA after 2019 (0.59, 0.38;0.94). VS at 48 weeks was lower in SSA compared to NBHIV (0.66, 0.52;0.86), a difference observed only among men (0.55, 0.41;0.75). CD4 cell count increases were also lower in SSA (adjusted mean difference: -77.0; 95%CI: -94.9;-59.1) and LA (-21.3, -30.6;-11.9). Disparities in viral suppression and immunological recovery between MHIV and NBHIV persisted over time. Loss to follow-up before week 48 after ART initiation was higher among LA (5.6%) and SSA (8.0%) compared to 2.7% in NBHIV.

Conclusions: MHIV of LA origin and men from SSA show higher LD prevalence and poorer immunological recovery, with disparities mainly affecting men. Lower VS is observed specifically in men from SSA. The similar LD prevalence in Spanish women and higher-parity SSA women may partly reflect their regular use of maternal-child health services, which have never been restricted for migrants in Spain. Although ART initiation within 3 months of enrolment was high overall, LA have been less likely than NBHIV to start early since 2019.