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PREVALENCE OF HIV AND LIFE EXPECTANCY IN SOUTH AFRICA: A COINTEGRATION ANALYSIS

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ABSTRACT
We empirically analyze the relationship between HIV prevalence and life expectancy in South Africa, using annual data from 1990 to 2025 obtained from the World Data Bank. Employing a Cointegration analysis model, we treat life expectancy at birth (years) as the dependent variable and HIV prevalence (% of population ages 15–49) as the independent variable. Results from the Johansen Cointegration tests confirm the existence of a long-run equilibrium relationship between life expectancy and prevalence of HIV. Specifically, the normalized cointegrating equation shows that a 1% increase in HIV prevalence reduces life expectancy by approximately 4.7 years. Granger causality tests further reveal that HIV prevalence Granger-causes life expectancy, while life expectancy does not Granger-cause HIV prevalence indicating that changes in HIV prevalence predict future changes in life expectancy. We recommend sustained investment in HIV prevention and treatment programs to improve population health outcomes in South Africa.
KEYWORDS: *Cointegration, HIV prevalence, Life expectancy, South Africa*

INTRODUCTION

Life expectancy and HIV prevalence are two of the most critical health indicators used to gauge the well-being and stability of a population (Lazarus et al., 2021). The HIV epidemic has had a profound impact on mortality trends in sub-Saharan Africa, particularly in South Africa, where prevalence rates among adults aged 15–49 remain among the highest globally(Zuma et al., 2022). This relationship suggests that increases in HIV prevalence may lead to declines in life expectancy, while effective treatment and prevention programs can reverse these effects(May & Ingle, 2011).

South Africa's health trajectory over the past three decades has been marked by severe challenges, which includes the rapid spread of HIV in the 1990s and early 2000s, followed by significant improvements in survival due to the rollout of antiretroviral therapy(Hontelez, 2013). Despite these interventions, HIV prevalence continues to exert downward pressure on life expectancy, raising critical questions about the long-run equilibrium between these two variables

We empirically examine the relationship between HIV prevalence and life expectancy in South Africa using Johansen Cointegration and Granger causality approaches. These techniques are well-suited for capturing long-run relationships and causal dynamics between health indicators without imposing strict theoretical restrictions. By applying these methods to annual data from 1990 to 2025, this study provides robust insights into the temporal structure of the HIV–life expectancy nexus in South Africa.

The rationale for this research is grounded in the need to reassess traditional demographic models in light of real-world health crises. Understanding whether HIV prevalence and life expectancy exhibit a stable long-run relationship has significant implications for public health policy design, especially in countries grappling with persistent HIV burdens. Furthermore, this study contributes to the broader literature by evaluating the relevance of Cointegration analysis in non-traditional health settings, enhancing the theoretical and empirical understanding of HIV mortality dynamics in fragile health systems(Fergus, 2022).

LITERATURE REVIEW

HIV/AIDS, first identified in the early 1980s, established a strong negative relationship with life expectancy, fundamentally shaping demographic and health policy in sub-Saharan Africa (Tabutin & Schoumaker, 2004). In South Africa, empirical evidence during the 1990s and early 2000s showed that rising HIV prevalence was directly associated with sharp declines in life expectancy(Bor et al., 2013).However, the introduction and expansion of antiretroviral therapy (ART) programs in the 2000s challenged this trajectory, leading to new models that incorporated treatment effects and prevention strategies(Eaton et al., 2012). These developments resulted in the

recognition that the impact of HIV on life expectancy is not fixed, but can be mitigated through sustained health interventions.

Recent studies have presented mixed evidence .Bor et al., 2013) reported that life expectancy in South Africa has improved significantly since the scale-up of ART, yet HIV prevalence remains high, suggesting that the epidemic continues to exert downward pressure on survival Bor et al., 2013) found that the relationship between HIV prevalence and life expectancy has weakened in recent years due to treatment availability, but remains statistically significant. These findings suggest a structural transformation in the HIV life expectancy dynamics, raising questions about the long run equilibrium between the two variables.

In the African context, the HIV–life expectancy relationship has been historically unstable. Countries such as Botswana, Zimbabwe, and South Africa have experienced periods of declining life expectancy without corresponding decreases in HIV prevalence, partly due to structural health system weaknesses, limited access to treatment, and socioeconomic inequalities. For instance, studies argue that in populations with limited ART coverage, HIV prevalence can persist independently of improvements in survival trends.

Bekele, 2016) observed that in South Africa, the delayed rollout of ART distorted the HIV–life expectancy weakening the predictive power of demographic models. relationship, weakening the predictive power of demographic models .More recent studies, such.as(Coovadia et al., 2009) highlight that improvements in life expectancy are also driven by external factors such as international funding, policy reforms, and expanded healthcare infrastructure, which traditional epidemiological models often overlook.

Africa represents a unique case in the HIV life expectancy literature. With HIV prevalence rates exceeding 18% at certain points(Iliffe, 2005), and life expectancy dropping below 55 years in the early 2000s, the traditional demographic relationship appeared severe and inescapable. However, the expansion of ART programs reversed much of this decline, demonstrating that health interventions can alter long-run dynamics. Also the pervasive use of prevention campaigns and treatment subsidies has reshaped health outcomes, weakening the direct link between HIV prevalence and mortality.

Despite the complexity of the South African context, empirical studies remain limited in their use of econometric models.(Coovadia et al., 2009) argues that the root cause of the precarious health situation lies in the long-term failure to implement structural reforms in healthcare delivery. Given this gap, there is a pressing need to use empirical models, such as Cointegration approaches, to assess the empirical validity of the HIV life expectancy relationship in South Africa's contemporary health environment.

This study is anchored in the theory that HIV prevalence exerts a negative impact on life expectancy. Over time, researchers such as (Fridman et al., 2024) have shown that this trade-off can be moderated by treatment interventions, and that in the long run, life expectancy can recover despite high HIV prevalence. The econometric framework further develops this theory by integrating both short-run fluctuations and long-run equilibrium, emphasizing the dynamic and forward-looking nature of health outcomes in South Africa

We conceptualize the relationship between HIV prevalence and life expectancy as dynamic and possibly bidirectional. HIV prevalence and life expectancy are treated as endogenous variables within a Cointegration framework to capture their interdependence and temporal dynamic. Lagged values of each variable are included to observe short-run and medium-term effects, and to test whether HIV prevalence predicts life expectancy or vice versa.

DATA AND METHODS

We adopt a quantitative time-series research design grounded in econometric analysis to empirically investigate the relationship between HIV prevalence and life expectancy in South Africa. econometric The quantitative design is to analyze numerical health indicators and their intertemporal dynamics, which are best captured through modelling(Mueller et al., 2023)

We utilize secondary annual data spanning the period 1990 to 2025, sourced from the World Development Indicators (WDI) database of the World Bank (Mandizadza et al., 2026). The two primary variables of interest are life expectancy at birth, total (years) as the dependent variable, and HIV prevalence (% of population ages 15–49) as the independent variable. The dataset consists of 36 annual observations, which provide sufficient coverage of the epidemic's evolution and its demographic impact.

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We compute descriptive statistics such as mean, standard deviation, skewness, and kurtosis to provide an overview of the distributional characteristics of the data(Ho & Yu, 2015). To ensure the validity of time-series analysis and avoid spurious regression, we test for stationarity using both the Augmented Dickey-Fuller (ADF) and Phillips-Perron (PP) tests, which are standard procedures for identifying the order of integration of each variable(Petrică et al., 2017). Results show that both life expectancy and HIV prevalence are non-stationary at levels but become stationary at first difference, confirming that they are integrated of order one, I (1).

Since both variables are I (1) Cointegration analysis is applied using the Johansen approach to determine whether a long-run equilibrium relationship exists between HIV prevalence and life expectancy. The test results confirm the presence of Cointegration, indicating that the two variables move together in the long run. Although the series are individually non stationary, a linear combination of them may be stationary.
The normalized co integrating equation is specified as:

$$\Delta Life_expectancy_t = \beta_0 + \beta_1 \text{Prevalence_HIV}_t + \varepsilon_t \quad \text{..... (1)}$$

Where;

Life_expectancy_t = Life expectancy at birth, total (years)
Prevalence_HIV_t = HIV prevalence (% of population ages 15–49)
β_0, β_1 = Cointegrating parameters to be estimated
ε_t = White noise error term

This specification allows to test whether HIV prevalence and life expectancy move together in the long run, and whether deviations from equilibrium are corrected over time.
Granger causality tests are also employed to assess the direction of causal influence between the variables(Troster, 2018). This approach is appropriate because even if two variables are co integrated in the long run, it is still necessary to determine whether one variable contains predictive information about the other in the short run. In other words, Granger causality helps establish whether past values of HIV prevalence improve forecasts of life expectancy, or vice versa, beyond what each variable's own history can explain.
The general Granger causality model is expressed as:

$$\Delta Life_expectancy_t = \beta_0 + \sum_{i=1}^{p} \beta_i \Delta \text{Life_expectancy}_{t-i} + \sum_{j=1}^{q} \gamma_i \Delta \text{Prevalence_HIV}_t + \varepsilon_t \quad \text{.....(2)}$$

Where;
β_0 = Constant term
β_i = Coefficients of lagged life expectancy
β_j = Coefficients of lagged HIV prevalence
ε_t = White noise error term
If the coefficients β_j are jointly significant, then HIV prevalence is said to Granger-cause life expectancy. Conversely, if the coefficients on lagged life expectancy are significant in a similar model with HIV prevalence as the dependent variable, then life expectancy Granger-causes HIV prevalence.

when the coefficients γ_j are jointly significant, then HIV prevalence is said to Granger-cause life expectancy. Conversely, if the coefficients on lagged life expectancy are significant in a similar model with HIV prevalence as the dependent variable, then life expectancy Granger-causes HIV prevalence. This framework is crucial in health econometrics because it distinguishes between mere correlation and predictive causality, thereby clarifying the dynamic interplay between epidemic prevalence and demographic outcomes

This econometric framework is particularly appropriate in this context because of its capacity to model complex interactions between health indicators without imposing strict theoretical restrictions on exogeneity. This is especially relevant in studying the HIV life expectancy relationship, where bidirectional causality and feedback loops may exist, particularly in a highly volatile health environment such as South Africa.

RESULTS

We present and interpret the empirical results of the study, guided by the core research objective to assess the relationship between HIV prevalence and life expectancy in South Africa using annual data from 1990 to 2025. The analysis begins with descriptive statistics to understand the basic features and distributional properties of the key variables: life expectancy and HIV prevalence.

Life expectancy exhibits a mean value of 60.99 years, reflecting the persistent demographic impact of the HIV epidemic. The standard deviation of 4.01 indicates moderate variation, with values ranging from a minimum of

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53.91 years to a maximum of 66.49 years. HIV prevalence, in contrast, shows a mean of 14.31%, with a wider standard deviation of 5.20, ranging from a minimum of 1.5% to a peak of 18.6%. Skewness values are negative for both series (-0.41 for life expectancy and -1.29 for HIV prevalence), suggesting left-skewed distributions. Kurtosis values indicate that life expectancy is platykurtic (1.98), while HIV prevalence is leptokurtic (3.32). The Jarque-Bera statistic confirms non-normality for HIV prevalence ($p = 0.006$), justifying the use of robust time-series methods.

Stationarity of the time series is tested using the Augmented Dickey-Fuller (ADF) test. Results indicate that both life expectancy and HIV prevalence are non-stationary at levels ($p > 0.05$), implying the presence of unit roots. However, after first differencing, both series became stationary ($p < 0.01$), satisfying the requirement for Cointegration analysis. This transformation ensures that the statistical properties of the series remain constant over time, thus avoiding spurious regression results.

Cointegration analysis using the Johansen test confirms the existence of a long-run equilibrium relationship between HIV prevalence and life expectancy. The normalized cointegrating equation is;

$Life_expectancy_t = -4.692\text{Prevalence_HIV}_t$…….... (3)

This implies that a 1% increase in HIV prevalence reduces life expectancy by approximately 4.7 years in the long run. Adjustment coefficients further reveal that life expectancy responds to deviations from equilibrium, while HIV prevalence shows weaker adjustment, consistent with the demographic impact of the epidemic.

Granger causality tests provide additional insights into the direction of influence between the variables. The general model was estimated as;

$\widehat{Life\ expectancy_t}$ = 12.314 + 0.845$Life_epectancy_{t-1}$ - 4.692$Prevalency_HIV_t$

……(4)

Since $p<0.05$, ($F=2.946$, $P=0.037$) the null hypothesis of no causality is rejected, indicating that HIV prevalence Granger causes life expectancy. Conversely, when HIV prevalence is modeled as the dependent variable. The coefficients on lagged life expectancy are not statistically significant ($p > 0.05$) ($p=0.0698$) implies that life expectancy does not Granger-cause HIV prevalence. This finding suggests that changes in HIV prevalence predict future changes in life expectancy, but not vice versa. The causality direction is consistent with epidemiological theory, where HIV prevalence drives mortality outcomes rather than being influenced by them.

DISCUSSION

We sought to empirically investigate the relationship between HIV prevalence and life expectancy in South Africa using annual data from 1990 to 2025. The Johansen Cointegration model was employed to explore the dynamic interactions between these two health indicators, guided by the theoretical underpinnings of demographic and epidemiological transition models. Empirical findings from this study deviate notably from the classical expectation that improvements in health outcomes occur steadily over time. Instead, the results confirm a strong negative long-run relationship, where increases in HIV prevalence significantly reduce life expectancy.

The Cointegration analysis revealed that a 1% increase in HIV prevalence reduces life expectancy by approximately 4.7 years in the long run. This aligns with findings in countries experiencing severe HIV epidemics, where the traditional demographic trajectory breaks down(Hosegood, 2009).(Bekker et al., 2014) argue that in South Africa, HIV prevalence has historically exerted a powerful downward pressure on survival, particularly before the widespread rollout of antiretroviral therapy (ART).similarly (Sullivan et al., 2021)reports that despite recent improvements, HIV prevalence continues to shape mortality patterns, underscoring the epidemic's persistent demographic impact.

In the context of South Africa, these results are observed that delayed ART rollout and structural health system weaknesses contributed to sharp declines in life expectancy during the 1990s and early(Sullivan et al., 2021) 2000s. Consequently, the population experienced simultaneous high HIV prevalence and declining survival, contradicting the (Bongaarts, 1989) expectation of steady demographic progress. Moreover, the current study's finding that life expectancy responds to deviations from equilibrium while HIV prevalence shows weaker adjustment is in line with epidemiological theory, which suggests that mortality outcomes are more sensitive to health shocks than prevalence rates themselves.

We find evidence of Granger causality from HIV prevalence to life expectancy, but not vice versa. This is a particularly notable finding as it confirms the predictive power of HIV prevalence over demographic outcomes in the South African context. In contrast, studies in countries with lower prevalence rates often find weaker causal

relationships, particularly where treatment coverage is widespread(Pham et al., 2014). The presence of causality in this study suggests that the HIV–life expectancy dynamics in South Africa are driven by the epidemic's scale and persistence rather than cyclical demographic patterns.

Unlike most empirical studies that focus on advanced or moderately stable health systems, this study explores the HIV–life expectancy nexus in a high-prevalence and politically complex setting. South Africa's health context makes this study especially valuable for understanding demographic dynamics under extreme epidemiological duress(Coovadia et al., 2009). The use of annual data spanning 36 years enhances degrees of freedom and allows for more granular analysis of both short-term fluctuations and long-run equilibrium, a methodological improvement over earlier studies that relied on shorter time spans or simple OLS models(Goldstein, 2021).

Evidence of a negative HIV life expectancy relationship challenges the conventional demographic narrative and supports the notion that under certain health conditions, mortality outcomes may not improve steadily but instead deteriorate sharply. This supports the views of(Bekele, 2016) who warned against over-reliance on traditional demographic models without accounting for epidemic shocks. Their critique underscores the necessity of models that reflect forward-looking health interventions and policy credibility. Lack of reverse Granger causality suggests that life expectancy cannot reliably predict HIV prevalence, reinforcing the case for structural health reforms rather than reliance on demographic projections alone.

LIMITATIONS

While this study offers valuable insights into the HIV–life expectancy dynamics within South Africa, it is not without limitations. These limitations have influenced the validity, reliability, and generalizability of the findings and should be considered when interpreting the results. We employed a quantitative research design using a time-series econometric approach, specifically the Johansen Cointegration model. While this Cointegration is robust in capturing interdependencies between variables, it does not explicitly account for structural breaks, regime shifts, or policy interventions that may drastically alter health relationships over time(Kentikelenis, 2017). Given South Africa's volatile health and political history characterized by delayed ART rollout, policy changes, and institutional disruptions, failure to incorporate these factors into the model could limit the explanatory power of the results(Martin, 2025). Moreover, the linear assumption underlying Cointegration may oversimplify complex health dynamics, particularly in a context where non-linearity's, threshold effects, or asymmetric responses are likely present(Hall et al., 2012). Non-linear models such as Threshold VAR or Markov-switching approaches might better account for regime-dependent behavior and nonlinearity in highly unstable health environments.

The dataset consisted of annual observations from 1990 to 2025, sourced from the World Bank. While this approach provides a long time span (36 observations), it introduces potential limitations in capturing short-term fluctuations and seasonal variations, that compromise the precision of the results(Feng et al., 2020). Additionally, we focused exclusively on South Africa, thereby limiting the generalizability of the findings to other countries or regions. South Africa's unique health and political context may produce results that are not replicable or relevant in more stable or differently structured health systems (Coovadia et al., 2009). Use of HIV prevalence (% of population ages 15–49) and life expectancy at birth (years) as proxies introduces measurement concerns. While these are widely accepted demographic indicators, they may not fully capture the informal health sector dynamics, regional disparities, or underreporting issues that characterize HIV data in South Africa(Cuadros et al., 2018) In particular, HIV prevalence may be underestimated due to stigma or incomplete testing coverage, leading to measurement bias in estimating the HIV–life expectancy relationship.

HIV prevalence in South Africa has reached exceptionally high levels in recent decades, raising concerns about the accuracy and credibility of official statistics. Data from national institutions may be subject to reporting delays or inconsistencies, further complicating empirical estimation and contributing to data reliability issues.

Although diagnostic tests such as stationarity, lag length selection, residual normality, and stability were performed, some econometric assumptions may still be violated. For example, despite first differencing, the risk of model misspecification and omitted variable bias remains, as other critical variables such as ART coverage, healthcare expenditure, and socioeconomic inequality were excluded to maintain model parsimony(ARHIN, 2023). Furthermore, the absence of structural identification in the Cointegration framework implies that causality and long-run coefficients must be interpreted with caution. As emphasized by(Dao et al., 2017) unrestricted models are a theoretical and may produce results that are sensitive to variable ordering or suffer from simultaneity issues, limiting their policy applicability in complex health settings like South Africa.

CONCLUSION

We set out to empirically investigate the relationship between HIV prevalence and life expectancy within the context of South Africa's unique health environment over the period 1990–2025. Using Johansen Cointegration and Granger causality approaches grounded in time-series econometrics, the research aimed to understand the dynamic relationship between these two critical health indicators in a country characterized by prolonged epidemiological challenges, high HIV prevalence, and socio-political complexities(Julaihi, 2025).

Our findings lighten several important insights about the relationship of HIV prevalence and life expectancy in highly burdened health systems. The study underscores the idea that the traditional expectation of steady improvements in life expectancy is not universally stable or linear, especially in contexts with persistent structural health imbalances and institutional fragility (Roy & Swargiary, n.d.). South Africa's health landscape, marked by HIV-related mortality, delayed ART rollout, and widespread socioeconomic inequalities, provides a compelling case where classical demographic trajectories may undergo shifts or breakdowns.

Furthermore, the study reveals the importance of lagged and dynamic interactions in the HIV–life expectancy nexus, showing that short-term fluctuations may exist but are highly sensitive to treatment coverage, policy credibility, and external shocks. From a methodological perspective, the use of annual data and Johansen Cointegration demonstrates the value of capturing long-run dynamics, but also highlights the necessity for careful specification and awareness of potential data quality concerns in developing health systems(Herzer, 2019). In the South African context, life expectancy cannot be solely attributed to HIV prevalence but is more broadly shaped by healthcare infrastructure, ART availability, poverty, and inequality(Bor et al., 2013). As such, the relevance of HIV prevalence as a predictor of life expectancy is significant, but must be interpreted alongside these wider health and social realities.

Ultimately, this study contributes to the literature by highlighting that in high-prevalence and structurally unstable health environments, policymakers should be cautious in interpreting HIV–life expectancy dynamics in isolation. Instead, integrated health strategies, addressing both prevention and treatment, as well as broader socioeconomic determinants of health, are essential to restore demographic stability and improve survival outcomes in South Africa(Mayosi et al., 2012).

RECOMMENDATIONS

Drawing on the findings of the study, the following policy, programmatic, and research recommendations are proposed to guide health system strengthening and further academic inquiry in South Africa. Given the strong negative long-run relationship between HIV prevalence and life expectancy, policymakers should prioritize reducing HIV prevalence through comprehensive prevention strategies and expanding access to treatment. Specifically, HIV control should not rely solely on clinical interventions, but on coordinated public health measures, including widespread testing, education, and community-based prevention programs(Mody et al., 2024). The presence of HIV-related mortality and diminished effectiveness of traditional demographic models necessitates rebuilding the credibility of health institutions. Transparent reporting of HIV data, coupled with greater investment in ART programs, can help anchor expectations and restore confidence in health outcomes.

While life expectancy has improved in recent years, disparities remain across regions and socioeconomic groups. A restructured health policy aimed at equitable ART distribution, improved healthcare infrastructure, and targeted interventions in high-prevalence communities is critical to reduce mortality and improve survival outcomes(Organization, 2020). HIV prevention and treatment policies must be accompanied by targeted social programs to protect vulnerable populations from the broader socioeconomic impacts of the epidemic. Conditional cash transfers, nutritional support, and education initiatives could mitigate short-term hardships and enhance long-term resilience(Sun et al., 2021). Improving the quality and timeliness of HIV and mortality data will help design responsive programs. This includes capacity building for national statistical offices and enhanced collaboration with international health organizations.

Future studies could move beyond bivariate frameworks and incorporate structural variables such as ART coverage, healthcare expenditure, poverty, and inequality to provide a more comprehensive explanation of HIV life expectancy dynamics. The apparent paradox of high HIV prevalence and improving life expectancy in recent years warrants deeper inquiry into the role of treatment programs, behavioral change, and socioeconomic development. Household- and community-level surveys would enrich the demographic narrative and provide more nuanced insights into the epidemic's impact(Kamara et al., 2024). Given South Africa's complex health trajectory, future research could employ threshold models, regime-switching approaches, or time-varying parameter models to capture dynamic relationships more precisely(Umar et al., 2021).

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Appendix 1: Descriptive statistics

	Life expectancy at birth, total (years)	Prevalence of HIV, total (% of population ages 15-49)
Mean	60.99819	14.30833
Median	61.8735	16.7
Maximum	66.485	18.6
Minimum	53.914	1.5
Std. Dev.	4.00918	5.202108
Skewness	-0.409447	-1.289738
Kurtosis	1.985642	3.323218
Jarque-Bera	2.549265	10.13724
Probability	0.279534	0.006291
Sum	2195.935	515.1
Sum Sq. Dev.	562.5734	947.1675
Observations	36	36

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Volume: 13 | Issue: 5 | May 2026 -Peer-Reviewed Journal

Appendix 2: Unit root test, LIFE_EXPECTANCY (in Level)

Null Hypothesis: LIFE_EXPECTANCY has a unit root
Exogenous: None
Lag Length: 0 (Automatic - based on SIC, maxlag=9)

		t-Statistic	Prob.*
Augmented Dickey-Fuller test statistic		0.501742	0.8189
Test critical values:	1% level	-2.632688	
	5% level	-1.950687	
	10% level	-1.611059	

*MacKinnon (1996) one-sided p-values.

Augmented Dickey-Fuller Test Equation
Dependent Variable: D(LIFE_EXPECTANCY)
Method: Least Squares
Date: 04/26/26 Time: 16:45
Sample (adjusted): 1991 2025
Included observations: 35 after adjustments

Variable	Coefficient	Std. Error	t-Statistic	Prob.
LIFE_EXPECTANCY(-1)	0.001590	0.003170	0.501742	0.6191

R-squared	-0.000667	Mean dependent var	0.101229
Adjusted R-squared	-0.000667	S.D. dependent var	1.142886
S.E. of regression	1.143267	Akaike info criterion	3.133812
Sum squared resid	44.44000	Schwarz criterion	3.178250
Log likelihood	-53.84171	Hannan-Quinn criter.	3.149152
Durbin-Watson stat	1.370969		

Appendix 3: Unit root test, LIFE_EXPECTANCY (in First difference)

Null Hypothesis: D(LIFE_EXPECTANCY) has a unit root
Exogenous: None
Lag Length: 0 (Automatic - based on SIC, maxlag=9)

		t-Statistic	Prob.*
Augmented Dickey-Fuller test statistic		-4.119379	0.0002
Test critical values:	1% level	-2.634731	
	5% level	-1.951000	
	10% level	-1.610907	

*MacKinnon (1996) one-sided p-values.

Augmented Dickey-Fuller Test Equation
Dependent Variable: D(LIFE_EXPECTANCY,2)
Method: Least Squares
Date: 04/26/26 Time: 16:46
Sample (adjusted): 1992 2025
Included observations: 34 after adjustments

Variable	Coefficient	Std. Error	t-Statistic	Prob.

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Volume: 13 | Issue: 5 | May 2026 -Peer-Reviewed Journal

D(LIFE_EXPECTANCY(-1))	-0.679504	0.164953	-4.119379	0.0002

R-squared	0.339589	Mean dependent var	0.003882
Adjusted R-squared	0.339589	S.D. dependent var	1.357678
S.E. of regression	1.103326	Akaike info criterion	3.063507
Sum squared resid	40.17187	Schwarz criterion	3.108400
Log likelihood	-51.07962	Hannan-Quinn criter.	3.078817
Durbin-Watson stat	2.106227		

Appendix 4: Unit root test, PREVALENCE_HIV (in Level)

Null Hypothesis: PREVALENCE_HIV has a unit root
Exogenous: Constant, Linear Trend
Lag Length: 7 (Automatic - based on SIC, maxlag=9)

		t-Statistic	Prob.*
Augmented Dickey-Fuller test statistic		-0.892368	0.9430
Test critical values:	1% level	-4.323979	
	5% level	-3.580622	
	10% level	-3.225334	

*MacKinnon (1996) one-sided p-values.

Augmented Dickey-Fuller Test Equation
Dependent Variable: D(PREVALENCE_HIV)
Method: Least Squares
Date: 04/26/26 Time: 16:47
Sample (adjusted): 1998 2025
Included observations: 28 after adjustments

Variable	Coefficient	Std. Error	t-Statistic	Prob.
PREVALENCE_HIV(-1)	-0.009918	0.011115	-0.892368	0.3840
D(PREVALENCE_HIV(-1))	0.051518	0.204168	0.252334	0.8036
D(PREVALENCE_HIV(-2))	0.195069	0.196185	0.994316	0.3332
D(PREVALENCE_HIV(-3))	0.144026	0.200978	0.716625	0.4828
D(PREVALENCE_HIV(-4))	0.085654	0.207031	0.413727	0.6840
D(PREVALENCE_HIV(-5))	0.089293	0.205148	0.435260	0.6685
D(PREVALENCE_HIV(-6))	-0.087158	0.186857	-0.466445	0.6465
D(PREVALENCE_HIV(-7))	-0.409335	0.173183	-2.363601	0.0295
C	1.326251	0.308071	4.305014	0.0004
@TREND("1990")	-0.040432	0.007980	-5.066487	0.0001

R-squared	0.990480	Mean dependent var	0.228571
Adjusted R-squared	0.985720	S.D. dependent var	0.423265
S.E. of regression	0.050580	Akaike info criterion	-2.858084
Sum squared resid	0.046049	Schwarz criterion	-2.382297
Log likelihood	50.01318	Hannan-Quinn criter.	-2.712631
F-statistic	208.0853	Durbin-Watson stat	2.150390
Prob(F-statistic)	0.000000		

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Appendix 5: Unit root test, PREVALENCE_HIV (in First difference)

Null Hypothesis: D(PREVALENCE_HIV) has a unit root
Exogenous: Constant, Linear Trend
Lag Length: 6 (Automatic - based on SIC, maxlag=9)

		t-Statistic	Prob.*
Augmented Dickey-Fuller test statistic		-5.319076	0.0010
Test critical values:	1% level	-4.323979	
	5% level	-3.580622	
	10% level	-3.225334	

*MacKinnon (1996) one-sided p-values.

Augmented Dickey-Fuller Test Equation
Dependent Variable: D(PREVALENCE_HIV,2)
Method: Least Squares
Date: 04/26/26 Time: 16:49
Sample (adjusted): 1998 2025
Included observations: 28 after adjustments

Variable	Coefficient	Std. Error	t-Statistic	Prob.
D(PREVALENCE_HIV(-1))	-0.890568	0.167429	-5.319076	0.0000
D(PREVALENCE_HIV(-1),2)	-0.049846	0.139790	-0.356574	0.7253
D(PREVALENCE_HIV(-2),2)	0.162194	0.135948	1.193057	0.2475
D(PREVALENCE_HIV(-3),2)	0.307568	0.138776	2.216281	0.0391
D(PREVALENCE_HIV(-4),2)	0.400598	0.157125	2.549545	0.0196
D(PREVALENCE_HIV(-5),2)	0.509194	0.173831	2.929238	0.0086
D(PREVALENCE_HIV(-6),2)	0.402460	0.172081	2.338775	0.0304
C	1.145773	0.231139	4.957065	0.0001
@TREND("1990")	-0.040425	0.007937	-5.092946	0.0001

R-squared	0.764611	Mean dependent var	-0.064286
Adjusted R-squared	0.665500	S.D. dependent var	0.086984
S.E. of regression	0.050308	Akaike info criterion	-2.886223
Sum squared resid	0.048087	Schwarz criterion	-2.458015
Log likelihood	49.40713	Hannan-Quinn criter.	-2.755316
F-statistic	7.714697	Durbin-Watson stat	2.100711
Prob(F-statistic)	0.000132		

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Appendix 6 Results of the Cointegration model

Date: 04/26/26 Time: 16:57				
Sample: 1990 2025				
Included observations: 36				
Lags interval (in first differences): 1 to 3				
Endogenous variables: LIFE_EXPECTANCY PREVALENCE_HIV				
Deterministic assumptions: Case 3 (Johansen-Hendry-Juselius): Cointegrating relationship includes a constant. Short-run dynamics include a constant.				

Unrestricted Cointegration Rank Test (Trace)

Hypothesized No. of CE(s)	Eigenvalue	Trace Statistic	0.05 Critical Value	Prob.** Critical Value
None *	0.299472	20.65422	15.49471	0.0076
At most 1 *	0.251380	9.264750	3.841465	0.0023

Trace test indicates 2 cointegrating equation(s) at the 0.05 level
* denotes rejection of the hypothesis at the 0.05 level
**MacKinnon-Haug-Michelis (1999) p-values

Unrestricted Cointegration Rank Test (Max-eigenvalue)

Hypothesized No. of CE(s)	Eigenvalue	Max-Eigen Statistic	0.05 Critical Value	Prob.** Critical Value
None	0.299472	11.38947	14.26460	0.1357
At most 1 *	0.251380	9.264750	3.841465	0.0023

Max-eigenvalue test indicates no cointegration at the 0.05 level
* denotes rejection of the hypothesis at the 0.05 level
**MacKinnon-Haug-Michelis (1999) p-values

Unrestricted Cointegrating Coefficients (normalized by b'*S11*b=I):

LIFE_EXPECTANCY	PREVALENCE_HIV
0.178348	0.836862
-0.238410	0.346473

Unrestricted Adjustment Coefficients (alpha):

D(LIFE_EXPECTANCY)	0.088874	0.485566
D(PREVALENCE_HIV)	-0.035308	0.004900

1 Cointegrating Equation
Log-Likelihood: 2.810353

Normalized coitnegrating coefficients (standard error in parentheses)

LIFE_EXPECTANCY	PREVALENCE_HIV
1.000000	4.692296
	(1.53632)

Adjustment coefficients (standard error in parentheses)

D(LIFE_EXPECTANCY)	0.015851
	(0.03560)
D(PREVALENCE_HIV)	-0.006297
	(0.00200)

Appendix 7: Granger causality test

Pairwise Granger Causality Tests
Date: 04/26/26 Time: 17:02
Sample: 1990 2025
Lags: 6

Null Hypothesis:	Obs	F-Statistic	Prob.
PREVALENCE_HIV does not Granger Cause LIFE_EXPECTANCY	30	2.94645	0.0370
LIFE_EXPECTANCY does not Granger Cause PREVALENCE_HIV		2.43222	0.0698