



Outness predicts greater leukocyte telomere length in sexually minoritized men with HIV who use methamphetamine

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ABSTRACT

Objective: This longitudinal study aimed to examine the associations of sexual minority stress and outness with leukocyte telomere length across time among sexually minority men (SMM) with HIV who use methamphetamine.

Methods: A sample of 91 SMM with HIV with biologically confirmed recent methamphetamine use completed measures of sexual minority stress and outness at the baseline visit in a randomized controlled trial. Telomere length was measured over 15 months using extracted leukocyte DNA. Statistical analyses were performed using bivariate analyses and generalized estimation equations to examine the independent association between baseline outness and leukocyte telomere length, adjusting for chronological age and recent stimulant use.

Results: Greater outness was significantly associated with longer telomere length (estimate = 0.008; CI: 0.001–0.008) after adjusting for chronological age and stimulant use. There was no evidence that stimulant use, intervention assignment, or race/ethnicity moderated the association between outness and greater leukocyte-telomere length. Sexual minority stress was not significantly associated with leukocyte-telomere length.

Conclusion: Findings are among the first to demonstrate that greater outness is associated with slower biological aging in SMM. Further research is needed to elucidate the bio-behavioral mechanisms linking outness and greater leukocyte-telomere length.

1. Introduction

The advent of modern antiretroviral therapy (ART) has led to a dramatic decrease in HIV-related mortality among people with HIV (PWH) in Western countries since 1996 (Yeni, 2006). Despite a continued narrowing of the gap in overall life-expectancy between PWH and those without HIV, disparities persist, even among those with treated HIV (Marcus, 2020). Indeed, PWH are more likely to develop age-associated non-communicable comorbidities, including

cardiovascular, metabolic, pulmonary, renal, bone, and malignant diseases, compared to those without HIV, who have comparable lifestyle and demographic factors (Schouten et al., 2014). Approximately half of PWH, including those who are virally suppressed by ART, experience some form of neurocognitive impairment (Wei et al., 2020). These comorbidities are believed to be the result of accelerated biological aging among PWH (Rodés et al., 2022; Rosenthal and Tyor, 2019). However, important gaps exist in our understanding of modifiable factors associated with biological aging, particularly among sexual

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minority men (SMM; gay, bisexual, and other non-heterosexual men), who continue to be the population most affected by HIV in the US (Ghanooi et al., 2022; Flentje et al., 2020).

Telomere attrition—the shortening of the protective end-complexes of chromosomes in humans and other eukaryotic organisms—is a widely regarded indicator of biological aging that has been shown to predict age-related disease and mortality among the broader population (Blackburn et al., 2015). When telomeres become critically shortened, such as progressively through repeated cellular division in tissues with limited telomerase availability or through greater exposure to oxidative stress, their capacity to protect against chromosomal damage is compromised. In such cases, intracellular signals indicative of telomere damage are triggered, leading to cessation of cell division and other downstream effects associated with senescence.

While there is individual variation in the rate of telomere shortening, a general trend is that older chronological age is associated with shorter telomeres (Mather et al., 2011). In addition to chronological age, stress (Flentje et al., 2020), male sex (Gutierrez-Rodriguez et al., 2022), racial and ethnic minoritized status (Pantesco et al., 2018), methamphetamine use (Ghanooi et al., 2022), and other physical, biological, and chemical agents (Fernandes et al., 2021) have all been negatively associated with leukocyte-telomere length. In a recent cross-sectional study, sexual minority stress was observed to be associated with elevations in several measures of biological aging derived from DNA methylation in SMM with HIV who use methamphetamine. On the other hand, outness (i.e., the degree to which someone is open about their sexual orientation) was associated with greater methylation-derived estimates of naïve CD4⁺ and CD8⁺ T-cells as well as lower plasma interleukin-6 (Ghanooi et al., 2022).

SMM are approximately three times more likely to report the use of methamphetamine, cocaine, and prescription stimulants in the past year compared to heterosexual men (Compton and Jones, 2021). SMM who use methamphetamine and other stimulants are at an elevated risk for HIV, which is supported by recent findings that one-in-three sexual and gender minority people who were newly diagnosed with HIV reported recent methamphetamine use (Groves et al., 2020). SMM with HIV who use stimulants are more likely to experience barriers to navigating the HIV care continuum, which leads to difficulties in achieving and maintaining viral suppression (i.e., viral load <200 copies/mL) and faster clinical HIV progression (Carrico et al., 1999, 2014; Ellis et al., 2003; Nolan et al., 2017). As such, SMM with HIV who use stimulants are a particularly high-priority population for studies examining the determinants of biological aging.

The Minority Stress Theory posits that sexual identity-based prejudice and stigmatization (i.e., sexual minority stress) experienced over and above general life stress (Brooks, 1981; Meyer, 1995, 2003) is a key driver of health disparities in SMM. Specific sexual minority stressors identified as salient and applicable to SMM include sexual identity-based discrimination, victimization, rejection, internalized and anticipated stigma, and difficulties with outness. Although findings are generally limited to small, cross-sectional studies, a recent evidence-based review of 26 studies indicated that there is some evidence that sexual minority stress is associated with biological outcomes, including immune response and HIV-specific outcomes (Flentje et al., 2020). An important gap addressed in the present study is that, to our knowledge, no longitudinal study has examined the association of sexual minority stress and outness with leukocyte telomere length. To this end, this study longitudinally examined the associations of sexual minority stress and outness at baseline with leukocyte telomere length in SMM with HIV who use methamphetamine.

2. Methods

The data used in this study were collected as part of a randomized control trial of a positive affect intervention delivered during contingency management for stimulant abstinence (Carrico et al., 2024). From

2013 to 2017, we enrolled a total of 184 SMM with HIV who had biologically confirmed, recent methamphetamine use in San Francisco, California. Eligible participants met the following inclusion criteria: 1) 18 years of age or older; 2) identify as a man with male sex assigned at birth 3) reported anal sex with a man in the past 12 months; 4) documentation of HIV-positive serostatus (i.e., letter of diagnosis or ART medications matched to photo identification); and 5) provided a urine and/or hair sample that was reactive to methamphetamine use. This longitudinal study examined de-identified data from 91 participants at baseline, six, 12, and 15 months. Participants included in the analytic sample ($n = 91$) did not substantially differ from those excluded ($n = 93$) on key baseline characteristics, including race and ethnicity, income, education, years since HIV diagnosis, telomere length, sexual minority stress, and treatment arm assignment. All procedures were approved by the Institutional Review Boards of the University of California, San Francisco with reliance agreements from the University of Miami and Northwestern University. All participants completed signed informed consent.

2.1. Measures

2.1.1. Demographics, health status, and recent stimulant use

Age (i.e., date of birth), race, ethnicity, and HIV diagnosis were calculated using self-reported demographic questionnaires. HIV viral load testing was performed to detect plasma HIV RNA using the Abbott Real Time HIV-1 assay (Abbott Molecular, Inc., Des Plaines, Illinois). The lower limit of detection was 40 copies/mL. CD4⁺ and CD8⁺ T cell counts were measured in whole blood using flow cytometry (Quest Diagnostics). Urine samples were collected for onsite toxicology screening for methamphetamine and cocaine using iCup (Redwood Biotech, Inc., Santa Rosa, California USA). The iCup can detect any stimulant use (i.e., methamphetamine or cocaine) within the past 72 h and is represented as a binary variable that indicates the presence (yes) or absence (no) of the substance.

2.1.2. Sexual minority stress

The degree to which participants experienced sexual minority stress was measured using five items derived from the sexual minority stress subscale of the Cultural Assessment of Risk for Suicide Scale (Chu et al., 2013). Sample items include, “The decision to hide or reveal my sexual orientation to others causes me significant distress,” and “I was rejected by a family member or friend after telling him/her my sexual orientation.” Likert-type response options ranged from 1 (strongly disagree) to 6 (strongly agree). Higher scores represented more sexual minority stress (Cronbach’s $\alpha = 0.70$).

2.1.3. Outness inventory

Outness was measured as the extent to which participants publicly identified their sexual orientation to friends, family, and the world (Mohr and Fassinger, 2000). Response options included a Likert-type scale with scores that ranged from 1 (person definitely does not know about your sexual orientation status) to 7 (person definitely knows about your sexual orientation status and it is openly talked about). This measure of outness had good internal consistency (Cronbach’s $\alpha = 0.91$). Higher scores indicated greater outness.

2.1.4. Leukocyte-telomere length

Leukocyte telomere length samples were preprocessed using standard procedures to yield validated estimates, as previously described (Zhang et al., 2017). The mean telomere length was determined by quantitative polymerase chain reaction (qPCR). This method calculates the ratio of the telomeric product to a single-copy gene, which appears once in the human genome. This single-copy gene served to standardize the estimated number of telomere repeats in the sample based on the number of genome equivalents, acting as a proxy for the number of cells in the sample. A QuantiNova SYBR Green PCR Kit (Qiagen) was used for

sample amplification. Each sample was measured in quintuplicate, with an average of at least three valid replicates used for subsequent analyses. Measurements deviating by ≥ 1.5 standard deviation units were considered outliers and excluded. If three valid replicates could not be obtained, the qPCR was repeated. The PCR template for each unknown sample consisted of 4 ng of genomic DNA. Each qPCR experiment included one intra-plate and one inter-plate control sample to evaluate variability using the coefficient of variation. A purchased control genomic DNA sample was used to create a standard curve and normalize the assay plates by calculating the geometric mean of the control DNA sample on each plate. For participants providing multiple blood samples over time, DNA from all repeated measurements was placed on the same assay plate to further reduce variability in relative telomere length assessment.

2.2. Statistical analyses

Bivariate analyses were conducted to assess the relationship between each predictor and leukocyte-telomere length chosen a priori (Ghanooni et al., 2022): sexual minority stress, outness, and recent stimulant use. This included correlation analysis, t-tests, ANOVA, and chi-square tests, as appropriate. Statistically significant bivariate results were included in a subsequent multivariable analysis. Multivariable analysis were performed using Generalized Estimating Equations (GEE) using the geeglm function of the geepack package in R (RFfs, 2024). We selected the exchangeable correlation structure for the analysis to account for autocorrelations between repeated measures of leukocyte-telomere length. Models were adjusted for age and stimulant use due to its theoretical relevance to biological aging among sexual minority men. This systematic stepwise approach ensured a robust and theoretically informed analysis. Results were mean-centered, 95 % confidence intervals (CI) were reported, and p-values ≤ 0.05 (two-tailed) were considered significant.

The potential moderating role of viral load, race/ethnicity, and intervention assignment were evaluated. Interaction terms for each moderator (viral load, race/ethnicity, intervention assignment) were added separately to the primary GEE models. Each interaction term was tested one at a time to assess whether the marginal associations between significant predictors identified in our bivariate analysis and leukocyte telomere length differed across levels of these moderators. Additionally, we explored socioeconomic indicators (e.g., education, income) and viral load as covariates given its linkage to biological aging.

3. Results

Among the 91 participants, the ages ranged from 22 to 59 years, with a mean age of 42.5 years (SD = 8.91). Over half of the participants had a positive urine test for stimulants (56 %), and the mean baseline telomere length was 0.949 (SD = 0.04) (Supplemental Table 1). Bivariate results indicated outness was marginally associated with telomere length (estimate = 0.005; $p = 0.014$), however we did not detect significant differences between stimulant use (estimate = -0.017 ; $p = 0.07$) and sexual minority stress (estimate = 0.001; $p = 0.101$).

Our GEE analysis indicated the passage of time was inversely associated with shorter leukocyte telomere length such that telomeres were significantly shorter at six months (estimate: -0.002 ; 95 % CI: 0.01, -0.001), 12 months (estimate: -0.011 ; 95 % CI: $(-0.02, -0.001)$), and 15 months (estimate: -0.030 ; 95 % CI: $-0.05, -0.026$) compared to baseline. Greater outness scores were independently associated with longer telomeres (estimate = 0.008; 95 % CI: 0.001, 0.008), after adjusting for chronological age and stimulant use (Table 1). Covariates such as socioeconomic indicators and viral load were not significantly associated with leukocyte telomere length and did not alter the association between outness and telomere length. The model-derived association between outness and leukocyte telomere length is shown in Fig. 1. There were no differences in leukocyte telomere length as a function of

Table 1

Longitudinal associations of outness and leukocyte telomere length (N = 91).

Factors	Estimate	Std. Error	Wald Statistic	95 % Confidence Intervals	p-value
Intercept	0.936	0.021	904.2	(0.88, 0.96)	<0.01
Outness (centered)	0.008	0.004	4.50	(0.001, 0.008)	0.001
Baseline (Ref)					
6 months	-0.002	0.002	0.411	$(-0.01, -0.001)$	<0.01
12 months	-0.011	0.003	11.4	$(-0.02, -0.001)$	<0.01
15 months	-0.030	0.003	28.76	$(-0.05, -0.026)$	<0.01
Chronological Age	-0.0006	0.0004	3.54	$(-0.0018, -0.0002)$	0.06
Recent Stimulant Use	-0.009	0.003	0.02	$(-0.005, 0.003)$	0.87

intervention assignment or race and ethnicity in the proposed study; thus, it was not included in the models. Although viral load was not directly associated with telomere length, a significant interaction was observed such that the association between outness and telomere length was stronger among individuals with detectable viral load (estimate = 0.0064, $p = 0.0119$) (Supplemental Table 2).

4. Discussion

To our knowledge, this study is the first to observe a longitudinal association between outness and longer leukocyte telomere length in SMM with HIV who use stimulants, a validated biomarker linked to accelerated aging. This association was not moderated by recent stimulant use, intervention assignment, or race or ethnicity. Although the measured effect size was small, these findings partially support the minority stress model, which suggest sexual minority stress processes, such as outness, are associated with biological outcomes (Flentje et al., 2020) which is consistent with other studies that indicate psychosocial health related to sexual identity is important in the biological aging process (Rivera et al., 2024). Furthermore, previous cross-sectional studies indicated that stimulant use was associated with shorter leukocyte telomere length (Ghanooni et al., 2022), there was no evidence that recent stimulant use was associated with changes in leukocyte telomere length. Taken together, these results support the need for further longitudinal research to examine the biobehavioral mechanisms, whereby outness could influence the rate of biological aging in SMM with HIV.

Although race/ethnicity did not moderate the association of outness with longer leukocyte telomere length in this sample, there is some evidence that the beneficial effects of outness are more pronounced among non-Hispanic White SMM. For example, one longitudinal study found that outness was associated with lower rates of tryptophan degradation over 15 months in non-Hispanic White SMM, but not among men of color (Vincent et al., 2021). Non-Hispanic White SMM have been shown to have greater social support after disclosing their sexual minority status, whereas racial and ethnic SMM reported less support after disclosing and more instances of discrimination (Vincent et al., 2017, 2021; Storholm et al., 2019). Further research is needed to examine the relevance of intersectional minority stress processes among SMM of color.

This longitudinal study should be interpreted in context of some limitations. Because findings were among SMM with HIV who use methamphetamine, future studies should examine the associations of sexual minority stress and outness in the broader population of SMM with and without HIV. Second, it is noteworthy that the beneficial association of outness with longer leukocyte telomere length was observed in a sample recruited in San Francisco, arguably one of the most affirmative cities in the United States for SMM. Further research is needed to

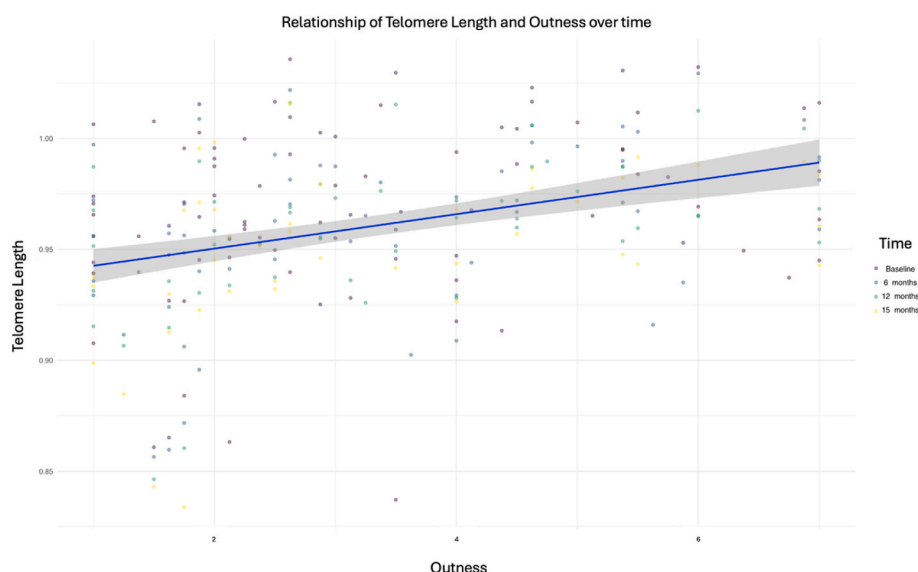


Fig. 1. The relationship between leukocyte telomere length and outness at baseline, 6-months, 12-months, and 15months. (N = 91).
Caption: Outness predicts greater leukocyte telomere length over 15 months.

determine whether and how the associations of sexual minority stress and outness with measures of biological aging differ across geographic regions with varying degrees of structural stigma. Third, the sample size was modest, which may have limited the statistical power for detecting smaller effect sizes. Fourth, we did not observe a significant association between sexual minority stress and telomere length, which may be due, in part, to limitations in the measurement of sexual minority stress. The instruments used, while validated, may not have fully captured the scope or chronicity of minority stress experiences, particularly structural or cumulative forms of stigma, discrimination, and marginalization that unfold over the life course. Measures focused primarily on proximal stressors (e.g., recent discrimination or internalized stigma) may not fully reflect the chronic, multi-level nature of sexual minority stress that is theorized to impact biological aging processes. Finally, all participants enrolled in a randomized controlled trial received contingency management, which is an evidence-based behavioral intervention that achieves moderate reductions in stimulant use (Prendergast et al., 2006). Declining rates of stimulant use over time may have limited our ability to detect associations between recent stimulant use and leukocyte-telomere length.

Despite these limitations, our findings provide further evidence to support the association between outness in relation to a validated biomarker of biological aging. Given the increasing prominence of laws, policies, and discrimination against sexual and gender minority communities, further investigation on the implications of telomere length and social environments in which individuals feel comfortable disclosing their sexual identity as well as same-sex attraction and behaviors are warranted.

CRediT authorship contribution statement

Renessa S. Williams: Writing – review & editing, Writing – original draft, Methodology, Conceptualization. **Ariana Johnson:** Writing – review & editing, Writing – original draft, Formal analysis. **Michael Miller-Perusse:** Writing – review & editing, Writing – original draft. **Annesa Flentje:** Writing – review & editing, Supervision, Funding acquisition. **Judy Moskowitz:** Writing – review & editing, Funding acquisition. **Samantha E. Dilworth:** Writing – review & editing, Formal analysis. **Keith J. Horvath:** Writing – review & editing, Funding acquisition. **Adam W. Carrico:** Writing – review & editing, Writing – original draft, Supervision, Formal analysis, Conceptualization. **Brad E. Aouizerat:** Writing – review & editing, Methodology, Formal analysis.

Delaram Ghanooni: Writing – review & editing, Writing – original draft.

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Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Renessa Williams reports financial support was provided by National Institute on Minority Health and Health Disparities. Annesa Flentje reports financial support was provided by National Institute on Drug Abuse. Annesa Flentje reports financial support was provided by National Institute on Minority Health and Health Disparities. Adam Carrico reports was provided by National Institute on Drug Abuse. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.bbih.2025.101086>.

Data availability

Data will be made available on request.

References

- Blackburn, E.H., Epel, E.S., Lin, J., 2015. Human telomere biology: a contributory and interactive factor in aging, disease risks, and protection. *Science* 350 (6265), 1193–1198.
- Brooks, V.R., 1981. *Minority Stress and Lesbian Women*. Lexington Books, Lexington, p. 219.
- Carrico, A.W., Riley, E.D., Johnson, M.O., Charlebois, E.D., Neilands, T.B., Remien, R.H., et al., 1999. Psychiatric risk factors for HIV disease progression: the role of inconsistent patterns of anti-retroviral therapy utilization. *J. Acquir. Immune Defic. Syndr.* 56 (2), 146–150.
- Carrico, A.W., Shoptaw, S., Cox, C., Stall, R., Li, X., Ostrow, D.G., et al., 2014. Stimulant use and progression to AIDS or mortality after the initiation of highly active antiretroviral therapy. *JAIDS J. Acquired Immune Deficiency Syndrom.* 67 (5), 508–513.
- Carrico, A.W., Cherenack, E.M., Flentje, A., Moskowitz, J.T., Asam, K., Ghanooni, D., et al., 2024. A positive affect intervention alters leukocyte DNA methylation in sexual minority men with HIV who use methamphetamine. *Brain Behav. Immun.* 120, 151–158.
- Chu, J., Floyd, R., Diep, H., Pardo, S., Goldblum, P., Bongar, B., 2013. A tool for the culturally competent assessment of suicide: the cultural assessment of risk for suicide (CARS) measure. *Psychol. Assess.* 25 (2), 424–434.
- Compton, W.M., Jones, C.M., 2021. Substance use among men who have sex with men. *N. Engl. J. Med.* 385 (4), 352–356.
- Ellis, R.J., Childers, M.E., Cherner, M., Lazzaretto, D., Letendre, S., HIVNRCC, the, 2003. Increased human immunodeficiency virus loads in active methamphetamine users are explained by reduced effectiveness of antiretroviral therapy. *J. Infect. Dis.* 188 (12), 1820–1826.
- Fernandes, S.G., Dsouza, R., Khattar, E., 2021. External environmental agents influence telomere length and telomerase activity by modulating internal cellular processes: implications in human aging. *Environ. Toxicol. Pharmacol.* 85, 103633.
- Flentje, A., Heck, N.C., Brennan, J.M., Meyer, I.H., 2020. The relationship between minority stress and biological outcomes: a systematic review. *J. Behav. Med.* 43 (5), 673–694.
- Ghanooni, D., Carrico, A.W., Williams, R., Glynn, T.R., Moskowitz, J.T., Pahwa, S., et al., 2022. Sexual minority stress and cellular aging in methamphetamine-using sexual minority men with treated HIV. *Psychosom. Med.* 84 (8), 949.
- Grov, C., Westmoreland, D., Morrison, C., Carrico, A.W., Nash, D., 2020. The crisis we are not talking about: one-in-Three annual HIV seroconversions among sexual and gender minorities were persistent methamphetamine users. *J. Acquir. Immune Defic. Syndr.* 85 (3), 272–279.
- Gutierrez-Rodriguez, F., Alves-Paiva, R.M., Scatena, N.F., Martinez, E.Z., Scheucher, P.S., Calado, R.T., 2022. Association between leukocyte telomere length and sex by quantile regression analysis. *Hematol. Trans. Cell Therapy* 44 (3), 346–351.
- Marcus, J. (Ed.), 2020. *Increased Overall Life Expectancy but Not Comorbidity-free Years for People with HIV*. CROI, Boston, Massachusetts.
- Mather, K.A., Jorm, A.F., Parslow, R.A., Christensen, H., 2011. Is telomere length a biomarker of aging? A review. *J. Geront. Series A: Biol. Sci. Med.* 66A (2), 202–213.
- Meyer, I.H., 1995. Minority stress and mental health in gay men. *J. Health Soc. Behav.* 36 (1), 38–56.
- Meyer, I.H., 2003. Prejudice, social stress, and mental health in lesbian, gay, and bisexual populations: conceptual issues and research evidence. *Psychol. Bull.* 129 (5), 674–697.
- Mohr, J., Fassinger, R., 2000. Measuring dimensions of Lesbian and Gay Male experience. *Meas. Eval. Counsel. Dev.* 33 (2), 66–90.
- Nolan, S., Walley, A.Y., Heeren, T.C., Patts, G.J., Ventura, A.S., Sullivan, M.M., et al., 2017. HIV-infected individuals who use alcohol and other drugs, and virologic suppression. *AIDS Care* 29 (9), 1129–1136.
- Pantesco, E.J., Leibel, D.K., Ashe, J.J., Waldstein, S.R., Katznel, L.I., Liu, H.B., et al., 2018. Multiple forms of discrimination, social status, and telomere length: interactions within race. *Psychoneuroendocrinology* 98, 119–126.
- Prendergast, M., Podus, D., Finney, J., Greenwell, L., Roll, J., 2006. Contingency management for treatment of substance use disorders: a meta-analysis. *Addiction* 101 (11), 1546–1560.
- RFFS, Computing, 2024. Package ‘Gee’ Pack. R Core Team, Vienna, Austria.
- Rivera, A.S., Chao, C.R., Hechter, R.C., 2024. Disparities in telomere length by sexual orientation in adults from the genetic epidemiology research on aging cohort. *Am. J. Epidemiol.* <https://doi.org/10.1093/aje/kwae352> kwae352.
- Rodés, B., Cadiñanos, J., Esteban-Cantos, A., Rodríguez-Centeno, J., Arribas, J.R., 2022. Ageing with HIV: challenges and biomarkers. *EBioMedicine* 77, 103896.
- Rosenthal, J., Tyor, W., 2019. Aging, comorbidities, and the importance of finding biomarkers for HIV-associated neurocognitive disorders. *J. Neurovirol.* 25 (5), 673–685.
- Schouten, J., Wit, F.W., Stolte, I.G., Kootstra, N.A., van der Valk, M., Geerlings, S.E., et al., 2014. Cross-sectional comparison of the prevalence of age-associated comorbidities and their risk factors between HIV-Infected and uninfected individuals: the AGEHIV cohort study. *Clin. Infect. Dis.* 59 (12), 1787–1797.
- Storholm, E.D., Huang, W., Siconolfi, D.E., Pollack, L.M., Carrico, A.W., Vincent, W., et al., 2019. Sources of resilience as mediators of the effect of minority stress on stimulant use and sexual risk behavior among young black men who have sex with men. *AIDS Behav.* 23 (12), 3384–3395.
- Vincent, W., Pollack, L.M., Huebner, D.M., Peterson, J.L., Steward, W.T., Rebchook, G. M., et al., 2017. HIV risk and multiple sources of heterosexism among young black men who have sex with men. *J. Consult. Clin. Psychol.* 85 (12), 1122–1130.
- Vincent, W., Carrico, A.W., Dilworth, S.E., Fuchs, D., Neilands, T.B., Moskowitz, J.T., et al., 2021. Intersecting minority statuses and tryptophan degradation among stimulant-using, sexual minority men living with HIV. *J. Consult. Clin. Psychol.* 89 (3), 156–165.
- Wei, J., Hou, J., Su, B., Jiang, T., Guo, C., Wang, W., et al., 2020. The prevalence of frascati-criteria-based HIV-associated neurocognitive disorder (HAND) in HIV-infected adults: a systematic review and meta-analysis. *Front. Neurol.* 11.
- Yeni, P., 2006. Update on HAART in HIV. *J. Hepatol.* 44, S100–S103.
- Zhang, X., Hu, Y., Justice, A.C., Li, B., Wang, Z., Zhao, H., et al., 2017. DNA methylation signatures of illicit drug injection and hepatitis C are associated with HIV frailty. *Nat. Commun.* 8 (1).