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ISBN

9798193262749

Author

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Publication Date

2026-09-08

Peer reviewed|Thesis/dissertation

Examining Health Disparities
in the use of Hematopoietic Cell Transplants for
HIV-related Lymphoma

By

KURT ANTHONY DAVID
DISSERTATION

Submitted in partial satisfaction of the requirements for the degree of

DOCTOR OF PHILOSOPHY

in

Nursing Science and Healthcare Leadership

in the

OFFICE OF GRADUATE STUDIES

of the

UNIVERSITY OF CALIFORNIA

DAVIS

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2026

ACKNOWLEDGEMENTS

In the context of sexual and gender minorities (SGM; similarly known as LGBTQ+), “coming out” refers to a dynamic and multifaceted process in which SGM individuals internally acknowledge, externally disclose, and continuously affirm their own sexual orientation and/or gender identity within their community and society at large.

The 2015 landmark case, *Obergefell v Hodges*, signaled a turning point in the U.S fight for SGM equality—a positive societal shift in the longstanding rejection of SGM individuals had occurred. With increased SGM representation and now legal recognition of same sex marriage, it seemed the stigma surrounding “coming out”, and the discrimination that often followed, was dissipating. However, today, the progress towards continued and actualized SGM liberation faces the threat of erasure.

This work is dedicated to the SGM pioneers who paved the way to make space where love can thrive. That built the underground gay havens where coming out was not just accepted but embraced, and who fought for a future where “coming out” wasn’t a traumatic life event.

Crucially as important, this work is dedicated to the revolutionaries of today. To those that will stand up and show up. To those that will rise to meet this moment.

Examining Health Disparities in the use of Hematopoietic Cell Transplants for HIV-related Lymphoma

Acknowledgments	ii
Table of Contents	iii
Abstract	iv
Chapter 1: Introduction and Problem Statement	1
Chapter 2: Review of the Literature and Specific Aims	16
Chapter 3: Utilization of Hematopoietic Cell Transplantation for HIV-lymphoma: The Role of Social and Clinical Characteristics in California, 1991-2016	24
Chapter 4: Differences in Hematopoietic Cell Transplantation Outcomes by HIV status, California, 1991-2016	49
Chapter 5: Implications and Conclusions	140

Examining Health Disparities in the use of Hematopoietic Cell Transplants for HIV-related Lymphoma

ABSTRACT

There are over 1.2 million people with HIV (PWH) in the United States, with inequities embedded in the HIV epidemic since its earliest years. Disparities persist across the HIV continuum including screening and diagnosis, treatment, viral suppression, and complications; and are influenced by social determinants of health including race/ethnicity, socioeconomic status, sexual and gender minority status, insurance, and geographic location. As combination antiretroviral therapy (cART) became FDA approved in 1996, HIV became a chronic, manageable disease, resulting in a growing and aging population of PWH. As overall survival in PWH has improved, cancer has emerged as a major comorbidity, yet there is a paucity in published evidence addressing the cancer needs of PWH. Lymphoma is among the most common cancers in PWH, with elevated risk due to underlying immunosuppression and viral co-infections (e.g., Epstein-Barr virus). HCT is a potentially curative treatment modality for relapsed or refractory lymphoma. Despite increased risk for lymphomas, PWH experience barriers to standard cancer care, with little known about access to hematopoietic cell transplantation (HCT) for PWH and the associated clinical care.

To address these gaps, this dissertation presents two population-based studies of adults aged 18-79 with first primary lymphoma diagnosed from 1991-2016, with cohorts drawn from a novel linkage between the Center for International Blood and Marrow Transplant Research (CIBMTR), the California Cancer Registry (CCR), and the California Patient Discharge Database (PDD). In Specific Aim 1, we characterize HCT utilization among 6,248 PWH diagnosed with lymphoma following HIV diagnosis.

Cumulative incidence methods accounted for the competing risk of death, and multivariable Cox proportional hazards models evaluated the sociodemographic and clinical factors associated with HCT use among PWH. Overall, despite improvements in HCT utilization in the modern cART era, HCT use in PWH remained low and was associated with older age, lower neighborhood socioeconomic status, being single/unmarried, and public insurance. In Specific Aim 2, we tested whether HIV status was associated with differences in treatment burden, hospital utilization, and survival after HCT. We identified 140 HCT recipients with HIV (cases) matched 1:3 to 409 HCT recipients without HIV (controls) on age, sex, lymphoma subtype, stage, HCT type, transplant year, and time from diagnosis to transplant. Adjusted cox models demonstrate no significant differences in 90-day acute complications or hospital utilization, while overall survival was similar at 90-days, 1 year, and 2 years post-HCT.

Collectively, the studies in this dissertation demonstrate that while HCT use among PWH with lymphoma has increased in California since the introduction of cART, though sociodemographic disadvantages contribute to lesser utilization. Additionally, the findings from these studies suggest that other factors not measured in these studies, like institutional barriers and societal attitudes (e.g. HIV-related stigma), may drive inequities in access. Importantly, we demonstrated that among otherwise eligible patients with HIV that undergo HCT, HIV was not associated with greater risk of acute renal or liver toxicities, infection/sepsis, increased hospital utilization, or inferior survival. These findings support equitable inclusion of PWH in standard eligibility and referral processes, whereby HIV alone should not preclude HCT therapy.

Chapter 1: Introduction and Problem Statement

INTRODUCTION

There are over 1.2 million people living with human immunodeficiency virus (HIV) in the United States, ^{1,2} with inequities deeply ingrained in the history of HIV since its discovery. ³ Among those affected, disparities exist across the major milestones of the HIV continuum—including screening, diagnosis, treatment, and complications. ^{4–12} These disparities are influenced by social determinants of health, which include structural and systemic factors that impact access to healthcare and overall well-being. ¹³ Specifically for people with HIV (PWH), these determinants may include sexual or gender minority status, race/ethnicity, socioeconomic status, and geographic location. ^{14–19} Existing literature highlights both the complexity and persistence of inequities across the HIV trajectory. Successful advancements in HIV treatment, particularly combination antiretroviral therapy (cART), have led to a growing population of aging PWH who are now at increased risk for cancer in general. ^{20–24} Currently, little evidence exists regarding the specific cancer-related needs of PWH. ^{25–27}

Considering the paucity of research at the intersection of HIV and cancer, my research as a PhD Candidate at the University of California, Davis focuses on cancer in people with HIV, also known as HIV-associated malignancies. My experience as an Oncology Clinical Nurse Specialist and Hematopoietic Cell Transplant Registered Nurse informed the design of this research, supported by the National Institute of Nursing Research with the National Institutes of Health (1F31NR021235). In this study we evaluate the utilization of, and sociodemographic and clinical factors associated with, hematopoietic cell transplants (HCTs) in PWH, and evaluate for differences in acute complications, hospital use, and survival following HCT based on HIV status.

EARLY PRESENTATIONS OF HIV

In 1981 in the San Francisco Bay Area and New York City, a rare cancer began to emerge among men who have sex with men (MSM).²⁸ The term “MSM” is used to describe men that engage in sexual acts with other men, that may occur without the internal self-identification as having a gay or bisexual sexual orientation,²⁹ and is also used to categorize a route of HIV transmission.¹ While Kaposi sarcoma had already been identified, a cancer rarely seen in the general population characterized by dermatologic signs and symptoms like purple rashes and easy bruising,^{30,31} the underlying cause of Kaposi sarcoma among the sudden outbreak in MSM in these metropolitan areas was still unknown. Patients presented with distinct symptom profiles across what would be discovered as the HIV lifecycle. During the acute phase of HIV infection, patients often experienced flu-like symptoms indicative of HIV seroconversion.²⁴ During the chronic phases of HIV infection, in particular prior to discovery of antiretrovirals targeting HIV, chronic inflammation occurs and uncontrolled HIV progresses to acquired immunodeficiency syndrome (AIDS) – as CD4 counts drop risk for infectious complications increase, contributing to sepsis-related mortality.²⁴ For many gay men during the early days of HIV/AIDS, the sudden septic deaths and AIDS-associated wasting of their partners due to the unknown etiology of AIDS was traumatic.

The political landscape resulting from the HIV epidemic and early hysteria was marked by both HIV-related stigma and advocacy,³² particularly within the LGBTQ+ community. Social activist groups such as the AIDS Coalition to Unleash Power (ACT UP) in New York City played a pivotal role in demanding government action, increasing

public awareness, and accelerating research funding.³³ While political activism for LGBTQ+ rights gained momentum in the streets, scientific efforts at the bench and in clinical studies were simultaneously advancing toward identifying the causative agent of AIDS.^{34,35} Theories regarding the cause of AIDS during this time varied widely, ranging from religious interpretations to biological hypotheses.^{36,37}

DISCOVERING HIV & cART

The discovery of HIV came as a result of groundbreaking research conducted on both sides of the Atlantic from competing labs.³⁶ In the U.S at the National Cancer Institute, Dr. Robert Gallo's lab utilized an approach focusing on the T-cell leukemia virus (HTLV) as the causative agent of AIDS.³⁸ Meanwhile, in Paris, France, Dr. Luc Montagnier's team at Pasteur Institute pursued a different viral etiology—lymphadenopathy-associated virus (LAV), ultimately leading to the identification of the HIV virus.³⁹ In 2008, Dr. Luc Montagnier was awarded the Nobel Prize for the discovery of HIV, confirming that the underlying mechanism of AIDS was caused by a retrovirus,^{40,41} that when left untreated progressively destroyed the immune system ultimately leading to opportunistic infections, sepsis, and death.

HIV is a retrovirus that contains two strands of RNA and binds to healthy CD4 T cells at the CD4 receptor to enter the cell.^{24,42} As an RNA virus, after anchoring to the cell membrane HIV utilizes reverse transcription to integrate itself into the host genome, the primary mechanism that establishes lifelong HIV infection.²⁴ Within the cytoplasm of the CD4 T cell, the HIV then forms a double-stranded HIV DNA that is integrated into the host CD4 T cell.^{24,42} T-cells are part of the humoral immunity whose primary

function is to recognize pathogens and mount an immune response to harmful infections. With a dampened immune system, people living with HIV are challenged to fight off infections due to the sustained infection and cell death of CD4 T cells by the HIV virus, making PWH particularly at risk for opportunistic infections and sepsis.

Understanding the viral replication mechanisms of HIV provided targets for pharmacological intervention. The emergence of combination antiretroviral therapy (cART) in 1996 (versus earlier single agent regimens),^{20,24} was the most significant milestone in HIV discovery and treatment. Combination antiretroviral therapy significantly reduced viral replication by targeting multiple timepoints in the HIV lifecycle necessary for viral replication, thus reducing HIV viral load in patients and later preventing further HIV transmission.⁴³ HIV was no longer a death sentence. The first available cART regimen included 3 separate pills multiple times per day—a protease inhibitor (saquinavir/Invirase) and two nucleoside reverse transcriptase inhibitors (zidovudine/Retrovir and lamivudine/Epivir).^{44–46} Later, in 2006, the first ‘one pill, once a day’ regimen came to market including tenofovir, FTC, and efavirenz (Atripla).²⁰ Despite substantial advances in modern HIV pharmacology, significant challenges to HIV care remain, including disparities in access to HIV testing, delays in initiating cART treatment, and cART adherence.^{47–52}

CURRENT PREVALENCE OF HIV

By the end of 2022 there were over 1.2 million people living with HIV in the United States, and significant inequities in access to HIV care and treatment continue to widen.^{1,53–55} While HIV mortality rates vary widely by population characteristics and

geography, ¹ one population-based study found that HIV mortality peaked in 1995 with an annual death rate of 10.3% (prior to widespread adoption of cART), and following the widespread adoption in the later 90's the annual death rate decreased to 0.2%. ⁵⁶

HIV disproportionately affects marginalized groups resulting in health disparities. ⁵⁷ In terms of HIV transmission categories within the U.S, men who have sex with men (MSM) represent the largest HIV transmission category (67%) followed by heterosexual contact (22%), injection drug use (7%), and lastly, MSM with injection drug use (4%).¹ In terms of racial and ethnic differences in HIV prevalence, Black/African American individuals represent the largest affected racial/ethnic group (39%), followed by Hispanic/Latino (27%) and White (27%), multiracial (5%), Asian (2%), American Indian/Alaska Native (1%), and Native Hawaiian/Pacific Islander (<1%). ^{1,2}

Among the largest affected subgroup (MSM; 67%), intersectional ^{58–60} disparities exist. Intersectionality refers to the intersecting power dynamics associated with multiple, co-occurring identities (e.g., race and sexual orientation); whereby the intersecting power dynamics are not only additive, but compounded when co-occurring simultaneously. ^{58–60} When looking at the intersection of race/ethnicity and sexual orientation and gender identity (SOGI), it is estimated that 40% of Black/African American MSM will acquire HIV by the age of 30, and that an estimated 60% will acquire HIV by the age of 40. ^{10,61} The lifetime risk of HIV in Black/African American MSM is 1:2 compared to 1:11 in White MSM.¹⁰ In 2022, Black/African American MSM represented 34% of new HIV diagnoses compared to 24% in White MSM.¹ One factor contributing to the disparities in new HIV diagnoses are the inequities in HIV preexposure prophylaxis (PrEP) uptake among Black/African American MSM.⁶² PrEP is

a daily oral combination antiretroviral that while typically used for HIV treatment, is given prophylactically to prevent HIV infection in high-risk individuals like SGMs.⁶²

The HIV prevalence and lifetime risk noted have clear implications on 1) who is most at risk for HIV, 2) who subsequently develops long-term HIV sequelae (like AIDS or HIV-associated malignancies), and 3) among those that are diagnosed with HIV and develop HIV complications like an HIV-associated malignancy, who then has access to relevant treatments.

CARE ACROSS THE HIV CONTINUUM

The HIV care continuum begins with prevention e.g., Pre-Exposure Prophylaxis [PrEP] using cART when undiagnosed, or condoms), though the additional five major milestones of the HIV continuum as conceptualized by the Centers for Disease Control include: 1) HIV infected but undiagnosed, 2) HIV diagnosed but not retained in medical care, 3) retained in care but not prescribed ART, 4) prescribed ART but not virally suppressed, and 5) virally suppressed.⁶³ HIV testing is currently performed using a variety of tests, including an HIV self-test and an at-home rapid test, along with blood tests performed in labs.⁶⁴ Retention in HIV care is defined as having received two or more CD4 or viral load tests performed at least three months apart.⁶⁵ Standard of care therapy for HIV antiretroviral currently includes oral combination therapy using a single integrase strand transfer inhibitor (INSTI) agent with one or two nucleoside reverse transcriptase inhibitor (NRTI) agents.^{23,66} Oral cART may contain a single pill or multiple pills, and dosed once daily or twice daily. Antiretroviral therapy is considered successful when the viral load is suppressed (defined as < 200 copies/ml) or undetectable.⁶⁵ If an

HIV viral load is poorly controlled, the patient's condition may progress to acquired immunodeficiency syndrome (AIDS), defined as a CD4 + T cell count of <200 cells per μ l. ²⁴ AIDS is the most severe stage of HIV disease that is characterized by a poorly functioning immune system with significantly increased risk for opportunistic infections.^{1,24} Certain cancer diagnoses are associated with the presentation of AIDS, known as AIDS-defining-malignancies (e.g. Kaposi sarcoma, cervical). ^{22,67}

ROLE OF HIV ON CANCER

HIV is hypothesized to influence cancer biology by creating an immunosuppressive environment that increases susceptibility to oncogenesis through multiple mechanisms. ^{24,68} The HIV virus directly weakens immune surveillance by depleting CD4+ T cells, impairing the body's ability to regulate abnormal cells before they develop into cancer. ^{24,68} This immunosuppressive mechanism provides an opportunity for other risk factors, such as viruses that are directly oncogenic—like Epstein-Barr virus (EBV), human papillomavirus (HPV), and Kaposi's sarcoma-associated herpes virus (KSHV) to proliferate more freely in PWH. ^{23,69} Additionally, behavioral and social risk factors, such as smoking and substance use further contribute to cancer risk. ^{70,71} While combination antiretroviral therapy (cART) has significantly reduced the incidence of AIDS-defining cancers, ^{72,73} non-AIDS-defining malignancies in the setting of well-controlled HIV are increasing in prevalence across different cancer diagnoses. ^{9,74}

Cancer diagnoses among PWH can be stratified into malignancies that occur in the context of AIDS (AIDS-defining malignancies), and those that occur in the absence

of AIDS (non-AIDS-defining malignancies).^{72,75} AIDS-defining malignancies include Kaposi's sarcoma, Hodgkin's lymphoma, and cervical cancer; while non-AIDS-defining malignancies common in PWH include anal cancer, lung cancer, and liver cancer.^{74,76} Within clinical oncology treatment guidelines, the region of cancer involvement and its clinical staging generally informs the subsequent treatment plan that consists of surgery, chemotherapy/immunotherapy, radiation, and/or transplant.

CARE ACROSS THE CANCER CONTINUUM

Much like HIV, cancer care can be mapped across a cancer continuum— this includes cancer prevention, detection/diagnosis, treatment, survivorship, and survival/mortality.⁷⁷ The National Comprehensive Cancer Network (NCCN) develops evidence-based, consensus-based clinical practice guidelines for patient care across the cancer continuum, including guidelines for those with HIV,⁷⁸ lymphoma,⁷⁹ and receiving HCT.⁸⁰ There is no current screening for lymphomas, though diagnoses often present with sudden weight loss, fevers, and night sweats that prompt further evaluation including a PET/CT scan.⁷⁹ First-line treatment for lymphomas include chemotherapy and radiotherapy, and hematopoietic stem cell transplants (HCT) may be utilized as first-line treatment for high-risk disease or in relapsed/refractory disease.^{79,80} A hematopoietic stem cell transplant (HCT) involves the collection of stem cells from a living host (allogeneic transplant) or the patient can collect and freeze their own cells for re-transplantation (autologous transplant). The predominant transplant type used for lymphoma is an autologous transplant.^{79,80} To ensure success prior to the transplant, the patient is evaluated for baseline organ function, functional status, infectious disease

markers (like HIV), and caregiver support (like from a spouse or family member).⁸¹ The patient subsequently undergoes consolidation chemotherapy, a process intended to eradicate the patient's own immune system creating physiologic space for a new immune system to be transplanted via stem cells.

While helpful, NCCN guidelines do not definitively guide cancer care in PWH due to limited evidence.⁷⁸ In a national survey of oncologists, 70% reported that current evidence supporting the treatment of cancer patients with HIV is insufficient.⁸² Despite being at greater risk for developing lymphoma, individuals with HIV are simultaneously at greater risk for not even receiving lymphoma treatment. The odds of not receiving chemotherapy or radiation for non-Hodgkin's lymphoma (NHL) in someone with HIV was 1.82 (1.65 - 2.00; $p < 0.001$) compared to someone without HIV.⁸³ Among patients with Hodgkin lymphoma (HL), the odds of not receiving chemotherapy or radiation in someone with HIV was 1.92 (1.66 - 2.22; $p < 0.001$) compared to someone without HIV.⁸³ While population-based studies demonstrate significantly greater odds of not receiving chemotherapy or radiation for patients with HIV that are diagnosed with NHL or HL, much less is known regarding the specific use of HCTs in patients with HIV-related lymphoma.

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Chapter 2: Literature Review

HIV-RELATED LYMPHOMA

Lymphoma is a white blood cell malignancy that has multiple subtypes depending on the specific cell lineage affected, and in the HIV setting can occur in the context of AIDS or non-AIDS.^{1,2} A non-Hodgkin lymphoma (NHL) diagnosis in an individual with HIV is indicative of the presentation of AIDS, while a Hodgkin lymphoma diagnosis can occur independent of AIDS.³ While HIV creates an immunosuppressive environment,^{2,4} HIV itself is not directly oncogenic and lacks the ability to cause cancer. However, PWH have higher rates of co-infection with oncogenic viruses, such as Epstein-Barr virus (EBV), hepatitis B virus (HBV), human T-lymphotropic virus-1 (HTLV-1), human papillomavirus (HPV), hepatitis C virus (HCV), Kaposi sarcoma herpesvirus (KSHV/HHV8), and Merkel cell polyomavirus.⁵ Specific to lymphomas, EBV is associated with both non-Hodgkin and Hodgkin lymphoma subtypes.⁵ In addition to viral etiologies of cancer in PWH, social factors associated with greater cancer risk like smoking, are prevalent in PWH.⁶

The National Institute of Health (NIH) National Cancer Institute conducted a multi-state population-based study (excluding CA) linking HIV and cancer registries to evaluate the prevalence and risk of cancer diagnosis in people living with HIV.⁷ The HIV/AIDS Cancer Match (HACM) Study estimated that overall cancer risk was 69% higher among individuals with HIV compared to the general population, and the most common cancer type was non-Hodgkin lymphoma (17.3%), while Hodgkin lymphoma represented 4.1% of cases.⁸ The risk for NHL was found to be 11.5 times greater in individuals with HIV compared to those without HIV, while the risk for HL was found to

be 7.7 times greater in an individual with HIV.⁸ Despite an elevated risk of lymphomas in PWH, evidence suggests PWH face barriers in accessing standard cancer treatment like chemotherapy and radiation (cite). However, the likelihood for receiving HCT as a treatment modality for relapsed/refractory lymphomas is unknown.

SOCIAL DETERMINANTS AND HCT UTILIZATION

Social determinants of health influence which patients with lymphoma receive a HCT, though few studies have evaluated the influence of social determinants on HCT use specifically in PWH.^{9,10} Factors like race/ethnicity, socioeconomic status, caregiver access, and insurance type have a significant influence on which patients receive HCT among the general population, though the contributions of these factors on use in PWH has never been studied.

Racial disparities in the use of HCT among the general population are well-documented. Black/African American patients are more likely to not receive chemotherapy, not be referred for HCT evaluation, and not receive HCT.^{11–13} At the state level among a cohort of California patients without HIV, Black patients were less likely than White patients to receive HCT for leukemia (OR 0.52; $p < 0.05$) and for lymphoma (OR 0.34; $p < 0.01$).¹⁴ At the national level, Black Medicare patients were less likely to receive an autologous HCT compared to White Medicare patients (HR 0.39; $p = 0.01$).¹²

Socioeconomic status and insurance similarly exert significant influence on HCT use among the general population.^{10,12,14,15} Among California patients with multiple

myeloma, patients with Medicaid/Medicare, those with no insurance, and those with low neighborhood socioeconomic status were significantly less likely to undergo HCT.¹⁶

Given these findings demonstrating sociodemographic barriers to HCT use in the general population and for other hematologic malignancies with HCT indications, a critical need for further research exists. Future studies must evaluate the influence of social determinants on HCT utilization in PWH, including race/ethnicity, insurance, and socioeconomic status to support the development of interventions that close the gap in access to curative cancer therapies, like HCT.

CLINICAL OUTCOMES IN HCT IN PWH

Clinical hesitancy regarding HCT use in PWH stems from the unique care needs of HIV. This includes combination antiretroviral therapy (cART) that may have drug-drug interactions resulting in metabolic toxicities like acute renal or liver complications, as well as immunosuppression resulting in opportunistic infections and sepsis. These complications overall can contribute to prolonged hospitalizations, increased hospital admissions, and poorer overall survival.¹⁷

Despite these concerns, several retrospective and prospective studies have demonstrated equivalent overall survival (OS) between HCT recipients with and without HIV. Two-year OS following HCT in PWH is approximately 75% with no significant difference compared to those without HIV,¹⁸ with an estimated one-year overall survival of HCT recipients with HIV between 83.5 - 87.3%.^{19,20} This demonstrates that PWH that are healthy enough to undergo transplant (i.e., adequate pre-transplant function and

immune status) have the potential to benefit from the life-saving intervention and quality of life afforded through HCT.

Evidence is limited in evaluating other relevant clinical concerns in PWH such as organ toxicities and infection, resulting in many providers remaining cautious about HCT use in PWH. Added clinical concerns include risk for increased infections due to immunosuppression from HIV,^{21–23} and concerns for drug-drug interactions between antiretrovirals and chemotherapy.^{19,24} Studies have demonstrated high levels of infections in HCT recipients with HIV (55% - 64.7% had at least 1 infection),^{20,25} with particular concerns for CMV in allogeneic HCTs,^{19,23,25,26} and HSV in autologous HCTs.²⁶ However, due to small samples in published studies, differences between HCT recipients with and without HIV have not been well described. Evidence to support the need for or against more protective prophylactic guidelines against opportunistic infections or organ toxicities in PLWH is limited. Larger, population-based studies are needed to clarify if HIV-specific complications actually result in clinically-meaningful outcomes in clinical practice.

GAPS IN KNOWLEDGE FOR HCT RECIPIENTS w/ HIV

Despite considerable advances in HIV management and HCT safety, there is insufficient evidence on HCT use specifically in PWH. Additionally, it is unknown which social and clinical factors influence HCT use among PWH that do receive HCT. Consequently, in the absence of definitive clinical guidelines many HCT programs have not widely adopted HCT protocols inclusive of patients with HIV. This results in the added patient burden of finding an HCT center that will treat them. While some HCT

centers may deny eligibility on the basis of HIV alone, other HCT centers take a more nuanced approach by requiring undetectable viral loads, cART adherence, and/or other indicators of well-controlled HIV. These accepting centers of PWH may be further from the patient's nearest treating center and be logistically unfeasible.

The relative rarity of HIV-related lymphoma further contributes to the evidence gap. In the U.S, HIV historically has predominantly affected men who have sex with men (MSM), a sexual minority population already limited in numbers. Therefore, using a population-based approach helps to adequately power studies for rigorous analysis. The majority of evidence evaluating HCT use in PWH is substantiated by small case series, in addition to a few prospective non-randomized controlled trials that use matched, registry-based controls. Additionally, most studies are conducted in centers with highly specialized clinical expertise in HCT, HIV, and infectious disease, such as MD Anderson, City of Hope, and Johns Hopkins.

Among a national U.S sample of approximately 39,500 HCT recipients, less than 1% of HCTs (n=108) were performed in PWH.²⁶ However, this study did not examine the role of social determinants on HCT utilization such as race/ethnicity, insurance, and socioeconomic status.²⁶ Although infection outcomes following HCT were reported in PWH,²⁶ it did not report on sepsis rates or characterize organ toxicities.^{19,24} Moreover, post-transplant hospitalization patterns in PWH, like overall length of stay and frequency of admissions, remain largely unstudied.

To address these critical gaps, this study uses a retrospective, population-based cohort in a novel data linkage to evaluate HCT utilization and outcomes among patients with HIV-related lymphoma.

SPECIFIC AIMS

Specific Aim 1: Measure the associations between sociodemographic and clinical factors on HCT utilization in patients with HIV-related lymphoma.

The goal of Specific Aim 1 is to evaluate associations between sociodemographic factors and clinical characteristics and the likelihood of receiving HCT among patients with HIV that are diagnosed with non-Hodgkin lymphoma (NHL) or Hodgkin lymphoma (HL). Elucidating the context in which patients do not receive HCT will provide targets for future intervention including policies, supportive programs, implementation studies, and controlled trials.

Specific Aim 2: Test for differences in treatment burden, hospital use, and survival outcomes between lymphoma HCT recipients with and without HIV.

The goal of Specific Aim 2 is to test for differences in clinical outcomes between lymphoma patients with HIV that received HCT compared to matched lymphoma patients without HIV that received HCT. By using a matched case-control analysis, differences in acute renal and liver complications, hospital admissions and length of stay, and overall survival specific to HIV will be elucidated, thereby supporting specific HIV-related care and updated clinical guidelines for HCT use in lymphoma patients with HIV.

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**Trends and Predictors of Hematopoietic Cell Transplant Utilization
for HIV-lymphoma in California, 1991-2016**

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CIBMTR CORE SUPPORT LIST

CIBMTR (Center for International Blood and Marrow Transplant Research®) is supported primarily by the Public Health Service U24CA076518 from the National Cancer Institute (NCI), the National Heart, Lung and Blood Institute (NHLBI), and the National Institute of Allergy and Infectious Diseases (NIAID); 75R60222C00011 from the Health Resources and Services Administration (HRSA). Additional federal support is provided by U01AI184132 from the National Institute of Allergy and Infectious Diseases (NIAID); and UG1HL174426 from the National Heart, Lung and Blood Institute (NHLBI). Support is also provided by the Medical College of Wisconsin, NMDP (National Marrow Donor Program), Gateway for Cancer Research, Pediatric Transplantation and Cellular Therapy Consortium and from the following commercial entities: AbbVie; Actinium Pharmaceuticals, Inc.; Adaptimmune LLC; Adaptive Biotechnologies Corporation; ADC Therapeutics; Adienne SA; Alexion; AlloVir, Inc.; Amgen, Inc.; Astellas Pharma US; AstraZeneca; Atara Biotherapeutics; Autolus Limited; Beam; BeiGene; BioLineRX; Blue Spark Technologies; Blueprint Medicines; Bristol Myers Squibb Co.; CareDx Inc.; CSL Behring; CytoSen Therapeutics, Inc.; DKMS; Eurofins Viracor, DBA Eurofins Transplant Diagnostics; Gamida-Cell, Ltd.; Genetix; Gift of Life Biologics; Gift of Life Marrow Registry; HistoGenetics; ImmunoFree; Incyte Corporation; Iovance; Janssen Research & Development, LLC; Janssen/Johnson & Johnson; Japan Hematopoietic Cell Transplantation Data Center; Jasper Therapeutics; Jazz Pharmaceuticals, Inc.; Karius; Kashi Clinical Laboratories; Kiadis Pharma; Kite, a Gilead Company; Kyowa Kirin International plc; Labcorp; Legend Biotech; Mallinckrodt Pharmaceuticals; Med Learning Group; Medac GmbH; Medexus; Merck & Co.; Mesoblast, Inc.; Millennium, the Takeda Oncology Co.; Miller Pharmacal Group, Inc.; Miltenyi Biotec, Inc.; MorphoSys; MSA-EDITLife; Neovii Pharmaceuticals AG; Novartis Pharmaceuticals Corporation; Omeros Corporation; Orca Biosystems, Inc.; OriGen BioMedical; Ossium Health, Inc.; Pfizer, Inc.; Pharmacyclics, LLC, An AbbVie Company; Pierre Fabre Pharmaceuticals; PPD Development, LP; Registry Partners; Rigel Pharmaceuticals; Sanofi; Sarah Cannon; Seagen Inc.; Servier; Sobi, Inc.; Sociedade Brasileira de Terapia Celular e Transplante de Medula Óssea (SBTMO); Stemcell Technologies; Stemline Technologies; STEMSOFT; Syndax; Takeda Pharmaceuticals; Talaris Therapeutics; Tscan Therapeutics; Vertex Pharmaceuticals; Vor Biopharma Inc.; Xenikos BV.

HIGHLIGHTS

1. Overall HCT utilization for HIV-lymphoma remained low despite advances in combination ART.
2. HCT utilization increased over 25 years from 1.9% to 7.2% across combination ART eras.
3. Older age, low socioeconomic status, public insurance, and being single predicted lower HCT use.
4. Persistent disparities among people with HIV may limit access to new and emerging cell therapies.

ABSTRACT

Background: Hematopoietic cell transplant (HCT) is a potentially curative treatment for relapsed or refractory lymphoma, and population-level patterns of HCT utilization among people with HIV are poorly characterized.

Objective(s): The purpose of this study is to quantify HCT utilization in people with HIV-lymphoma and examine the associations between sociodemographic factors and clinical characteristics of HCT utilization among people with HIV-lymphoma.

Study Design: We used a linkage between the Center for International Blood and Marrow Transplant Research registry, California Cancer Registry, and the California Patient Discharge Database and included adults (18-79) diagnosed with first primary lymphoma in 1991-2016 following an HIV diagnosis. Cumulative incidence of HCT use for lymphoma, accounting for the competing risk of death, was estimated overall and by combination antiretroviral therapy (ART) eras. Adjusted cox proportional hazards models evaluated sociodemographic and clinical factors associated with HCT use.

Results: Among 6,248 patients with HIV-lymphoma, the cumulative incidence of HCT use at 60 months was 4.8%. The cumulative incidence increased from 1.9% (95% confidence interval (CI): 1.0-3.5) in the pre-combination ART era (1991-1996), to 4.7% (CI: 3.5-6.1) in 1997-2005, and 7.2% (CI: 5.6-9.0) in 2006-2016 ($p < 0.0001$). Factors associated with lower HCT use included older age (Hazard Ratio [HR]=0.98, CI: 0.96-0.99), low socioeconomic status (HR=0.38, CI: 0.25-0.59), single/unmarried (HR=0.5, CI: 0.34-0.73), and public insurance (HR=0.64, CI: 0.44-0.93).

Conclusion(s): In this large, diverse population of people with HIV, HCT utilization increased after combination ART introduction, but remains low, with significant sociodemographic and clinical disparities that may limit access to current and future HCT therapies.

INTRODUCTION

Over 1.1 million people are living with human immunodeficiency virus (HIV) in the United States.¹ Given significant advancements in combination anti-retroviral therapy (ART) beginning in 1996,^{2,3} there is an unprecedented and growing population of aging people with HIV at risk for cancer.^{4–10} People with HIV suffer from an 11.5 times greater risk for developing non-Hodgkin lymphoma, and 7.7 times greater risk of developing Hodgkin lymphoma.⁸ According to National Comprehensive Cancer Network clinical practice guidelines, first-line therapy for lymphomas includes chemotherapy/immunotherapy with or without radiation for bulky disease.^{11,12} In the event of relapsed or refractory lymphomas following first-line therapy, patients may be treated using hematopoietic cell transplant (HCT) if eligible.^{12,13} Despite the increased risk of lymphoma among people with HIV,⁸ population-based studies have shown people with HIV are significantly less likely to receive cancer treatment, including chemotherapy or radiation therapy.^{14–16} While HCT is a potentially curative treatment option for lymphomas in the non-HIV population,¹² the likelihood of receiving an HCT in people with HIV is unknown.

A variety of factors associated with HCT utilization in non-HIV populations are known.^{17,18} Barriers to HCT include poor underlying health (e.g., comorbidities, organ dysfunction, lower functional status), older age (historically), psychosocial factors (e.g. lack of caregiver support or substance abuse), lack of insurance, lower socioeconomic status, late-stage disease, and lack of matched donors when an allogeneic HCT is indicated.^{17–19} Much less is known about factors associated with HCT utilization in the HIV population and the cumulative incidence of HCT use in people with HIV-lymphoma is unknown.^{20,21} Additionally, no studies have examined the specific social and clinical factors that influence HCT utilization in people with HIV, who often suffer from unique HIV-related burdens that existed prior to their cancer diagnosis,^{22–24} such as HIV-related stigma,^{25,26} daily adherence to combination ART,^{2,27} and engagement in HIV care including routine laboratory blood testing for HIV viral load and

kidney function.^{27,28} While one population-based U.S. study evaluated HCTs in people with HIV,²⁹ it did not examine or adjust for sociodemographic and clinical factors on HCT utilization. Given the paucity of data on HCT utilization in a growing population of aging, at-risk people with HIV, a critical need exists to characterize the social factors and clinical characteristics that influence HCT utilization to increase HCT access.

To address this gap, our study uses a novel data linkage between the California Cancer Registry (CCR), the Center for International Blood and Marrow Transplant Research® (CIBMTR), and the California Patient Discharge Database (PDD).³⁰ The purpose of this study is to quantify HCT utilization in people with HIV-lymphoma and examine the associations between sociodemographic factors and clinical characteristics of HCT utilization among people with HIV-lymphoma.

METHODS

Database and Cohort

This study used a retrospective, population-based cohort from a data linkage between the CCR, CIBMTR, and hospitalization data from the California PDD, as described previously.³⁰ The CCR is California's population-based cancer surveillance system, collecting cancer incidence on >99% of new cancer diagnoses since 1988. CIBMTR is a research collaboration between the Medical College of Wisconsin and the National Marrow Donor Program (NMDP). It comprises a voluntary working group of more than 310 participating centers worldwide that contribute detailed data on cellular and gene therapies.^{31,32} Since 1991, the California Department of Health Care Access and Information has mandated reporting of diagnostic and procedure codes on all inpatient hospitalization admissions in California from nonfederal hospitals across the state through the PDD.

The analysis included patients in the CCR who were diagnosed with a first primary lymphoma during 1991-2016, had HIV prior to lymphoma diagnosis, and were between the ages

of 18 and 79 at lymphoma diagnosis, given the rarity of HCT in patients >80 years. Patients were excluded if they did not have a valid social security number for linking to the PDD; follow-up date was missing or survival time was missing or zero.

HIV and HCT

The primary exposure in this cohort was HIV status and categorized as yes if HIV diagnosis occurred prior to lymphoma diagnosis date in the CCR or PDD. In the PDD, HIV diagnosis and associated date was ascertained using HIV ICD-9/ICD-10 codes in the primary or other diagnosis position. The primary outcome was HCT utilization and was captured from all three data sources independently. Considering HCT data (ex: HCT type/date) were available in multiple sources, we prioritized information from the CIBMTR where available, then used International Classification of Diseases (ICD) ICD-9/ICD-10 codes from the PDD, and lastly, reported treatment fields for the initial course of treatment from CCR. HCT use was receiving any transplant type for first primary lymphoma and further categorized as either autologous or allogeneic HCT. We further excluded 10 patients with an unknown HCT date or invalid survival time (e.g. <0) (Figure 1).

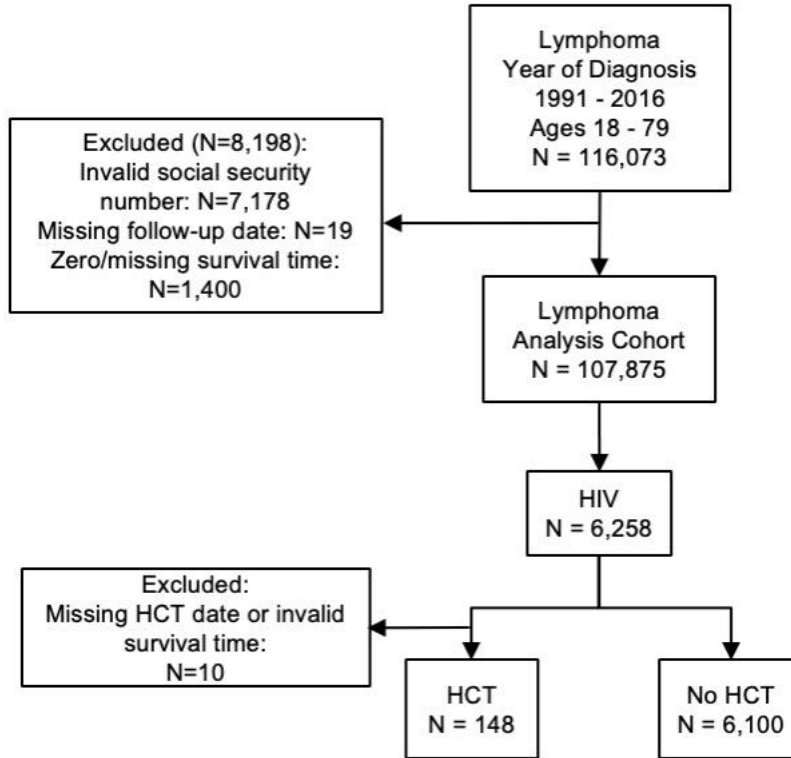


Figure 1: Cohort Selection Flow Diagram

Sociodemographic and Clinical Factors

Sociodemographic data (age, sex, race/ethnicity, marital status, neighborhood socioeconomic status, insurance) were obtained from the CCR. Age was measured continuously in years. Sex was categorized as male, female, and transgender.³³ Race/ethnicity was classified as non-Hispanic White (NHW), non-Hispanic Black/African American, Hispanic, Asian, Pacific Islander, and other/unknown. Marital status was categorized as married/partnered or not-married/partnered. Neighborhood socioeconomic status (nSES) was a composite measure comprising census and American Community Survey education, occupation, unemployment, household income, poverty, rent, and house values at the block group level, and grouped into quintiles based on the distribution of SES across all census block groups in California. For this study, nSES was divided into low (1st or 2nd quintiles) and high (3rd, 4th, or

5th quintiles), as done previously.³⁴ Type of health insurance at cancer diagnosis or initial treatment, which is available from 1996 forward in the CCR, was categorized as private, public/no insurance, and unknown/not available.

Clinical characteristics (cancer stage at diagnosis, lymphoma subtype, cancer diagnosis year, prior cancer treatment) were also obtained from the CCR. Cancer stage at diagnosis was categorized as localized, advanced, or unknown. Lymphoma subtypes were grouped as Hodgkin lymphoma (HL), diffuse large B-cell lymphoma (DLBCL), other non-Hodgkin lymphoma (NHL), and lymphoma (not otherwise specified). Cancer diagnosis year was categorized by applying cut points reflecting major advancements in HIV combination ART pharmacology – 1991-1996 (pre-combination ART), 1997-2005 (early combination ART), and 2006-2016 (late combination ART).²⁷ Receipt of any initial treatment for lymphoma prior to HCT included chemotherapy, immunotherapy, and/or radiation therapy.

Statistical Analysis

Descriptive statistics were used to summarize sociodemographic and clinical characteristics of lymphoma patients with HIV overall and among the HCT vs. no HCT groups. Differences between HCT and no HCT groups were examined using independent t-tests for continuous variables and chi-square for categorical variables. Dependent t-tests for continuous variables were used among the HCT group. The cumulative incidence and 95% confidence interval (CI) of HCT utilization were determined from initial lymphoma diagnosis until date of HCT, death date, last known date of contact, or study cutoff of 12/31/18, the end of available follow-up in the linked dataset, whichever occurred first. Cumulative incidence accounted for the competing risk of death.

Multivariable Cox proportional hazards regression models were used to evaluate sociodemographic and clinical characteristics associated with HCT utilization, using Fine and Gray's methods to account for the competing risk of death.³⁵ Proportional hazards assumptions

for all Cox models were evaluated using the Schoenfeld Residuals Test;³⁶ lymphoma subtype violated proportional hazards and was included as a stratification variable in the model.

All p-values were two-sided with significance set a priori to $p < 0.05$. All analyses were performed using SAS 9.4 (SAS, Cary, NC). This study was approved by the Institutional Review Board (IRB) of the University of California, Davis, the California Committee for the protection of human subjects, and the NMDP IRB (CIBMTR data).

RESULTS

Baseline Sociodemographic and Clinical Characteristics

Among all patients with HIV-lymphoma (N=6248), the mean age was 44 years (SD=10.8), and most were male (91.1%). There were no significant differences in age and sex between those that did and did not receive an HCT (Table 1). The majority of patients with HIV-lymphoma were NHW (57.2%). Non-Hispanic Black patients represented a greater proportion of those that did not receive a HCT (14.3%) vs. those who did (8.1%), while Asian patients represented a greater proportion of those that received HCT (7.4%) vs. those who did not (2.6%). There were more patients with high nSES among those that received HCT (77.0%) compared to those that did not (60.6%). A greater proportion of HCT recipients were married/partnered (29.1% HCT vs. 15.6% no HCT) and had private insurance (64.9% vs. 34% no HCT).

Overall, the most common subtype was DLBCL (46.6%), followed by other NHL (24.1%), other lymphomas (16.9%), and lastly HL (12.5%). Among those that received an HCT, the majority were diagnosed with DLBCL (60.1%) and HL (16.9%), compared to DLBCL (46.3%) and NHL, NOS (24.4%) among those that did not receive a transplant. The majority had advanced stage disease at diagnosis (65.1%). There were significant differences in stage at diagnosis between those that did and did not receive an HCT ($p < 0.001$), including advanced stage (82.4% HCT vs 64.7% no HCT). Most patients were diagnosed with lymphoma 1991-1996

(40.1%), followed by 1997-2005 (34.8%), and 2006-2016 (25.1%). Among those that received an HCT, cancer diagnoses increased over time ($p<0.001$): 1991-1996 (10.15% HCT vs 40.8% no HCT), 1997-2005 (41.2% HCT vs 34.7% no HCT), and 2006-2016 (48.6% HCT vs 24.5% no HCT). The majority of patients received initial lymphoma treatment with chemotherapy, immunotherapy, or radiotherapy overall (73.6%), and was higher among those who received an HCT (>95% HCT vs 73% no HCT).

Table 1. Sociodemographic and Clinical Characteristics of Patients with HIV-Related Lymphoma, by Hematopoietic Cell Transplantation (HCT), California, 1991-2016

Characteristics	All (N=6248)	No HCT (N=6100)	HCT (N=148)	P-value
Age				0.609
Mean $\pm$ SD	44.0 $\pm$ 10.8	44.0 $\pm$ 10.9	44.4 $\pm$ 9.9	
Range	18.0-79.0	18.0-79.0	20.0-69.0	
Median (IQR)	43.0 (36.0-50.0)	43.0 (36.0-50.0)	45.0 (38.0- 51.0)	
Sex				0.766
Male	5692 (91.1)	5558 (91.1)	134 (90.5)	
Female	535 (8.6)	522 (8.6)	13 (8.8)	
Transgender	21 (0.3)	< 21 (0.3)	< 5	
Race/ethnicity				0.002
Non-Hispanic White	3573 (57.2)	3489 (57.2)	84 (56.8)	
Non-Hispanic Black	887 (14.2)	875 (14.3)	12 (8.1)	
Hispanic	1558 (24.9)	1518 (24.9)	40 (27.0)	
Asian	170 (2.7)	159 (2.6)	11 (7.4)	
Pacific Islander/Unknown	60 (1.0)	< 60	< 5	
Neighborhood SES				<.001
Low	2233 (35.7)	2207 (36.2)	26 (17.6)	
High	3812 (61.0)	3698 (60.6)	114 (77.0)	
Unknown	203 (3.2)	195 (3.2)	8 (5.4)	
Marital status				<.001
Single/Not Married or Partnered	5256 (84.1)	5151 (84.4)	105 (70.9)	
Married/Partnered	992 (15.9)	949 (15.6)	43 (29.1)	

Health Insurance				<.001
Private	2168 (34.7)	2072 (34.0)	96 (64.9)	
Public/No Insurance	1814 (29.0)	1772 (29.0)	42 (28.4)	
Unknown/NA < 1996	2266 (36.3)	2256 (37.0)	10 (6.8)	
Lymphoma subtype				<.001
HL	780 (12.5)	755 (12.4)	25 (16.9)	
DLBCL	2912 (46.6)	2823 (46.3)	89 (60.1)	
NHL, NOS	1503 (24.1)	1487 (24.4)	16 (10.8)	
Lymphoma, Other	1053 (16.9)	1035 (17.0)	18 (12.2)	
Stage at diagnosis				<.001
Local	1745 (27.9)	1725 (28.3)	20 (13.5)	
Advanced	4066 (65.1)	3944 (64.7)	122 (82.4)	
Unknown	437 (7.0)	431 (7.1)	6 (4.1)	
Cancer Diagnosis Era				<.001
1991-1996	2504 (40.1)	2489 (40.8)	15 (10.1)	
1997-2005	2176 (34.8)	2115 (34.7)	61 (41.2)	
2006-2016	1568 (25.1)	1496 (24.5)	72 (48.6)	
Prior Cancer Therapy				<.001
Yes	4599 (73.6)	4455 (73.0)	> 95%	
No/Unknown	1649 (26.4)	1645 (27.0)	< 5	
Abbreviations: SES=socioeconomic status; HL=Hodgkin lymphoma; DLBCL=Diffuse large B-cell lymphoma; NHL=non-Hodgkin lymphoma; IQR = interquartile range; NOS =Not otherwise specified; NA < 1996=Information not available prior to 1996.				

HCT Cumulative Incidence By Cancer Diagnosis Era

The overall cumulative incidence of HCT at 60 months was 4.8%. When stratified by treatment era, the cumulative incidence of HCT at 60 months increased over time, from 1.9% (95% CI: 1.0-3.5) in the pre-combination ART era (1991-1996), to 4.7% (95% CI: 3.5-6.1) in the early post-combination ART era (1997-2005), and 7.2% (95% CI: 5.6 - 9.0) in the late post-combination ART era (2006-2016) [Figure 2; Table 2].

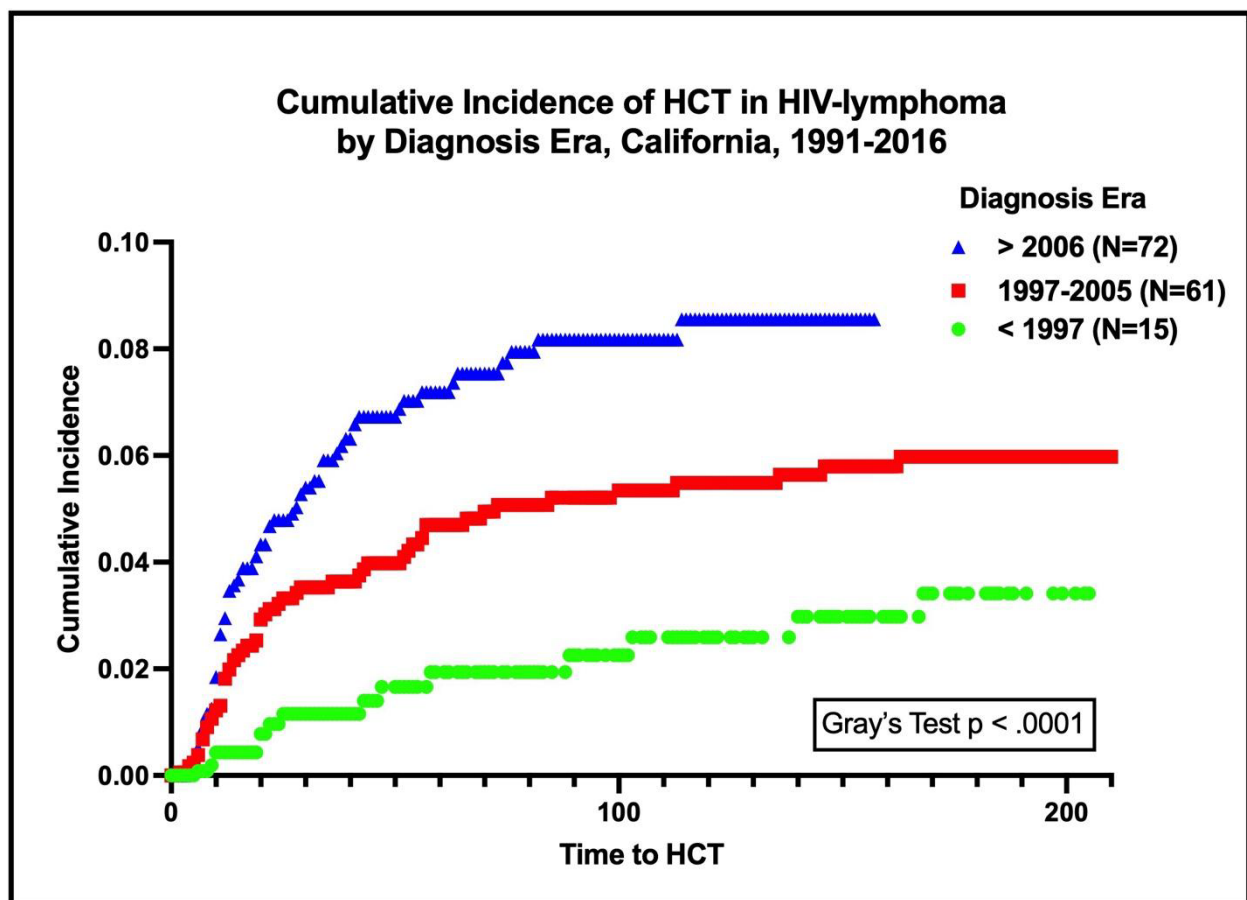


Figure 2

Table 2. Five-year Cumulative Incidence of Hematopoietic Cell Transplantation at 60 Months in HIV-Lymphoma, by Diagnosis Era, California, 1991-2016

Diagnosis Era	Years	N	60-Month Cumulative Incidence, % (95% CI)
Overall	1991–2016	148	4.8% (4.0%–5.7%)
Pre-combination ART	1991-1996	15	1.9% (1.0% - 3.5%)
Early post-combination ART	1997–2005	61	4.7% (3.5% – 6.1%)
Late post-combination ART	2006-2016	72	7.2% (5.6% - 9.0%)
Abbreviations: combination ART=combination antiretroviral therapy			

Factors Associated with HCT Utilization

In multivariable models, increasing age at diagnosis was associated with decreased utilization of HCTs (HR=0.98; 95% CI: 0.96-0.99) in people with HIV-lymphoma (Figure 3; Table 3). Asian patients were over twice as likely than NHW patients to receive an HCT (HR=2.09, 95% CI 1.1-3.97). Compared to patients with high nSES, patients with low nSES were less likely to receive HCT (HR=0.38, 95% CI 0.25-0.59). Patients that were not-married/partnered were less likely to receive HCT (HR=0.50, 95% CI 0.34-0.73) than those who were married/partnered. Patients with public/no (vs. private) insurance (HR=0.64, 95% CI 0.44-0.93) or unknown/not available insurance (HR=0.25, 95% CI 0.11-0.59) were less likely to receive HCT.

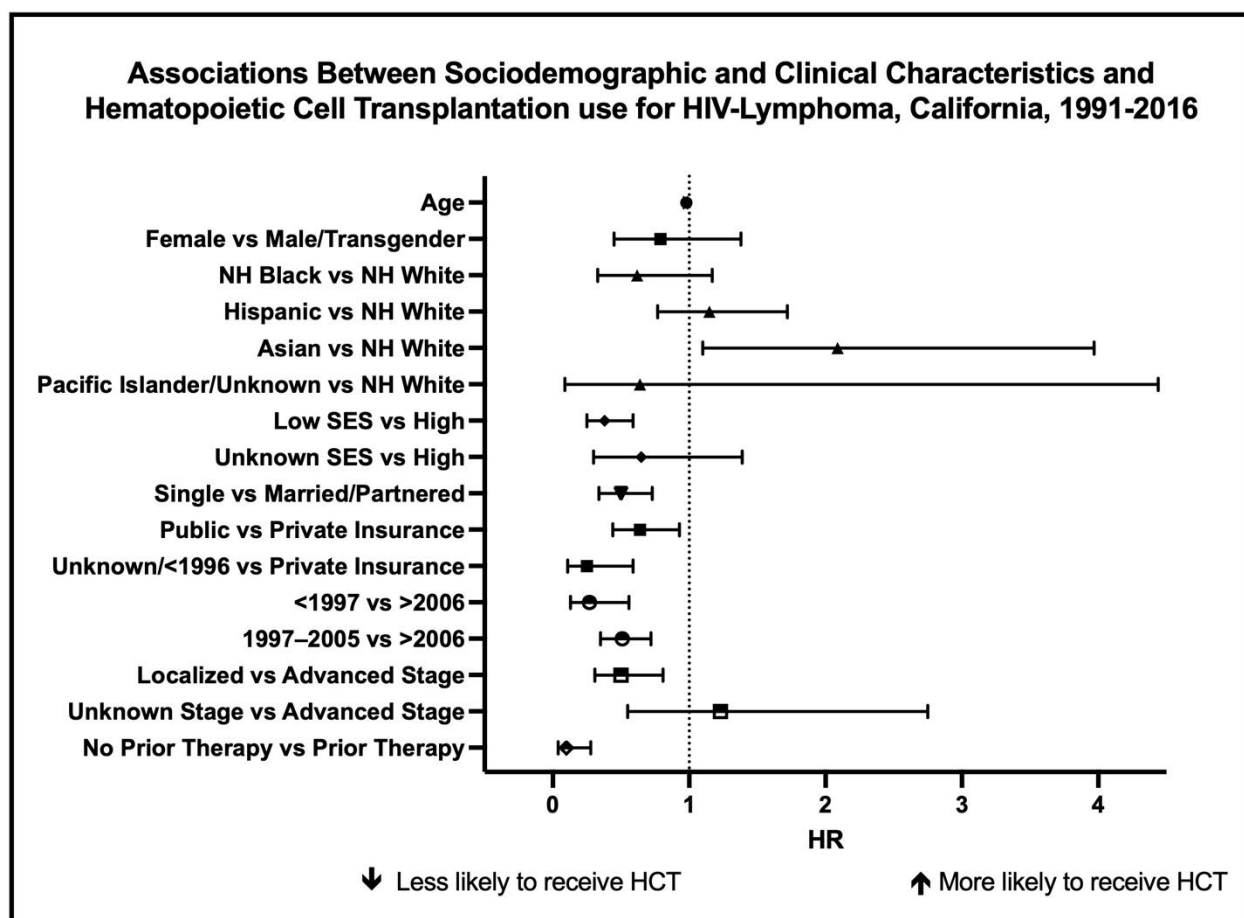


Figure 3

Table 3. Associations Between Sociodemographic and Clinical Characteristics and Hematopoietic Cell Transplantation use for HIV-Lymphoma, California, 1991-2016

Characteristics	HR	CI 95%		P-value
Age	0.98	0.96	0.99	0.0043
Sex				
Male/Transgender	Reference			
Female	0.79	0.45	1.38	0.4095
Race/ethnicity				
Non-Hispanic White	Reference			
Non-Hispanic Black/African American	0.62	0.33	1.17	0.1428
Hispanic	1.15	0.77	1.72	0.504
Asian	2.09	1.1	3.97	0.0254

Pacific Islander/Unknown	0.64	0.09	4.44	0.65
Neighborhood SES				
High	Reference			
Low	0.38	0.25	0.59	<.0001
Unknown	0.65	0.3	1.39	0.2655
Marital Status				
Married/Partnered	Reference			
Single	0.5	0.34	0.73	0.0003
Health Insurance				
Private	Reference			
Public	0.64	0.44	0.93	0.019
Unknown/ <1996	0.25	0.11	0.59	0.0015
Cancer Diagnosis Era				
2006-2016	Reference			
1997-2005	0.51	0.35	0.72	0.0005
1991-1996	0.27	0.13	0.56	0.0002
Stage				
Advanced	Reference			
Localized	0.5	0.31	0.81	0.0043
Unknown	1.23	0.55	2.75	0.619
Prior Cancer Therapy				
Yes	Reference			
No	0.1	0.04	0.28	<.0001

Final adjusted model included age, sex, race/ethnicity, nSES, marital status, insurance, cancer diagnosis era, stage, and prior cancer therapy. Lymphoma subtype violated the proportional hazards assumption and was included as a stratification variable in the model. Male and transgender participants were grouped together based on similar human immunodeficiency virus (HIV) transmission mechanisms, consistent with established HIV literature.

Abbreviations: SES=socioeconomic status

Compared to patients diagnosed in the late combination ART era (2006-2016), patients diagnosed in the pre-combination ART era (1991-1996; HR=0.27 95% CI: 0.13-0.56) and in the early combination ART era (1997-2005; HR=0.51 95% CI: 0.35-0.72) were less likely to receive HCT. Patients that did not receive any prior cancer therapy were less likely to receive HCT (HR=0.1; 95% CI: 0.04-0.28) than those who received therapy.

DISCUSSION

In one of the largest population-based studies of individuals with HIV-related lymphoma who received HCT, we demonstrate increased HCT utilization in California since market entry of combination antiretroviral therapy in 1996. Additionally, we found HCT utilization for HIV-related lymphoma in California was greater than national estimates for overall lymphoma (0.98% - 1.1%), though disparities in HCT use exist within the diverse populations of people with HIV in California.^{37,38} Patients with HIV of older age (in an already aging HIV population), low nSES, and single were significantly less likely to receive HCT. Our findings contribute to the growing body of evidence demonstrating barriers to cancer treatment access in people with HIV.^{14,15,39,40}

We observed a significant increase in the utilization of HCT across cancer diagnosis eras, mirroring the growing body of HIV evidence over time, suggesting the safety and feasibility of HCT in people with HIV and the increase in clinician and organizational willingness to transplant people with HIV.^{20,21,41,42} Despite increased utilization of HCT in people with HIV over time, the overall low proportion of patients with HIV who receive HCT suggests other persistent barriers to HCT may exist despite advances in combination ART since 1996.

Patients with no prior cancer therapy were significantly less likely to receive HCT. This is important, as people with HIV are less likely to receive any first-line cancer therapy.^{14,15} These findings highlight barriers to HIV-lymphoma treatment across the cancer therapy continuum, including first-line therapy at initial diagnosis and subsequent therapies, like HCT, for relapsed/refractory disease.

We observed that Asian patients with HIV were more likely than NHW patients to receive an HCT, although prior literature evaluating race/ethnicity among the general HCT population found Hispanic and non-Hispanic Black patients were less likely to receive HCT after controlling for socioeconomic status.³⁸ This may reflect the sample limitations of our cohort, in addition to the racial and ethnic diversity of people with HIV in California, and state-level influences from regional HIV advocacy in the San Francisco Bay Area and Los Angeles metropolitan areas.⁴³ Our model controls for nSES; prior multivariable models evaluating HCT use in the general population have demonstrated that race/ethnicity (as a proxy to systemic racism) lost statistical significance when controlling for socioeconomic status,³⁸ highlighting methodological challenges in quantitative health equity and social epidemiology. The observation that individuals with low nSES and public insurance are less likely to receive HCT highlight the financial challenges of accessing specialized care like HCT when clinically indicated.

In addition to the barriers caused by financial resources, social and caregiver support among people with HIV lymphoma must be evaluated during HCT eligibility.¹⁹ Our study showed that people with HIV lymphoma that were single or unpartnered were significantly less likely to receive HCT. While 67% of people with HIV identify as a sexual or gender minority,¹ many of these patients may rely more on close friends or paid caregivers for caregiver support during the HCT process when single/unpartnered due to estranged familial relationships from “coming out”.^{44,45} Consistent with prior literature among the general HCT population,^{46,47} advanced age was associated with lesser HCT utilization among people with HIV lymphoma in California. A dose-reduced or non-myeloablative chemotherapy regimen may be appropriate for patients with advanced age in general,^{48,49} with added consideration on the appropriate level of caregiver support when spousal support may not be available.

HCT centers and transplant leadership teams not already familiar with the HCT care of people with HIV should discuss their regional prevalence of HIV and how this translates to inclusivity of people with HIV within their transplant programs, in particular for autologous HCT

protocols for relapsed/refractory diffuse large B-cell lymphoma. If patients with HIV are excluded from institutional protocols, HCT teams should question why. HIV-related stigma may present at individual and organizational levels in both low and high HIV prevalence settings— this may present in interpersonal interactions with people with HIV (referred elsewhere by an individual clinician) or codified in organizational HCT protocols that systemically exclude people with HIV for unsubstantiated reasons.^{24–26}

This study has several limitations. First, the retrospective, observational design precluded analysis of several patient-level clinical factors that may influence HCT eligibility and utilization. Although increasing age was independently associated with lower HCT utilization, our data did not include measures of comorbidity burden, polypharmacy, frailty, or functional status. Therefore, we could not determine whether lower HCT utilization with increasing age reflected chronological age itself or age-associated clinical factors that may influence HCT eligibility. Similarly, individual-level HIV clinical characteristics, including ART use and adherence, HIV viral load, CD4 count, and virologic suppression, were not available; consequently, we could not evaluate whether HIV disease control influenced HCT eligibility or utilization.

Second, we were unable to assess several provider-, social-, and health system-level factors that may contribute to HCT access. These included provider referral patterns and perceptions of HIV prognosis or transplant eligibility, geographic access to centers with expertise in HIV-associated malignancies and transplantation, and health system factors such as patient navigation and care coordination. Fragmentation between HIV, oncology, and transplant services may create additional barriers to HCT referral and access. We also could not directly measure caregiver involvement and used marital/partnered status as a proxy for caregiver access. Because HCT requires substantial caregiver support and longitudinal engagement with care, marital status may not fully capture the availability of social support, and we may therefore underestimate caregiver access.

Third, the relatively small number of patients who received HCT limited additional stratified analyses of sociodemographic and clinical characteristics by diagnosis era, including further characterization of trends within the most recent era. In addition, substantial advances in HIV, lymphoma, and HCT care occurred over the 25-year study period beyond the introduction of combination ART and may have contributed to increasing HCT utilization. As these and other unmeasured factors could not be included in our multivariable models, residual confounding is possible.

Finally, evolving treatment paradigms for relapsed or refractory lymphoma, including CAR T-cell therapy and bispecific T-cell engagers, may alter the role of and referral patterns for HCT. Future studies should evaluate whether advances in cellular and immune-based therapies reduce or exacerbate disparities in treatment access among people with HIV. Despite these limitations, our population-based findings spanning 25 years demonstrate persistent sociodemographic and clinical differences in HCT utilization among people with HIV-lymphoma and identify important factors for future investigation.

In summary, this study demonstrates increasing, but overall low utilization of HCT among people with HIV-lymphoma across combination ART eras, reflecting broader advancements in HIV research and an evolving understanding of HCT clinical practices for this population. However, significant sociodemographic barriers to HCT persist, and may continue to hinder access to people with HIV-lymphoma as new treatments become standard of care, like chimeric antigen receptor t-cell therapy (CAR-T) and bispecific T-cell engagers. Future efforts should focus on evaluating additional factors not able to be measured in this study, including individual and organizational HIV-related stigma and HIV clinical factors (CD4, viral load, combination ART adherence), and the effect of sociodemographic and clinical factors on the next era of cellular therapies (CAR-T and bispecific T-cell engagers).

ACKNOWLEDGMENTS

This work was supported by the National Institute of Nursing Research of the National Institutes of Health under Award Number F31NR021235. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health. The collection of cancer incidence data used in this study was supported by the California Department of Public Health pursuant to California Health and Safety Code Section 103885; Centers for Disease Control and Prevention's (CDC) National Program of Cancer Registries, under cooperative agreement 5NU58DP007156; the National Cancer Institute's SEER under contract HHSN261201800032I awarded to the University of California, San Francisco, contract HHSN261201800015I awarded to the University of Southern California, and contract HHSN261201800009I awarded to the Public Health Institute. The ideas and opinions expressed herein are those of the author(s) and do not necessarily reflect the opinions of the State of California, Department of Public Health, the National Cancer Institute, and the Centers for Disease Control and Prevention or their Contractors and Subcontractors. This research was supported in part by the Intramural Research Program of the National Institutes of Health (NIH). The contributions of the NIH author(s) are considered Works of the United States Government. The findings and conclusions presented in this paper are those of the author(s) and do not necessarily reflect the views of the NIH or the U.S. Department of Health and Human Services.

AUTHOR CONTRIBUTIONS

Conceptualization and Study Design: KD, AJF, SC, RZ, LM, LMM, SLC, THMK; **Data Acquisition / Data Curation:** KD, AB, LM, LMM, THMK; **Methodology and Statistical Analysis:** KD, AB, AJF, SJS, SC, CM, JJA, LM, LMM, SLC, THMK; **Interpretation of Data:** KD, AB, AJF, SJS, SC, RZ, LF, CM, JJA, LM, LMM, SLC, THMK; **Drafting of the Manuscript:** KD, AB, AJF, SJS, SC, RZ, LF, CM, JJA, LM, LMM, SLC, THMK; **Critical Revision for Important Intellectual Content:** KD, AB, AJF, SJS, SC, RZ, LF, CM, JJA, LM, LMM, SLC, THMK; **Supervision:** AB, AJF, LM, LMM; **Funding Acquisition:** KD, AJF, SC, RZ, LF, LM, LMM, SLC, THMK; **Final Approval of the Manuscript:** All authors.

CONFLICT OF INTEREST DISCLOSURES

The authors 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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**Differences in Hematopoietic Cell Transplantation Outcomes by HIV status,
California, 1991-2016**

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CIBMTR CORE SUPPORT LIST

CIBMTR is supported primarily by the Public Health Service U24CA076518 from the National Cancer Institute (NCI), the National Heart, Lung and Blood Institute (NHLBI), and the National Institute of Allergy and Infectious Diseases (NIAID); 75R60222C00011 from the Health Resources and Services Administration (HRSA). Additional federal support is provided by U01AI184132 from the National Institute of Allergy and Infectious Diseases (NIAID); and UG1HL174426 from the National Heart, Lung and Blood Institute (NHLBI). Support is also provided by the Medical College of Wisconsin, NMDP, Gateway for Cancer Research, Pediatric Transplantation and Cellular Therapy Consortium and from the following commercial entities: AbbVie; Actinium Pharmaceuticals, Inc.; Adaptimmune LLC; Adaptive Biotechnologies Corporation; ADC Therapeutics; Adienne SA; Alexion; AlloVir, Inc.; Amgen, Inc.; Astellas Pharma US; AstraZeneca; Atara Biotherapeutics; Autolus Limited; Beam; BeiGene; BioLineRX; Blue Spark Technologies; Blueprint Medicines; Bristol Myers Squibb Co.; CareDx Inc.; CSL Behring; CytoSen Therapeutics, Inc.; DKMS; Eurofins Viracor, DBA Eurofins Transplant Diagnostics; Gamida-Cell, Ltd.; Genetix; Gift of Life Biologics; Gift of Life Marrow Registry; HistoGenetics; ImmunoFree; Incyte Corporation; Iovance; Janssen Research & Development, LLC; Janssen/Johnson & Johnson; Japan Hematopoietic Cell Transplantation Data Center; Jasper Therapeutics; Jazz Pharmaceuticals, Inc.; Karius; Kashi Clinical Laboratories; Kiadis Pharma; Kite, a Gilead Company; Kyowa Kirin International plc; Labcorp; Legend Biotech; Mallinckrodt Pharmaceuticals; Med Learning Group; Medac GmbH; Medexus; Merck & Co.; Mesoblast, Inc.; Millennium, the Takeda Oncology Co.; Miller Pharmacal Group, Inc.; Miltenyi Biotec, Inc.; MorphoSys; MSA-EDITLife; Neovii Pharmaceuticals AG; Novartis Pharmaceuticals Corporation; Omeros Corporation; Orca Biosystems, Inc.; OriGen BioMedical; Ossium Health, Inc.; Pfizer, Inc.; Pharmacyclics, LLC, An AbbVie Company; Pierre Fabre Pharmaceuticals; PPD Development, LP; Registry Partners; Rigel Pharmaceuticals; Sanofi; Sarah Cannon; Seagen Inc.; Servier; Sobi, Inc.; Sociedade Brasileira de Terapia Celular e Transplante de Medula Óssea (SBTMO); Stemcell Technologies; Stemline Technologies; STEMSOFT; Syndax; Takeda Pharmaceuticals; Talaris Therapeutics; Tscan Therapeutics; Vertex Pharmaceuticals; Vor Biopharma Inc.; Xenikos BV.

Highlights

- We compared post-HCT outcomes in matched cohorts, including 140 lymphoma patients with HIV and 409 lymphoma patients without HIV.
- HIV status was not associated with increased risk of acute renal or liver complications, infection, or sepsis within 90 days after HCT.
- HCT recipients with HIV had similar risk of hospital admission and length of stay compared to matched HCT recipients without HIV.
- Overall survival was comparable by HIV status at 90 days, 1 year, and 2 years following HCT.
- Our findings support safe HCT use for otherwise eligible patients with HIV.

ABSTRACT

Hematopoietic cell transplantation (HCT) can be curative for lymphoma, yet population-level HCT outcomes by HIV status are not well studied, contributing to variable access to HCT for people with HIV. In this population-based study, we used data from a linkage between California Cancer Registry, Center for International Blood and Marrow Transplant Research, and the California Patient Discharge Database to identify HCT recipients between 18-79 with first primary lymphoma between 1991-2016. HIV and non-HIV cohorts matched 1:3 on age, sex, lymphoma subtype, stage at diagnosis, HCT type, year of transplant ± 3 years, and time from diagnosis to transplant ± 2 years resulted in 140 HIV and 409 non-HIV (N=549) patients. Clinical outcomes (acute renal and liver complications), hospital utilization (number of admissions and total length of stay), and overall survival were compared between the matched cohorts using adjusted cox proportional hazards regression models. Between HIV and non-HIV cohorts, there were no significant differences in 90-day acute complications or hospital use (acute renal complications hazard ratio (HR)=1.05, 95% confidence interval (CI): 0.66–1.69; acute liver complications HR=0.69, CI: 0.09–5.04; sepsis HR=1.15, CI: 0.48–2.79; infection HR=0.92, CI: 0.56–1.52; any admission HR=0.92, CI: 0.71–1.20). Overall survival at 90 days (94% HIV vs 93% non-HIV), 1 year (78% HIV vs 73% non-HIV) and 2 years (73% HIV and 69% non-HIV) following HCT was similar between groups ($p=0.33$). Our findings suggest that HIV status does not confer higher risk of acute complications, greater risk of hospital utilization, or lower overall survival, contributing to the evidence that HCT can be safely and equitably used in people with HIV that are otherwise eligible.

INTRODUCTION

Patients with human immunodeficiency virus (HIV) suffer from an 11.5 times greater risk for non-Hodgkin lymphoma (NHL) and 7.7 times greater risk of developing Hodgkin lymphoma (HL), yet the impact of HIV on general cancer treatment remains poorly understood.^{1–4} While hematopoietic cell transplant (HCT) has become an increasingly viable and curative treatment modality for lymphoma patients, it is unknown if people with HIV require different cancer care standards based on comorbid HIV treatment, like combination antiretroviral therapy (cART). According to the National Comprehensive Cancer Network clinical practice guidelines, clinicians specialized in infectious disease and the co-management of chemotherapy and combination anti-retroviral for HIV should be consulted to mitigate the risk of adverse outcomes during HCT.^{5–7} Specific HCT concerns in patients with HIV include drug-drug interactions between cART and intensive chemotherapy regimens,^{8,9} and underlying immunosuppression from HIV,^{10,11} potentially resulting in organ toxicities, sepsis, greater hospital use, and increased mortality.

Existing evidence, including a recent meta-analysis of autologous HCT for HIV-lymphoma,¹² supports the feasibility and safety of HCT in people with HIV-lymphoma, though much of the literature has focused on overall survival.^{13–18} Evidence characterizing other post-HCT clinical outcomes remains limited, hindering the ability to meaningfully discern if adapted care standards for HCT in patients with HIV are warranted.^{5,19,20} As a result, HIV-related complications and institutional expertise may hinder transplant eligibility and use, for reasons such as treatment-related toxicities, opportunistic infections, drug-drug interactions, and the need for specialized HIV management.^{5,6,12} Among the existing evidence evaluating HCT outcomes in patients with HIV, many studies are limited by small sample sizes or single-center studies in highly specialized HCT centers with immunocompromised infectious disease teams,^{6,8,13,21–23} thus not capturing HCT approaches for people with HIV at the population-level, particularly in diverse state-level populations such as California.

To address this gap, our study used a novel data linkage between the California Cancer Registry (CCR), the Center for International Blood and Marrow Transplant Research (CIBMTR), and the California Patient Discharge Database (PDD),²⁴ to identify a cohort of HIV lymphoma patients that received an HCT and compare them to a matched cohort of lymphoma patients without known HIV that received an HCT. The purpose of this study is to evaluate if there are differences in clinical outcomes, hospital utilization, and overall survival following HCT between the cohorts. While prior HIV and HCT literature may allude to greater risk, a better understanding of this can inform the need for specialized cancer care based on HIV status.

METHODS

Database

This study used a population-based, retrospective cohort from a data linkage between the CIBMTR, CCR, and hospitalization data from the California PDD, previously described elsewhere.²⁴ The CCR is California's population-based cancer surveillance system, collecting cancer incidence on >99% of new cancer diagnoses since 1988. CIBMTR® (Center for International Blood and Marrow Transplant Research®) is a research collaboration between the Medical College of Wisconsin and NMDP. It comprises a voluntary working group of more than 310 participating centers worldwide that contribute detailed data on cellular and gene therapies. Since 1991, the California Department of Health Care Access and Information has mandated reporting of procedure and diagnostic codes on all inpatient hospitalization admissions in California from nonfederal hospitals across the state through the PDD.

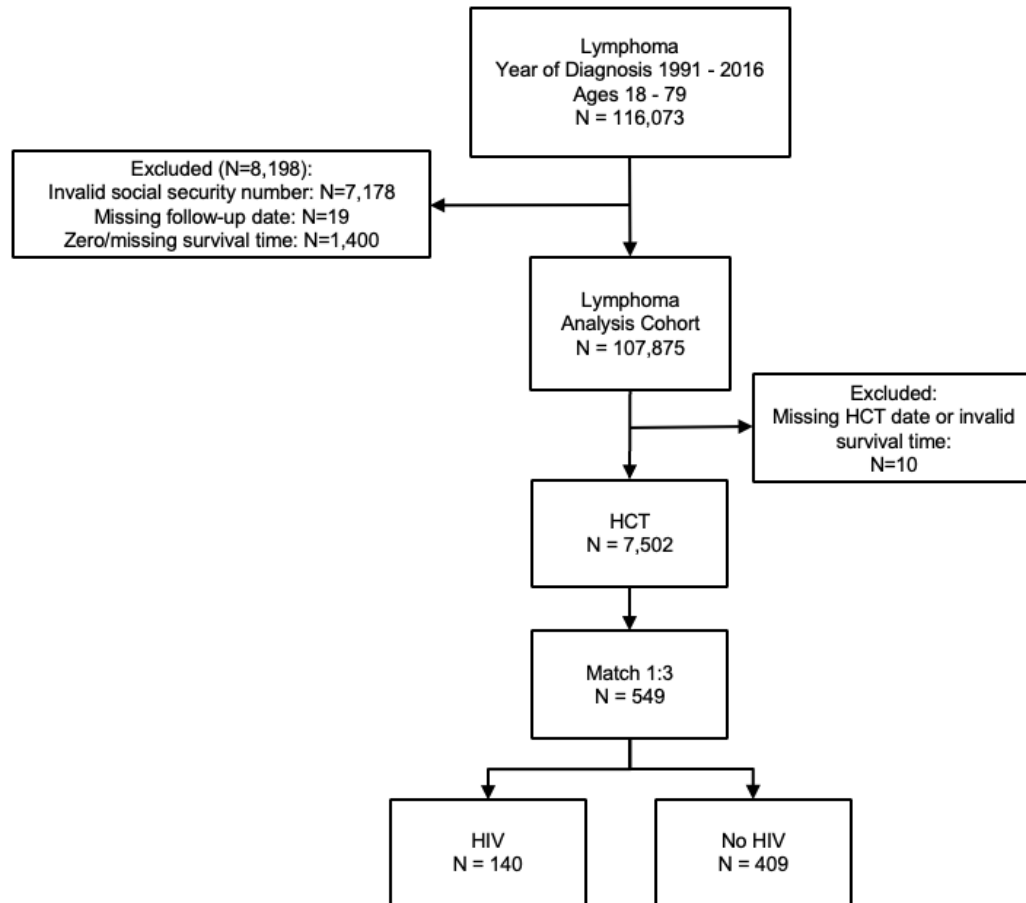
Study Population

This analysis included patients in the CCR who were diagnosed with lymphoma between 1991-2016, received an HCT for their lymphoma, and were between the ages of 18 and 79 at diagnosis. HCT utilization was captured from all three data sources independently. Considering

most HCT data were available in multiple sources, we prioritized using information from the CIBMTR where available, then used specific International Classification of Diseases (ICD) ICD-9/ICD-10 codes from the PDD, and, lastly, reported treatment fields for the initial course of treatment from the CCR. HCT receipt was categorized as either autologous HCT or allogeneic HCT. Patients were excluded if they did not have a valid social security number for linking to the PDD, survival time was zero, HCT date or follow-up date were unknown, or HCT survival time was <0.

Among the 7,502 lymphoma patients with an HCT, we identified the human immunodeficiency virus (HIV) and non-HIV cohorts. The HIV lymphoma cohort included all patients with an HIV diagnosis prior to lymphoma diagnosis date in the CCR or PDD. HIV status at time of lymphoma diagnosis was abstracted for lymphomas in the CCR from 1990 onwards. From the PDD, an HIV diagnosis was identified using HIV ICD-9/ICD-10 codes in the primary or other diagnosis position and associated dates. The non-HIV cohort was created by individually matching HCT recipients with HIV to HCT recipients without HIV matched (1:3) on age, sex, lymphoma subtype, stage at diagnosis, HCT type, year of transplant +/- 3 years, and time from diagnosis to transplant $\pm$ 2 years as an indicator of lymphoma progression using the greedy matching protocol.²⁵ There were 8 HCT recipients with HIV that did not match and were excluded from analysis. This resulted in 140 HCT recipients with HIV and 409 HCT recipients without HIV that were included for analysis (Figure 1).

Figure 1: Cohort Selection Flow Diagram



Demographic and Clinical Factors

Baseline demographic and lymphoma characteristics and HCT related information were obtained from the CIBMTR, CCR, and PDD as previously described.²⁴ Age was analyzed categorically (18-39, 40-64, 65+ years). Sex was categorized as male/transgender and female.²⁶ Lymphoma subtypes were grouped as HL, diffuse large B-cell lymphoma (DLBCL), other NHL, and lymphoma not otherwise specified (NOS). Cancer stage at diagnosis was categorized as localized, advanced, or unknown. Year of transplant was categorized as <1997, 1997-2005, and ≥2006, corresponding to HIV treatment eras.²⁷ Comorbidities were identified using the Elixhauser comorbidity index (excluding AIDS and lymphoma), and were captured up to 2

years prior to the date of cancer diagnosis.²⁸ Comorbidities were classified based on admissions in PDD as no admission (and thus, no information), or 0, 1-2, or ≥ 3 comorbidities. Time from diagnosis to transplant was measured as the difference in time between lymphoma diagnosis and HCT date.

Clinical Outcomes and Hospital Utilization

Acute complications within 90 days of HCT were captured from the PDD, including acute renal complications, acute liver complications, infection, and sepsis.^{29,30} These complications were identified using relevant ICD-9/ICD-10 codes in primary or other diagnosis positions (**Supplemental Materials 3 - ICD codes**).

Hospital use was captured from the PDD and evaluated using total number of admissions and combined length of stay within the first 90 days following HCT. Hospitalization prior to HCT date was excluded. Total number of admissions were categorized as never admitted, admitted once or twice, and admitted 3+ times as well as ever versus never admitted. Length of stay was categorized as never admitted, admitted <1 week, and admitted ≥ 1 week.

Overall survival at 90 days, 1 year and 2 years were examined to compare our population-based cohort to seminal single-center studies.¹³ Survival following HCT was measured from HCT date to death date, last known date of contact, or study cutoff (12/31/18), whichever occurred first within the initial 90-days, 1-year or 2-years following HCT date.

Statistical Analysis

Descriptive statistics summarized baseline characteristics of HCT recipients by HIV status. Differences between those with and without HIV were examined using independent t-

tests for continuous variables and chi-square for categorical variables among the matched cohorts.

Matched Cox proportional hazards regression models were performed to evaluate the association of HIV status with each acute complication (yes/no) and being admitted, using Fine and Gray's methods to account for the competing risk of death. Proportional hazard assumptions for all Cox models were evaluated using the Schoenfeld Residuals Test. Results are presented as adjusted hazard ratios (HR) and associated 95% confidence intervals (CI).

Kaplan-Meier survival analyses were performed between the matched cohorts to measure 90-day, one-year, and two-year overall survival following HCT. Log-rank test was performed for overall differences in survival between the matched cohorts across all time-points limited to two years following HCT.

All P-values were two-sided with significance set a priori to <0.05 . All analyses were performed using SAS 9.4 (SAS, Cary, NC). This study was approved by the Institutional Review Board of the University of California, Davis, the California Committee for the protection of human subjects, and the NMDP.

RESULTS

Characteristics in HIV and Matched non-HIV Cohorts

A total of 549 HCT recipients were included in the matched cohort, 140 HCT recipients with HIV and 409 without HIV. There were no significant differences in match criteria (age, sex, lymphoma subtype, stage at diagnosis, HCT type, year of transplant, and time from diagnosis to HCT) or comorbidities between those with and without HIV. Overall, the majority of HCT recipients were between 40-64 (70.3%), male/transgender (91.3%), diagnosed with DLBCL (60.8%), had advanced disease (87.4%), received an autologous transplant (94.2%), received an HCT in ≥ 2006 (61.7%), and received a transplant at a median of 450 days after their lymphoma diagnosis (interquartile range: 301-1118; **Table 1**).

Table 1. Patient and Hematopoietic Cell Transplantation (HCT) Characteristics in the HIV and Matched* Non-HIV Cohorts, California, 1991-2016

Characteristics	All (N=549)	No HIV (N=409)	HIV (N=140)
Age			
18-39	147 (26.8)	108 (26.4)	39 (27.9)
40-64	386 (70.3)	289 (70.7)	97 (69.3)
65+	16 (2.9)	12 (2.9)	4 (2.9)
Sex			
Male/Transgender**	501 (91.3)	373 (91.2)	128 (91.4)
Female	48 (8.7)	36 (8.8)	12 (8.6)
Diagnosis			
Hodgkin Lymphoma	98 (17.9)	73 (17.8)	25 (17.9)
DLBCL	334 (60.8)	249 (60.9)	85 (60.7)
NHL, NOS	47 (8.6)	35 (8.6)	12 (8.6)
Lymphoma, Other***	70 (12.8)	52 (12.7)	18 (12.9)
Stage at Diagnosis			
Localized, Regional	65 (11.8)	48 (11.7)	17 (12.1)
Advanced	480 (87.4)	359 (87.8)	121 (86.4)
Unknown	4 (0.7)	2 (0.5)	2 (1.4)
Elixhauser Comorbidities ($\leq$ 2 years prior to diagnosis)			
No admission	322 (58.7)	245 (59.9)	77 (55.0)
0	80 (14.6)	60 (14.7)	20 (14.3)
1-2	91 (16.6)	67 (16.4)	24 (17.1)
3+	56 (10.2)	37 (9.0)	19 (13.6)
HCT Type			
Allogeneic	32 (5.8)	24 (5.9)	8 (5.7)
Autologous	517 (94.2)	385 (94.1)	132 (94.3)
Year of HCT			
< 1997	28 (5.1)	23 (5.6)	5 (3.6)

1997 - 2005	182 (33.2)	131 (32.0)	51 (36.4)
> 2006	339 (61.7)	255 (62.3)	84 (60.0)
Time to HCT (days)			
Mean $\pm$ SD	873.4 $\pm$ 984.4	868.6 $\pm$ 985.0	887.3 $\pm$ 986.0
Min-Max	68.0-5653.0	97.0-5653.0	68.0-5094.0
Median (IQR)	450.0 (301.0-1118.0)	448.0 (299.0-1118.0)	470.5 (305.0-1133.0)

*Matched on age, sex, lymphoma subtype, stage at diagnosis, HCT type, year of transplant +/- 3 years, and time from diagnosis to transplant +/- 720 days (all p-values > 0.05).

**Male and transgender participants were grouped together based on similar HIV transmission mechanisms, consistent with established HIV literature.

***Lymphoma, Other includes CNS lymphoma (n=14 non-HIV; n=4 HIV).

Abbreviations: HIV= human immunodeficiency virus. DLBCL=diffuse large B-cell lymphoma; CNS, central nervous system; NHL, NOS=non-Hodgkin lymphoma not otherwise specified; SD=standard deviation; IQR=interquartile range.

Clinical and Hospital Use Outcomes

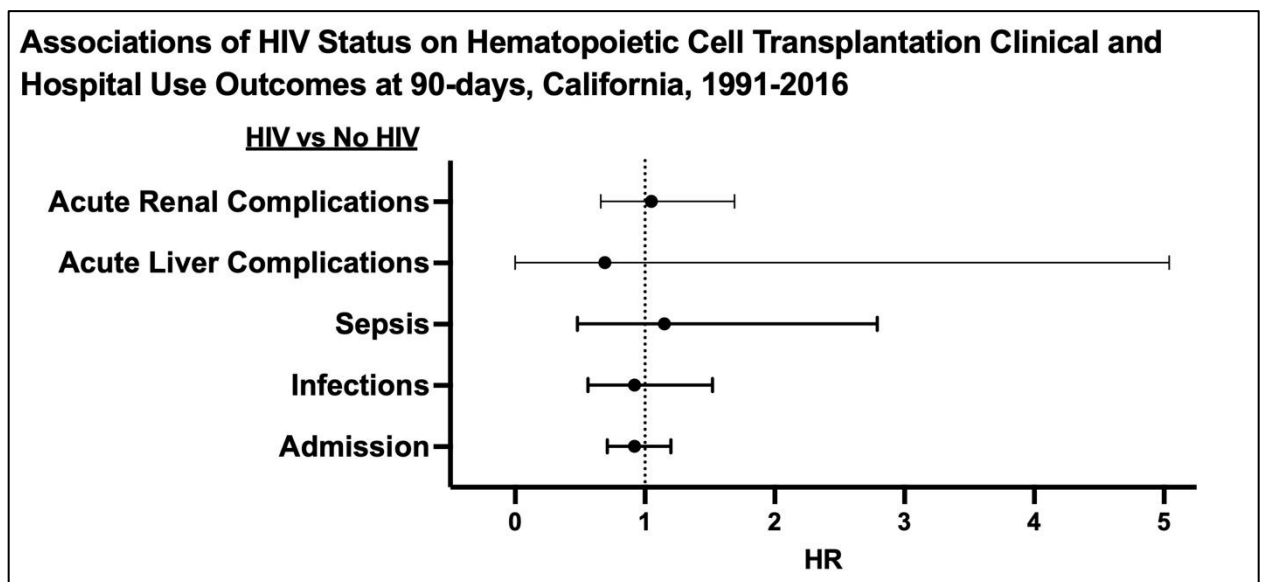
In HIV and non-HIV cohorts combined, a small proportion of patients experienced 90-day acute renal complications (10.9%), acute liver complications (0.9%), sepsis (3.3%), or infection (11.5%; **Table 2**). Between the HIV and non-HIV matched cohort, the proportion of 90-day acute renal complications (11.4% HIV vs 10.8% non-HIV), acute liver complications (<1% HIV vs 1% non-HIV), sepsis (3.6% HIV vs 3.2% non-HIV), or infections (10.7% HIV vs 11.7% non-HIV) were similar. In regression models, HIV was not associated with 90-day acute renal complications (HR=1.05, 95% CI: 0.66-1.69), acute liver complications (HR=0.69, 95% CI: 0.09-5.04), sepsis (HR=1.15, 95% CI: 0.48-2.79), or infections (HR=0.92, 95% CI: 0.56-1.52) (**Table 3, Figure 2**).

Table 2. Hematopoietic Cell Transplantation Clinical and Hospital Use Outcomes at 90-days in the HIV and Matched* Non-HIV cohorts, California, 1991-2016

Outcomes	All (N=549)	No HIV (N=409)	HIV (N=140)	P-value
Infections				0.743
No	486 (88.5)	361 (88.3)	125 (89.3)	
Yes	63 (11.5)	48 (11.7)	15 (10.7)	
Sepsis				0.822
No	531 (96.7)	396 (96.8)	135 (96.4)	
Yes	18 (3.3)	13 (3.2)	5 (3.6)	
Acute Renal Complications				0.826
No	489 (89.1)	365 (89.2)	124 (88.6)	
Yes	60 (10.9)	44 (10.8)	16 (11.4)	
Acute Liver Complications				0.777
No	544 (99.1)	405 (99.0)	139 (99.3)	
Yes	5 (0.9)	4 (1.0)	1 (0.7)	
Hospital Admissions				0.688
Never admitted	390 (71.0)	287 (70.2)	103 (73.6)	
Admitted once or twice	148 (27.0)	113 (27.6)	35 (25.0)	

Admitted 3+	11 (2.0)	9 (2.2)	2 (1.4)	
Combined Length of Stay				0.354
Never admitted	390 (71.0)	287 (70.2)	103 (73.6)	
Admitted, < 1 week	51 (9.3)	36 (8.8)	15 (10.7)	
≥ 1 week	108 (19.7)	86 (21.0)	22 (15.7)	
*Matched on age, sex, lymphoma subtype, stage at diagnosis, HCT type, year of transplant +/- 3 years, and time from diagnosis to transplant +/- 720 days. Abbreviations: HIV= human immunodeficiency virus				

Figure 2



HIV=human immunodeficiency virus

Table 3. Associations of HIV Status on Hematopoietic Cell Transplantation Clinical and Hospital Use Outcomes at 90-days, California, 1991-2016

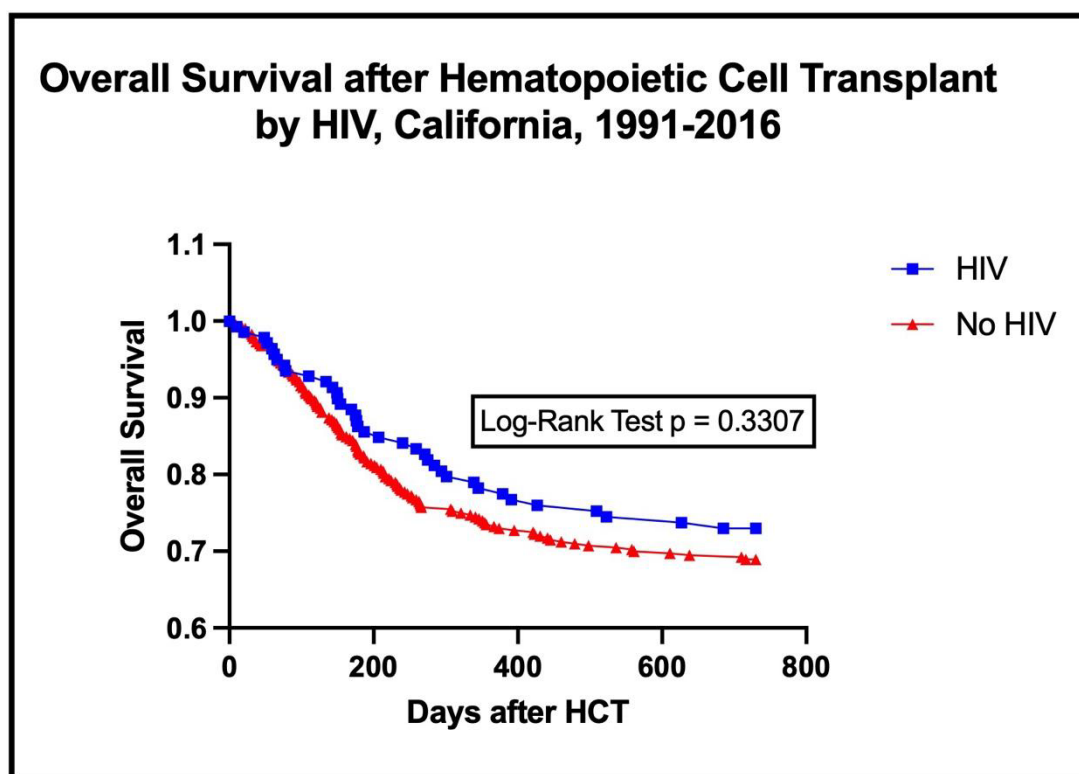
	Counts	HR	CI 95%		P-value
Acute Renal Complications					0.8313
No HIV	44	Reference			
HIV	16	1.05	0.66	1.69	
Acute Liver Complications					0.7113
No HIV	4	Reference			
HIV	1	0.69	0.09	5.04	
Sepsis					0.7507
No HIV	13	Reference			
HIV	5	1.15	0.48	2.79	
Infections					0.748
No HIV	48	Reference			
HIV	15	0.92	0.56	1.52	
Any Admission					0.555
No HIV	122	Reference			
HIV	37	0.92	0.71	1.20	
Human immunodeficiency virus (HIV) and non-HIV cohorts matched on age, sex, lymphoma subtype, stage at diagnosis, HCT type, year of transplant +/- 3 years, and time from diagnosis to transplant +/- 720 days. Match variables controlled using strata match identification number.					

In HIV and non-HIV cohorts combined, 27% were admitted once or twice and 2% were admitted three or more times to the hospital within 90 days following transplant; 9% were admitted for <1 week and 20% for ≥ 1 week (**Table 2**). The proportion of those admitted once or twice (25% HIV vs. 27.6% non-HIV) and for a combined length of stay for >1 week (15.7% HIV vs. 21% non-HIV) were similar between the HIV and non-HIV matched cohorts. In regression models, HIV was not significantly associated with being admitted within 90 days following transplant (HR=0.92 95% CI: 0.71-1.20).

Overall Survival

In HIV and non-HIV cohorts combined, the overall survival was 93% at 90-days, 74% at 1-year, and 69% at 2-years (**Table 4**, HIV=human immunodeficiency virus). Overall survival at 90-days was similar between HCT recipients with and without HIV (94% HIV vs 93% non-HIV), at 1-year (78% HIV vs 73% non-HIV), and 2-years was similar between HCT recipients with and without HIV (73% HIV vs 69% non-HIV).

Figure 3



HIV=human immunodeficiency virus

Table 4. Overall Survival at 90-day, 1-year, and 2-years after Hematopoietic Cell Transplant in the HIV and Matched* non-HIV cohorts, California, 1991-2016

Group	N	90-Day	1-year	2-year	P-value**
Overall	549	93%	74%	69%	
HIV	140	94%	78%	73%	0.3307
No HIV	409	93%	73%	69%	

*Matched on age, sex, lymphoma subtype, stage at diagnosis, HCT type, year of transplant +/- 3 years, and time from diagnosis to transplant +/- 720 days.
**Log-rank test was performed for differences in survival between HIV and non-HIV matched cohorts.
Abbreviations: HIV= human immunodeficiency virus

DISCUSSION

In this matched cohort of lymphoma patients with and without HIV who received HCT, we found that HIV status was not associated with increased acute complications, higher hospital utilization, or reduced survival following HCT. Our findings confirm existing evidence from smaller clinical trials suggesting that HCT can be safely and effectively performed in otherwise eligible HCT recipients with HIV and result in comparable outcomes.^{8,12–15,22,23} This lack of association suggests that HIV clinical management, including combination antiretroviral therapy (cART) and general HIV supportive care,^{31,32} does not contribute to worse clinical outcomes, hospital use, or overall survival following HCT in recipients with HIV.

Rates of acute renal complications, liver complications, sepsis, and infection at 90-days, were low overall and did not differ by HIV status. Specifically, the lack of association seen in sepsis and infections suggests that underlying immunosuppression from well-controlled HIV does not contribute to worse opportunistic infections,^{23,33} and that drug-drug interactions between combination antiretroviral therapy and high-dose chemotherapy do not contribute to clinically-meaningful acute renal complications and liver complications.⁸ Using a drug-drug interaction table between common HCT chemo/biotherapies and modern cART (**Supplemental Material 2 - cART and chemo/antineoplastic therapy drug-drug interaction table**),^{5,34} we

only found evidence to support significant DDIs between cART and cladribine. Contemporary combination ART regimens have fewer clinically meaningful drug-drug interactions than earlier regimens, simplifying HIV clinical management during the HCT period. While our study only includes patients diagnosed through 2016, outcomes observed in this cohort may not fully reflect more recent advancements in HIV medicine.

Hospital utilization within 90 days following HCT, including admissions and length of stay, was similar between those with and without HIV, with most patients overall never being admitted or admitted for >1 week. This hospital use pattern may reflect a neutropenic fever or engraftment syndrome following HCT resulting in hospital admission through count recovery,³⁵ considering the low rates of confirmed infection and sepsis. The lack of differences in admission and length of stay further contribute to the evidence base suggesting HCT can be safely used in people with HIV without added hospital burden.

Our findings extend the existing survival evidence by using a population-based study with one of the largest populations of HCT recipients with HIV. We demonstrate equivalent OS at 90-days, 1-year, and 2-years, consistent with smaller, seminal studies evaluating HCT survival by HIV status.^{13,14,22} The comparable survival rates further support that HCT outcomes based on HIV status are not different, and that HIV infection alone, while all other HCT eligibility criteria are met, should not exclude a patient with HIV from HCT candidacy. Hesitancy surrounding HCT use in people with HIV thus may be influenced by additional factors beyond survival, acute complications, and hospital burden, such as unfamiliarity with recent HIV literature, low prevalence of HIV in relative geographic regions, or HIV-related stigma at the individual and organizational levels.

Our study has several implications for clinical practice. First, well-controlled HIV should not preclude consideration of HCT in patients with HIV lymphoma. Second, HCT centers should advocate for equitable access to HCT as codified in HCT inclusion/exclusion protocols (see

Supplemental Materials 1 - Additional Inclusion Criteria Guidelines for HIV + Individuals

for one example from a large academic medical center), as people with HIV continue to experience barriers to cancer treatment.^{36,37} In particular, autologous HCT protocols for diffuse large B-cell lymphoma should include people with HIV. Progress has been made in more broadly including patients with HIV in cancer clinical trials and by using implementation science approaches in closing the gap in unmet care needs for people with HIV.^{38,39} Lastly, HCT centers within California included both larger academic medical centers and smaller tertiary medical centers within community HMO/PPO networks, demonstrating that HCT care across a variety of HCT center sizes was sufficient in the care of HCT recipients with HIV.

While the matched design balanced available confounders across key clinical and demographic factors, several clinically meaningful pre-HCT characteristics were unavailable, including chemosensitivity, remission status at HCT, and performance/functional status. Thus, confounding and selection of healthier HCT patients, particularly among people with HIV, cannot be ruled out. Additionally, HIV-specific variables, including viral load, CD4 count, combination ART adherence, and prophylactic support during the HCT period, were unavailable but may influence HCT outcomes. However, the comparability across these matched factors (age, sex, lymphoma subtype, disease stage, type of transplant, year of transplant, and time to transplant) supports interpretation and minimizes confounding, thereby strengthening the validity of our model findings using 25 years of data. Overall survival does not distinguish cause-specific mortality, thereby limiting mortality inferences on the contributions of cancer, transplant, or HIV on death, though the relatively small number of HCT recipients with HIV limited further stratification by cause of death. While our study includes one of the largest HIV HCT cohorts compared to published reports, the population was modest relative to the broader HCT population, thus limiting power and limiting further stratification by transplant type to protect the privacy of patients within our statistical analysis. Generalizability is limited to novel therapies for relapsed/refractory lymphoma, such as chimeric antigen receptor t-cell therapy (CAR-T). Additionally, the majority of the patients were <65, limiting generalizability to older populations.

In conclusion, our findings demonstrate that HCT recipients with HIV experience comparable rates of acute complications, hospital utilization, and overall survival as HCT recipients without HIV. These results contribute strong evidence in support of safe and equitable use of HCT in the HIV population and argue against the exclusion of patients with HIV from HCT eligibility. Future efforts should focus on ensuring equitable HCT access to eligible HCT patients with HIV when clinically indicated, and examining HIV lymphoma outcomes with emerging cellular therapies, such as chimeric antigen receptor t-cell therapy (CAR-T).

ACKNOWLEDGMENTS

This work was supported by the National Institute of Nursing Research of the National Institutes of Health under Award Number F31NR021235. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health. The collection of cancer incidence data used in this study was supported by the California Department of Public Health pursuant to California Health and Safety Code Section 103885; Centers for Disease Control and Prevention's (CDC) National Program of Cancer Registries, under cooperative agreement 5NU58DP007156; the National Cancer Institute's SEER under contract HHSN261201800032I awarded to the University of California, San Francisco, contract HHSN261201800015I awarded to the University of Southern California, and contract HHSN261201800009I awarded to the Public Health Institute. The ideas and opinions expressed herein are those of the author(s) and do not necessarily reflect the opinions of the State of California, Department of Public Health, the National Cancer Institute, and the Centers for Disease Control and Prevention or their Contractors and Subcontractors. This research was supported in part by the Intramural Research Program of the National Institutes of Health (NIH). The contributions of the NIH author(s) are considered Works of the United States Government. The findings and conclusions presented in this paper are those of the author(s) and do not necessarily reflect the views of the NIH or the U.S. Department of Health and Human Services.

AUTHOR CONTRIBUTIONS

Conceptualization and Study Design: KD, AJF, SC, RZ, LM, LMM, SLC, THMK; **Data Acquisition / Data Curation:** KD, AB, LM, LMM, THMK; **Methodology and Statistical Analysis:** KD, AB, AJF, SJS, SC, CM, JJA, LM, LMM, SLC, THMK; **Interpretation of Data:** KD, AB, AJF, SJS, SC, RZ, LF, CM, JJA, LM, LMM, SLC, THMK; **Drafting of the Manuscript:** KD, AB, AJF, SJS, SC, RZ, LF, CM, JJA, LM, LMM, SLC, THMK; **Critical Revision for Important Intellectual Content:** KD, AB, AJF, SJS, SC, RZ, LF, CM, JJA, LM, LMM, SLC, THMK; **Supervision:** AB, AJF, LM, LMM; **Funding Acquisition:** KD, AJF, SC, RZ, LF, LM, LMM, SLC, THMK; **Final Approval of the Manuscript:** All authors.

CONFLICT OF INTEREST DISCLOSURES

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Supplemental Material 1 – Additional Inclusion Criteria Guidelines for HIV + Individuals

Below is one example from an HCT center offering HCT to individuals with HIV using additional HIV-related inclusion criteria.

Additional Inclusion Criteria Guidelines for HIV + Individuals

Patients on antiviral therapies (ARVs) can either have

_____ Undetectable HIV viral load (VL less than 50 copies/mL)

Or

_____ If VL detectable at less than 2000 copies/mL must have a review of previous ARV therapies or previous genotypic or phenotypic testing indicating the ability to fully suppress the virus by the addition of sensitive drugs. Review must be conducted by the ID specialist caring for the patient.

Or

_____ if VL greater than 2000 copies/mL, a current HIV genotype and/or phenotype must be obtained. If ARV therapy to which the virus is sensitive can be determined based on genotype and previous ARV therapy, then the patient is eligible. Review must be conducted by the ID specialist caring for the patient.

_____ Evaluated or scheduled in Stanford Positive Care clinic

Supplemental Materials 2 – cART (x-axis) and chemo/antineoplastic therapy (y-axis) drug-drug interaction table

	abacavir/la mivudine	abacavir/dol utegravir/la	abacavir/la mivudine/zi	atazanavir/c obicistat	bictegravir/ emtricitabin	cabotegravi r/rilpivirine	darunavir/c obicistat	darunavir/c obicistat/em	dolutegravir /lamivudine	dolutegravir /rilpivirine	doravirine/l amivudine/t	efavirenz/e mtricitabine	efavirenz/la mivudine/te	elvitegravir/ cobicistat/e	elvitegravir/ cobicistat/e	emtricitabin e/rilpivirine/	emtricitabin e/rilpivirine/	emtricitabin e/tenofovir	emtricitabin e/tenofovir	lamivudine/t enofovir	lamivudine/ zidovudine	lopinavir/rit onavir
ATG (rabbit)																						
Bendamustine																						
Busulfan																						
Carboplatin																						
Carmustine																						
Cladribine																						
Clofarabine																						
Cyclophosphamide																						
Cytarabine																						
Etoposide																						
Fludarabine																						
Gemtuzumab																						
Inotuzumab																						
Melphalan																						
Pentostatin																						
Rituximab																						
Thiotepa																						
Treosulfan																						

ATG = antithymocyte globulin
Corresponding antiretroviral brand names: abacavir/lamivudine (Epzicom); abacavir/dolutegravir/lamivudine (Triumeq);
abacavir/lamivudine/zidovudine (Trizivir); atazanavir/cobicistat (Evotaz); bictegravir/emtricitabine/tenofovir alafenamide (Biktarvy);
cabotegravir/rilpivirine (Cabenuva); darunavir/cobicistat (Prezcobix); darunavir/cobicistat/emtricitabine/tenofovir alafenamide (Symtuza);
dolutegravir/lamivudine (Dovato); dolutegravir/rilpivirine (Juluca); doravirine/lamivudine/tenofovir disoproxil fumarate (Delstrigo);
efavirenz/emtricitabine/tenofovir disoproxil fumarate (Atripla); efavirenz/lamivudine/tenofovir disoproxil fumarate (Symfi);
elvitegravir/cobicistat/emtricitabine/tenofovir alafenamide (Genvoya); elvitegravir/cobicistat/emtricitabine/tenofovir disoproxil fumarate (Stribild);
emtricitabine/rilpivirine/tenofovir alafenamide (Odefsey); emtricitabine/rilpivirine/tenofovir disoproxil fumarate (Complera); emtricitabine/tenofovir
alafenamide (Descovy); emtricitabine/tenofovir disoproxil fumarate (Truvada); lamivudine/tenofovir disoproxil fumarate (Cimduo);
lamivudine/zidovudine (Combivir); lopinavir/ritonavir (Kaletra).

	No known interaction
	Avoid combination
	Monitor therapy
	No action needed
	Consider modification

Supplemental Materials – single agent ART (x-axis) and chemo/antineoplastic therapy (y-axis) drug-drug interaction table

	NRTIs						NNRTIs					INSTIs			PIs				
	abacavir	emtricitabine	lamivudine	tenofovir disoproxil	tenofovir alafenamide	zidovudine	doravirine	efavirenz	etravirine	nevirapine	rilpivirine	cabotegravir	dolutegravir	raltegravir	atazanavir	darunavir	fosamprenavir	ritonavir	tipranavir
ATG (rabbit)																			
Bendamustine																			
Busulfan																			
Carboplatin																			
Carmustine																			
Cladribine																			
Clofarabine																			
Cyclophosphamide																			
Cytarabine																			
Etoposide																			
Fludarabine																			
Gemtuzumab																			
Inotuzumab																			
Melphalan																			
Pentostatin																			
Rituximab																			
Thiotepa																			
Treosulfan																			

NRTI = Nucleoside Reverse Transcriptase Inhibitors; NNRTI = Non-Nucleoside Reverse Transcriptase Inhibitors; INSTI = Integrase Strand Transfer Inhibitors; PI = Protease Inhibitors

Corresponding antiretroviral brand names: abacavir (Ziagen); emtricitabine (Emtriva); lamivudine (Epivir); tenofovir disoproxil fumarate (Viread); tenofovir alafenamide (Vemlidy); zidovudine (Retrovir); doravirine (Pifeltro); efavirenz (Sustiva); etravirine (Intelligence); nevirapine (Viramune); rilpivirine (Edurant); cabotegravir (Apretude); dolutegravir (Tivicay); raltegravir (Isentress); atazanavir (Reyataz); darunavir (Prezista); fosamprenavir (Lexiva); ritonavir (Norvir); tipranavir (Aptivus).

No known interaction

	Avoid combination
	Monitor therapy
	No action needed
	Consider modification

Supplemental Materials – single agent ART (x-axis) and chemo/antineoplastic therapy (y-axis) drug-drug interaction table

	FI	CCR5A	AI	PAI	CI	PE
	enfuvirtide	maraviroc	fostemsavir	ibalizumab- uiyk	lenacapavir	cobicistat
ATG (rabbit)						
Bendamustine						
Busulfan						
Carboplatin						
Carmustine						
Cladribine						
Clofarabine						
Cyclophosphamide						
Cytarabine						
Etoposide						
Fludarabine						
Gemtuzumab						
Inotuzumab						
Melphalan						
Pentostatin						
Rituximab						
Thiotepa						
Treosulfan						
FI = Fusion Inhibitors; CCR5A = CCR5 Antagonists; AI = Attachment Inhibitors; PAI = Post-Attachment Inhibitors; CI = Capsid Inhibitors; PE = Pharmacokinetic Enhancers Corresponding antiretroviral brand names: enfuvirtide (Fuzeon); maraviroc (Selzentry); fostemsavir (Rukobia); ibalizumab-uiyk (Trogarzo); lenacapavir (Sunlenca); cobicistat (Tybost).						

No known interaction

	Avoid combination
	Monitor therapy
	No action needed
	Consider modification

Supplemental Materials 3 – ICD codes

Description	ICD Code
Acute glomerulonephritis	580
Acute glomerulonephritis with lesion of proliferative glomerulonephritis	580
Acute glomerulonephritis with lesion of rapidly progressive glomerulonephritis	580.4
Acute glomerulonephritis with other specified pathological lesion in kidney	580.8
Acute glomerulonephritis in diseases classified elsewhere	580.81
Acute glomerulonephritis with other specified pathological lesion in kidney	580.89
Acute glomerulonephritis with unspecified pathological lesion in kidney	580.9
Nephrotic syndrome	581
Nephrotic syndrome with lesion of proliferative glomerulonephritis	581
Nephrotic syndrome with lesion of membranous glomerulonephritis	581.1
Nephrotic syndrome with lesion of membranoproliferative glomerulonephritis	581.2
Nephrotic syndrome with lesion of minimal change glomerulonephritis	581.3
Nephrotic syndrome with other specified pathological lesion in kidney	581.8
Nephrotic syndrome in diseases classified elsewhere	581.81
Nephrotic syndrome with other specified pathological lesion in kidney	581.89
Nephrotic syndrome with unspecified pathological lesion in kidney	581.9
Nephritis and nephropathy not specified as acute or chronic	583
Nephritis and nephropathy, not specified as acute or chronic, with lesion of proliferative glomerulonephritis	583
Nephritis and nephropathy, not specified as acute or chronic, with lesion of membranous glomerulonephritis	583.1

Nephritis and nephropathy, not specified as acute or chronic, with lesion of membranoproliferative glomerulonephritis	583.2
Nephritis and nephropathy, not specified as acute or chronic, with lesion of rapidly progressive glomerulonephritis	583.4
Nephritis and nephropathy, not specified as acute or chronic, with lesion of renal cortical necrosis	583.6
Nephritis and nephropathy, not specified as acute or chronic, with lesion of renal medullary necrosis	583.7
Nephritis and nephropathy not specified as acute or chronic with other specified pathological lesion in kidney	583.8
Nephritis and nephropathy, not specified as acute or chronic, in diseases classified elsewhere	583.81
Nephritis and nephropathy, not specified as acute or chronic, with other specified pathological lesion in kidney	583.89
Nephritis and nephropathy, not specified as acute or chronic, with unspecified pathological lesion in kidney	583.9
Acute kidney failure	584
Acute kidney failure with lesion of tubular necrosis	584.5
Acute kidney failure with lesion of renal cortical necrosis	584.6
Acute kidney failure with lesion of renal medullary [papillary] necrosis	584.7
Acute kidney failure with other specified pathological lesion in kidney	584.8
Acute kidney failure, unspecified	584.9
Renal failure, unspecified	586
Renal sclerosis, unspecified	587
Disorders resulting from impaired renal function	588
Renal osteodystrophy	588
Nephrogenic diabetes insipidus	588.1
Other specified disorders resulting from impaired renal function	588.8
Secondary hyperparathyroidism (of renal origin)	588.81
Other specified disorders resulting from impaired renal function	588.89

Unspecified disorder resulting from impaired renal function	588.9
Small kidney of unknown cause	589
Unilateral small kidney	589
Bilateral small kidneys	589.1
Small kidney, unspecified	589.9
Hydronephrosis	591
Calculus of kidney and ureter	592
Calculus of kidney	592
Calculus of ureter	592.1
Urinary calculus, unspecified	592.9
Other disorders of kidney and ureter	593
Nephroptosis	593
Hypertrophy of kidney	593.1
Cyst of kidney, acquired	593.2
Stricture or kinking of ureter	593.3
Other ureteric obstruction	593.4
Hydroureter	593.5
Postural proteinuria	593.6
Vesicoureteral reflux	593.7
Vesicoureteral reflux unspecified or without reflux nephropathy	593.7
Vesicoureteral reflux with reflux nephropathy, unilateral	593.71
Vesicoureteral reflux with reflux nephropathy, bilateral	593.72
Other vesicoureteral reflux with reflux nephropathy NOS	593.73
Other specified disorders of kidney and ureter	593.8
Vascular disorders of kidney	593.81
Ureteral fistula	593.82
Other specified disorders of kidney and ureter	593.89
Unspecified disorder of kidney and ureter	593.9
Disorders of fluid electrolyte and acid-base balance	276
Hyperosmolality and/or hypernatremia	276
Hyposmolality and/or hyponatremia	276.1
Acidosis	276.2
Alkalosis	276.3
Mixed acid-base balance disorder	276.4

Volume depletion disorder	276.5
Volume depletion, unspecified	276.5
Dehydration	276.51
Hypovolemia	276.52
Fluid overload disorder	276.6
Other fluid overload	276.69
Hyperpotassemia	276.7
Hypopotassemia	276.8
Electrolyte and fluid disorders not elsewhere classified	276.9
Acute and subacute necrosis of liver	570
Hepatic encephalopathy	572.2
Portal hypertension	572.3
Hepatorenal syndrome	572.4
Cholelithiasis	574
Calculus of gallbladder with acute cholecystitis	574
Calculus of gallbladder with acute cholecystitis, without mention of obstruction	574
Calculus of gallbladder with acute cholecystitis, with obstruction	574.01
Calculus of gallbladder with other cholecystitis	574.1
Calculus of gallbladder with other cholecystitis, without mention of obstruction	574.1
Calculus of gallbladder with other cholecystitis, with obstruction	574.11
Calculus of gallbladder without mention of cholecystitis	574.2
Calculus of gallbladder without mention of cholecystitis, without mention of obstruction	574.2
Calculus of gallbladder without mention of cholecystitis, with obstruction	574.21
Calculus of bile duct with acute cholecystitis	574.3
Calculus of bile duct with acute cholecystitis, without mention of obstruction	574.3
Calculus of bile duct with acute cholecystitis, with obstruction	574.31
Calculus of bile duct with other cholecystitis	574.4
Calculus of bile duct with other cholecystitis, without mention of obstruction	574.4

Calculus of bile duct with other cholecystitis, with obstruction	574.41
Calculus of bile duct without mention of cholecystitis	574.5
Calculus of bile duct without mention of cholecystitis, without mention of obstruction	574.5
Calculus of bile duct without mention of cholecystitis, with obstruction	574.51
Calculus of gallbladder and bile duct with acute cholecystitis	574.6
Calculus of gallbladder and bile duct with acute cholecystitis, without mention of obstruction	574.6
Calculus of gallbladder and bile duct with acute cholecystitis, with obstruction	574.61
Calculus of gallbladder and bile duct with other cholecystitis	574.7
Calculus of gallbladder and bile duct with other cholecystitis, without mention of obstruction	574.7
Calculus of gallbladder and bile duct with other cholecystitis, with obstruction	574.71
Calculus of gallbladder and bile duct with acute and chronic cholecystitis	574.8
Calculus of gallbladder and bile duct with acute and chronic cholecystitis, without mention of obstruction	574.8
Calculus of gallbladder and bile duct with acute and chronic cholecystitis, with obstruction	574.81
Calculus of gallbladder and bile duct without cholecystitis	574.9
Calculus of gallbladder and bile duct without cholecystitis, without mention of obstruction	574.9
Calculus of gallbladder and bile duct without cholecystitis, with obstruction	574.91
Other disorders of gallbladder	575
Acute cholecystitis	575
Other cholecystitis	575.1
Cholecystitis, unspecified	575.1
Acute and chronic cholecystitis	575.12
Obstruction of gallbladder	575.2
Hydrops of gallbladder	575.3
Perforation of gallbladder	575.4
Fistula of gallbladder	575.5
Cholesterolosis of gallbladder	575.6

Other specified disorders of gallbladder	575.8
Unspecified disorder of gallbladder	575.9
Other disorders of biliary tract	576
Postcholecystectomy syndrome	576
Cholangitis	576.1
Obstruction of bile duct	576.2
Perforation of bile duct	576.3
Fistula of bile duct	576.4
Spasm of sphincter of Oddi	576.5
Other specified disorders of biliary tract	576.8
Unspecified disorder of biliary tract	576.9
Nonspecific elevation of levels of transaminase or lactic acid dehydrogenase [LDH]	790.4
Sepsis due to streptococcus, group A	A40.0
Sepsis due to streptococcus, group B	A40.1
Sepsis due to Streptococcus pneumoniae	A40.3
Other streptococcal sepsis	A40.8
Streptococcal sepsis, unspecified	A40.9
Sepsis due to Staphylococcus aureus	A41.0
Sepsis due to other specified staphylococcus	A41.1
Sepsis due to unspecified staphylococcus	A41.2
Sepsis due to Hemophilus influenzae	A41.3
Sepsis due to anaerobes	A41.4
Gram-negative sepsis, unspecified	A41.50
Sepsis due to Escherichia coli [E. coli]	A41.51
Sepsis due to Pseudomonas	A41.52
Sepsis due to Serratia	A41.53
Other Gram-negative sepsis	A41.59
Sepsis due to Enterococcus	A41.81
Other specified sepsis	A41.89
Sepsis, unspecified organism	A41.9
Severe sepsis without septic shock	R65.20
Severe sepsis with septic shock	R65.21
Septicemia	38
Streptococcal septicemia	38
Staphylococcal septicemia	38.1
Staphylococcal septicemia, unspecified	38.1
Methicillin susceptible Staphylococcus aureus septicemia	38.11

Methicillin resistant Staphylococcus aureus septicemia	38.12
Other staphylococcal septicemia	38.19
Pneumococcal septicemia [Streptococcus pneumoniae septicemia]	38.2
Septicemia due to anaerobes	38.3
Septicemia due to other gram-negative organisms	38.4
Septicemia due to gram-negative organism, unspecified	38.4
Septicemia due to hemophilus influenzae [H. influenzae]	38.41
Septicemia due to escherichia coli [E. coli]	38.42
Septicemia due to pseudomonas	38.43
Septicemia due to serratia	38.44
Other septicemia due to gram-negative organisms	38.49
Other specified septicemias	38.8
Unspecified septicemia	38.9
Septic shock	785.52
Systemic inflammatory response syndrome (sirs)	995.9
Systemic inflammatory response syndrome, unspecified	995.9
Sepsis	995.91
Severe sepsis	995.92
Cholera	001
Cholera due to Vibrio cholerae	001.0
Cholera due to Vibrio cholerae el tor	001.1
Cholera, unspecified	001.9
Typhoid and paratyphoid fevers	002
Typhoid fever	002.0
Paratyphoid fever A	002.1
Paratyphoid fever B	002.2
Paratyphoid fever C	002.3
Paratyphoid fever, unspecified	002.9
Other salmonella infections	003
Salmonella gastroenteritis	003.0
Salmonella septicemia	003.1
Localized salmonella infections	003.2
Localized salmonella infection, unspecified	003.20
Salmonella meningitis	003.21
Salmonella pneumonia	003.22
Salmonella arthritis	003.23
Salmonella osteomyelitis	003.24

Other localized salmonella infections	003.29
Other specified salmonella infections	003.8
Salmonella infection, unspecified	003.9
Shigellosis	004
Shigella dysenteriae	004.0
Shigella flexneri	004.1
Shigella boydii	004.2
Shigella sonnei	004.3
Other specified shigella infections	004.8
Shigellosis, unspecified	004.9
Other food poisoning (bacterial)	005
Staphylococcal food poisoning	005.0
Botulism food poisoning	005.1
Food poisoning due to Clostridium perfringens (C. welchii)	005.2
Food poisoning due to other Clostridia	005.3
Food poisoning due to Vibrio parahaemolyticus	005.4
Other bacterial food poisoning	005.8
Food poisoning due to Vibrio vulnificus	005.81
Other bacterial food poisoning	005.89
Food poisoning, unspecified	005.9
Amebiasis	006
Acute amebic dysentery without mention of abscess	006.0
Amebic nondysenteric colitis	006.2
Amebic liver abscess	006.3
Amebic lung abscess	006.4
Amebic brain abscess	006.5
Amebic skin ulceration	006.6
Amebic infection of other sites	006.8
Amebiasis, unspecified	006.9
Other protozoal intestinal diseases	007
Balantidiasis	007.0
Giardiasis	007.1
Coccidiosis	007.2
Intestinal trichomoniasis	007.3
Cryptosporidiosis	007.4
Cyclosporiasis	007.5
Other specified protozoal intestinal diseases	007.8

Unspecified protozoal intestinal disease	007.9
Intestinal infections due to other organisms	008
Intestinal infection due to <i>E. coli</i>	008.0
Intestinal infection due to <i>E. coli</i> , unspecified	008.00
Intestinal infection due to enteropathogenic <i>E. coli</i>	008.01
Intestinal infection due to enterotoxigenic <i>E. coli</i>	008.02
Intestinal infection due to enteroinvasive <i>E. coli</i>	008.03
Intestinal infection due to enterohemorrhagic <i>E. coli</i>	008.04
Intestinal infection due to other intestinal <i>E. coli</i> infections	008.09
Intestinal infection due to Arizona group of paracolon bacilli	008.1
Intestinal infection due to <i>Aerobacter aerogenes</i>	008.2
Intestinal infection due to <i>Proteus (mirabilis) (morganii)</i>	008.3
Intestinal infection due to other specified bacteria	008.4
Intestinal infection due to <i>Staphylococcus</i>	008.41
Intestinal infection due to <i>Pseudomonas</i>	008.42
Intestinal infection due to <i>Campylobacter</i>	008.43
Intestinal infection due to <i>Yersinia enterocolitica</i>	008.44
Intestinal infection due to <i>Clostridium difficile</i>	008.45
Intestinal infection due to other anaerobes	008.46
Intestinal infection due to other gram-negative bacteria	008.47
Intestinal infection due to other organisms	008.49
Bacterial enteritis, unspecified	008.5
Enteritis due to specified virus	008.6
Enteritis due to rotavirus	008.61
Enteritis due to adenovirus	008.62
Enteritis due to Norwalk virus	008.63
Enteritis due to other small round viruses (SRVs)	008.64
Enteritis due to calicivirus	008.65
Enteritis due to astrovirus	008.66
Enteritis due to enterovirus NEC	008.67
Enteritis due to other viral enteritis	008.69
Intestinal infection due to other organism, not elsewhere classified	008.8
Ill-defined intestinal infections	009
Infectious colitis, enteritis, and gastroenteritis	009.0

Colitis, enteritis, and gastroenteritis of presumed infectious origin	009.1
Infectious diarrhea	009.2
Diarrhea of presumed infectious origin	009.3
Primary tuberculous infection	010
Primary tuberculous complex	010.0
Primary tuberculous infection, unspecified	010.00
Primary tuberculous infection, bacteriological or histological examination not done	010.01
Primary tuberculous infection, bacteriological or histological examination unknown (at present)	010.02
Primary tuberculous infection, tubercle bacilli found (in sputum) by microscopy	010.03
Primary tuberculous infection, bacilli not found by microscopy but found by bacterial culture	010.04
Primary tuberculous infection, bacilli not found bacteriologically, confirmed histologically	010.05
Primary tuberculous infection, not found bacteriologically/histologically, confirmed by other methods	010.06
Tuberculous pleurisy in primary progressive tuberculosis	010.1
Tuberculous pleurisy, unspecified	010.10
Tuberculous pleurisy, exam not done	010.11
Tuberculous pleurisy, exam unknown	010.12
Tuberculous pleurisy, bacilli found by microscopy	010.13
Tuberculous pleurisy, bacilli not found by microscopy but found by culture	010.14
Tuberculous pleurisy, not found bacteriologically, confirmed histologically	010.15
Tuberculous pleurisy, not found by exam, confirmed by other methods	010.16
Other primary progressive tuberculosis	010.8
Other primary progressive tuberculosis, unspecified	010.80
Other primary progressive TB, exam not done	010.81
Other primary progressive TB, exam unknown	010.82
Other primary progressive TB, bacilli found by microscopy	010.83
Other primary progressive TB, bacilli not found by microscopy but found by culture	010.84

Other primary progressive TB, not found bacteriologically, confirmed histologically	010.85
Other primary progressive TB, not found by exam, confirmed by other methods	010.86
Primary tuberculous infection, unspecified type	010.9
Primary TB infection, unspecified, unspecified	010.90
Primary TB infection, unspecified, exam not done	010.91
Primary TB infection, unspecified, exam unknown	010.92
Primary TB infection, unspecified, bacilli found by microscopy	010.93
Primary TB infection, unspecified, bacilli not found by microscopy but found by culture	010.94
Primary TB infection, unspecified, not found bacteriologically, confirmed histologically	010.95
Primary TB infection, unspecified, not found by exam, confirmed by other methods	010.96
Pulmonary tuberculosis	011
Tuberculosis of lung infiltrative	011.0
Tuberculosis of lung, infiltrative, unspecified	011.00
Tuberculosis of lung, infiltrative, bacteriological or histological examination not done	011.01
Tuberculosis of lung, infiltrative, bacteriological or histological examination unknown	011.02
Tuberculosis of lung, infiltrative, tubercle bacilli found (in sputum) by microscopy	011.03
Tuberculosis of lung, infiltrative, bacilli not found by microscopy, found by culture	011.04
Tuberculosis of lung, infiltrative, bacilli not found, confirmed histologically	011.05
Tuberculosis of lung, infiltrative, bacilli not found, confirmed by other methods	011.06
Tuberculosis of lung nodular	011.1
Tuberculosis of lung, nodular, unspecified	011.10
Tuberculosis of lung, nodular, bacteriological or histological examination not done	011.11
Tuberculosis of lung, nodular, bacteriological or histological examination unknown	011.12
Tuberculosis of lung, nodular, tubercle bacilli found (in sputum) by microscopy	011.13
Tuberculosis of lung, nodular, bacilli not found by microscopy, but found by bacterial culture	011.14

Tuberculosis of lung, nodular, bacilli not found, confirmed histologically	011.15
Tuberculosis of lung, nodular, bacilli not found, confirmed by other methods	011.16
Tuberculosis of lung with cavitation	011.2
Tuberculosis of lung with cavitation, unspecified	011.20
Tuberculosis of lung with cavitation, bacteriological or histological examination not done	011.21
Tuberculosis of lung with cavitation, bacteriological or histological examination unknown	011.22
Tuberculosis of lung with cavitation, tubercle bacilli found (in sputum) by microscopy	011.23
Tuberculosis of lung with cavitation, bacilli not found by microscopy, but found by bacterial culture	011.24
Tuberculosis of lung with cavitation, bacilli not found, confirmed histologically	011.25
Tuberculosis of lung with cavitation, bacilli not found, confirmed by other methods	011.26
Tuberculosis of bronchus	011.3
Tuberculosis of bronchus, unspecified	011.30
Tuberculosis of bronchus, bacteriological or histological examination not done	011.31
Tuberculosis of bronchus, bacteriological or histological examination unknown	011.32
Tuberculosis of bronchus, tubercle bacilli found (in sputum) by microscopy	011.33
Tuberculosis of bronchus, bacilli not found by microscopy, but found in bacterial culture	011.34
Tuberculosis of bronchus, bacilli not found, confirmed histologically	011.35
Tuberculosis of bronchus, bacilli not found, confirmed by other methods	011.36
Tuberculous fibrosis of lung	011.4
Tuberculous fibrosis of lung, unspecified	011.40
Tuberculous fibrosis of lung, bacteriological or histological examination not done	011.41
Tuberculous fibrosis of lung, bacteriological or histological examination unknown	011.42
Tuberculous fibrosis of lung, tubercle bacilli found (in sputum) by microscopy	011.43

Tuberculous fibrosis of lung, bacilli not found by microscopy, but found by bacterial culture	011.44
Tuberculous fibrosis of lung, bacilli not found, confirmed histologically	011.45
Tuberculous fibrosis of lung, bacilli not found, confirmed by other methods	011.46
Tuberculous pneumonia (any form)	011.6
Tuberculous pneumonia, unspecified	011.60
Tuberculous pneumonia, bacteriological or histological examination not done	011.61
Tuberculous pneumonia, bacteriological or histological examination unknown	011.62
Tuberculous pneumonia, tubercle bacilli found (in sputum) by microscopy	011.63
Tuberculous pneumonia, bacilli not found by microscopy, but found by bacterial culture	011.64
Tuberculous pneumonia, bacilli not found, confirmed histologically	011.65
Tuberculous pneumonia, bacilli not found by exam, but tuberculosis confirmed by other methods (e.g., inoculation of animals)	011.66
Tuberculous pneumothorax	011.7
Tuberculous pneumothorax, unspecified	011.70
Tuberculous pneumothorax, bacteriological or histological examination not done	011.71
Tuberculous pneumothorax, bacteriological or histological examination unknown	011.72
Tuberculous pneumothorax, tubercle bacilli found (in sputum) by microscopy	011.73
Tuberculous pneumothorax, bacilli not found by microscopy, but found by bacterial culture	011.74
Tuberculous pneumothorax, bacilli not found, confirmed histologically	011.75
Tuberculous pneumothorax, bacilli not found by exam, but tuberculosis confirmed by other methods (e.g., inoculation of animals)	011.76
Other specified pulmonary tuberculosis	011.8
Other specified pulmonary TB, unspecified	011.80
Other specified pulmonary TB, bacteriological or histological examination not done	011.81

Other specified pulmonary TB, bacteriological or histological examination unknown	011.82
Other specified pulmonary TB, tubercle bacilli found (in sputum) by microscopy	011.83
Other specified pulmonary TB, bacilli not found by microscopy, but found by bacterial culture	011.84
Other specified pulmonary TB, bacilli not found, confirmed histologically	011.85
Other specified pulmonary TB, bacilli not found, confirmed by other methods (e.g., inoculation of animals)	011.86
Unspecified pulmonary tuberculosis	011.9
Pulmonary tuberculosis, unspecified, unspecified	011.90
Pulmonary tuberculosis, unspecified, bacteriological or histological examination not done	011.91
Pulmonary tuberculosis, unspecified, bacteriological or histological examination unknown (at present)	011.92
Pulmonary tuberculosis, unspecified, tubercle bacilli found (in sputum) by microscopy	011.93
Pulmonary tuberculosis, unspecified, bacilli not found by microscopy, but found by bacterial culture	011.94
Pulmonary tuberculosis, unspecified, bacilli not found by bacteriological exam, but tuberculosis confirmed histologically	011.95
Pulmonary tuberculosis, unspecified, bacilli not found by bacteriological or histological exam, but confirmed otherwise	011.96
Other respiratory tuberculosis	012
Tuberculous pleurisy	012.0
Tuberculous pleurisy, unspecified	012.00
Tuberculous pleurisy, bacteriological or histological examination not done	012.01
Tuberculous pleurisy, bacteriological or histological examination unknown	012.02
Tuberculous pleurisy, tubercle bacilli found (in sputum) by microscopy	012.03
Tuberculous pleurisy, bacilli not found by microscopy, but found by bacterial culture	012.04
Tuberculous pleurisy, bacilli not found, confirmed histologically	012.05

Tuberculous pleurisy, bacilli not found, confirmed by other methods (e.g., inoculation of animals)	012.06
Tuberculosis of intrathoracic lymph nodes	012.1
Tuberculosis of intrathoracic lymph nodes, unspecified	012.10
Tuberculosis of intrathoracic lymph nodes, bacteriological or histological examination not done	012.11
Tuberculosis of intrathoracic lymph nodes, bacteriological or histological examination unknown	012.12
Tuberculosis of intrathoracic lymph nodes, tubercle bacilli found (in sputum) by microscopy	012.13
Tuberculosis of intrathoracic lymph nodes, bacilli not found by microscopy, but found by bacterial culture	012.14
Tuberculosis of intrathoracic lymph nodes, bacilli not found, confirmed histologically	012.15
Tuberculosis of intrathoracic lymph nodes, bacilli not found, confirmed by other methods (e.g., animal inoculation)	012.16
Isolated tracheal or bronchial tuberculosis	012.2
Isolated tracheal or bronchial tuberculosis, unspecified	012.20
Isolated tracheal or bronchial tuberculosis, bacteriological or histological examination not done	012.21
Isolated tracheal or bronchial tuberculosis, bacteriological or histological examination unknown	012.22
Isolated tracheal or bronchial tuberculosis, tubercle bacilli found (in sputum) by microscopy	012.23
Isolated tracheal or bronchial tuberculosis, bacilli not found by microscopy, but found by bacterial culture	012.24
Isolated tracheal or bronchial tuberculosis, bacilli not found, confirmed histologically	012.25
Isolated tracheal or bronchial tuberculosis, bacilli not found, confirmed by other methods (e.g., inoculation of animals)	012.26
Tuberculous laryngitis	012.3
Tuberculous laryngitis, unspecified	012.30
Tuberculous laryngitis, bacteriological or histological examination not done	012.31
Tuberculous laryngitis, bacteriological or histological examination unknown	012.32

Tuberculous laryngitis, tubercle bacilli found (in sputum) by microscopy	012.33
Tuberculous laryngitis, bacilli not found by microscopy, but found by bacterial culture	012.34
Tuberculous laryngitis, bacilli not found, confirmed histologically	012.35
Tuberculous laryngitis, bacilli not found, confirmed by other methods (e.g., inoculation of animals)	012.36
Other specified respiratory tuberculosis	012.8
Other specified respiratory tuberculosis, unspecified	012.80
Other specified respiratory tuberculosis, bacteriological or histological examination not done	012.81
Other specified respiratory tuberculosis, bacteriological or histological examination unknown	012.82
Other specified respiratory tuberculosis, tubercle bacilli found (in sputum) by microscopy	012.83
Other specified respiratory tuberculosis, bacilli not found by microscopy, but found by bacterial culture	012.84
Other specified respiratory tuberculosis, bacilli not found, confirmed histologically	012.85
Other specified respiratory tuberculosis, bacilli not found, confirmed by other methods (e.g., inoculation of animals)	012.86
Tuberculosis of meninges and central nervous system	013
Tuberculous meningitis	013.0
Tuberculous meningitis, unspecified	013.00
Tuberculous meningitis, bacteriological or histological examination not done	013.01
Tuberculous meningitis, bacteriological or histological examination unknown	013.02
Tuberculous meningitis, tubercle bacilli found (in sputum) by microscopy	013.03
Tuberculous meningitis, bacilli not found by microscopy, but found by bacterial culture	013.04
Tuberculous meningitis, bacilli not found, confirmed histologically	013.05
Tuberculous meningitis, bacilli not found, confirmed by other methods (e.g., inoculation of animals)	013.06
Tuberculoma of meninges	013.1
Tuberculoma of meninges, unspecified	013.10

Tuberculoma of meninges, bacteriological or histological examination not done	013.11
Tuberculoma of meninges, bacteriological or histological examination unknown	013.12
Tuberculoma of meninges, tubercle bacilli found (in sputum) by microscopy	013.13
Tuberculoma of meninges, bacilli not found by microscopy, but found by bacterial culture	013.14
Tuberculoma of meninges, bacilli not found by bacteriological exam, but confirmed histologically	013.15
Tuberculoma of meninges, bacilli not found by bacteriological or histological exam, but confirmed by other methods	013.16
Tuberculoma of brain	013.2
Tuberculoma of brain, unspecified	013.20
Tuberculoma of brain, bacteriological or histological examination not done	013.21
Tuberculoma of brain, bacteriological or histological examination unknown	013.22
Tuberculoma of brain, tubercle bacilli found (in sputum) by microscopy	013.23
Tuberculoma of brain, bacilli not found by microscopy, but found by bacterial culture	013.24
Tuberculoma of brain, bacilli not found by bacteriological exam, but confirmed histologically	013.25
Tuberculoma of brain, bacilli not found by bacteriological or histological exam, but confirmed by other methods	013.26
Tuberculous abscess of brain	013.3
Tuberculous abscess of brain, unspecified	013.30
Tuberculous abscess of brain, bacteriological or histological examination not done	013.31
Tuberculous abscess of brain, bacteriological or histological examination unknown	013.32
Tuberculous abscess of brain, tubercle bacilli found (in sputum) by microscopy	013.33
Tuberculous abscess of brain, bacilli not found by microscopy, but found by bacterial culture	013.34
Tuberculous abscess of brain, bacilli not found by bacteriological exam, but confirmed histologically	013.35

Tuberculous abscess of brain, bacilli not found by bacteriological or histological exam, but confirmed by other methods	013.36
Tuberculoma of spinal cord	013.4
Tuberculoma of spinal cord, unspecified	013.40
Tuberculoma of spinal cord, bacteriological or histological examination not done	013.41
Tuberculoma of spinal cord, bacteriological or histological examination unknown	013.42
Tuberculoma of spinal cord, tubercle bacilli found (in sputum) by microscopy	013.43
Tuberculoma of spinal cord, bacilli not found by microscopy, but found by bacterial culture	013.44
Tuberculoma of spinal cord, bacilli not found by bacteriological exam, but confirmed histologically	013.45
Tuberculoma of spinal cord, bacilli not found by bacteriological or histological exam, but confirmed by other methods	013.46
Tuberculous abscess of spinal cord	013.5
Tuberculous abscess of spinal cord, unspecified	013.50
Tuberculous abscess of spinal cord, bacteriological or histological examination not done	013.51
Tuberculous abscess of spinal cord, bacteriological or histological examination unknown	013.52
Tuberculous abscess of spinal cord, tubercle bacilli found (in sputum) by microscopy	013.53
Tuberculous abscess of spinal cord, bacilli not found by microscopy, but found by bacterial culture	013.54
Tuberculous abscess of spinal cord, bacilli not found by bacteriological exam, but confirmed histologically	013.55
Tuberculous abscess of spinal cord, bacilli not found by bacteriological or histological exam, but confirmed by other methods	013.56
Tuberculous encephalitis or myelitis	013.6
Tuberculous encephalitis or myelitis, unspecified	013.60
Tuberculous encephalitis or myelitis, bacteriological or histological examination not done	013.61
Tuberculous encephalitis or myelitis, bacteriological or histological examination unknown	013.62

Tuberculous encephalitis or myelitis, tubercle bacilli found (in sputum) by microscopy	013.63
Tuberculous encephalitis or myelitis, bacilli not found by microscopy, but found by bacterial culture	013.64
Tuberculous encephalitis or myelitis, bacilli not found by bacteriological exam, but confirmed histologically	013.65
Tuberculous encephalitis or myelitis, bacilli not found by bacteriological or histological exam, but confirmed by other methods	013.66
Other specified tuberculosis of central nervous system	013.8
Other specified tuberculosis of CNS, unspecified	013.80
Other specified tuberculosis of CNS, bacteriological or histological examination not done	013.81
Other specified tuberculosis of CNS, bacteriological or histological examination unknown	013.82
Other specified tuberculosis of CNS, tubercle bacilli found (in sputum) by microscopy	013.83
Other specified tuberculosis of CNS, bacilli not found by microscopy, but found by bacterial culture	013.84
Other specified tuberculosis of CNS, bacilli not found by bacteriological exam, but confirmed histologically	013.85
Other specified tuberculosis of CNS, bacilli not found by bacteriological or histological exam, but confirmed by other methods	013.86
Unspecified tuberculosis of central nervous system	013.9
Unspecified tuberculosis of CNS, unspecified	013.90
Unspecified tuberculosis of CNS, bacteriological or histological examination not done	013.91
Unspecified tuberculosis of CNS, bacteriological or histological examination unknown	013.92
Unspecified tuberculosis of CNS, tubercle bacilli found (in sputum) by microscopy	013.93
Unspecified tuberculosis of CNS, bacilli not found by microscopy, but found by bacterial culture	013.94
Unspecified tuberculosis of CNS, bacilli not found by bacteriological exam, but confirmed histologically	013.95
Unspecified tuberculosis of CNS, bacilli not found by bacteriological or histological exam, but confirmed by other methods	013.96

Tuberculosis of intestines, peritoneum, and mesenteric glands	014
Tuberculous peritonitis	014.0
Tuberculous peritonitis, unspecified	014.00
Tuberculous peritonitis, bacteriological or histological examination not done	014.01
Tuberculous peritonitis, bacteriological or histological examination unknown (at present)	014.02
Tuberculous peritonitis, tubercle bacilli found (in sputum) by microscopy	014.03
Tuberculous peritonitis, bacilli not found by microscopy but found by bacterial culture	014.04
Tuberculous peritonitis, bacilli not found by bacteriological exam, but tuberculosis confirmed histologically	014.05
Tuberculous peritonitis, bacilli not found by bacteriological or histological exam, but confirmed by other methods (e.g., inoculation)	014.06
Other tuberculosis of intestines, peritoneum, and mesenteric glands	014.8
Other TB of intestines, peritoneum, and mesenteric glands, unspecified	014.80
Other TB of intestines, peritoneum, and mesenteric glands, bacteriological or histological exam not done	014.81
Other TB of intestines, peritoneum, and mesenteric glands, bacteriological or histological exam unknown	014.82
Other TB of intestines, peritoneum, and mesenteric glands, tubercle bacilli found by microscopy	014.83
Other TB of intestines, peritoneum, and mesenteric glands, bacilli not found by microscopy, but by culture	014.84
Other TB of intestines, peritoneum, and mesenteric glands, bacilli not found by exam, confirmed histologically	014.85
Other TB of intestines, peritoneum, and mesenteric glands, bacilli not found by exam, confirmed by other methods	014.86
Tuberculosis of bones and joints	015
Tuberculosis of vertebral column	015.0
Tuberculosis of vertebral column, unspecified	015.00

Tuberculosis of vertebral column, bacteriological or histological examination not done	015.01
Tuberculosis of vertebral column, bacteriological or histological examination unknown (present)	015.02
Tuberculosis of vertebral column, tubercle bacilli found by microscopy	015.03
Tuberculosis of vertebral column, bacilli not found by microscopy, but found by culture	015.04
Tuberculosis of vertebral column, bacilli not found by exam, confirmed histologically	015.05
Tuberculosis of vertebral column, bacilli not found by exam, confirmed by other methods	015.06
Tuberculosis of hip	015.1
Tuberculosis of hip, unspecified	015.10
Tuberculosis of hip, bacteriological or histological examination not done	015.11
Tuberculosis of hip, bacteriological or histological examination unknown (present)	015.12
Tuberculosis of hip, tubercle bacilli found by microscopy	015.13
Tuberculosis of hip, bacilli not found by microscopy, but found by culture	015.14
Tuberculosis of hip, bacilli not found by exam, confirmed histologically	015.15
Tuberculosis of hip, bacilli not found by exam, confirmed by other methods	015.16
Tuberculosis of knee	015.2
Tuberculosis of knee, unspecified	015.20
Tuberculosis of knee, bacteriological or histological examination not done	015.21
Tuberculosis of knee, bacteriological or histological examination unknown (present)	015.22
Tuberculosis of knee, tubercle bacilli found by microscopy	015.23
Tuberculosis of knee, bacilli not found by microscopy, but found by culture	015.24
Tuberculosis of knee, bacilli not found by exam, confirmed histologically	015.25
Tuberculosis of knee, bacilli not found by exam, confirmed by other methods	015.26
Tuberculosis of limb bones	015.5

Tuberculosis of limb bones, unspecified	015.50
Tuberculosis of limb bones, bacteriological or histological examination not done	015.51
Tuberculosis of limb bones, bacteriological or histological examination unknown (at present)	015.52
Tuberculosis of limb bones, tubercle bacilli found (in sputum) by microscopy	015.53
Tuberculosis of limb bones, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	015.54
Tuberculosis of limb bones, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	015.55
Tuberculosis of limb bones, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	015.56
Tuberculosis of mastoid	015.6
Tuberculosis of mastoid, unspecified	015.60
Tuberculosis of mastoid, bacteriological or histological examination not done	015.61
Tuberculosis of mastoid, bacteriological or histological examination unknown (at present)	015.62
Tuberculosis of mastoid, tubercle bacilli found (in sputum) by microscopy	015.63
Tuberculosis of mastoid, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	015.64
Tuberculosis of mastoid, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	015.65
Tuberculosis of mastoid, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	015.66
Tuberculosis of other specified bone	015.7
Tuberculosis of other specified bone, unspecified	015.70
Tuberculosis of other specified bone, bacteriological or histological examination not done	015.71
Tuberculosis of other specified bone, bacteriological or histological examination unknown (at present)	015.72

Tuberculosis of other specified bone, tubercle bacilli found (in sputum) by microscopy	015.73
Tuberculosis of other specified bone, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	015.74
Tuberculosis of other specified bone, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	015.75
Tuberculosis of other specified bone, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	015.76
Tuberculosis of other specified joint	015.8
Tuberculosis of other specified joint, unspecified	015.80
Tuberculosis of other specified joint, bacteriological or histological examination not done	015.81
Tuberculosis of other specified joint, bacteriological or histological examination unknown (at present)	015.82
Tuberculosis of other specified joint, tubercle bacilli found (in sputum) by microscopy	015.83
Tuberculosis of other specified joint, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	015.84
Tuberculosis of other specified joint, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	015.85
Tuberculosis of other specified joint, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	015.86
Tuberculosis of unspecified bones and joints	015.9
Tuberculosis of unspecified bones and joints, unspecified	015.90
Tuberculosis of unspecified bones and joints, bacteriological or histological examination not done	015.91
Tuberculosis of unspecified bones and joints, bacteriological or histological examination unknown (at present)	015.92
Tuberculosis of unspecified bones and joints, tubercle bacilli found (in sputum) by microscopy	015.93

Tuberculosis of unspecified bones and joints, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	015.94
Tuberculosis of unspecified bones and joints, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	015.95
Tuberculosis of unspecified bones and joints, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	015.96
Tuberculosis of genitourinary system	016
Tuberculosis of kidney	016.0
Tuberculosis of kidney, unspecified	016.00
Tuberculosis of kidney, bacteriological or histological examination not done	016.01
Tuberculosis of kidney, bacteriological or histological examination unknown (at present)	016.02
Tuberculosis of kidney, tubercle bacilli found (in sputum) by microscopy	016.03
Tuberculosis of kidney, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	016.04
Tuberculosis of kidney, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	016.05
Tuberculosis of kidney, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	016.06
Tuberculosis of bladder	016.1
Tuberculosis of bladder, unspecified	016.10
Tuberculosis of bladder, bacteriological or histological examination not done	016.11
Tuberculosis of bladder, bacteriological or histological examination unknown (at present)	016.12
Tuberculosis of bladder, tubercle bacilli found (in sputum) by microscopy	016.13
Tuberculosis of bladder, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	016.14

Tuberculosis of bladder, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	016.15
Tuberculosis of bladder, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	016.16
Tuberculosis of ureter	016.2
Tuberculosis of ureter, unspecified	016.20
Tuberculosis of ureter, bacteriological or histological examination not done	016.21
Tuberculosis of ureter, bacteriological or histological examination unknown (at present)	016.22
Tuberculosis of ureter, tubercle bacilli found (in sputum) by microscopy	016.23
Tuberculosis of ureter, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	016.24
Tuberculosis of ureter, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	016.25
Tuberculosis of ureter, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	016.26
Tuberculosis of other urinary organs	016.3
Tuberculosis of other urinary organs, unspecified	016.30
Tuberculosis of other urinary organs, bacteriological or histological examination not done	016.31
Tuberculosis of other urinary organs, bacteriological or histological examination unknown (at present)	016.32
Tuberculosis of other urinary organs, tubercle bacilli found (in sputum) by microscopy	016.33
Tuberculosis of other urinary organs, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	016.34
Tuberculosis of other urinary organs, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	016.35

Tuberculosis of other urinary organs, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	016.36
Tuberculosis of epididymis	016.4
Tuberculosis of epididymis, unspecified	016.40
Tuberculosis of epididymis, bacteriological or histological examination not done	016.41
Tuberculosis of epididymis, bacteriological or histological examination unknown (at present)	016.42
Tuberculosis of epididymis, tubercle bacilli found (in sputum) by microscopy	016.43
Tuberculosis of epididymis, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	016.44
Tuberculosis of epididymis, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	016.45
Tuberculosis of epididymis, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	016.46
Tuberculosis of other male genital organs	016.5
Tuberculosis of other male genital organs, unspecified	016.50
Tuberculosis of other male genital organs, bacteriological or histological examination not done	016.51
Tuberculosis of other male genital organs, bacteriological or histological examination unknown (at present)	016.52
Tuberculosis of other male genital organs, tubercle bacilli found (in sputum) by microscopy	016.53
Tuberculosis of other male genital organs, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	016.54
Tuberculosis of other male genital organs, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	016.55
Tuberculosis of other male genital organs, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	016.56

Tuberculous oophoritis and salpingitis	016.6
Tuberculous oophoritis and salpingitis, unspecified	016.60
Tuberculous oophoritis and salpingitis, bacteriological or histological examination not done	016.61
Tuberculous oophoritis and salpingitis, bacteriological or histological examination unknown (at present)	016.62
Tuberculous oophoritis and salpingitis, tubercle bacilli found (in sputum) by microscopy	016.63
Tuberculous oophoritis and salpingitis, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	016.64
Tuberculous oophoritis and salpingitis, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	016.65
Tuberculous oophoritis and salpingitis, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	016.66
Tuberculosis of other female genital organs	016.7
Tuberculosis of other female genital organs, unspecified	016.70
Tuberculosis of other female genital organs, bacteriological or histological examination not done	016.71
Tuberculosis of other female genital organs, bacteriological or histological examination unknown (at present)	016.72
Tuberculosis of other female genital organs, tubercle bacilli found (in sputum) by microscopy	016.73
Tuberculosis of other female genital organs, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	016.74
Tuberculosis of other female genital organs, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	016.75
Tuberculosis of other female genital organs, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	016.76
Genitourinary tuberculosis unspecified	016.9
Genitourinary tuberculosis, unspecified, unspecified	016.90

Genitourinary tuberculosis, unspecified, bacteriological or histological examination not done	016.91
Genitourinary tuberculosis, unspecified, bacteriological or histological examination unknown (at present)	016.92
Genitourinary tuberculosis, unspecified, tubercle bacilli found (in sputum) by microscopy	016.93
Genitourinary tuberculosis, unspecified, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	016.94
Genitourinary tuberculosis, unspecified, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	016.95
Genitourinary tuberculosis, unspecified, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	016.96
Tuberculosis of other organs	017
Tuberculosis of skin and subcutaneous cellular tissue	017.0
Tuberculosis of skin and subcutaneous cellular tissue, unspecified	017.00
Tuberculosis of skin and subcutaneous cellular tissue, bacteriological or histological examination not done	017.01
Tuberculosis of skin and subcutaneous cellular tissue, bacteriological or histological examination unknown (at present)	017.02
Tuberculosis of skin and subcutaneous cellular tissue, tubercle bacilli found (in sputum) by microscopy	017.03
Tuberculosis of skin and subcutaneous cellular tissue, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	017.04
Tuberculosis of skin and subcutaneous cellular tissue, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	017.05
Tuberculosis of skin and subcutaneous cellular tissue, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	017.06

Erythema nodosum with hypersensitivity reaction in tuberculosis	017.1
Erythema nodosum with hypersensitivity reaction in tuberculosis, unspecified	017.10
Erythema nodosum with hypersensitivity reaction in tuberculosis, bacteriological or histological examination not done	017.11
Erythema nodosum with hypersensitivity reaction in tuberculosis, bacteriological or histological examination unknown (at present)	017.12
Erythema nodosum with hypersensitivity reaction in tuberculosis, tubercle bacilli found (in sputum) by microscopy	017.13
Erythema nodosum with hypersensitivity reaction in tuberculosis, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	017.14
Erythema nodosum with hypersensitivity reaction in tuberculosis, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	017.15
Erythema nodosum with hypersensitivity reaction in tuberculosis, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	017.16
Tuberculosis of peripheral lymph nodes	017.2
Tuberculosis of peripheral lymph nodes, unspecified	017.20
Tuberculosis of peripheral lymph nodes, bacteriological or histological examination not done	017.21
Tuberculosis of peripheral lymph nodes, bacteriological or histological examination unknown (at present)	017.22
Tuberculosis of peripheral lymph nodes, tubercle bacilli found (in sputum) by microscopy	017.23
Tuberculosis of peripheral lymph nodes, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	017.24
Tuberculosis of peripheral lymph nodes, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	017.25

Tuberculosis of peripheral lymph nodes, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	017.26
Tuberculosis of eye	017.3
Tuberculosis of eye, unspecified	017.30
Tuberculosis of eye, bacteriological or histological examination not done	017.31
Tuberculosis of eye, bacteriological or histological examination unknown (at present)	017.32
Tuberculosis of eye, tubercle bacilli found (in sputum) by microscopy	017.33
Tuberculosis of eye, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	017.34
Tuberculosis of eye, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	017.35
Tuberculosis of eye, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	017.36
Tuberculosis of ear	017.4
Tuberculosis of ear, unspecified	017.40
Tuberculosis of ear, bacteriological or histological examination not done	017.41
Tuberculosis of ear, bacteriological or histological examination unknown (at present)	017.42
Tuberculosis of ear, tubercle bacilli found (in sputum) by microscopy	017.43
Tuberculosis of ear, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	017.44
Tuberculosis of ear, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	017.45
Tuberculosis of ear, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	017.46
Tuberculosis of thyroid gland	017.5
Tuberculosis of thyroid gland, unspecified	017.50

Tuberculosis of thyroid gland, bacteriological or histological examination not done	017.51
Tuberculosis of thyroid gland, bacteriological or histological examination unknown (at present)	017.52
Tuberculosis of thyroid gland, tubercle bacilli found (in sputum) by microscopy	017.53
Tuberculosis of thyroid gland, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	017.54
Tuberculosis of thyroid gland, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	017.55
Tuberculosis of thyroid gland, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	017.56
Tuberculosis of adrenal glands	017.6
Tuberculosis of adrenal glands, unspecified	017.60
Tuberculosis of adrenal glands, bacteriological or histological examination not done	017.61
Tuberculosis of adrenal glands, bacteriological or histological examination unknown (at present)	017.62
Tuberculosis of adrenal glands, tubercle bacilli found (in sputum) by microscopy	017.63
Tuberculosis of adrenal glands, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	017.64
Tuberculosis of adrenal glands, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	017.65
Tuberculosis of adrenal glands, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	017.66
Tuberculosis of spleen	017.7
Tuberculosis of spleen, unspecified	017.70
Tuberculosis of spleen, bacteriological or histological examination not done	017.71
Tuberculosis of spleen, bacteriological or histological examination unknown (at present)	017.72
Tuberculosis of spleen, tubercle bacilli found (in sputum) by microscopy	017.73

Tuberculosis of spleen, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	017.74
Tuberculosis of spleen, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	017.75
Tuberculosis of spleen, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	017.76
Tuberculosis of esophagus	017.8
Tuberculosis of esophagus, unspecified	017.80
Tuberculosis of esophagus, bacteriological or histological examination not done	017.81
Tuberculosis of esophagus, bacteriological or histological examination unknown (at present)	017.82
Tuberculosis of esophagus, tubercle bacilli found (in sputum) by microscopy	017.83
Tuberculosis of esophagus, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	017.84
Tuberculosis of esophagus, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	017.85
Tuberculosis of esophagus, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	017.86
Tuberculosis of other specified organs	017.9
Tuberculosis of other specified organs, unspecified	017.90
Tuberculosis of other specified organs, bacteriological or histological examination not done	017.91
Tuberculosis of other specified organs, bacteriological or histological examination unknown (at present)	017.92
Tuberculosis of other specified organs, tubercle bacilli found (in sputum) by microscopy	017.93
Tuberculosis of other specified organs, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	017.94

Tuberculosis of other specified organs, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	017.95
Tuberculosis of other specified organs, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	017.96
Miliary tuberculosis	018
Acute miliary tuberculosis	018.0
Acute miliary tuberculosis, unspecified	018.00
Acute miliary tuberculosis, bacteriological or histological examination not done	018.01
Acute miliary tuberculosis, bacteriological or histological examination unknown (at present)	018.02
Acute miliary tuberculosis, tubercle bacilli found (in sputum) by microscopy	018.03
Acute miliary tuberculosis, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	018.04
Acute miliary tuberculosis, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	018.05
Acute miliary tuberculosis, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	018.06
Other specified miliary tuberculosis	018.8
Other specified miliary tuberculosis, unspecified	018.80
Other specified miliary tuberculosis, bacteriological or histological examination not done	018.81
Other specified miliary tuberculosis, bacteriological or histological examination unknown (at present)	018.82
Other specified miliary tuberculosis, tubercle bacilli found (in sputum) by microscopy	018.83
Other specified miliary tuberculosis, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	018.84
Other specified miliary tuberculosis, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	018.85

Other specified miliary tuberculosis, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	018.86
Unspecified miliary tuberculosis	018.9
Miliary tuberculosis, unspecified, unspecified	018.90
Miliary tuberculosis, unspecified, bacteriological or histological examination not done	018.91
Miliary tuberculosis, unspecified, bacteriological or histological examination unknown (at present)	018.92
Miliary tuberculosis, unspecified, tubercle bacilli found (in sputum) by microscopy	018.93
Miliary tuberculosis, unspecified, tubercle bacilli not found (in sputum) by microscopy, but found by bacterial culture	018.94
Miliary tuberculosis, unspecified, tubercle bacilli not found by bacteriological examination, but tuberculosis confirmed histologically	018.95
Miliary tuberculosis, unspecified, tubercle bacilli not found by bacteriological or histological examination, but tuberculosis confirmed by other methods [inoculation of animals]	018.96
Plague	020
Bubonic plague	020.0
Cellulocutaneous plague	020.1
Septicemic plague	020.2
Primary pneumonic plague	020.3
Secondary pneumonic plague	020.4
Pneumonic plague, unspecified	020.5
Other specified types of plague	020.8
Plague, unspecified	020.9
Tularemia	021
Ulceroglandular tularemia	021.0
Enteric tularemia	021.1
Pulmonary tularemia	021.2
Oculoglandular tularemia	021.3
Other specified tularemia	021.8
Unspecified tularemia	021.9
Anthrax	022
Cutaneous anthrax	022.0

Pulmonary anthrax	022.1
Gastrointestinal anthrax	022.2
Anthrax septicemia	022.3
Other specified manifestations of anthrax	022.8
Anthrax, unspecified	022.9
Brucellosis	023
Brucella melitensis	023.0
Brucella abortus	023.1
Brucella suis	023.2
Brucella canis	023.3
Other brucellosis	023.8
Brucellosis, unspecified	023.9
Glanders	024
Melioidosis	025
Rat-bite fever	026
Spirillary fever	026.0
Streptobacillary fever	026.1
Unspecified rat-bite fever	026.9
Other zoonotic bacterial diseases	027
Listeriosis	027.0
Erysipelothrix infection	027.1
Pasteurellosis	027.2
Other specified zoonotic bacterial diseases	027.8
Unspecified zoonotic bacterial disease	027.9
Leprosy	030
Lepromatous leprosy [type L]	030.0
Tuberculoid leprosy [type T]	030.1
Indeterminate leprosy [group I]	030.2
Borderline leprosy [group B]	030.3
Other specified leprosy	030.8
Leprosy, unspecified	030.9
Diseases due to other mycobacteria	031
Pulmonary diseases due to other mycobacteria	031.0
Cutaneous diseases due to other mycobacteria	031.1
Disseminated due to other mycobacteria	031.2
Other specified mycobacterial diseases	031.8
Unspecified diseases due to mycobacteria	031.9
Diphtheria	032

Faucial diphtheria	032.0
Nasopharyngeal diphtheria	032.1
Anterior nasal diphtheria	032.2
Laryngeal diphtheria	032.3
Other specified diphtheria	032.8
Conjunctival diphtheria	032.81
Diphtheritic myocarditis	032.82
Diphtheritic peritonitis	032.83
Diphtheritic cystitis	032.84
Cutaneous diphtheria	032.85
Other specified diphtheria	032.89
Diphtheria, unspecified	032.9
Whooping cough	033
Whooping cough due to bordetella pertussis [B. pertussis]	033.0
Whooping cough due to bordetella parapertussis [B. parapertussis]	033.1
Whooping cough due to other specified organism	033.8
Whooping cough, unspecified organism	033.9
Streptococcal sore throat and scarlet fever	034
Streptococcal sore throat	034.0
Scarlet fever	034.1
Erysipelas	035
Meningococcal infection	036
Meningococcal meningitis	036.0
Meningococcal encephalitis	036.1
Meningococemia	036.2
Waterhouse-Friderichsen syndrome, meningococcal	036.3
Meningococcal carditis	036.4
Meningococcal carditis, unspecified	036.40
Meningococcal pericarditis	036.41
Meningococcal endocarditis	036.42
Meningococcal myocarditis	036.43
Other specified meningococcal infections	036.8
Meningococcal optic neuritis	036.81
Meningococcal arthropathy	036.82
Other specified meningococcal infections	036.89

Meningococcal infection, unspecified	036.9
Tetanus	037
Actinomycotic infections	039
Cutaneous actinomycotic infection	039.0
Pulmonary actinomycotic infection	039.1
Abdominal actinomycotic infection	039.2
Cervicofacial actinomycotic infection	039.3
Madura foot	039.4
Actinomycotic infection of other specified sites	039.8
Actinomycotic infection of unspecified site	039.9
Other bacterial diseases	040
Gas gangrene	040.0
Rhinoscleroma	040.1
Whipple's disease	040.2
Necrobacillosis	040.3
Other specified botulism	040.4
Infant botulism	040.41
Wound botulism	040.42
Other specified bacterial diseases	040.8
Tropical pyomyositis	040.81
Toxic shock syndrome	040.82
Other specified bacterial diseases	040.89
Bacterial infection in conditions classified elsewhere and of unspecified site	041
Streptococcus infection in conditions classified elsewhere and of unspecified site	041.0
Streptococcus infection in conditions classified elsewhere and of unspecified site, streptococcus, unspecified	041.00
Streptococcus infection in conditions classified elsewhere and of unspecified site, streptococcus, group A	041.01
Streptococcus infection in conditions classified elsewhere and of unspecified site, streptococcus, group B	041.02
Streptococcus infection in conditions classified elsewhere and of unspecified site, streptococcus, group C	041.03

Streptococcus infection in conditions classified elsewhere and of unspecified site, streptococcus, group D [Enterococcus]	041.04
Streptococcus infection in conditions classified elsewhere and of unspecified site, streptococcus, group G	041.05
Streptococcus infection in conditions classified elsewhere and of unspecified site, other streptococcus	041.09
Staphylococcus infection in conditions classified elsewhere and of unspecified site	041.1
Staphylococcus infection in conditions classified elsewhere and of unspecified site, staphylococcus, unspecified	041.10
Methicillin susceptible Staphylococcus aureus in conditions classified elsewhere and of unspecified site	041.11
Methicillin resistant Staphylococcus aureus in conditions classified elsewhere and of unspecified site	041.12
Staphylococcus infection in conditions classified elsewhere and of unspecified site, other staphylococcus	041.19
Pneumococcus infection in conditions classified elsewhere and of unspecified site	041.2
Friedländer's bacillus infection in conditions classified elsewhere and of unspecified site	041.3
Escherichia coli [e. coli] infection in conditions classified elsewhere and of unspecified site	041.4
Shiga toxin-producing Escherichia coli [E. coli] (STEC) O157	041.41
Other specified Shiga toxin-producing Escherichia coli [E. coli] (STEC)	041.42
Shiga toxin-producing Escherichia coli [E. coli] (STEC), unspecified	041.43
Other and unspecified Escherichia coli [E. coli]	041.49
Hemophilus influenzae [H. influenzae] infection in conditions classified elsewhere and of unspecified site	041.5
Proteus (mirabilis) (morganii) infection in conditions classified elsewhere and of unspecified site	041.6

Pseudomonas infection in conditions classified elsewhere and of unspecified site	041.7
Other specified bacterial infections in conditions classified elsewhere and of unspecified site	041.8
Other specified bacterial infections in conditions classified elsewhere and of unspecified site, mycoplasma	041.81
Bacteroides fragilis	041.82
Other specified bacterial infections in conditions classified elsewhere and of unspecified site, Clostridium perfringens	041.83
Other specified bacterial infections in conditions classified elsewhere and of unspecified site, other anaerobes	041.84
Other specified bacterial infections in conditions classified elsewhere and of unspecified site, other gram-negative organisms	041.85
Helicobacter pylori [H. pylori]	041.86
Other specified bacterial infections in conditions classified elsewhere and of unspecified site, other specified bacteria	041.89
Bacterial infection, unspecified, in conditions classified elsewhere and of unspecified site	041.9
Acute poliomyelitis	045
Acute paralytic poliomyelitis specified as bulbar	045.0
Acute paralytic poliomyelitis specified as bulbar, poliovirus, unspecified type	045.00
Acute paralytic poliomyelitis specified as bulbar, poliovirus type I	045.01
Acute paralytic poliomyelitis specified as bulbar, poliovirus type II	045.02
Acute paralytic poliomyelitis specified as bulbar, poliovirus type III	045.03
Acute poliomyelitis with other paralysis	045.1
Acute poliomyelitis with other paralysis, poliovirus, unspecified type	045.10
Acute poliomyelitis with other paralysis, poliovirus type I	045.11
Acute poliomyelitis with other paralysis, poliovirus type II	045.12

Acute poliomyelitis with other paralysis, poliovirus type III	045.13
Acute nonparalytic poliomyelitis	045.2
Acute nonparalytic poliomyelitis, poliovirus, unspecified type	045.20
Acute nonparalytic poliomyelitis, poliovirus type I	045.21
Acute nonparalytic poliomyelitis, poliovirus type II	045.22
Acute nonparalytic poliomyelitis, poliovirus type III	045.23
Unspecified acute poliomyelitis	045.9
Acute poliomyelitis, unspecified, poliovirus, unspecified type	045.90
Acute poliomyelitis, unspecified, poliovirus type I	045.91
Acute poliomyelitis, unspecified, poliovirus type II	045.92
Acute poliomyelitis, unspecified, poliovirus type III	045.93
Slow virus infection and prion diseases of central nervous system	046
Kuru	046.0
Jakob-creutzfeldt disease	046.1
Variant Creutzfeldt-Jakob disease	046.11
Other and unspecified Creutzfeldt-Jakob disease	046.19
Subacute sclerosing panencephalitis	046.2
Progressive multifocal leukoencephalopathy	046.3
Other specified prion diseases of central nervous system	046.7
Gerstmann-Sträussler-Scheinker syndrome	046.71
Fatal familial insomnia	046.72
Other and unspecified prion disease of central nervous system	046.79
Other specified slow virus infection of central nervous system	046.8
Unspecified slow virus infection of central nervous system	046.9
Meningitis due to enterovirus	047
Meningitis due to coxsackie virus	047.0
Meningitis due to echo virus	047.1
Other specified viral meningitis	047.8
Unspecified viral meningitis	047.9
Other enterovirus diseases of central nervous system	048

Other non-arthropod-borne viral diseases of central nervous system	049
Lymphocytic choriomeningitis	049.0
Meningitis due to adenovirus	049.1
Other specified non-arthropod-borne viral diseases of central nervous system	049.8
Unspecified non-arthropod-borne viral diseases of central nervous system	049.9
Smallpox	050
Variola major	050.0
Alastrim	050.1
Modified smallpox	050.2
Smallpox, unspecified	050.9
Cowpox and paravaccinia	051
Cowpox and vaccinia not from vaccination	051.0
Cowpox	051.01
Vaccinia not from vaccination	051.02
Pseudocowpox	051.1
Contagious pustular dermatitis	051.2
Paravaccinia, unspecified	051.9
Chickenpox	052
Postvaricella encephalitis	052.0
Varicella (hemorrhagic) pneumonitis	052.1
Postvaricella myelitis	052.2
Chickenpox with other specified complications	052.7
Chickenpox with unspecified complication	052.8
Varicella without mention of complication	052.9
Herpes zoster	053
Herpes zoster with meningitis	053.0
Herpes zoster with other nervous system complications	053.1
Herpes zoster with unspecified nervous system complication	053.10
Geniculate herpes zoster	053.11
Postherpetic trigeminal neuralgia	053.12
Postherpetic polyneuropathy	053.13
Herpes zoster myelitis	053.14
Herpes zoster with other nervous system complications	053.19

Herpes zoster with ophthalmic complications	053.2
Herpes zoster dermatitis of eyelid	053.20
Herpes zoster keratoconjunctivitis	053.21
Herpes zoster iridocyclitis	053.22
Herpes zoster with other ophthalmic complications	053.29
Herpes zoster with other specified complications	053.7
Otitis externa due to herpes zoster	053.71
Herpes zoster with other specified complications	053.79
Herpes zoster with unspecified complication	053.8
Herpes zoster without mention of complication	053.9
Herpes simplex	054
Eczema herpeticum	054.0
Genital herpes	054.1
Genital herpes, unspecified	054.10
Herpetic vulvovaginitis	054.11
Herpetic ulceration of vulva	054.12
Herpetic infection of penis	054.13
Other genital herpes	054.19
Herpetic gingivostomatitis	054.2
Herpetic meningoencephalitis	054.3
Herpes simplex with ophthalmic complications	054.4
Herpes simplex with unspecified ophthalmic complication	054.40
Herpes simplex dermatitis of eyelid	054.41
Dendritic keratitis	054.42
Herpes simplex disciform keratitis	054.43
Herpes simplex iridocyclitis	054.44
Herpes simplex with other ophthalmic complications	054.49
Herpetic septicemia	054.5
Herpetic whitlow	054.6
Herpes simplex with other specified complications	054.7
Visceral herpes simplex	054.71
Herpes simplex meningitis	054.72
Herpes simplex otitis externa	054.73
Herpes simplex myelitis	054.74
Herpes simplex with other specified complications	054.79
Herpes simplex with unspecified complication	054.8
Herpes simplex without mention of complication	054.9

Measles	055
Postmeasles encephalitis	055.0
Postmeasles pneumonia	055.1
Postmeasles otitis media	055.2
Measles with other specified complications	055.7
Measles keratoconjunctivitis	055.71
Measles with other specified complications	055.79
Measles with unspecified complication	055.8
Measles without mention of complication	055.9
Rubella	056
Rubella with neurological complications	056.0
Rubella with unspecified neurological complication	056.00
Encephalomyelitis due to rubella	056.01
Rubella with other neurological complications	056.09
Rubella with other specified complications	056.7
Arthritis due to rubella	056.71
Rubella with other specified complications	056.79
Rubella with unspecified complications	056.8
Rubella without mention of complication	056.9
Other viral exanthemata	057
Erythema infectiosum (fifth disease)	057.0
Other specified viral exanthemata	057.8
Viral exanthem, unspecified	057.9
Other human herpesvirus	058
Roseola infantum	058.1
Roseola infantum, unspecified	058.10
Roseola infantum due to human herpesvirus 6	058.11
Roseola infantum due to human herpesvirus 7	058.12
Other human herpesvirus encephalitis	058.2
Human herpesvirus 6 encephalitis	058.21
Other human herpesvirus encephalitis	058.29
Other human herpesvirus infections	058.8
Human herpesvirus 6 infection	058.81
Human herpesvirus 7 infection	058.82
Other human herpesvirus infection	058.89
Other poxvirus infections	059
Other orthopoxvirus infections	059.0
Orthopoxvirus infection, unspecified	059.00

Monkeypox	059.01
Other orthopoxvirus infections	059.09
Other parapoxvirus infections	059.1
Parapoxvirus infection, unspecified	059.10
Bovine stomatitis	059.11
Sealpox	059.12
Other parapoxvirus infections	059.19
Yatapoxvirus infections	059.2
Yatapoxvirus infection, unspecified	059.20
Tanapox	059.21
Yaba monkey tumor virus	059.22
Other poxvirus infections	059.8
Poxvirus infections, unspecified	059.9
Yellow fever	060
Sylvatic yellow fever	060.0
Urban yellow fever	060.1
Yellow fever, unspecified	060.9
Dengue	061
Mosquito-borne viral encephalitis	062
Japanese encephalitis	062.0
Western equine encephalitis	062.1
Eastern equine encephalitis	062.2
St. Louis encephalitis	062.3
Australian encephalitis	062.4
California virus encephalitis	062.5
Other specified mosquito-borne viral encephalitis	062.8
Mosquito-borne viral encephalitis, unspecified	062.9
Tick-borne viral encephalitis	063
Russian spring-summer [taiga] encephalitis	063.0
Louping ill	063.1
Central european encephalitis	063.2
Other specified tick-borne viral encephalitis	063.8
Tick-borne viral encephalitis, unspecified	063.9
Viral encephalitis transmitted by other and unspecified arthropods	064
Arthropod-borne hemorrhagic fever	065
Crimean hemorrhagic fever [CHF Congo virus]	065.0
Omsk hemorrhagic fever	065.1

Kyasanur forest disease	065.2
Other tick-borne hemorrhagic fever	065.3
Mosquito-borne hemorrhagic fever	065.4
Other specified arthropod-borne hemorrhagic fever	065.8
Arthropod-borne hemorrhagic fever, unspecified	065.9
Other arthropod-borne viral diseases	066
Phlebotomus fever	066.0
Tick-borne fever	066.1
Venezuelan equine fever	066.2
Other mosquito-borne fever	066.3
West nile fever	066.4
West Nile Fever, unspecified	066.40
West Nile Fever with encephalitis	066.41
West Nile Fever with other neurologic manifestation	066.42
West Nile Fever with other complications	066.49
Other specified arthropod-borne viral diseases	066.8
Arthropod-borne viral disease, unspecified	066.9
Viral hepatitis	070
Viral hepatitis A with hepatic coma	070.0
Viral hepatitis A without mention of hepatic coma	070.1
Viral hepatitis b with hepatic coma	070.2
Viral hepatitis B with hepatic coma, acute or unspecified, without mention of hepatitis delta	070.20
Viral hepatitis B with hepatic coma, acute or unspecified, with hepatitis delta	070.21
Viral hepatitis b without mention of hepatic coma	070.3
Viral hepatitis B without mention of hepatic coma, acute or unspecified, without mention of hepatitis delta	070.30
Viral hepatitis B without mention of hepatic coma, acute or unspecified, with hepatitis delta	070.31
Other specified viral hepatitis with hepatic coma	070.4
Acute hepatitis C with hepatic coma	070.41
Hepatitis delta without mention of active hepatitis B disease with hepatic coma	070.42
Hepatitis E with hepatic coma	070.43
Other specified viral hepatitis with hepatic coma	070.49
Other specified viral hepatitis without mention of hepatic coma	070.5

Acute hepatitis C without mention of hepatic coma	070.51
Hepatitis delta without mention of active hepatitis B disease or hepatic coma	070.52
Hepatitis E without mention of hepatic coma	070.53
Other specified viral hepatitis without mention of hepatic coma	070.59
Unspecified viral hepatitis with hepatic coma	070.6
Unspecified viral hepatitis c	070.7
Unspecified viral hepatitis C without hepatic coma	070.70
Unspecified viral hepatitis C with hepatic coma	070.71
Unspecified viral hepatitis without mention of hepatic coma	070.9
Rabies	071
Mumps	072
Mumps orchitis	072.0
Mumps meningitis	072.1
Mumps encephalitis	072.2
Mumps pancreatitis	072.3
Mumps with other specified complications	072.7
Mumps hepatitis	072.71
Mumps polyneuropathy	072.72
Other mumps with other specified complications	072.79
Mumps with unspecified complication	072.8
Mumps without mention of complication	072.9
Ornithosis	073
Ornithosis with pneumonia	073.0
Ornithosis with other specified complications	073.7
Ornithosis with unspecified complication	073.8
Ornithosis, unspecified	073.9
Specific diseases due to coxsackie virus	074
Herpangina	074.0
Epidemic pleurodynia	074.1
Coxsackie carditis	074.2
Coxsackie carditis, unspecified	074.20
Coxsackie pericarditis	074.21
Coxsackie endocarditis	074.22
Coxsackie myocarditis	074.23
Hand, foot, and mouth disease	074.3

Other specified diseases due to Coxsackie virus	074.8
Infectious mononucleosis	075
Trachoma	076
Trachoma, initial stage	076.0
Trachoma, active stage	076.1
Trachoma, unspecified	076.9
Other diseases of conjunctiva due to viruses and chlamydiae	077
Inclusion conjunctivitis	077.0
Epidemic keratoconjunctivitis	077.1
Pharyngoconjunctival fever	077.2
Other adenoviral conjunctivitis	077.3
Epidemic hemorrhagic conjunctivitis	077.4
Other viral conjunctivitis	077.8
Unspecified diseases of conjunctiva due to viruses and chlamydiae	077.9
Unspecified diseases of conjunctiva due to chlamydiae	077.98
Unspecified diseases of conjunctiva due to viruses	077.99
Other diseases due to viruses and chlamydiae	078
Molluscum contagiosum	078.0
Viral warts	078.1
Viral warts, unspecified	078.10
Condyloma acuminatum	078.11
Plantar wart	078.12
Other specified viral warts	078.19
Sweating fever	078.2
Cat-scratch disease	078.3
Foot and mouth disease	078.4
Cytomegaloviral disease	078.5
Hemorrhagic nephrosonephritis	078.6
Arenaviral hemorrhagic fever	078.7
Other specified diseases due to viruses and chlamydiae	078.8
Epidemic vertigo	078.81
Epidemic vomiting syndrome	078.82
Other specified diseases due to chlamydiae	078.88
Other specified diseases due to viruses	078.89

Viral and chlamydial infection in conditions classified elsewhere and of unspecified site	079
Adenovirus infection in conditions classified elsewhere and of unspecified site	079.0
Echo virus infection in conditions classified elsewhere and of unspecified site	079.1
Coxsackie virus infection in conditions classified elsewhere and of unspecified site	079.2
Rhinovirus infection in conditions classified elsewhere and of unspecified site	079.3
Human papillomavirus in conditions classified elsewhere and of unspecified site	079.4
Retrovirus in conditions classified elsewhere and of unspecified site	079.5
Retrovirus, unspecified	079.50
Human T-cell lymphotropic virus, type I [HTLV-I]	079.51
Human T-cell lymphotropic virus, type II [HTLV-II]	079.52
Other specified retrovirus	079.59
Respiratory syncytial virus (RSV)	079.6
Other specified viral and chlamydial infections	079.8
Hantavirus infection	079.81
SARS-associated coronavirus	079.82
Parvovirus B19	079.83
Other specified chlamydial infection	079.88
Other specified viral infection	079.89
Unspecified viral and chlamydial infections	079.9
Unspecified chlamydial infection	079.98
Unspecified viral infection	079.99
Louse-borne (epidemic) typhus	080
Other typhus	081
Murine (endemic) typhus	081.0
Brill's disease	081.1
Scrub typhus	081.2
Typhus, unspecified	081.9
Tick-borne rickettsioses	082
Spotted fevers	082.0
Boutonneuse fever	082.1
North Asian tick fever	082.2
Queensland tick typhus	082.3

Ehrlichiosis	082.4
Ehrlichiosis, unspecified	082.40
Ehrlichiosis chafeensis [E. chafeensis]	082.41
Other ehrlichiosis	082.49
Other specified tick-borne rickettsioses	082.8
Tick-borne rickettsiosis, unspecified	082.9
Other rickettsioses	083
Q fever	083.0
Trench fever	083.1
Rickettsialpox	083.2
Other specified rickettsioses	083.8
Rickettsiosis, unspecified	083.9
Malaria	084
Falciparum malaria [malignant tertian]	084.0
Vivax malaria [benign tertian]	084.1
Quartan malaria	084.2
Ovale malaria	084.3
Other malaria	084.4
Mixed malaria	084.5
Malaria, unspecified	084.6
Induced malaria	084.7
Blackwater fever	084.8
Other pernicious complications of malaria	084.9
Leishmaniasis	085
Visceral [kala-azar] leishmaniasis	085.0
Cutaneous leishmaniasis, urban	085.1
Cutaneous leishmaniasis, Asian desert	085.2
Cutaneous leishmaniasis, Ethiopian	085.3
Cutaneous leishmaniasis, American	085.4
Mucocutaneous leishmaniasis, (American)	085.5
Leishmaniasis, unspecified	085.9
Trypanosomiasis	086
Chagas' disease with heart involvement	086.0
Chagas' disease with other organ involvement	086.1
Chagas' disease without mention of organ involvement	086.2
Gambian trypanosomiasis	086.3
Rhodesian trypanosomiasis	086.4

African trypanosomiasis, unspecified	086.5
Trypanosomiasis, unspecified	086.9
Relapsing fever	087
Relapsing fever, louse-borne	087.0
Relapsing fever, tick-borne	087.1
Relapsing fever, unspecified	087.9
Other arthropod-borne diseases	088
Bartonellosis	088.0
Other specified arthropod-borne diseases	088.8
Lyme Disease	088.81
Babesiosis	088.82
Other specified arthropod-borne diseases, other	088.89
Arthropod-borne disease, unspecified	088.9
Congenital syphilis	090
Early congenital syphilis, symptomatic	090.0
Early congenital syphilis, latent	090.1
Early congenital syphilis, unspecified	090.2
Syphilitic interstitial keratitis	090.3
Juvenile neurosyphilis	090.4
Juvenile neurosyphilis, unspecified	090.40
Congenital syphilitic encephalitis	090.41
Congenital syphilitic meningitis	090.42
Other juvenile neurosyphilis	090.49
Other late congenital syphilis, symptomatic	090.5
Late congenital syphilis, latent	090.6
Late congenital syphilis, unspecified	090.7
Congenital syphilis, unspecified	090.9
Early syphilis symptomatic	091
Genital syphilis (primary)	091.0
Primary anal syphilis	091.1
Other primary syphilis	091.2
Secondary syphilis of skin or mucous membranes	091.3
Adenopathy due to secondary syphilis	091.4
Uveitis due to secondary syphilis	091.5
Syphilitic uveitis, unspecified	091.50
Syphilitic chorioretinitis (secondary)	091.51
Syphilitic iridocyclitis (secondary)	091.52
Secondary syphilis of viscera and bone	091.6

Secondary syphilitic periostitis	091.61
Secondary syphilitic hepatitis	091.62
Secondary syphilis of other viscera	091.69
Secondary syphilis, relapse	091.7
Other forms of secondary syphilis	091.8
Acute syphilitic meningitis (secondary)	091.81
Syphilitic alopecia	091.82
Other forms of secondary syphilis	091.89
Unspecified secondary syphilis	091.9
Early syphilis latent	092
Early syphilis, latent, serological relapse after treatment	092.0
Early syphilis, latent, unspecified	092.9
Cardiovascular syphilis	093
Aneurysm of aorta, specified as syphilitic	093.0
Syphilitic aortitis	093.1
Syphilitic endocarditis	093.2
Syphilitic endocarditis of valve, unspecified	093.20
Syphilitic endocarditis of mitral valve	093.21
Syphilitic endocarditis of aortic valve	093.22
Syphilitic endocarditis of tricuspid valve	093.23
Syphilitic endocarditis of pulmonary valve	093.24
Other specified cardiovascular syphilis	093.8
Syphilitic pericarditis	093.81
Syphilitic myocarditis	093.82
Other specified cardiovascular syphilis	093.89
Cardiovascular syphilis, unspecified	093.9
Neurosyphilis	094
Tabes dorsalis	094.0
General paresis	094.1
Syphilitic meningitis	094.2
Asymptomatic neurosyphilis	094.3
Other specified neurosyphilis	094.8
Syphilitic encephalitis	094.81
Syphilitic parkinsonism	094.82
Syphilitic disseminated retinochoroiditis	094.83
Syphilitic optic atrophy	094.84
Syphilitic retrobulbar neuritis	094.85

Syphilitic acoustic neuritis	094.86
Syphilitic ruptured cerebral aneurysm	094.87
Other specified neurosyphilis	094.89
Neurosyphilis, unspecified	094.9
Other forms of late syphilis with symptoms	095
Syphilitic episcleritis	095.0
Syphilis of lung	095.1
Syphilitic peritonitis	095.2
Syphilis of liver	095.3
Syphilis of kidney	095.4
Syphilis of bone	095.5
Syphilis of muscle	095.6
Syphilis of synovium, tendon, and bursa	095.7
Other specified forms of late symptomatic syphilis	095.8
Late symptomatic syphilis, unspecified	095.9
Late syphilis, latent	096
Other and unspecified syphilis	097
Late syphilis, unspecified	097.0
Latent syphilis, unspecified	097.1
Syphilis, unspecified	097.9
Gonococcal infections	098
Gonococcal infection (acute) of lower genitourinary tract	098.0
Gonococcal infection (acute) of upper genitourinary tract	098.1
Gonococcal infection (acute) of upper genitourinary tract, site unspecified	098.10
Gonococcal cystitis (acute)	098.11
Gonococcal prostatitis (acute)	098.12
Gonococcal epididymo-orchitis (acute)	098.13
Gonococcal seminal vesiculitis (acute)	098.14
Gonococcal cervicitis (acute)	098.15
Gonococcal endometritis (acute)	098.16
Gonococcal salpingitis, specified as acute	098.17
Other gonococcal infection (acute) of upper genitourinary tract	098.19
Gonococcal infection of eye	098.4
Gonococcal conjunctivitis (neonatorum)	098.40

Gonococcal iridocyclitis	098.41
Gonococcal endophthalmitis	098.42
Gonococcal keratitis	098.43
Other gonococcal infection of eye	098.49
Gonococcal infection of joint	098.5
Gonococcal arthritis	098.50
Gonococcal synovitis and tenosynovitis	098.51
Gonococcal bursitis	098.52
Gonococcal spondylitis	098.53
Other gonococcal infection of joint	098.59
Gonococcal infection of pharynx	098.6
Gonococcal infection of anus and rectum	098.7
Gonococcal infection of other specified sites	098.8
Gonococcal keratosis (blennorrhagica)	098.81
Gonococcal meningitis	098.82
Gonococcal pericarditis	098.83
Gonococcal endocarditis	098.84
Other gonococcal heart disease	098.85
Gonococcal peritonitis	098.86
Gonococcal infection of other specified sites	098.89
Other venereal diseases	099
Chancroid	099.0
Lymphogranuloma venereum	099.1
Granuloma inguinale	099.2
Reiter's disease	099.3
Other nongonococcal urethritis	099.4
Other nongonococcal urethritis, unspecified	099.40
Other nongonococcal urethritis, chlamydia trachomatis	099.41
Other nongonococcal urethritis, other specified organism	099.49
Other venereal diseases due to chlamydia trachomatis	099.5
Other venereal diseases due to chlamydia trachomatis, unspecified site	099.50
Other venereal diseases due to chlamydia trachomatis, pharynx	099.51
Other venereal diseases due to chlamydia trachomatis, anus and rectum	099.52

Other venereal diseases due to chlamydia trachomatis, lower genitourinary sites	099.53
Other venereal diseases due to chlamydia trachomatis, other genitourinary sites	099.54
Other venereal diseases due to chlamydia trachomatis, unspecified genitourinary site	099.55
Other venereal diseases due to chlamydia trachomatis, peritoneum	099.56
Other venereal diseases due to chlamydia trachomatis, other specified site	099.59
Other specified venereal diseases	099.8
Venereal disease, unspecified	099.9
Leptospirosis	100
Leptospirosis icterohemorrhagica	100.0
Other specified leptospiral infections	100.8
Leptospiral meningitis (aseptic)	100.81
Other specified leptospiral infections	100.89
Leptospirosis, unspecified	100.9
Vincent's angina	101
Yaws	102
Initial lesions of yaws	102.0
Multiple papillomata due to yaws and wet crab yaws	102.1
Other early skin lesions of yaws	102.2
Hyperkeratosis due to yaws	102.3
Gummata and ulcers due to yaws	102.4
Gangosa	102.5
Bone and joint lesions due to yaws	102.6
Other manifestations of yaws	102.7
Latent yaws	102.8
Yaws, unspecified	102.9
Pinta	103
Primary lesions of pinta	103.0
Intermediate lesions of pinta	103.1
Late lesions of pinta	103.2
Mixed lesions of pinta	103.3
Pinta, unspecified	103.9
Other spirochetal infection	104
Nonvenereal endemic syphilis	104.0
Other specified spirochetal infections	104.8

Spirochetal infection, unspecified	104.9
Dermatophytosis	110
Dermatophytosis of scalp and beard	110.0
Dermatophytosis of nail	110.1
Dermatophytosis of hand	110.2
Dermatophytosis of groin and perianal area	110.3
Dermatophytosis of foot	110.4
Dermatophytosis of the body	110.5
Deep seated dermatophytosis	110.6
Dermatophytosis of other specified sites	110.8
Dermatophytosis of unspecified site	110.9
Dermatomycosis other and unspecified	111
Pityriasis versicolor	111.0
Tinea nigra	111.1
Tinea blanca	111.2
Black piedra	111.3
Other specified dermatomycoses	111.8
Dermatomycosis, unspecified	111.9
Candidiasis	112
Candidiasis of mouth	112.0
Candidiasis of vulva and vagina	112.1
Candidiasis of other urogenital sites	112.2
Candidiasis of skin and nails	112.3
Candidiasis of lung	112.4
Disseminated candidiasis	112.5
Candidiasis of other specified sites	112.8
Candidal endocarditis	112.81
Candidal otitis externa	112.82
Candidal meningitis	112.83
Candidal esophagitis	112.84
Candidal enteritis	112.85
Other candidiasis of other specified sites	112.89
Candidiasis of unspecified site	112.9
Coccidioidomycosis	114
Primary coccidioidomycosis (pulmonary)	114.0
Primary extrapulmonary coccidioidomycosis	114.1
Coccidioidal meningitis	114.2
Other forms of progressive coccidioidomycosis	114.3

Pulmonary coccidioidomycosis, unspecified	114.5
Coccidioidomycosis, unspecified	114.9
Histoplasmosis	115
Infection by histoplasma capsulatum	115.0
Infection by Histoplasma capsulatum, without mention of manifestation	115.00
Infection by Histoplasma capsulatum, meningitis	115.01
Infection by Histoplasma capsulatum, retinitis	115.02
Infection by Histoplasma capsulatum, pericarditis	115.03
Infection by Histoplasma capsulatum, endocarditis	115.04
Infection by Histoplasma capsulatum, pneumonia	115.05
Infection by Histoplasma capsulatum, other	115.09
Infection by histoplasma duboisii	115.1
Infection by Histoplasma duboisii, without mention of manifestation	115.10
Infection by Histoplasma duboisii, meningitis	115.11
Infection by Histoplasma duboisii, retinitis	115.12
Infection by Histoplasma duboisii, pericarditis	115.13
Infection by Histoplasma duboisii, endocarditis	115.14
Infection by Histoplasma duboisii, pneumonia	115.15
Infection by Histoplasma duboisii, other	115.19
Histoplasmosis unspecified	115.9
Histoplasmosis, unspecified, without mention of manifestation	115.90
Histoplasmosis, unspecified, meningitis	115.91
Histoplasmosis, unspecified, retinitis	115.92
Histoplasmosis, unspecified, pericarditis	115.93
Histoplasmosis, unspecified, endocarditis	115.94
Histoplasmosis, unspecified, pneumonia	115.95
Histoplasmosis, unspecified, other	115.99
Blastomycotic infection	116
Blastomycosis	116.0
Paracoccidioidomycosis	116.1
Lobomycosis	116.2
Other mycoses	117
Rhinosporidiosis	117.0
Sporotrichosis	117.1
Chromoblastomycosis	117.2

Aspergillosis	117.3
Mycotic mycetomas	117.4
Cryptococcosis	117.5
Allescheriosis [Petriellidosis]	117.6
Zygomycosis [Phycomycosis or Mucormycosis]	117.7
Infection by dematiaceous fungi [Phaeohyphomycosis]	117.8
Other and unspecified mycoses	117.9
Opportunistic mycoses	118
Schistosomiasis (bilharziasis)	120
Schistosomiasis due to schistosoma haematobium	120.0
Schistosomiasis due to schistosoma mansoni	120.1
Schistosomiasis due to schistosoma japonicum	120.2
Cutaneous schistosomiasis	120.3
Other specified schistosomiasis	120.8
Schistosomiasis, unspecified	120.9
Other trematode infections	121
Opisthorchiasis	121.0
Clonorchiasis	121.1
Paragonimiasis	121.2
Fascioliasis	121.3
Fasciolopsiasis	121.4
Metagonimiasis	121.5
Heterophyiasis	121.6
Other specified trematode infections	121.8
Trematode infection, unspecified	121.9
Echinococcosis	122
Echinococcus granulosus infection of liver	122.0
Echinococcus granulosus infection of lung	122.1
Echinococcus granulosus infection of thyroid	122.2
Echinococcus granulosus infection, other	122.3
Echinococcus granulosus infection, unspecified	122.4
Echinococcus multilocularis infection of liver	122.5
Echinococcus multilocularis infection, other	122.6
Echinococcus multilocularis infection, unspecified	122.7
Echinococcosis, unspecified, of liver	122.8
Echinococcosis, other and unspecified	122.9
Other cestode infection	123

Taenia solium infection, intestinal form	123.0
Cysticercosis	123.1
Taenia saginata infection	123.2
Taeniasis, unspecified	123.3
Diphyllobothriasis, intestinal	123.4
Sparganosis [larval diphyllobothriasis]	123.5
Hymenolepiasis	123.6
Other specified cestode infection	123.8
Cestode infection, unspecified	123.9
Trichinosis	124
Filarial infection and dracontiasis	125
Bancroftian filariasis	125.0
Malayan filariasis	125.1
Loiasis	125.2
Onchocerciasis	125.3
Dipetalonemiasis	125.4
Mansonella ozzardi infection	125.5
Other specified filariasis	125.6
Dracontiasis	125.7
Unspecified filariasis	125.9
Ancylostomiasis and necatoriasis	126
Ancylostomiasis due to ancylostoma duodenale	126.0
Necatoriasis due to necator americanus	126.1
Ancylostomiasis due to ancylostoma braziliense	126.2
Ancylostomiasis due to ancylostoma ceylanicum	126.3
Other specified ancylostoma	126.8
Ancylostomiasis and necatoriasis, unspecified	126.9
Other intestinal helminthiasis	127
Ascariasis	127.0
Anisakiasis	127.1
Strongyloidiasis	127.2
Trichuriasis	127.3
Enterobiasis	127.4
Capillariasis	127.5
Trichostrongyliasis	127.6
Other specified intestinal helminthiasis	127.7
Mixed intestinal helminthiasis	127.8
Intestinal helminthiasis, unspecified	127.9

Other and unspecified helminthiasis	128
Toxocariasis	128.0
Gnathostomiasis	128.1
Other specified helminthiasis	128.8
Helminth infection, unspecified	128.9
Intestinal parasitism, unspecified	129
Toxoplasmosis	130
Meningoencephalitis due to toxoplasmosis	130.0
Conjunctivitis due to toxoplasmosis	130.1
Chorioretinitis due to toxoplasmosis	130.2
Myocarditis due to toxoplasmosis	130.3
Pneumonitis due to toxoplasmosis	130.4
Hepatitis due to toxoplasmosis	130.5
Toxoplasmosis of other specified sites	130.7
Multisystemic disseminated toxoplasmosis	130.8
Toxoplasmosis, unspecified	130.9
Trichomoniasis	131
Urogenital trichomoniasis	131.0
Urogenital trichomoniasis, unspecified	131.00
Trichomonal vulvovaginitis	131.01
Trichomonal urethritis	131.02
Trichomonal prostatitis	131.03
Other urogenital trichomoniasis	131.09
Trichomoniasis of other specified sites	131.8
Trichomoniasis, unspecified	131.9
Pediculosis and phthirus infestation	132
Pediculus capitis [head louse]	132.0
Pediculus corporis [body louse]	132.1
Phthirus pubis [pubic louse]	132.2
Mixed pediculosis infestation	132.3
Pediculosis, unspecified	132.9
Acariasis	133
Scabies	133.0
Other acariasis	133.8
Acariasis, unspecified	133.9
Other infestation	134
Myiasis	134.0
Other arthropod infestation	134.1

Hirudiniasis	134.2
Other specified infestations	134.8
Infestation, unspecified	134.9
Sarcoidosis	135
Other and unspecified infectious and parasitic diseases	136
Ainhum	136.0
Behcet's syndrome	136.1
Specific infections by free-living amebae	136.2
Specific infection due to acanthamoeba	136.21
Other specific infections by free-living amebae	136.29
Pneumocystosis	136.3
Psorospermiasis	136.4
Sarcosporidiosis	136.5
Other specified infectious and parasitic diseases	136.8
Unspecified infectious and parasitic diseases	136.9
Late effects of tuberculosis	137
Late effects of respiratory or unspecified tuberculosis	137.0
Late effects of central nervous system tuberculosis	137.1
Late effects of genitourinary tuberculosis	137.2
Late effects of tuberculosis of bones and joints	137.3
Late effects of tuberculosis of other specified organs	137.4
Late effects of acute poliomyelitis	138
Late effects of other infectious and parasitic diseases	139
Late effects of viral encephalitis	139.0
Late effects of trachoma	139.1
Late effects of other and unspecified infectious and parasitic diseases	139.8
Urinary tract infection, site not specified	599.0
Abscess of liver	572.0
Portal pyemia	572.1

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Chapter 5: Implications and Conclusion

The findings from these studies have important implications for HCT utilization in people with HIV (PWH). In our first of two population-based studies of hematopoietic cell transplantation (HCT) among PWH in California, using 25 years of data we demonstrated that HCT use has increased since combination antiretroviral therapy (cART) came to market in 1996, however overall use remains low and is significantly associated with certain sociodemographic and clinical factors. We found that among PWH, older age, lower neighborhood socioeconomic status (nSES), and being single/unpartnered were associated with lower likelihood of HCT, highlighting the broader sociodemographic and clinical factors that add to the complexity of HIV diagnosis on HCT eligibility.

In our second study using a matched cohort analysis comparing HCT recipients with and without HIV, we demonstrated that HIV status was not associated with increased risk of acute renal or liver complications, infection, or sepsis; or increased risk of hospital admission. Additionally, we demonstrated that there were no significant differences in overall survival between matched cohorts at 90 days, one year, and two years following HCT. Taken together, our results offer strong support for the use of HCT in people with well-controlled HIV in the current cART era, and that HIV alone should not preclude PWH from HCT when all other eligibility criteria are met.

The findings from our two studies together suggest that inequities in HCT access and use among PWH may be attributed to social and institutional factors, and that fears of added risk for clinically-meaningful acute renal and liver complications, infection/sepsis, and added hospital burden may be unsubstantiated in the modern

cART era. Other contributing barriers to HCT use may include provider lack of familiarity with recent HIV literature, relatively low HIV prevalence in some geographic regions, and HIV-related stigma.

IMPLICATIONS FOR PRACTICE

Our findings have several implications for clinical practice. Overall, people with HIV should have equitable access to undergo the same HCT eligibility processes as the general population, including assessment of infectious disease markers.¹ People with HIV should not be excluded from HCT based on HIV infection alone. Similar to all HCT recipients during the recovery process, immune reconstitution will be monitored using routine labs. During prolonged periods of neutropenia across all patients, signs and symptoms of infection and sepsis must be monitored by the patient and their caregiver.¹ All HCT recipients are required to identify a caregiver prior to the HCT process, and PWH should be supported in identifying qualified caregivers within their social network, especially if the patient is not married.

Similar to all HCT recipients, the HCT conditioning regimen must be evaluated for drug-drug interactions as a standard. As noted in Specific Aim 2 supplemental materials, the majority of chemotherapy/immunotherapy used in HCT do not have significant drug-drug interactions with the most common HIV antiretrovirals, thus not likely to contribute to acute renal or liver complications during HCT. Lab values indicative of organ toxicity, such as creatinine and LFTs for renal and liver complications, are routinely monitored across all patients as a standard following HCT thus capturing potential toxicities early. Specifically for PWH, patients should stay

adherent to their cART regimen to stay undetectable, with aggressive symptom management for chemotherapy side effects that may hinder cART adherence—like mucositis and nausea/vomiting.

IMPLICATIONS FOR RESEARCH

More research is needed to examine HCT access among PWH in different U.S. regions to identify potential disparities. Provider referral patterns from oncologists for HCT in PWH, which precede discussions on HCT eligibility, remain poorly understood and warrant further research. More broadly, the role of HIV-related stigma within HCT remains poorly understood. This is important as CAR-T cell therapy is increasingly replacing autologous transplantation as first-line therapy for lymphomas, where the barriers to utilization among PWH may be mirrored. Within population-based research approaches, which leverage aggregation of a relatively small cohort of PWH receiving HCT, additional studies are needed to evaluate other predictors that influence utilization, clinical outcomes, and hospital use outcomes. While sampling in prospective studies remains a challenge, more patient-reported outcomes are needed to evaluate how HIV-related behaviors shape the HCT experience for PWH.

IMPLICATIONS FOR POLICY

Legislators must advocate for equitable access to HCT across all insurance types, including state, federal, and private payors. Insurance coverage must include uninterrupted access to HIV care and cART before, during, and after HCT completion. Professional organizations like the National Comprehensive Cancer Network (NCCN

and the American Society of Transplantation and Cell Therapy (ASTCT) should review and adapt existing guidelines to reflect emerging evidence on the safety and efficacy of HCT in PWH. Lastly, state transplant registries across the U.S should expand data collection to allow better tracking for subgroup analyses of PWH, enabling easier segmentation and focused equity research.

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