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Review Article

Impact of Psychological Stress on Immunity in Chronic Infections (HIV and TB)

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Abstract: Human immunodeficiency virus (HIV) infection and tuberculosis (TB) remain major global health challenges, affecting not only morbidity and mortality but also patients' quality of life and everyday functioning. In both chronic infections, psychological stress and common mental disorders—particularly depression and anxiety—often coexist and are strongly shaped by social determinants. These psychological conditions may, in turn, influence immune function and contribute to poorer clinical outcomes. This literature review explores the relationship between psychological stress, mental disorders, and immune alterations through a psychoneuroimmunology perspective. The review highlights how chronic stress activates the hypothalamic–pituitary–adrenal (HPA) axis and the sympathetic nervous system, increasing cortisol and catecholamines and forming a neuroendocrine–immune network that may suppress cellular immunity when stress is persistent. Additionally, prolonged immune dysregulation may worsen disease progression and reduce treatment adherence among affected individuals. The findings support the importance of integrating medical care with psychological and social interventions to improve long-term outcomes among people living with HIV and TB.

Keywords: HIV; Immune Function; Psychoneuroimmunology; Psychological Stress; Tuberculosis.

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1. Introduction

HIV and TB remain major health global priorities. By the end of 2021, an estimated 38.4 million people worldwide were living with HIV, with 1.5 million new infections and 650,000 deaths per year. HIV is also strongly associated with an increased risk of mental disorders, particularly depression and anxiety; depressive symptoms are reported in about 30–50% of individuals living with HIV, with depression risk roughly twice that of the general population (Fabrazzo et al., 2023). TB on the other hand, continues to be a leading cause of infectious morbidity and mortality, with a particularly high burden in low- and middle-income countries, including Indonesia (Sabrina, 2025). Systematic reviews describe a bidirectional relationship between TB and mental disorders: depression and schizophrenia may increase the risk of active TB, and TB itself can contribute to psychological problems (Hayward et al., 2022). In Indonesia, where both HIV and TB burdens are high, the combined clinical and social implications are substantial, although national figures are not yet fully documented (Sabrina, 2025).

The burden of HIV and TB is not only reflected in organ damage or declining immune status, but also in broader impacts on quality of life (QoL) and daily functioning. In HIV, the availability of antiretroviral therapy (ART) has shifted the disease trajectory from fatal to chronic, moving care priorities beyond survival toward preserving QoL. Many patients live

longer but face persistent symptoms, fatigue, cognitive difficulties, and functional disability. Lower QoL is associated with poorer immune responses, non-adherence to therapy, and worsening disease severity (Obeagu and Alsadi, 2025). In TB, chronic respiratory symptoms, long treatment courses with adverse effects, and social stigma can reduce physical and social functioning, limiting daily activity and social roles. These realities show that functional and psychosocial dimensions cannot be separated from biomedical assessments when evaluating the burden of HIV and TB (Hayward et al., 2022).

Social context also plays a major role in shaping both psychological responses and immune outcomes. Stigma, discrimination, social isolation, economic insecurity, and trauma related to diagnosis are linked to high rates of depression, anxiety, and psychological stress among people with HIV and TB. These mental health problems are not only psychosocial; they correlate with measurable immune changes. In HIV, mental disorders are associated with chronic immune activation, increased proinflammatory cytokines, HPA-axis dysfunction, and immunologic pathway changes that can influence mood and cognition (Obeagu and Alsadi, 2025). Clinically, depression and anxiety are linked to immune dysregulation, including faster CD4 decline, higher viral load, and more rapid disease progression (Obeagu and Alsadi, 2025). In TB, stress and depression are thought to reduce cellular immunity, disrupt T-lymphocyte function and cytokine balance, and facilitate reactivation of latent TB or progression from subclinical infection to active disease; psychological stress is therefore a meaningful part of chronic infection pathophysiology and influences clinical outcomes (Hayward et al., 2022).

This review examines how psychological stress affects immunity in chronic HIV and TB, clarifies the biopsychosocial mechanisms involved, and highlights implications for integrated interventions such as medical, psychological, and social especially in Indonesia.

2. Method

Psychological stress and mental disorders have been widely recognized as factors that can influence immune functioning through psychoneuroimmunological mechanisms. Chronic psychological stress activates the hypothalamic–pituitary–adrenal (HPA) axis and the sympathetic nervous system, resulting in increased secretion of cortisol and catecholamines that can suppress cellular immune responses when stress is prolonged (Segerstrom & Miller, 2004). In individuals with chronic infectious diseases such as HIV and tuberculosis, persistent stress may weaken T-cell–mediated immunity and reduce the body's ability to control infection and inflammation (Dhabhar, 2014). Moreover, mental health conditions such as depression and anxiety are associated with increased production of pro-inflammatory cytokines, including interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), which may further contribute to immune dysregulation (Irwin & Cole, 2011). These biological interactions indicate that psychological factors can play a significant role in shaping disease progression and treatment outcomes in chronic infections (Glaser & Kiecolt-Glaser, 2005).

A literature search was conducted using scientific articles published within the last five years relevant to HIV/TB, stress/depression/anxiety, inflammatory biomarkers and/or immune parameters (e.g., CD4, cytokines, CRP, viral load), and clinical outcomes. Articles were screened by title and abstract, then reviewed in full text to confirm mechanistic and clinical relevance. Key findings were extracted and synthesized to summarize biological pathways (neuroendocrine and immune/inflammatory) and social-behavioral pathways (therapy adherence, quality of life, stigma/social support) that may explain how stress and mental disorders worsen outcomes in people living with HIV and in TB patients, and to identify implications for integrated interventions.

3. Results and Discussion

Basic Concepts of Psychological Stress, Mental Disorders, and Immune System in Chronic Disease

Psychosocial stress is a psychological and biological response that occurs when environmental demands are perceived as threatening or beyond an individual's coping capacity. In chronic disease, stressors are often persistent, including social isolation, poverty, limited access to treatment, financial burden, trauma, and uncertainty about the future (Obeagu and Alsadi, 2025; Vadakkiniath, 2023). People with chronic illnesses also report higher rates of mental health problems—especially stress, anxiety, and depression—which affect emotional and social functioning. Studies in chronic-disease populations show that these problems are common and are associated with psychosocial determinants

such as social isolation, quality of social relationships, economic status, education, length of hospitalization, and frequency of healthcare visits (Vadakkiniath, 2023).

Clinically, persistent psychological stress is not only emotional; it involves activation of neuroendocrine and autonomic nervous systems (Clark, 2024). Prolonged stress may negatively affect immune, cardiovascular, neuroendocrine, and central nervous system functioning (Vadakkiniath, 2023). The main biological pathways used to explain stress-related bodily changes include activation of the HPA axis and autonomic nervous responses, which increase stress hormones such as cortisol and catecholamines (Obeagu and Alsadi, 2025). When chronic stress is not addressed, these neuroendocrine changes can contribute to physical and mental health problems (Vadakkiniath, 2023).

Mental disorders in chronic disease refer to a broad spectrum of psychopathological symptoms and syndromes that accompany illness and influence social functioning and quality of life. Anxiety and depression frequently co-occur and are often difficult to separate in both practice and research because they are interrelated and can worsen each other. Depression comorbid with chronic disease is also associated with poorer outcomes than either condition alone, supporting the view that mental disorders should be understood as part of the total disease burden rather than as a separate issue (Vadakkiniath, 2023).

The relationship between stress, mental disorders, and immunity is commonly explained through psychoneuroimmunology—the interaction of the nervous, endocrine, and immune systems (Ricky, 2021). Chronic stress can reduce immune competence through persistent neuroendocrine changes while also increasing vulnerability to mood disorders. Conversely, mental disorders can reduce medication adherence, worsen quality of life, and undermine disease control through behavioral and social pathways, forming a reciprocal relationship between mental health and chronic disease progression (Obeagu and Alsadi, 2025).

Psychoneuroimmunological Mechanisms in Chronic Infections (HIV and TB)

HPA-axis activation is a central pathway linking psychosocial stress with immune changes in chronic infection. Psychosocial stress increases corticotropin-releasing hormone (CRH) release from the hypothalamus, which stimulates adrenocorticotropic hormone (ACTH) secretion from the pituitary and results in glucocorticoid (cortisol) release from the adrenal cortex. In parallel, HPA-axis activation is associated with sympathetic nervous system activation, increasing catecholamine production such as adrenaline and noradrenaline. Together these processes create a bidirectional nerve–endocrine–immune network connecting the central nervous system and immune system. When stress becomes chronic, this network can suppress immune function and sustain neuroendocrine–immune dysfunction through feedback mechanisms, worsening the overall condition (Clark, 2024).

In HIV, the relationship between mental health and immunity is often described as bidirectional: immune dysfunction and chronic inflammation may contribute to affective symptoms, while chronic stress and depression can reduce immune competence via HPA-axis and sympathetic pathways. Inflammation and psychological stress may disrupt the HPA axis, leading to abnormal cortisol secretion, which can suppress immune function and intensify depressive symptoms in a self-reinforcing cycle. Persistent stress is associated with elevated cortisol and catecholamines, which may suppress cellular immunity and reduce the activity of key immune cells, including natural killer cell activity and lymphocyte proliferation (Clark, 2024).

At the biomolecular level, HIV is marked by persistent systemic immune activation and increased proinflammatory mediators, which have been linked in several studies to depressive symptoms. Reviews of depression biomarkers in HIV describe associations with inflammatory markers such as IL-6, TNF-α, monocyte activation markers, and D-dimer, although evidence varies. In treated populations, psychological stress has also been correlated with systemic inflammation (e.g., CRP) and carotid arterial inflammation, indicating that stress–inflammation pathways are not only theoretical but can be observed in biological and vascular indicators (Fabrazzo et al., 2023).

A particularly important feature of HIV is neuroinflammation. HIV can enter the central nervous system early and establish reservoirs in immune cells such as microglia/macrophages, triggering persistent neuroinflammation linked to mood and cognitive impairment. Biomarker-focused reviews suggest that microglial activation may connect CNS HIV infection to depressive symptoms through cytokine/chemokine release, immunometabolic dysfunction, and neurotrophic pathways, including the role of brain-derived neurotrophic factor (BDNF) and the kynurenine pathway (Obeagu and Alsadi, 2025; Clark, 2024).

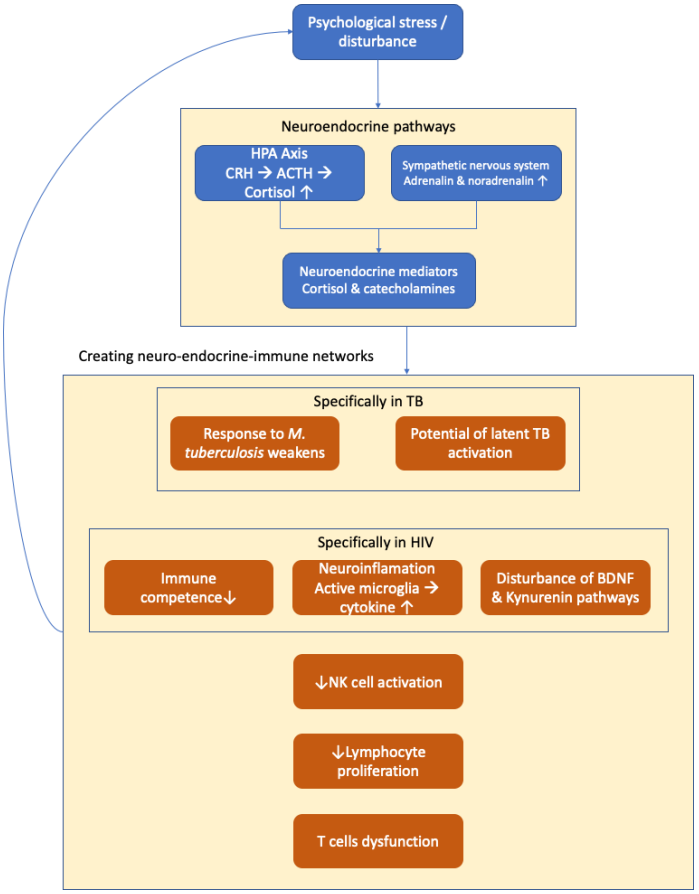

Figure 1. Diagram of the neuro-endocrine-immune mechanisms involved in psychological disturbance and chronic diseases such as HIV and TB

In TB, psychoneuroimmunology is often discussed through the concept of a tuberculosis–depression syndemic: a two-way synergy between depression/stress, social determinants, and TB vulnerability and course. Chronic depressive episodes may increase cortisol production and alter IL-6 production, potentially weakening antigen responses and disrupting inflammation needed to control bacterial infection, and may contribute to activation of latent TB (Fabrazzo et al., 2023; Sabrina, 2025).

From the standpoint of cellular immunity, which is crucial for intracellular pathogens such as *M. tuberculosis*, depression and chronic stress are associated with impaired T-cell availability and function. Depression/anxiety in TB has also been linked to dysregulation of cytokines and inflammatory mediators, including IL-2, IL-6, IL-1β, and macrophage migration inhibitory factor (MIF). TB activates proinflammatory cytokines (e.g., IL-1β, IL-6, TNF-α) that may affect the central nervous system and contribute to neuroinflammation and alterations in mood-regulating circuits, alongside serotonin suppression associated with depressive symptoms. Major depression is also associated with altered T-cell function, including reduced CD4+ T cells. When stress and depression persist, TB literature describes an "immune risk" phenotype—such as inversion of the CD4/CD8 ratio with reduced lymphocyte counts—reflecting relative immunosuppression and higher vulnerability to progression from latent to active TB (Sabrina, 2025; Clark, 2024).

Psychological Stress, Mental Disorders and Immunity in HIV

1) Spectrum of Mental Disorders in People Living with HIV/AIDS

Psychological stress and mental disorders commonly accompany people living with HIV/AIDS (PLWHA), ranging from mood disorders to neurocognitive disturbances. The literature indicates that PLWHA carry a heavier burden of psychological morbidity than the general population, often with chronic, co-occurring conditions driven by the interaction of biological and psychosocial challenges. Factors frequently linked to psychological distress include stress related to diagnosis, fear of disease progression, internalized stigma, social isolation, and financial hardship (Obeagu and Alsadi, 2025).

Depression is the most commonly reported mood disorder in PLWHA. WHO data at the end of 2021 reported approximately 38.4 million people living with HIV, and the global prevalence of depressive symptoms in PLWHA is estimated at 30–50%, far higher than the general population (around 7%). Depression risk in PLWHA is often described as roughly double that of the general population and has meaningful functional consequences, including reduced daily functioning, poorer health-related quality of life, and decreased engagement in care. Beyond psychological comorbidity, depression is also considered biologically relevant because of its association with faster disease progression through effects on immune function (Obeagu and Alsadi, 2025).

Anxiety is also common in PLWHA, including generalized anxiety disorder, panic disorder, and social anxiety. Anxiety symptoms may be triggered by uncertainty around living with a chronic condition, fear of disclosure, and experiences of discrimination and social rejection. These symptoms can impair decision-making, reduce adherence, and increase maladaptive coping behaviors such as substance use, which can worsen prognosis. Anxiety frequently coexists with depression and can create a mutually reinforcing psychological burden (Obeagu and Alsadi, 2025).

Post-traumatic stress disorder (PTSD) is often under-recognized but clinically important in PLWHA. PTSD may relate to traumatic experiences before or after diagnosis, including disclosure-related violence, loss of social support, or histories of trauma such as violence and homelessness. Symptoms such as hyperarousal, flashbacks, and emotional numbing may disrupt therapeutic relationships and reduce adherence and long-term engagement in care. Reviews also note associations between PTSD and markers of inflammation and immune activation among PLWHA with controlled viremia (Clark, 2024).

Substance use disorders also appear within the mental health spectrum of PLWHA and may function as both risk factors and psychosocial consequences after diagnosis. Substance use may begin as a coping strategy but can worsen clinical outcomes through reduced ART adherence, increased viral replication, and barriers to accessing and staying in care. Coexisting substance use and psychiatric disorders complicate management and increase clinical risk; this clustering is often described as syndemic and shaped by social determinants such as stigma, marginalization, and poverty (Obeagu and Alsadi, 2025).

A relatively distinctive aspect of HIV is HIV-associated neurocognitive disorders (HAND), which range from mild cognitive impairment to severe forms such as HIV-associated dementia. HAND can affect memory, attention, executive function, and psychomotor speed, directly limiting daily functioning and medication adherence. Although severe forms are less common in the ART era, milder impairment remains frequent, especially among individuals with delayed diagnosis, low CD4 counts, or substance-use history. Mechanistically, early CNS invasion and persistent neuroinflammation are considered important contributors (Shchaslyvyi et al., 2024).

2) Impact of Stress and Mental Disorders on Clinical Outcomes in HIV

Stress and mental disorders are not merely comorbidities in HIV; they are important determinants of impaired daily functioning and poorer health-related quality of life. HIV's early CNS involvement and persistent neuroinflammation also contribute to neurocognitive complaints and reduced quality of life. Clinically, these neurocognitive aspects matter because inflammation and neuroendocrine dysfunction are associated with neuropsychiatric outcomes in PLWHA (Fabrazzo et al., 2023).

Stress and depression may worsen HIV outcomes through behavioral pathways that directly undermine treatment success. Mental disorders can disrupt engagement in care, medication adherence, and symptom management, thereby increasing the risk of disease progression indirectly through failure to maintain virological suppression. This is clinically important because ART adherence is a key prerequisite for viral suppression; when depression/anxiety is untreated, individuals may struggle to keep consistent treatment routines and follow-up visits, contributing to increased viral load and decreased CD4+ (Fabrazzo et al., 2023).

Depression and stress have also been linked to cardiovascular and cerebrovascular risk (including stroke) through systemic inflammation and arterial activation. FDG-PET studies in PLWHA show that perceived stress correlates with systemic inflammation and carotid artery inflammation, independent of standard vascular risk factors and health behaviors.

These findings support the framework that stress may contribute to atherosclerosis and cerebrovascular risk through inflammatory pathways, making psychological stress clinically relevant even among virologically suppressed individuals. Depression has also been discussed as a risk factor for ischemic stroke in cohorts of older adults living with HIV (Fabrazzo et al., 2023; Chow et al., 2023).

Psychosocial factors—particularly stigma, social support, and hardiness (coping capacity)—also influence outcomes. Stigma and discrimination can present as rejection, exclusion, and barriers in daily life, worsening emotional distress. Internalized stigma is associated with perceived social support and psychological well-being and may ultimately affect immune status in PLWHA (Mirzaian, 2021).

Psychological Stress, Mental Disorders, and Immunity in TB

1) Spectrum of Mental Disorders in TB

Globally, TB and mental disorders frequently coexist, most commonly depression and psychosis/schizophrenia. TB caused an estimated 1.4 million deaths in 2019 (Hayward et al., 2022). Studies describe mental disorders in TB using screening tools (e.g., PHQ-9) and broader diagnostic interviews (e.g., Composite International Diagnostic Interview) to capture conditions ranging from mild to previously undiagnosed (Clark, 2024).

Strong epidemiological evidence for associations between mental disorders and TB largely comes from Asia. Systematic reviews report that depression can increase TB incidence: nationally databased cohort studies in South Korea and Taiwan reported hazard ratios indicating higher TB risk among individuals with depression. Cross-country analyses in low- and middle-income countries based on WHO survey data also found higher odds of depressive episodes in individuals reporting TB symptoms compared to those without TB symptoms. These findings support the tuberculosis–depression syndemic concept, describing two-way synergy between TB and depression within a framework shaped by biological and social determinants (Clark, 2024).

For psychotic disorders, cohort evidence also suggests increased TB risk. A cohort study in Taiwan reported that schizophrenia was associated with higher TB incidence. In an MDR-TB service in Nigeria, psychosis prevalence among MDR-TB patients was reported to be markedly higher than controls, and remained high even after excluding anti-TB drug-induced psychosis; schizophrenia prevalence was also higher in TB patients than controls (Clark, 2024). These findings broaden the psychiatric comorbidity spectrum in TB beyond mood disorders.

People at high risk for TB; including individuals experiencing homelessness, alcohol and drug misuse, or migration are also more likely to experience mental disorders than the general population. Systematic reviews emphasize that crowded living conditions and social vulnerability among people with severe mental disorders can further increase TB risk, making institutional settings important targets for TB prevention and screening (Hayward et al., 2022).

2) Impact of Stress and Mental Disorders on Clinical Outcomes in TB

As in HIV, psychological stress and mental disorders in TB are associated with poorer outcomes, including delayed treatment-seeking and long-term non-adherence to medication regimens—factors that can increase disease severity, mortality, and the risk of drug resistance. TB treatment is also lengthy and can involve potent drug combinations with substantial side effects that may worsen mental health. For instance, isoniazid and cycloserine may trigger depressive symptoms, complicating treatment and potentially interfering with therapy success (Sabrina, 2025).

Stress Reduction Interventions in Chronic Infection

1) Stress-Reduction Interventions in HIV

Stress-reduction interventions for PLWHA include mindfulness-based approaches, meditation, and cognitive-behavioral interventions aimed at chronic stress and affective symptoms, with the goal of improving psychological outcomes and, in some cases, biological outcomes related to inflammation.

Behavioral Stress Reduction Programs (BSRPs) are structured interventions that teach skills such as relaxation, coping management, and lifestyle modification to help individuals manage stress. Studies in PLWHA suggest these programs can reduce depression and

perceived stress and improve certain quality-of-life domains, although immune outcomes do not always change significantly (Shchaslyvyi et al., 2024).

Mindfulness-Based Stress Reduction (MBSR) is an eight-week program that trains non-judgmental, present-moment awareness to reduce stress and improve well-being. A controlled trial in adults with HIV-1 reported reduced depression, reduced perceived stress, increased mindfulness, and increased positive affect, but did not show significant changes in CD4+ T-cell count, HIV-1 viral load, or inflammatory biomarkers such as IL-6, CRP, and D-dimer. In older PLWHA, MBSR was associated with improved depression and later improvement in quality of life, without changes in cognitive performance. Overall, psychological and quality-of-life outcomes appear to improve more consistently than immune outcomes in some MBSR studies (Shchaslyvyi et al., 2024).

Other approaches are also discussed. Transcendental Meditation has been described as a behavioral-based stress reduction option in PLWHA. Cognitive-behavioral stress management has been reported to reduce anxiety and 24-hour urinary catecholamine output, indicating measurable changes in neuroendocrine stress responses (Ricky, 2021). Immunologic markers such as IL-6 and CRP do not always change, although it remains biologically plausible that stress reduction could influence inflammatory pathways associated with psychological stress (Shchaslyvyi et al., 2024).

Supportive and non-pharmacological interventions have also been evaluated. A small quasi-experimental study reported increased NK cell activity and NK cell counts following MBSR, although psychological or endocrine variables did not significantly change (Shchaslyvyi et al., 2024). Massage therapy in adolescents with HIV has been associated with improved immune function, suggesting touch-based supportive therapy may have a role in immune modulation. In clinical and nursing practice, mentoring and coping strategies are also positioned as practical psychological supports to reduce stress, with the rationale that stress management may help prevent accelerated immune decline through psychoneuroimmunological pathways (Obeagu and Alsadi, 2025).

2) Stress Reduction Interventions in TB

Stress-reduction interventions are increasingly viewed as an important element of TB care because psychological burden is high and can affect engagement in treatment (Hayward et al., 2022). Strengthening psychosocial support within TB services can help reduce distress and improve treatment continuity, especially as TB–depression is often framed as a syndemic involving two-way interactions among social, psychological, and disease processes (Sabrina, 2025; Clark, 2024).

In TB and MDR-TB, psychosocial education and psychological support are relevant because mental disorders, social stress, and reduced quality of life are frequently reported. Systematic reviews and meta-analyses in MDR-TB populations identify mental health disorders and social stressors as key factors associated with quality of life, making integrated psychosocial services a practical need in long-term care. Policy and practice discussions also emphasize that individuals with mental disorders represent a high-risk group for TB, supporting a holistic approach that integrates TB and mental health services to enable timely diagnosis and treatment of both active and latent TB while also improving mental health support (Sabrina, 2025; Clark, 2024).

Structured psychological interventions have also been examined. Cognitive Behavioral Therapy (CBT) in pulmonary TB has been evaluated as an approach to reduce psychological stress and improve quality of life. Evidence from a community-based randomized controlled trial suggests CBT can be used alongside pharmacological TB treatment to reduce distress and improve daily functioning (Clark, 2024).

At the health system level, the literature points to the need to strengthen primary mental health services within TB programs using a biosocial and integrated framework. Because the relationship between mental health and TB is multidirectional, social determinants such as poverty, substance use, homelessness, and institutionalization should be addressed as part of prevention and care. As the previous discussion of the immune mechanisms, it also supports consideration of host-directed approaches that include psychological factors as one target to reduce progression from latent infection to active TB, alongside strengthened social interventions and primary-care mental health services (Sabrina, 2025; Clark, 2024).

5. Conclusion

Psychological stress and mental disorders in both HIV and TB are important determinants of worse clinical outcomes through two main pathways: (1) biological pathways involving neuroendocrine–immune dysfunction and inflammation that can suppress cellular immunity, and (2) social-behavioral pathways involving reduced engagement in care, poorer quality of life, and decreased adherence; ultimately increasing disease progression, severity, mortality, and the risk of drug resistance. Routine screening for distress, depression, and anxiety should be embedded in HIV/TB services, followed by integrated interventions combining medical management, psychosocial support, and evidence-based therapies such as CBT or structured stress-reduction programs, while also addressing social determinants (stigma, discrimination, isolation, and economic vulnerability) to support consistent treatment and improve long-term outcomes.

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