Incidence and Persistence of Depression Among Women Living with and Without HIV in South Africa: A Longitudinal Study

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Abstract

Depression and trauma are common among women living with HIV. This is the first study to track the longitudinal course of depression and examine the relationship between depression and trauma over time among women in South Africa. HIV-infected and uninfected women (N = 148) were assessed at baseline and one year later. Results of a path analysis show the multi-directional and entwined influence of early life stress, other life-threatening traumas across the lifespan, depression and PTSD over the course of HIV. We also observed higher rates of depressive symptomatology and more persistent cases among infected women compared to uninfected women, as well as a more consistent and enduring relationship between childhood trauma and depression among women living with HIV. The present study is unique in documenting the course of untreated depression and PTSD in women with and without HIV infection with a high prevalence of early childhood trauma.

Keywords

HIV; Depression; PTSD; Childhood trauma

Introduction

Sub-Saharan Africa remains the centre of the HIV epidemic. An estimated 7.03 million South Africans were living with HIV in South Africa by 2016 [1]. People living with HIV (PLHIV) face many challenges, including psychiatric comorbidities. As the life expectancy of PLHIV improves, increased attention has been paid to the impact of comorbid non-communicable diseases such as cardiovascular disease [2] and cancer [3]. However, little

attention has been paid to the significant mental health burden associated with HIV [4]. PLHIV show higher rates of depression [5]. Epidemiological data from the US indicate that nearly half of PLHIV are affected by at least one psychiatric disorder, with depression being the most frequent diagnosis [6]. In South Africa, rates of psychopathologies such as mood disorders among PLHIV are higher compared to the general population [7–9]. Worldwide rates of depression ranging from 14 to 26% have been reported [6, 10–15]. A recent study conducted in South Africa showed that mental disorders like major depression are common in individuals presenting for HIV testing, irrespective of the test result, providing tentative evidence that for some PLHIV, receipt of a positive test result was not cause of their mental disorder [16]. Irrespective of whether HIV is the cause of depression in infected individuals, depression in HIV has a negative impact on the course of infection, reducing adherence to treatment [17–21], accelerating disease progression, and increasing mortality [22–25]. Moreover, there is evidence that depressive symptoms adversely affect responsiveness to antiretroviral therapy (ART) [10, 26]. Depression is often unrecognised and untreated in PLHIV [27]. Various interventions for the treatment of depression in PLHIV are available and may lead to improved quality of life and better prognosis [28, 29]. A limitation of this work is that the majority of studies have been cross-sectional in design, with few studies investigating the longitudinal course of depression among PLHIV [10, 13, 18, 19, 24].

Women tend to be particularly affected by the HIV epidemic. In South Africa, the prevalence of HIV was 22.3% among women in 2016, with approximately one-fifth of South African women in their reproductive ages living with HIV [1]. Many of these infected women also have developmental trajectories characterised by trauma. High rates of intimate partner violence, rape and childhood abuse have been reported [30–33]. Traumatic experiences and life adversity may predispose individuals to impulsive and risky behaviours, which increase the risk of HIV infection [34–36]. In South Africa, rates of posttraumatic stress disorder (PTSD) among PLHIV ranging from 5% to 14.8% have been reported [7, 8] and symptoms have been shown to persist, with follow-up rates of 20% reported [8]. There is substantial and consistent evidence that chronic depression, stressful events, and trauma may negatively affect HIV disease progression in terms of decreased CD4 T lymphocytes, increased viral load, and greater risk for clinical decline and mortality [25, 37, 38].

To our knowledge, there are no published studies tracking the longitudinal course of depression or the relationship between depression and trauma over time among women in South Africa. The present study is the first to investigate this. Studies have shown strong evidence for a link between early life trauma, depression and PTSD, with an increased risk for development of depression among women abused in childhood than non-abused women [39]. A substantial influence of multiple childhood trauma on a severe and chronic course of depression in adulthood has been documented [40]. Moreover, in a mouse model, evidence for a mechanism by which early life stress encodes lifelong susceptibility to stress has been shown [41]. In light of these findings, we hypothesised an entwined influence of early life stress, other life-threatening traumas across the lifespan, depression and PTSD over the course of HIV and therefore included these as predictors of depression in our models.

We carried out a longitudinal study with a 12-month follow-up period, collecting demographic, clinical, laboratory and psychiatric data from a cohort of South African

women. The study sought to document: (1) rates of incident and persistent depression and posttraumatic stress disorder over time and (2) the relationship between depression, traumatic experiences, posttraumatic stress disorder and HIV infection over time. Previous studies in South Africa investigating the persistence of psychiatric disorders in PLHIV reported baseline rates of depression of 35 and 26% by 6-month follow-up [8]. The incidence of depression was 8% [8]. In the South African Stress and Health (SASH) study, a 12-month and lifetime prevalence study of common mental disorders, depression in HIV-negative women was found to be around 5% at both time points [42]. Therefore, for the present study, we expected to find similar rates of incident and persistent depression as these previous South African studies.

The examination of predictors of depression at follow-up in the present study is an examination of predictors of *persistent and emergent depression* in a sample of relatively healthy infected women. The primary purpose of this study was to compare the incidence and persistence of depression in HIV-positive compared with HIV-negative women. Documenting differences in incidence and persistence of depression in HIV-positive women has implications for the assessment and management of this comorbid condition in HIV.

Methods

Participants

A total of 148 women were included in a larger longitudinal genetics, cognitive and neuroimaging study at the Department of Psychiatry of Stellenbosch University [43–45]. Sixty-eight women (46%) were living with HIV, of whom 46 (67%) were on ART. Eligibility criteria included: willingness and ability to provide written informed consent, ability to read and write in either English or Afrikaans at 5th grade level, aged between 18 and 65 years, medically well enough to undergo neuropsychological testing and MRI scanning. Exclusions were a current or past history of schizophrenia, bipolar disorder or other psychotic disorders, current substance or alcohol abuse or dependence, significant previous head injury, demonstrated frank dementia on the International HIV Dementia Scale, current seizure disorders of any cause, history of CNS infections of neoplasms, hepatitis B or C positive status, and current use (within the past month) of any psychotropic medication.

Procedure

All participants were recruited by a team comprising a researcher, a research assistant and a professional nurse, with the help of doctors and adherence counselors from community health care facilities in and around the Cape Town area. All participants who consented were screened for eligibility either in person at the clinic or telephonically. Those who met initial screening criteria subsequently underwent behavioural, neuromedical, neurocognitive and neuroimaging assessments at Stellenbosch University at baseline and one year later. Participants were reimbursed for their travel costs to the University for all study visits and provided with refreshments at each visit. All participants screening positive for depression at baseline or at follow-up were referred to their local health care facility for further care.

Measures

Demographic and Clinical Characteristics

A comprehensive history was obtained from, and a general physical examination conducted in, all participants. Virologic markers of HIV disease progression (CD4 T cell count and viral loads) were obtained from blood samples. Age, gender, marital status, ethnicity, years of education and employment status were captured.

Psychiatric Morbidity

Current and lifetime psychiatric disorders were assessed using the MINI-International Neuropsychiatric Interview-Plus (M.I.N.I.-Plus) [46].

Depression

Participants were assessed for depressive symptomatology using the Center for Epidemiologic Studies Depression Scale (CES-D) [47]. The CES-D is a 20-item widely used self-report instrument designed to measure depressive symptomatology. The scale was specifically designed to measure depressive symptomatology in the general population, unlike previous depression scales which have been predominantly used in clinical populations. The CES-D emphasises the affective component of depressive symptomatology, namely depressed mood. Each item comprises a Likert scale ranging from 0 to 3. A total score for the 20 items is obtained, with the lowest possible score being 0 and the highest possible score being 60. Higher scores are indicative of more severe depression.

Traumatic Life Events

Exposure to traumatic life events were assessed for using the life events checklist (LEC) [48]. The LEC is a widely used measure of exposure to potentially traumatic events and was developed to facilitate the diagnosis of PTSD. One of the LEC's unique features is that it enquires about various types of exposure to each potentially traumatic event. Therefore, the LEC elicits whether the participant experienced, witnessed, or learned of the traumatic event, a feature that other traumatic event measures do not possess. Participants rate their experience of each traumatic event listed on a Likert scale. A total score is derived from the sum of experiencing and/or witnessing the event. Higher scores are indicative of the experience of more traumatic life events.

Posttraumatic Stress Symptomatology

Posttraumatic stress symptomatology was assessed using the Davidson trauma scale (DTS) [49]. The DTS is a widely used 17-item self-report and measures symptoms of PTSD on frequency and severity scales. The items are categorised according to the criteria set out in the DSM-IV: criteria B (intrusive re-experiencing) criteria C (avoidance and numbness) and criteria D (hyper-arousal). For each item, the participant rates the frequency and severity during the previous week on 5-point (0–4) scales, with the lowest possible score being 0 and the highest possible score being 136. Higher scores are indicative of more PTSD symptoms.

Childhood Trauma

The childhood trauma questionnaire short form (CTQ-SF) [50], a 28-item self-report inventory that provides valid screening for histories of abuse and neglect was administered. It assesses five types of maltreatment including, emotional, physical, and sexual abuse, and emotional and physical neglect. These five subscales each consist of 5 items with scores ranging from 5 to 25. The overall trauma score ranges from 25 to 125 with higher scores indicating higher levels of childhood trauma (score of 25–31 = no trauma, score of 41–51 = low to moderate, 56–68 = moderate to severe, and 73–125 = severe to extreme).

Data Analysis

Data were analysed using the Statistical Package for the Social Sciences (SPSS) for Windows, version 24.0 and Statistica, version 13.0. Basic statistical analyses including descriptive statistics were performed. To ensure that the present study was adequately powered for the regression and PLS SEM path analyses performed, power calculations were determined using G*Power [51]. The power calculations confirmed that the study was adequately powered to perform the multivariate modelling. The number of baseline, emergent and persistent cases of depression and PTSD were computed using cut-off scores on the CES-D and DTS self-report measures. A conceptual framework guided all analyses (see Fig. 1). Multiple linear regression analyses were carried out to assess the relationship between the variables of interest. Predictor variables were entered simultaneously into the model using the enter method. Lastly, a PLS SEM path analysis was performed to confirm the results of the aforementioned analyses.

Results

Demographic Characteristics of the Sample

All participants were African black, Xhosa (African indigenous language) speaking (98%) women, with a mean age of 32.5 years, SD = 7.9 (range 18–50). The mean highest level of education was 10.5 years, SD = 1.7, ranging from five to 14 years. The majority were single (68%), unemployed (70%), and reported a combined annual household income of less than ZAR10 000 ($730) per year (89%).

Clinical Characteristics of the Sample

The mean CD4 T-cell count was 436 cells/mm^3, SD = 244, (range 25–1053 cells/mm^3). The mean HIV viral load was 65740.55 copies/ml, SD = 175212, and ranged from below the detectable limit to 1,052,380 copies/ml. The lower limit for detection was 40 copies/ml. The predominant HIV clade was subtype C. Forty-six (67%) of the 68 women living with HIV were on ART. Five cases seroconverted between baseline and follow-up. None of the women were on treatment for depression and/or PTSD at baseline or at follow-up.

Depression

At baseline, the mean CES-D score was 10.8, with a minimum of 0 and a maximum of 60. A year later, the mean CES-D scores was 8.7, with a minimum of 0 and a maximum of 60. At baseline, the mean CES-D scores for women living with HIV was 14 compared

to a CES-D score of 8 for uninfected women. A t-test revealed a significant difference between groups on the CES-D score at baseline ($p = 0.04$). Based on a cut-off score of 16 on the CES-D, the number of baseline, emergent and persistent cases of depression was calculated (see Table 1). Baseline cases refer to the number of women with depression at baseline only (i.e. remitted at the follow-up visit). Emergent cases refer to the number of women with new onset depression at follow-up (i.e. these women did not have depression at baseline). Persistent cases refers to the number of women with depression at baseline persisting through to follow-up.

PTSD

At baseline, the mean DTS score was 13.8, with a minimum of 0 and a maximum of 136. A year later, the mean DTS scores was 12, with a minimum of 0 and a maximum of 93. The mean DTS scores for women living with HIV was 17 compared to a DTS score of 11.5 for uninfected women. There were no significant differences between groups on the DTS score ($p > 0.31$). Based on a cut-off score of 40 on the DTS, the number of baseline, emergent and persistent cases of PTSD was calculated (see Table 1).

Trauma Exposure

Childhood trauma was determined with a score of > 40 on the CTQ-SF. In total, 79 women (53%) reported experiencing childhood trauma. The mean score on the CTQ-SF was 50, with a minimum of 25 and a maximum of 114. Of the 68 women living with HIV, 53 (78%) reported experiencing childhood trauma, with the majority of these cases reporting trauma in the severe to extreme range (n = 28). Of 80 uninfected women, 26 (32%) reported experiencing childhood trauma, with the majority of these cases in the mild to moderate range (n = 17). These women were also exposed to other discrete lifetime traumas regarded as index events for the development of PTSD. At baseline, the mean number of traumatic life events experienced was five with a maximum of 18 life events reported.

Regression

Socio-demographic and clinical characteristics shown in the conceptual framework (Fig. 1) were not significantly associated with baseline, follow-up or persistent depression in univariate analyses and were therefore not included in multivariate regression models.

For the group as a whole (N = 148), multiple linear regression revealed that childhood trauma (CTQ) and posttraumatic stress symptomatology (DTS) significantly predicted depression at baseline (Table 2). This model explained 28% of the variance of depressive symptomatology at baseline ($R^2 = 0.28$). Multiple linear regression revealed that for the group as a whole (N = 148), childhood trauma and PTSD symptoms at follow-up significantly predicted depression at follow-up and that this model explained 34% of the variance of depressive symptomatology at follow-up ($R^2 = 0.34$). See Table 3.

For women living with HIV (n = 68), multiple linear regression revealed that childhood trauma and posttraumatic stress symptomatology significantly predicted depression at baseline (Table 4). This model explained 26% of the variance of depressive symptomatology at baseline ($R^2 = 0.26$). Childhood trauma, traumatic life events at baseline and PTSD

symptoms at follow-up significantly predicted depression at follow-up and that this model explained 44% of the variance of depressive symptomatology at follow-up ($R^2 = 0.44$). See Table 5.

For uninfected women (n = 80), multiple linear regression revealed that childhood trauma, traumatic life events at baseline and posttraumatic stress symptomatology at baseline significantly predicted depression at baseline (Table 6). This model explained 30% of the variance of depressive symptomatology at baseline ($R^2 = 0.30$). PTSD symptoms at follow-up significantly predicted depression at follow-up with this model explaining 28% of the variance of depressive symptomatology at follow-up ($R^2 = 0.28$). See Table 7.

For the group as a whole (N = 148), multiple linear regression revealed that childhood trauma and posttraumatic stress symptomatology at follow-up significantly predicted persistent depression (Table 8). This model explained 24% of the variance of persistent depression ($R^2 = 0.24$).

PLS SEM Path Analysis

For the group as a whole (N = 148), path analysis revealed significant effects between depression at baseline and depression at follow-up ($p = 0.03$), childhood trauma and depression at baseline ($p < 0.01$), posttraumatic stress symptoms at baseline and depression at baseline ($p < 0.01$), and posttraumatic stress symptoms at follow-up with depression at follow-up ($p < 0.01$).

For women living with HIV, there was a significant relationship between childhood trauma and depression at baseline ($p = 0.002$), posttraumatic stress symptoms at baseline and depression at baseline ($p = 0.014$), and posttraumatic stress symptoms at follow-up with depression at follow-up ($p < 0.001$).

For uninfected women, there was a significant relationship between childhood trauma and depression at baseline ($p < 0.001$), posttraumatic stress symptoms at baseline and depression at baseline ($p = 0.005$), posttraumatic stress symptoms at follow-up with depression at follow-up ($p < 0.001$), posttraumatic stress symptoms at baseline and posttraumatic stress symptoms at follow-up ($p = 0.002$), and traumatic life events at baseline with depression at baseline ($p = 0.04$).

No significant differences ($p > 0.05$) were found when testing for differences in path coefficients between HIV-positive and HIV-negative women. However, there was a trend between life events at baseline and depression at both baseline and follow-up, with these coefficients approaching significance. See Fig. 2.

Discussion

The present study provides prevalence data on depression and PTSD, over time, in a cohort of South African women allowing us to investigate the dynamics of these disorders and their relation to biographical and socioeconomic factors (traumatic life events and childhood trauma) and HIV status. Results of the path analysis in this sample over a 12-month period

shows the multi-directional and entwined influence of early life stress, other life-threatening traumas across the lifespan, depression and PTSD over the course of HIV.

This sample represents South African women who are socioeconomically disadvantaged and face several challenges such as increased rates of childhood and adult traumatic experiences. There were no significant demographic differences between HIV infected and uninfected women in this sample. The women were Black African, poor (mean annual combined household income of less than 750 dollars), and mostly single and unemployed. Given that poverty and low education correlate with lower access to information and appropriate health care, and with greater exposure to violence, these factors can be broadly considered as contributors to disease and poor quality of life [52]. As proposed by the 'relative income hypothesis' (psychosocial hypothesis), income inequality has a greater impact on health than absolute low income as such, because social comparisons lead to stress and shame, which, in turn, enhance the propensity for several diseases [53].

As expected, and in line with existing literature, traumatic life events, childhood trauma and PTSD were associated with depression [54–56]. It is widely recognised that adversity arising from long-lasting social hardship, neglect, violence and sexual abuse, present risk factors for various neuropsychiatric disorders [55, 57–59]. The autonomic stress response system and resulting HPA axis changes are the most frequently understood underlying mechanisms [56, 60, 61]. Even though the stress response provides acute benefits for survival, when long lasting, it may create enduring neuroplastic changes that predispose to maladaptive neurobiological and emotional reactions to future negative events, such as socioeconomic challenges, poverty, low education and stigma [61, 62].

Childhood trauma and traumatic life events at baseline predicted depression at follow-up only for women living with HIV and not uninfected women. This finding suggests that there may be a more consistent and enduring relationship between childhood trauma and depression for PLHIV than for those uninfected by HIV. Because adverse early life experiences may control patterns of gene expression in adolescence and adulthood that modulate neurodevelopmental and neuroplastic changes [63–65], victims of adversity may be more vulnerable to additional adversity. Early life stress may induce a vulnerability to stress later in life, resulting in an increased risk for stress-related disorders [39]. Individuals with depression who have experienced early life adversity have a poorer response to antidepressant treatment [66] and increased relapse rates [67]. This highlights the need for screening and early detection of childhood trauma and the management of the effects thereof.

Considering the difficult life circumstances reported by the women in the present study, high rates of depression in these women at baseline could be expected. Indeed, with rates of 19% at baseline, 13.5% for emergent cases and 7% for persistent cases, these women have rates that are comparable to the general population of the US and South Africa [9, 68]. However, it should be noted that the depression rates were based on CES-D scores and not on clinical diagnosis. The CES-D was developed to identify populations at risk of developing depressive disorders but is not a clinical diagnostic tool [47]. Previous studies have found that the majority of depression cases resolved within six months [13, 69].

However, the aforementioned studies included non-traumatised individuals and the course of depression in individuals with early life stress is often different and characterised by chronicity [39]. Early life stress influences both response to treatment for depression as well as relapse rate [39, 67]. Early life stress and PTSD may be associated with treatment resistance and increased relapse rates of depression. Depression in HIV-infected women can emerge at any time following a diagnosis of HIV or initiation of ART. While there may be natural recovery of incident depressive episodes within a 12-month time frame, emergence and natural resolution of a major depressive episode may occur even within a 6-month time window as the course of major depressive disorder is highly variable. In the present study, the M.I.N.I.-Plus was administered at both baseline and 12-month follow up and the MDD module assessed for lifetime depression (which included the past 12 months). Item A6 in the M.I.N.I. module specifically asks "How many episodes of depression did you have in your lifetime? At the 12 month assessment, we were therefore able to capture any depressive episodes in the past year that may have occurred and had a natural recovery between the baseline and 12-month intervals. In this sample, no such episodes were reported. Providing some contrast, the rates of PTSD (10% at baseline, 6% for emergent cases and 4% for persistent cases) are below what we expected in this sample, with a 53% rate of reported childhood traumatic experiences. This suggests that mechanisms of resilience providing protection against the development of clinical depression and PTSD may be at play. This deserves further investigation. Notably, we previously reported that higher levels of resilience were associated with lower levels of self-reported depression in this sample of women [70].

Results of the present study revealed differences in depression between infected and uninfected women, with a higher endorsement of depressive symptomatology (CES-D score) among infected women at baseline compared to uninfected women. The finding of higher levels of depressive symptomatology among South African women living with HIV is consistent with studies documenting higher rates of psychiatric disorders among PLHIV in South Africa [8, 9, 71]. Stigma, fear of disclosure and lack of social support represent severe social and cultural problems [72]. HIV-related symptoms present another challenge, whether they arise directly from HIV or opportunistic infections, are due to comorbid illness, or manifest as side effects of cART [73]. Interestingly, among the ten cases of persistent depression, eight were women living with HIV. This finding may suggest that depression associated with HIV may be singular in some respect, and more resistant to remission. Longitudinal studies have been scarce but the few studies conducted report persistence of depression in HIV-infected individuals [8, 13, 74]. Substantial evidence indicates that among PLHIV, depression arises at least in part from virally mediated chronic neuroinflammation and neurotoxicity [75–77].

Despite not statistically differing in the rate of PTSD, infected and uninfected women in the present study have differential rates and severity of reported childhood trauma. Among uninfected women, 32% reported having experienced trauma during childhood, with the majority of these cases in the mild to moderate range, whereas 78% of women living with HIV experienced childhood trauma, with the majority of these cases reporting trauma in the severe to extreme range. These results point to a possible link between early life trauma and HIV infection. The HIV epidemic occurs largely within a context of considerable

social inequality, which leads people with a history of childhood abuse and adversity to be overrepresented among PLHIV [6, 26]. As a result of these prenatal and early life experiences, neuroplastic and epigenetic changes with lifelong and persisting effects emerge, in turn predisposing PLHIV to wide-ranging neuropsychiatric conditions [63, 65, 78–80].

A limitation of the present study was the retrospective assessment of childhood trauma and its contribution to recall bias. Moreover, the CTQ-SF does not include any questions around whether the abuse reported was a single isolated event(s) or repeated exposures. Moreover, depression and PTSD were self-reported and not based on a clinical diagnosis. Another limitation of the present study is that the factors associated with emergent depression in HIV are likely to differ from the factors associated with persistent depression. Ideally, the regressions for persistent and emergent depression should be parsed out and should be a consideration for other longitudinal studies of depression in HIV. Sub-analyses by HIV group may also be underpowered and should be considered as exploratory. Future studies are needed.

Conclusion

The present study, which is unique in documenting the course of untreated depression and PTSD in women with and without HIV infection with a high prevalence of early childhood trauma, provides evidence for increased depressive symptomatology among women living with HIV and persistence over time. Findings also suggest that there may be a more consistent and enduring relationship between childhood trauma and depression among women living with HIV compared to uninfected counterparts, highlighting the need to detect childhood trauma and intervene early.

Acknowledgements

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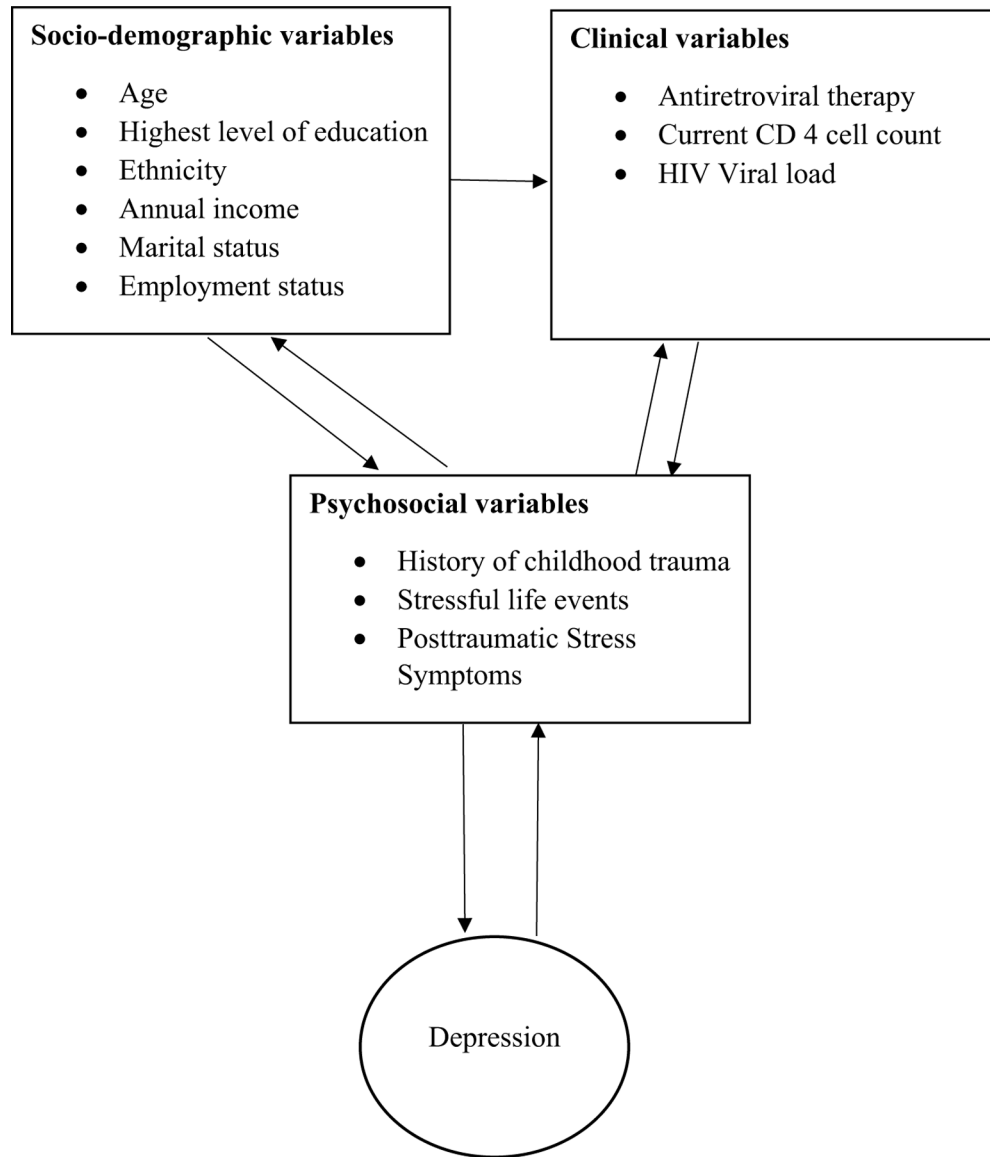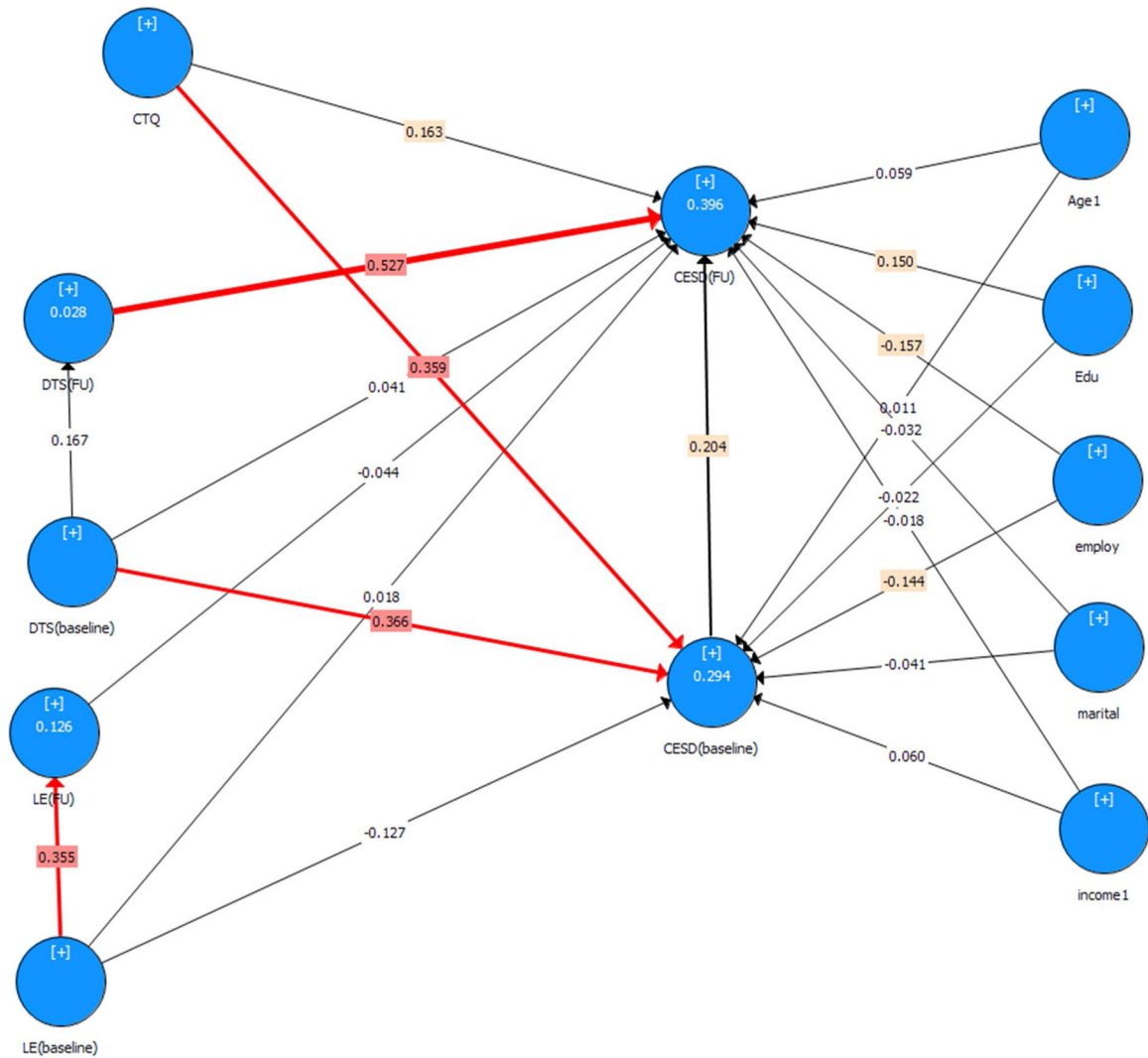
Fig. 1.
Conceptual framework

Fig. 2.
PLS SEM. *CTQ* childhood trauma, *DTS (baseline)* PTSD at baseline, *DTS (FU)* PTSD at
follow-up, *CESD (baseline)* depression at baseline, *CESD (FU)* depression at follow-up,
LE (baseline) traumatic life experiences at baseline, *LE (FU)* traumatic life experiences
at follow-up, *Edu* highest level of education, *employ* current employment status, *marital*
marital status, *income1* combined annual household income

Table 1

Baseline, persistent and emergent cases of depression and PTSD

	Total sample (N = 148)	HIV + (n = 68)	HIV − (n = 80)
Baseline depression	28 (19%)	13	15
Persistent depression	10 (7%)	8	2
Emergent depression	20 (13.5%)	9	11
Baseline PTSD	15 (10%)	10	5
Emergent PTSD	9 (6%)	5	4
Persistent PTSD	6 (4%)	2	4

Table 2

Regression summary for depression at baseline (N = 148)

Variable	β	SE of β	p	R^2	Adjusted R^2
Model	–	–	0.000	0.275	0.260
Childhood trauma	0.260	0.051	0.000		
PTSD (baseline)	0.171	0.039	0.000		
Life events (baseline)	−0.502	0.343	0.146		

Table 3

Regression summary for depression at follow-up (N = 148)

Variable	β	SE of β	p	R^2	Adjusted R^2
Model	–	–	0.000	0.311	0.335
Childhood trauma	0.138	0.046	0.003		
PTSD (baseline)	0.035	0.034	0.308		
Life events (baseline)	0.054	0.314	0.861		
PTSD (follow-up)	0.353	0.047	0.000		

Table 4

Regression summary for depression at baseline among women living with HIV (n = 68)

Variable	β	SE of β	p	R^2	Adjusted R^2
Model	–	–	0.000	0.257	0.222
Childhood trauma	0.274	0.093	0.004		
PTSD (baseline)	0.173	0.060	0.005		
Life events (baseline)	0.080	0.722	0.911		

Table 5

Regression summary for depression at follow-up among women living with HIV (n = 68)

Variable	β	SE of β	*p*	R^2	Adjusted R^2
Model	–	–	0.000	0.442	0.397
Childhood trauma	0.144	0.069	0.041		
PTSD (baseline)	0.057	0.044	0.203		
Life events (baseline)	0.131	0.549	0.043		
PTSD (follow-up)	0.395	0.069	0.000		

Table 6

Regression summary for depression at baseline among women without HIV (n = 80)

Variable	β	SE of β	p	R^2	Adjusted R^2
Model	–	–	0.000	0.301	0.273
Childhood trauma	0.353	0.090	0.000		
PTSD (baseline)	0.169	0.048	0.000		
Life events (baseline)	−0.920	0.336	0.007		

Table 7

Regression summary for depression at follow-up among women without HIV (n = 80)

Variable	β	SE of β	*p*	R^2	Adjusted R^2
Model	–	–	0.000	0.278	0.228
Childhood trauma	0.079	0.105	0.455		
PTSD (baseline)	0.020	0.058	0.732		
Life events (baseline)	−0.429	0.392	0.277		
PTSD (follow-up)	0.328	0.068	0.000		

Table 8

Regression summary for persistent depression (N = 148)

Variable	β	SE of β	p	R^2	Adjusted R^2
Model	–	–	0.000	0.241	0.220
Childhood trauma	0.004	0.001	0.000		
PTSD (baseline)	0.001	0.006	0.161		
Life events (baseline)	−0.001	0.001	0.816		
PTSD (follow-up)	0.004	0.001	0.000		