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CONFERENCE SUMMARY

The Current State of HIV and Aging: Findings Presented at the 10th International Workshop on HIV and Aging

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Abstract

With increasing effectiveness of antiretroviral therapy, people with HIV (PWH) are living longer and the prevalence of older PWH continues to increase. Accordingly, PWH are experiencing an increased burden of age-related comorbidities. With this shifting demographics, clinicians and researchers face additional challenges in how to identify, address, and manage the complex intersections of HIV- and aging-related conditions. Established in 2009, the International Workshop on HIV and Aging brings together clinicians and researchers in cross-disciplinary fields along with community advocates and PWH to address the multidisciplinary nature of HIV and aging. This article summarizes plenary talks from the 10th Annual International Workshop on HIV and Aging, which took place in New York City on October 10 and 11, 2019. Presentation topics included the following: the burdens of HIV-associated comorbidities, aging phenotypes, community engagement, and loneliness; these issues are especially important for older PWH, considering the current COVID-19 pandemic. We also discuss broad questions and potential directions for future research necessary to better understand the interaction between HIV and aging.

Keywords: HIV, aging, comorbidities, multidisciplinary

Introduction

Due to the increased effectiveness of antiretroviral therapy (ART), life expectancy has increased for people with HIV (PWH). Although disparities in life expectancy among PWH continue to persist, there is an increasing prevalence of PWH 50 years of age and older.[1] In addition, a proportion of incident HIV infections is occurring in older adults.[2–4] As a result, some estimates indicate that over 70% of PWH will be 50 years of age or older by 2030.[5–7] Compared to people aging without HIV, people aging with HIV experience a greater burden of aging-related conditions, including neurocognitive impairment, kidney disease, liver disease, osteoporosis, cardiovascular disease, and frailty.[8–14] Understanding the interaction between HIV infection and aging is a high priority to best manage care and treatment for older PWH.

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In 2009, the annual International Workshop on HIV and Aging began as an effort to address the needs of aging PWH and as a unique opportunity to engage in scientific dialog about the clinical care of, and research with, people aging with HIV. The workshop has three goals: (1) to stimulate and guide research that will enable better treatment methods and strategies for older PWH, (2) to encourage young investigators to engage in research and clinical care of older PWH, and (3) to foster collaborations among investigators, clinicians, advocates, and PWH. For the past decade, the workshop has brought together experts in pertinent cross-disciplinary fields, including basic mechanisms of aging, HIV biology and pathogenesis, clinical geriatrics, endocrinology, pharmacology, neurology, psychology, and social work. The 10th annual International Workshop on HIV and Aging was held on October 10 and 11, 2019, in New York, NY.

In this study, we present a summary of the key oral presentations from the workshop, beginning with the current HIV epidemic both in high-income countries and in Sub-Saharan Africa, and then reviewing advances in understanding phenotypes that overlap between aging and HIV, such as frailty. We also summarize presentations related to factors that contribute to these aging phenotypes, including pathogenesis such as increased coagulation and social factors such as loneliness. We discuss implications of potential unique therapies of cannabis and cure and conclude with reflections of a long-term HIV advocate and his contributions to the field. More detailed information on the workshop can be found on the conference website: https://www.virology-education.com/event/previous/10th-hiv-aging-workshop-2019/.

Current Burdens of HIV-Associated Comorbidities, Coinfections, and Complications and Influences on Future Burden

Keri N. Althoff, PhD, MPH

Current morbidity for PWH can be categorized into physical health morbidity (e.g., cardiovascular disease, liver disease, and metabolic syndromes), mental health morbidity (e.g., neurocognitive disorders, mental health conditions, and substance use), coinfections (e.g., tuberculosis, viral hepatitis B and C, and sexually transmitted infections), and syndromes (e.g., frailty, decreased mobility, and falls).[2,9,12–25] Prevalence estimates of these morbidities in PWH vary. For example, globally, COPD prevalence is estimated to be 10.5%, 3% of PWH were coinfected with tuberculosis in 2017, 7% of PWH are coinfected with hepatitis B virus, and 6% of PWH are coinfected with hepatitis C virus.[26–28] A systems biology approach over the life course will consider how factors earlier in life affect future burden of HIV-associated comorbidities, coinfections, and complications.[29,30] In addition, a ''geroprotectors approach'' to devise interventions that target common mechanisms of aging and delay the onset of more than one age-related disease at the same time may be especially relevant for PWH.[31]

With effective test and treat interventions now stimulated by Ending the HIV Epidemic in cities and countries worldwide, the risk profiles for comorbidities among PWH will likely shift.[32,33] Overall, PWH will continue to age, but simulation models suggest that risk profiles and burden of outcomes will differ for subpopulations of PWH (e.g., for Hispanic heterosexual women vs. white men who inject drugs vs. black men who have sex with men).[1,34] Changing exposures to duration of uncontrolled viremia before ART initiation, antiretroviral drugs, and early- and mid-life intervention opportunities may also affect future morbidity. Those who have been infected more recently and have benefited from test and treat initiatives in the Treat All era, initiating ART immediately after HIV diagnosis with less toxic ART, may have a reduced burden of comorbidities as they age, compared to those with prior exposure to more toxic ART (such as stavudine and zidovudine) and longer durations of pretreatment viremia. In addition, there may be more opportunity for early- and mid-life interventions to reduce the prevalence of traditional risk factors for age-related comorbidities through HIV clinical care.[12] Following the initiation of HIV treatment, long-term viral suppression, longer-term effects of current antiviral drugs (including weight gain and diabetes with integrase inhibitors[35,36] and tenofovir alafenamide[37,38]), and changes in lifestyle behaviors, including substance, use will also influence the future burden of morbidity in PWH.[35,36,39–41] In conclusion, interventions to address HIV-associated comorbidities, coinfections, and complications remain essential to reduce future morbidity for PWH and improve quality of life, even as efforts progress toward ending HIV epidemics around the world[32,41]; it will be important to consider how these interventions may need to be tailored for different subpopulations of PWH.

HIV and Aging in Sub-Saharan Africa

Jean B. Nachega, MD, PhD

As ART rollout continues to expand in low- and middle-income countries, the aging of the HIV epidemic will be mirrored in sub-Saharan Africa, which is home to 70% of the world's HIV epidemic. The associated increased life expectancy of PWH in this setting will lead to increases in HIV prevalence among older adults.[42] Indeed, modeling by Hontelez *et al.* using South African data suggests that HIV prevalence among people older than 50 years will nearly double in the next 30 years, and the absolute number of similarly aged PWH will triple in the same period.[43]

To sustain the benefits of global investments in HIV care in Africa, there is a need for increased research on determinants of health and quality of life for older PWH in sub-Saharan Africa. To date, most studies of aging with HIV in the region have been cross-sectional, have focused on single comorbidity domains (e.g., hypertension or obesity), and lack insight about local preferences for quality of life (e.g., self-reliance and social and cultural norms of well-being). In addition, available evidence suggests that some determinants of HIV-associated comorbidities among older PWH in Africa differ from those in the United States and Europe.[44] For example, increased exposure to biomass cooking fuel commonly used in sub-Saharan Africa has been found to be associated with higher odds of metabolic syndrome among PWH in the Eastern Democratic Republic of the Congo.[45] Host genetic predictors of kynurenine (K) pathway of tryptophan (T) metabolism and increase in K/T ratio also have been associated with an increased risk of atherosclerosis, depression, AIDS-related cancer, and all-cause mortality in Ugandan PWH.[46] Elucidating these determinants and their relative contributions to comorbidities among older PWH in sub-Saharan Africa is essential to developing effective

interventions to optimize health for a growing population of older PWH in this region.[44] This will require investment in training and research infrastructure for HIV and aging in sub-Saharan Africa.

Ten Years of Aging Research: Frailty and Aging

Linda P. Fried, MD, MPH

Understanding the evolution of frailty can help researchers identify the implications of and interventions for frailty in the context of HIV and aging. In the 1980s, frailty and disability were often considered synonymous, which caused problems in geriatric care and research due to the lack of specificity.[47] The following decade saw an effort by researchers and clinicians to differentiate aging from disease, and further distinguish multimorbidity, disability, and frailty, which were thought to be different from aging itself. Furthermore, frailty began to emerge as distinct from all of these. Over the last two decades, researchers have identified frailty not only as a unique medical syndrome linked to a particular underlying pathobiology that is aging related but also likely accelerated by catabolic disease.[48] More recently, through the efforts of the National Institute on Aging leadership in geroscience, there is an emerging central thesis of shared biologic pathways that are aging associated and aging driven, which emerge in the presentation of a frailty syndrome and in disease development.[49–51]

The frailty phenotype,[48] the theory for which was operationalized in the Cardiovascular Health Study and later validated in U.S. community-dwelling cohorts,[48,52] includes five primary characteristics: shrinking, weakness, slowness, poor endurance, and low activity. Individuals with none of the five characteristics were classified as nonfrail, those with one or two characteristics as prefrail, and those with three or more as frail. This identification of a constellation of symptoms and signs as diagnostic is consistent with the definition of a clinical syndrome. In subsequent studies, frail individuals had the highest risk of adverse health outcomes when compared to those who were nonfrail or prefrail, independent of disease.[48,52,53] When examined together, studies from 1998 to 2008 show that frailty is clinically observable; is not synonymous with multimorbidity, disability, or extreme old age; increases with age and varies by race and gender; behaves as a clinical syndrome; predicts disability and mortality independent of disease; and is associated with inflammation, and dysregulation of each of the core physiologic systems that regulate stress response and maintain homeostasis.[54] It has a natural progression, with those who are prefrail at the highest risk for becoming frail and those who are frail at highest risk of dying within the next 6–36 months, depending on severity of frailty.[52,53,55] Energy is a key factor in frailty, including energy homeostasis, energy production and utilization, and energy dysregulation.

Energy is a driver at every level of the syndrome, cellular, physiologic, and phenotypic. When the individual is stressed, such as in challenge studies (e.g., glucose tolerance tests and phosphocreatinine recovery following isometric calf exercises), frail, prefrail, and nonfrail can be clearly differentiated by the degree of response and rapidity in return to baseline, with the response to stressors in frail delayed and exaggerated, compared to the nonfrail.[55,56] Increasing evidence indicates multiple physiologic regulatory systems are dysregulated in frailty, with a greater number of dysregulated systems increasing the odds of frailty. Frailty appears to be the result of a breakdown in the homeostasis that is required for an organism to remain resilient.

Despite the growing body of research concerning frailty, questions remain, particularly for older PWH. There is evidence of an interplay between frailty and disease, especially catabolic diseases. The specific system drivers that start the syndrome of frailty, such as inflammation, need to be better understood. It also is unclear if there are shared etiologic factors between frailty and disease and how these interact with one another, especially in the case of HIV. The implications for the prevention and treatment of frailty are complicated by multiple co-occurring events: treatments that can alter multiple pathways, such as physical activity, will be most effective in reducing or preventing frailty. Ultimately, successful frailty prevention will involve interventions that target the underlying physiology and biology, which might be unique for older PWH. Frailty provides a window into the biology of vulnerability by allowing us to examine the resources and resilience in PWH who can be rebound in the face of stressors to the system.

The Pathway of Altered Coagulation as a Pathophysiological Mechanism in HIV Disease

Jason V. Baker, MD, MS

A key contributor to the current spectrum of end-organ disease risk among older PWH entails persistent abnormalities in coagulation activation, despite effective ART.[57–59] Older PWH are well known to be at excess risk for venous thromboembolism (VTE), and atherosclerotic cardiovascular disease is now a leading cause of morbidity and mortality among PWH.[60] Still, beyond these classic manifestations of macrolevel venous or arterial thrombosis, elevations in circulating D-dimer levels also are associated with increased risk for end-stage liver or renal disease, the frailty phenotype, all-cause mortality, and other grade 4 adverse events related to end-organ injury among PWH.[59,61,62] In this context, a central underlying question is how low-level persistent hyperactivation of the coagulation system contributes to excess risk across a wide spectrum of disease, beyond macrolevel thrombosis.

When studying potential causal associations between HIV-associated coagulopathy and end-organ disease risk, there are several important considerations and limitations when interpreting data from observational studies. Two examples are potential influence of confounders and mediators on associations between host factors and clinical risk. Confounders are not on the causal pathway, and associate with both the outcome and a biomarker or the exposure of interest. Age is one such confounder that is well known to associate with coagulation and risk for disease outcomes. Mediators, however, are more informative in this context as they are directly or partially on the causal pathway, such that they may account, at least in part, for the association between HIV-associated coagulopathy and end-organ disease risk.

Mediators specific to HIV disease that may contribute to coagulopathy include direct effects from viral replication as well as persistent immune depletion and loss of a protective barrier at the level of mucosal surfaces.[63–66] We have previously shown that HIV viremia increases procoagulant

factors (e.g., factor VIII), and concurrently decreases anticoagulant factors (e.g., antithrombin and protein C), with the resulting alterations in coagulation factor composition then associated with greater predicted thrombin generation and mortality risk.[63,66] These HIV-associated changes are very similar to changes in coagulation profiles that occur with advancing age.[54,67] HIV disease is also characterized by immunologic depletion at effector sites in the gastrointestinal tract, and other secondary lymphatic tissues contribute to the loss of mucosal integrity, which largely persists despite ART.[68] This loss of mucosal integrity contributes to ongoing immune activation and low-level hypercoagulation due, in part, to microbial antigens translocating across mucosal surfaces resulting in endotoxin-mediated activation of tissue factor pathways.[58,64,68,69] Pathologic alterations to coagulation profiles and low-level endotoxemia then represent potential mediators on the causal pathway from HIV disease to coagulopathy.

If an HIV-associated coagulopathy increases risk for thrombosis, it follows that risk for ischemic cardiovascular disease and mortality may be increased in this context. However, other end-organ diseases cannot be explained by macrovessel thrombosis and other explanations must be sought. Additional pathways have therefore been explored, whereby HIV coagulopathy could cause end-organ disease by driving inflammation-associated tissue injury. This hypothesis entails a cross-talk between coagulation and inflammation that is mediated by clotting factors (e.g., factor X) activating protease-activated receptors (PARs), which are expressed on leukocytes and on vascular surfaces.[70,71] Activation of PAR-1 and/or PAR-2 signaling, in part, drives inflammation and injury within end-organ tissues. However, this hypothesis has been tested and not supported in two proof-of-concept randomized trials, studying direct acting oral anticoagulants edoxaban (inhibition of factor X) and vorapaxar (inhibition of PAR-1).[72,73] Alternatively, it is possible that a disease state may contribute to alterations in coagulation (i.e., referred to as reverse causality), such that the development of end-organ dysfunction, whether HIV related or not, would itself further contribute to a coagulopathy. In summary, current data support that chronic HIV disease contributes to a coagulopathy, but further research is needed to better understand the mechanisms by which this coagulopathy contributes to end-organ pathology and clinical manifestations resembling those of aging among older PWH.

The Promise and Challenge of Cannabis and Cannabinoids as Medicine

Thomas D. Marcotte, PhD

There is a long history of medicinal use of cannabis, going back millenia. Political shifts in the early 20th century resulted in the criminalization of cannabis. In the 1990s, there was persistent anecdotal evidence that cannabis mitigated HIV-related symptoms, such as nausea, vomiting, and wasting, with simultaneous political shifts to favor access to medical cannabis. In 1996, the Compassionate Use Act was passed in California, which allowed the use of medicinal cannabis and spurred a call for greater research about the positive and negative effects of cannabis. In 1999, the Medical Marijuana Research Act was passed in California, which provided resources and pathways to conduct rigorous studies on cannabis, including development of the Center for Medicinal Cannabis Research at the University of California, San Diego.

In the decade that followed, cannabis researchers identified over 100 different cannabinoids in cannabis, the main two being tetrahydrocannabinol (THC), which is psychoactive, and cannabidiol (CBD), which is nonintoxicating. These are the two compounds most often discussed and studied in cannabis research, including those examining possible medicinal effects. In addition, the plant contains terpenoids, which contribute to the aroma and may act on serotonin, dopamine, and other receptors, and flavonoids, which contribute to the color of the plant and might have antioxidant and anti-inflammatory properties.

To date, two primary cannabinoid receptors have been noted: CB1, which is highly prevalent in the brain, as well as other body systems, and CB2. The body also has an endocannabinoid system, with two primary constituents being anandamide and 2-arachidonylglycerol. This system, unlike some other neurotransmitters, can be synthesized on demand and serve as signaling messengers to promote homeostasis.

In 2017, The Health Effects of Cannabis and Cannabinoids National Academies Report was released, and stated that in humans there is (1) conclusive evidence that cannabis benefits chronic pain, spasticity associated with multiple sclerosis, and control of nausea[74]; (2) moderate evidence that cannabis helps improve sleep in those with chronic conditions; (3) limited evidence that cannabis helps anxiety disorders and posttraumatic stress disorder (PTSD); and (4) no evidence (to date) that cannabis is effective as a treatment for diseases such as cancer, epilepsy, or schizophrenia. In most cases, the lack of evidence was based upon a lack of substantive research having been completed, rather than necessarily negative findings.

There are a number of studies, typically small, which provide data on the potential benefits of cannabis in PWH. A study by Abrams *et al.* found evidence that smoking cannabis reduces HIV neuropathic pain[75] and another by Wilsey *et al.* showed that both low (1.29% THC) and medium (3.53% THC) doses of vaporized cannabis were equally effective with neuropathy in other conditions.[76] In a population without HIV, Wallace *et al.* proposed a ''window'' for cannabis pain relief, such that too low or too high dose of THC may have no effect, or even exacerbate pain, indicating that dosage is very important.[77] Based on a retrospective observational study of PWH, there is a possibility of neuroprotective effects among moderate cannabis users compared to infrequent and frequent users.[78] If true, this is likely a benefit seen in individuals with an inflammatory condition, such as HIV, and in which the anti-inflammatory effects may be helpful, rather than in individuals not with such conditions. In one study of PWH, having a diagnosis of cannabis use disorder was predictive of higher odds of being a ''superager,'' an adult who performs better than his or her peers, and at par with younger individuals, on cognitive tests.[79]

While there are data supporting positive effects of cannabis use, several challenges to conducting cannabis research remain. First, smoking as a delivery method is a challenge due to concerns regarding the safety of using combustible materials (e.g., in medical settings), second hand smoke as an irritant, and difficulties in the standardization of dosing, to name a few. Second, the Drug Enforcement Agency (DEA) scheduling criteria and access regulations limits access to

cannabis for research. Currently, plant-based THC is schedule 1 (the most restrictive), and the scheduling of synthetic THC and synthetic and plant-based CBD vary widely (including CBD from hemp now being ''descheduled,'' whereas CBD from other sources remains on the schedule). Furthermore, currently the University of Mississippi remains the sole source for plant-based cannabis (non-hemp) in the United States. There is promise that the DEA will propose new regulations to expand the policy to allow additional sources. Finally, there has been a proliferation of CBD products (e.g., ointments, edibles, and capsules); however, these are not available to researchers because they are federally illegal, and therefore no study can assess their efficacy or safety.

Questions regarding HIV, aging, and cannabis remain unanswered, and are of particular importance due to the alterations in body composition, reduction in hepatic and renal drug clearance, cardiovascular and pulmonary changes, and so on, which can occur with both aging and HIV and interact with the effects of cannabis.[80] Method of administration will also play a role in evaluating the risks and benefits of cannabis, as there are indications that long-term smoking may result in increased risk of lung-related diseases in PWH.[81] More studies on the potential anti-inflammatory and neuroprotective qualities of cannabis are also needed, to inform their potential uses among older PWH.

Loneliness and Social Isolation in Older People with HIV

Maile Y. Karris

Loneliness, or the discrepancy between one's preferred and actual social relationships, is different from social isolation (a lack of contact between an individual and society) and is hypothesized to serve as an evolutionary cue.[82] Like hunger, the discomfort of loneliness encourages persons to seek out meaningful relationships, ultimately to enhance survival. Loneliness and social isolation are common in the United States, with estimates suggesting that nearly half of Americans report sometimes or always feeling alone and 40% sometimes or always reporting their social relationships are not meaningful.[83] Older PWH may experience slightly higher rates of loneliness than HIV-seronegative persons, with estimates ranging from 39% to 58% depending on the population evaluated.[84,85] Older PWH who report loneliness are more likely to smoke cigarettes, use alcohol or other substances, and have low social support, depressive symptoms, and poor to fair quality of life.[85] Loneliness and social isolation increase the odds of an early death by 26%–45%, an impact similar to that of smoking 15 cigarettes a day.[86–88]

The effect of loneliness and social isolation on health appears secondary to stress-induced cortisol dysregulation. Persons who are lonely demonstrate higher total peripheral vascular resistance and lower cardiac contractility.[89] Immunologically, persons who are lonely display less natural killer cell activity, poorer immune responses to influenza vaccination, and increased circulating levels of cortisol.[89–91] It is now apparent that chronic higher than usual levels of cortisol mediate the transcriptional response of glucocorticoid receptor pathways.[92] Clinically, these conserved transcriptional responses to adversity result in a mixed picture of excess inflammation and immunosuppression that moderate the association between loneliness and social isolation and health.[93,94] Protective factors that mitigate the impact of loneliness also exist and include wisdom, resilience, nostalgia and eudaimonia (sense of purpose in the world),[95,96] although the prevalence and impact of these factors in older PWH have not been studied. Overall, evaluation of loneliness in older PWH remains a significantly understudied topic. Further work that enhances our understanding of the true impact of loneliness and social isolation on quality of life, health, and function of older PWH is needed to ultimately develop effective interventions for this potentially modifiable condition.

HIV Cure Research in an Aging Population: Unique Opportunities and Challenges

Steven Deeks, MD

Finding a cure for HIV is an important consideration for all PWH. However, there are many unique challenges for cure research within the aging HIV population, including the impact of immunosenescence, increased rates of medical comorbidities, polypharmacy, and frailty. The potential benefits of cure for older PWH are also numerous: curing HIV could reduce stigma, improve psychosocial outcomes, and reduce harm associated with long-term ART toxicity and polypharmacy. An HIV cure could reduce inflammation, immune dysfunction, and tissue fibrosis, resulting in a significant reduction in morbidity for older PWH.[97] At present, PWH older than 65 years are routinely excluded from HIV cure research, potentially limiting advances in the field. Despite the unique needs of older PWH that might be addressed by a cure, study exclusion is often due to concerns that older PWH are less likely to tolerate and respond to emerging therapies. The use of this chronological cutoff means that little is known about the impact of age-associated changes in T cell dynamics on the size and activity of the HIV reservoir or the impact of immunosenescence on the prospects for cure. HIV cure research is a prime example of an arena in which collaborations between the fields of HIV and aging are essential.

The cure agenda can be advanced by drawing on parallels from the field of oncology, where numerous therapies have been developed to target pathways involved in immune function (immune checkpoint inhibitors, myeloid-derived suppressor cells, T-regulatory cells, and inhibitors of indoleamine 2,3-dioxygenase-1), or from the field of gerontology related to health span. An example of this cross-specialty pollination of ideas can be seen with drugs from the rapamycin (mTOR) inhibitor class. When given to mice, the mTOR inhibitor rapamycin extends life expectancy, likely through reduced inflammation and autophagy.[98] It is therefore possible that low-dose mTOR inhibitors could be used to improve health in aging and older PWH. Data suggest that blocking mTOR has a wide variety of potentially beneficial effects on the immune system, including inhibition of T cell proliferation and enhancement of immune responses to vaccines.[99,100] These data from the gerontology field have led to studies using mTOR inhibitors in the HIV cure arena, with positive results.[101] For instance, use of the mTOR inhibitor sirolimus by PWH on successful ART resulted in reductions in proliferating CD4+ T cells, immune activation, and HIV reservoir size.[102] Whether these benefits can be extended to decrease complications of aging in PWH has yet to be

established, but this cross-disciplinary research demonstrates how breakthroughs in other fields should be applied to further advances in HIV cure among PWH of all ages.

Being a Researcher and Community Advocate in HIV and Aging

Stephen Karpiak, PhD

Having a background in basic science, I studied the effects of antibodies and lipids on central nervous system recovery, while on faculty at Columbia University Medical School, offering me the opportunity to collaborate with researchers across the globe. In 1995, I transitioned away from basic science (neuroscience) and became involved in HIV for the first time. I became the Executive Director of ''A Place Called Home,'' which provided congregate housing for the homeless with HIV in Phoenix, Arizona. Through a series of transitions, I have found my place as a researcher-advocate on behalf of people aging with HIV. With the support of researchers in gerontology, I showed that older PWH did not have functional social networks and lacked access to caregivers, who can be critical in providing support as people age.[103] Data show that socially isolated seniors face higher mortality rates. My research has focused on the care issues of older PWH who face high risk for multimorbidity and associated polypharmacy, as well as psychosocial challenges, such as loneliness and depression. Like all individuals, PWH would benefit from strong social networks to help maintain good quality of life and stable mental health. Many community programs are starting to expand their services and focus on older adults.

To coalesce these important issues, my colleagues and I launched ''The Research on Older Adults with HIV (ROAH) Study,'' in 2005, to study the comprehensive psychosocial needs of older PWH in close collaboration with the community. This study, along with the ROAH 2.0 multisite study (2017–2020), has helped generate data to inform the development of programs and policies that best address the needs of older adults with HIV. Findings from this study indicate that access to and availability of mental health treatment is a significant problem for some older PWH with serious mental health issues. In addition, many older PWH exhibit high rates of loneliness and desire more social opportunities. The creation of more opportunities for older PWH to come together, whether building on existing programs or creating new ones, is a worthy aim. Many older PWH struggle with high housing costs and hunger. As a result, solutions for easing financial strains should be considered. Finally, given high levels of comorbidity and polypharmacy in older PWH, many would likely benefit from care guided by geriatric medicine's precepts and a coordinated care approach. Expanding combined HIV/geriatric care programs and training HIV care providers in geriatric care principles may be ways to increase access to this treatment approach. Working closely with the community has allowed for the identification of these future care directions.

Conclusions

Over the past decade, the annual International Workshop on HIV and Aging has provided a valuable platform to bring together researchers, clinicians, community advocates, and PWH to collaborate on ways to improve research and care for people aging with HIV. In this study, we summarized plenary talks from the 10th annual International Workshop on HIV and Aging 2019, which covered topics ranging from the burdens of HIV-associated comorbidities in high-income countries and in Sub-Saharan Africa, phenotypes that overlap between aging and HIV such as frailty, as well as research on the mechanistic and psychosocial aspects of aging with HIV. A key theme from the workshop was that understanding the intersections of HIV and biological aging remains complex and requires innovative interdisciplinary teamwork from researchers, clinicians, patients, and community partners to move the field forward. Creative teamwork approaches are particularly essential to confront new challenges that may emerge for a growing population of aging PWH, such as COVID-19. Building partnerships that engage the community and bridge different areas of expertise, from infectious diseases to nursing to gerontology, as well as different methods of data collection from multiple levels, will be essential to study the impact of the COVID-19 pandemic on the physical and mental health of aging PWH, and to prepare for future pandemics.[104]

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