Original Research Article

A Panel Vector Error Correction Modeling of the Impacts of Demographic Indicators on Human Development in Nigeria

ABSTRACT

This study examined the impact of key demographic indicators on human development in Nigeria using an advanced panel econometric framework. Annual panel data covering the period 1994-2024 across Nigeria's six geopolitical regions were employed to analyze how life expectancy at birth (LEB), fertility rate (FR), urbanization rate (UR), dependency ratio (DR), and female labour force participation (FLP) influence the Human Development Index (HDI). The study employed descriptive statistics, first- and second-generation panel unit root tests, Johansen Fisher panel cointegration tests, Dynamic Panel Fully Modified Ordinary Least Squares (FMOLS), Panel Vector Error Correction Models (VECM), and Dumitrescu–Hurlin as well as panel pairwise Granger causality tests. Descriptive statistics revealed considerable volatility and non-stationary behaviour in the level series of all variables. However, stationarity was achieved after first differencing, indicating integration of order one, I(1). This was confirmed by both first-generation (IPS) and second-generation (CCE-ADF) unit root tests. The Johansen Fisher panel cointegration results provided strong evidence of a stable long-run equilibrium relationship between HDI and the demographic variables. Long-run estimates obtained through dynamic panel FMOLS showed that life expectancy at birth and female labour force participation exert significant positive effects on human development, while fertility rate, urbanization rate, and dependency ratio have negative long-run impacts. These findings highlight the growth-enhancing role of improved health outcomes and women's economic participation, alongside the development constraints imposed by high fertility, demographic dependency, and unplanned urban expansion. Short-run dynamics analyzed through the Panel VECM revealed that increases in life expectancy and female labour participation immediately enhance HDI, whereas higher fertility and dependency ratios reduce human development in the short term. Urbanization exhibited mixed effects, with negative contemporaneous impacts but positive lagged effects, suggesting that rapid urban growth initially strains infrastructure and social services but contributes to development once adjustment occurs. The error-correction term was negative and highly significant, indicating a strong annual convergence speed of about 70% toward long-run equilibrium. Finally, Dumitrescu-Hurlin panel causality tests revealed strong unidirectional causality from all demographic indicators to HDI, alongside bidirectional causality between HDI and both life expectancy and female labour force participation. The study concluded that demographic factors played a critical and enduring role in shaping Nigeria's human development path and recommended improvement in health investment, gender-inclusive labour policies, fertility reduction, strategic urban planning, and effective management of demographic dependency for sustainable development. *(My suggestion: the abstract should be brief include background, objective, methods, and result. This is due to the abstract is not briefly)*

Keywords: Demographic Indicator, Panel Modeling, Vector Error Correction, Johansen Fisher Cointegration, Granger Causality, Nigeria. *(My suggestion: the key words should be five words only)*

1. INTRODUCTION

The global population has undergone profound demographic transformations characterized by rapid population growth, declining fertility and mortality rates, and significant changes in age structure. Historically, it took more than 50,000 years for the world population to reach one billion; however, the twentieth century witnessed an unprecedented acceleration in population growth, with billions added within a few decades. Although the global population growth rate has declined from over 2 percent in the 1960s to approximately 1 percent in recent years, projections suggest that the world population will exceed nine billion in the coming decades. These demographic transitions have far-reaching implications for economic performance, social welfare, and human development outcomes across countries (Figueiras et al., 2020).

Despite improvements in global income levels, life expectancy, and educational attainment, rapid population growth continues to pose substantial challenges, particularly in developing regions. Increasing population pressure intensifies the demand for employment opportunities, healthcare services, education, housing, and basic infrastructure, while also contributing to environmental degradation. A significant proportion of the global population resides in developing countries, where future population growth is expected to be concentrated. This demographic pattern highlights the importance of understanding how population dynamics interact with development processes, especially in low- and middle-income economies (Balcı & Özcan, 2019).

Demographic transitions manifest differently across countries, resulting in varied development trajectories. While many developed economies are experiencing declining population growth and aging populations, several countries in Sub-Saharan Africa continue to experience high fertility rates and youthful age structures. Nigeria represents a prominent example of this trend and is projected to become one of the most populous countries in the world in the coming decades. A rapidly expanding working-age population has the potential to generate a demographic dividend, whereby increased labour supply and productivity can stimulate economic growth and improve human development outcomes if supported by adequate investments in education, healthcare, and employment creation (Adeyemi & Ogunsola, 2016).

One of the key demographic indicators used in analyzing population dynamics is the dependency ratio, which measures the proportion of dependents relative to the working-age population. A declining dependency ratio can stimulate economic growth by increasing labour participation, productivity, and savings, while a rising dependency ratio may impose fiscal burdens on governments and households. Current projections indicate that, unlike many developed regions experiencing rising dependency ratios due to aging populations, Africa is expected to witness declining dependency ratios in the future. This trend presents a potential demographic window of opportunity for improving human development outcomes, provided that effective policies are implemented to harness this demographic advantage (Aksentijević & Ježić, 2017).

However, demographic changes have not automatically translated into improvements in human development outcomes in many developing countries, including Nigeria. Human development, as defined by the United Nations Development Programme, emphasizes expanding people's capabilities and opportunities in health, education, and living standards rather than focusing solely on economic growth (UNDP, 2023). Despite its abundant natural and human resources, Nigeria continues to perform poorly in key human development indicators and ranks low on the Human Development Index. Persistent challenges such as high illiteracy rates, inadequate healthcare systems, and widespread poverty highlight the disconnect between demographic expansion and improvements in human well-being (Onyedinefu, 2022; Singh & Kamra, 2016).

Although several studies have explored the relationship between demographic variables and development outcomes, empirical findings remain mixed and often inconclusive. Many existing studies rely on static econometric methods that fail to capture the complex dynamic interactions and long-run relationships among demographic indicators and human development. Furthermore, Nigeria exhibits substantial regional heterogeneity in demographic structures and socio-economic conditions across its states and geopolitical zones. To address these gaps, this study employs a Panel Vector Error Correction Model (PVECM) to examine both the short-run and long-run effects of selected demographic indicators—urbanization rate, dependency ratio, female labour force participation rate, fertility rate, and life expectancy—on the Human Development Index across Nigeria. Using panel data covering the period 1994–2024 and incorporating cross-sectional and temporal variations, the study provides a comprehensive and policy-relevant analysis that can guide evidence-based and region-specific strategies for improving human development outcomes in Nigeria (Baltagi, 2008).

Empirical studies linking demographic indicators, human development, and economic performance particularly in Nigeria remain limited and fragmented. Much of the existing literature focuses on the nexus between human capital components (education, health, income) and economic growth rather than directly modeling demographic indicators and human development outcomes. Early studies such as Isola and Alani (2012) and Adeyemi and Ogunsola (2016) provide strong evidence that human capital development proxied by education, life expectancy, and literacy positively influences economic growth in Nigeria. These studies generally report long-run relationships and, in some cases, bidirectional causality between human capital and growth. However, their reliance on single-equation or static time-series frameworks limits their ability to capture feedback effects and dynamic interactions inherent in demographic and human development processes.

A substantial body of Nigerian literature has examined income inequality, poverty, and economic growth using time-series econometric techniques such as OLS, ARDL, ECM, VAR, and Granger causality tests (Ade-omonijo, 2021; Chinonye, 2022; Omolua & Tamunowariye, 2021). Findings from these studies are mixed: while some report negative effects of income inequality and poverty on economic growth, others find positive or statistically insignificant relationships. Similar inconsistencies are observed in cross-country and regional studies (Manyeki & Balázs, 2020; Ramirez et al., 2017), suggesting that the growth effects of inequality and poverty are context-specific and sensitive to model specification. Importantly, most of these studies focus on GDP growth rather than human development indicators such as HDI, thereby limiting their relevance for broader development analysis.

Health- and education-focused studies further underscore the importance of demographic-related variables for development outcomes. Research on education expenditure and

enrolment generally confirms positive long-run effects on economic growth and human capital formation, although issues of funding instability, policy inconsistency, and governance failures weaken these impacts (Aighokhan *et al*., 2015; Omojimite, 2019; Lawal & Wahab, 2018). Similarly, studies on health expenditure and life expectancy report positive but often weak or indirect effects on economic growth, with causality patterns varying across countries and time periods (Ibikunle, 2019; Ogungbenle *et al*., 2022; Bedir, 2016). These mixed results suggest that health and education investments influence development largely through complex transmission mechanisms that are not adequately captured by static models.

More recent studies have explicitly examined population dynamics and demographic structure in relation to economic growth in Nigeria. Findings from VECM, VAR, and ARDL-based studies indicate the existence of long-run relationships between population growth, age structure, fertility, and economic performance, with education emerging as a critical channel for realizing the demographic dividend (Borji *et al*., 2019; Ademola, 2019; Sakiru *et al*., 2017). Nonetheless, these studies predominantly focus on growth outcomes and treat demographic variables as exogenous, without adequately accounting for their endogenous interaction with human development indicators. Moreover, most analyses are conducted at the national level, ignoring significant sub-national heterogeneity across Nigeria's states and geopolitical zones.

Overall, the empirical literature reveals three major gaps. First, there is a scarcity of studies directly modeling the dynamic relationship between demographic indicators and human development in Nigeria. Second, existing studies rely heavily on static or single-equation approaches that fail to capture short-run adjustments, long-run equilibrium relationships, and feedback effects. Third, limited attention is given to panel-based and sub-national analyses that account for regional heterogeneity. These gaps justify the adoption of a Panel Vector Error Correction Model (PVECM) in the present study to simultaneously examine the short-run dynamics, long-run relationships, and causal interactions between demographic indicators and human development across Nigeria.

2. MATERIAL AND METHODS

2.1 Data Source

The data utilized in this study are demographic indicators as independent variables and Human Development Index (HDI) as dependent variable. The demographic indicators are Life Expectancy at Birth (LEB), Fertility Rate (FR), Urbanization Rate (UR), Female Labour Force Participation Rate (FLP) and Dependency Ratio (DR). The data is obtained from National Bureau Statistics (NBS), World Bank, Harmonized Nigeria Living Standard Survey (HNLSS), Nigerian Demographic and Health Survey (NDHSS) and National Population Commission (NPC) and spanned from 1994 to 2024.

2.2 Preliminary Tests

The following preliminary test are employed in this study.

2.2.1 Im, Pesaran and Shin (IPS) panel unit root test

The Im, Pesaran and Shin (IPS) panel unit root test is one of the first generation unit root test (Im, Pesaran and Shin, 2003). It is designed to determine whether a panel of time-series variables contains a unit root. Unlike the Levin–Lin–Chu (LLC) test, which imposes a homogeneous autoregressive coefficient across panel units, the IPS test allows for heterogeneous autoregressive processes, making it suitable for empirical studies involving diverse cross-sectional units.

The IPS test is estimated using the following individual Augmented Dickey–Fuller (ADF) regressions for each cross-sectional unit $i = 1,2,\dots,N$ over time $t = 1,2,\dots,T$ given in Equation (1) as shown:

$$\Delta y_{it} = \alpha_i + \beta_i y_{it-1} + \sum_{j=1}^{p_i} \gamma_{ij} \Delta y_{it-j} + \varepsilon_{it} \qquad (1)$$

where y_{it} is the variable of interest, $\Delta y_{it} = y_{it} - y_{it-1}$, α_i is the individual fixed effects, β_i is the autoregressive coefficient, p_i is the lag length chosen for each unit and $\varepsilon_{it}$is the white noise error term. The IPS framework permits $\beta_i \neq \beta_j \forall\ i \neq j$ allowing heterogeneous unit root behaviour across individuals.

The IPS test examines whether *all* (null) or *at least one* (alternative) cross-sectional units are stationary.

H_0: $\beta_i = 0$ for all $i = 1, 2, \ldots, N$ (Unit root for all panels) versus

H_1: $\begin{cases} \beta_i < 0 \ \text{for at least some } i \\ \beta_i = 0 \ \text{for the remaining units} \end{cases}$ (Some units are stationary)

This alternative is heterogeneous, meaning only a fraction of the panels must be stationary.

The individual ADF $t-$statistic for each cross-sectional unit i, is estimated as given in Equation (2).

$$t_{iT} = \frac{\hat{\beta}_i}{\mathrm{SE}(\hat{\beta}_i)} \tag{2}$$

The cross-sectional average of ADF statistic is computed as given in Equation (3):

$$\bar{t}_{NT} = \frac{1}{N}\sum_{i=1}^{N} t_{iT} \tag{3}$$

Im, Pesaran and Shin (2003) show that the Z_{NT} is given as shown in Equation (4).

$$Z_{NT} = \frac{\sqrt{N}(\bar{t}_{NT} - E(t_{iT}|H_0))}{\sqrt{Var(t_{iT}|H_0)}} \tag{4}$$

Under the null hypothesis and as $N, T \to \infty$: $Z_{NT} \xrightarrow{d} N(0,1)$. The values of $E(t_{iT}|H_0)$ and $Var(t_{iT}|H_0)$ are provided in IPS simulation tables.

Decision Rule: Using the standardized statistic Z_{NT}, we reject H_0 if $Z_{NT} < Z_\alpha$ for a one-sided test, typically $Z_{0.05} = -1.645$. We fail to reject H_0 if $Z_{NT} \geq Z_\alpha$. The rejection of H_0 implies that at least one panel is stationary (no panel-wide unit root). Failure to reject H_0 means that all panels contain a unit root (variables are non-stationary).

2.2.2 Pesaran's CCE (common correlated effects) approach

Pesaran (2015) proposes a second generation unit root test called the Common Correlated Effects (CCE) estimator to account for a general multifactor error structure in heterogeneous panels. For unit-root testing the same principle is used: augment each individual regression with cross-section averages of the dependent variable and (if present) regressors to proxy the unobserved common factors.

Consider a general heterogeneous panel model (no regressors or with exogenous regressors $x_{i,t}$. For unit-root testing of $y_{i,t}$ the augmented ADF (CCE-augmented) regression is given in Equation (5):

$$\Delta y_{i,t} = \alpha_i + \beta_i y_{i,t-1} + \boldsymbol{\pi}_i^{'}\bar{\mathbf{z}}_{t-1} + \sum_{j=1}^{p_i}\phi_{i,j}\Delta y_{i,t-j} + \sum_{j=0}^{q}\gamma_{i,j}^{'}\Delta\bar{\mathbf{z}}_{t-j} + \varepsilon_{i,t} \tag{5}$$

where $\bar{\boldsymbol{z}}_t$ is a vector of cross-section averages used as factor proxies, typical choices:

$$\bar{\mathbf{z}}_t = \begin{bmatrix} \bar{y}_t \\ \bar{x}_t \end{bmatrix}, \qquad \bar{x}_t = \frac{1}{N}\sum_i x_{i,t}$$

$\Delta\bar{\boldsymbol{z}}_{t-j}$ are lagged differences of cross-section averages, p_i is the lag order for the individual ADF to soak up serial correlation, q is a (small) number of lags for the averaged differences (often 0 or 1), β_i is the parameter of interest.

The mean of individual t-statistics are computed as given in Equation (6):

$$\bar{t}_N^{CCE} = \frac{1}{N}\sum_{i=1}^{N} t_i^{CCE} \tag{6}$$

The individual estimates, common corrected mean group (CCEMG), β_i are computed as given in Equation (7):

$$\hat{\beta}_{CCEMG} = \frac{1}{N}\sum_{i=1}^{N}\beta_i \tag{7}$$

The standard error is computed as shown in Equation (8):

$$se(\hat{\beta}_{CCEMG}) = \sqrt{\frac{1}{N^2}\sum_{i=1}^{N}\widehat{Var}(\hat{\beta}_i)} \tag{8}$$

Then the test statistic is computed as given by Equation (9):

$$t_{CCEMG} = \frac{\hat{\beta}_{CCEMG}}{se(\hat{\beta}_{CCEMG})} \quad (9)$$

The test checks the following pair of hypothesis:
H_0: $\beta_i = 0$ for all i (panel contains unit roots)
H_1: $\beta_i < 0$ for at least some i(at least some series are stationary after CCE control).
Decision rules: For mean-t approach, reject H_0 if $\bar{t}_N^{CCE}$ is less than the left-tail critical value (more negative) and fail to reject H_0 if $\bar{t}_N^{CCE}$ is greater than the left-tail critical value. For CCEMG approach, reject H_0 if $t_{CCEMG} < Z_\alpha$ (left tail). Always report one-sided p-values (left-tailed) because the alternative is $\beta < 0$.

2.3 Johansen Fisher Panel Cointegration Test

Johansen Fisher Panel Cointegration Test is a panel data extension of the Johansen cointegration method developed by Maddala and Wu (1999) and later adapted by others (e.g., Larsson *et al*., 2001). It combines individual Johansen cointegration test results across cross-sections using Fisher's formula.
The Johansen Fisher Panel Cointegration Test aims to determine whether there is a long-run equilibrium relationship among a set of non-stationary variables across multiple cross-sectional units (countries, regions, etc.) in a panel dataset (Johansen, 1988, 1991).
Let the panel data structure be represented as shown in Equation (10):

$$\boldsymbol{Y}_{it} = \begin{bmatrix} y_{1it} \\ y_{2it} \\ \vdots \\ y_{kit} \end{bmatrix}, \qquad i = 1, 2, \dots, N, t = 1, 2, \dots, T \quad (10)$$

where Y_{it} is a vector of k non-stationary I(1) variables for cross-section i at time t, N is the number of cross-sections (e.g., countries), T is the number of time periods. For each cross-section i, the Johansen cointegration test is applied through the Johansen's Vector Error Correction Model (VECM) is given as shown in Equation (11):

$$\Delta \boldsymbol{Y}_{it} = \boldsymbol{\Pi}_i \boldsymbol{Y}_{i,t-1} + \sum_{j=1}^{p-1} \boldsymbol{\Gamma}_{ij} \Delta \boldsymbol{Y}_{i,t-j} + \boldsymbol{\varepsilon}_{it} \quad (11)$$

where Δ denotes the first difference operator, $\boldsymbol{\Pi}_i = \boldsymbol{\alpha}_i \boldsymbol{\beta}_i^{'}$ where $\boldsymbol{\beta}_i$ is the cointegration vector (long-run relationship), α_i is the adjustment coefficient (speed of adjustment to equilibrium), $\boldsymbol{\Gamma}_{ij}$ is the short-run dynamics coefficients, ε_{it} is the white noise error term. For each i, the Johansen test provides two statistics namely, Trace statistic and Maximum eigenvalue statistic are computed as given in Equations (12) and (13):

$$Trace_i(r) = -T \sum_{j=r+1}^{k} \ln\left(1 - \hat{\lambda}_{ij}\right) \quad (12)$$

$$MaxEigen_i(r, r+1) = -T \ln\left(1 - \hat{\lambda}_{i,r+1}\right) \quad (13)$$

where $\hat{\lambda}_{ij}$ are the estimated eigenvalues from the Johansen procedure for cross-section i. To extend Johansen's test to panel data, we combine the p-values of the individual Johansen tests across all N cross-sections using Fisher's formula (Maddala and Wu, 1999) is given by Equations (14) and (15):

$$Fisher_{Trace} = -2 \sum_{i=1}^{N} \ln(p_i, Trace) \quad (14)$$

$$Fisher_{MaxEigen} = -2 \sum_{i=1}^{N} \ln(p_i, MaxEigen) \quad (15)$$

where $p_i, Trace$ and $p_i, MaxEigen$ are the p-values of the Johansen trace and maximum eigenvalue statistics for each cross-section. Under the null hypothesis (no cointegration in any cross-section), the Fisher statistics follow a chi-square distribution, $Fisher \sim \mathcal{X}_{2N}^2$.
The test checks the following pair of hypothesis:
H_0: $\Pi_i = 0$ for all i (No cointegration in any of the panel units)
H_1: $\text{rank}(\Pi_i) > 0$ for some i (At least one panel unit exhibits cointegration).

If Fisher statistic $> \mathcal{X}^2_{\alpha,2N}$, reject H_0 (i.e., evidence of cointegration), otherwise fail to reject H_0.

2.4 Panel dynamic ordinary least squares (Panel DOLS) model

The Panel Dynamic Fully Modified Ordinary Least Squares (Panel FMOLS) estimator is a robust econometric technique used for the estimation of long-run co-integrating relationships in panel data environments where variables are non-stationary and potentially co-integrated across cross-sectional units. Originally developed for time series analysis by Stock and Watson (1993), the DOLS method was later extended to panel settings by Kao and Chiang (2000) and Pedroni (2000). The key innovation of Panel DOLS lies in its ability to correct for endogeneity and serial correlation by augmenting the co-integrating regression with leads and lags of the first differences of the explanatory variables.

Let $i = 1, \dots, N$ index cross-sectional units and $t = 1, \dots, T$ index time. The Panel DOLS method can be developed under two major assumptions:

i. Homogeneous co-integrating vectors across individuals (Pedroni, 2000)
ii. Heterogeneous co-integrating vectors (Kao, 1999)

We now develop the detailed mathematical methodology under both frameworks.

For panel co-integrating regression model, let $y_{it} \in \mathbb{R}$ be the dependent variable, $\boldsymbol{x}_{it} = \left(x_{1,it}, x_{2,it}, \dots, x_{k,it}\right)' \in \mathbb{R}^k$ be a $k \times 1$ vector of explanatory variables. Assume all variables are integrated of order one, i.e., $y_{it} \sim I(1)$, $\boldsymbol{x}_{it} \sim I(1)$. Further assume there exists a cointegration relationship between y_{it} and $\boldsymbol{x}_{it}$. The co-integrating model for each unit i is given in Equation (16):

$$y_{it} = \alpha_i + \boldsymbol{x}_{it}'\boldsymbol{\beta}_i + u_{it} \tag{16}$$

where $\alpha_i$is the individual-specific intercept, $\boldsymbol{\beta}_i$ is cointegrating vector, $u_{it} \sim I(0)$ is stationary cointegrating residual. To correct for endogeneity and serial correlation, the DOLS model augments the cointegration equation with leads and lags of the first differences of $\boldsymbol{x}_{it}$ given in Equation (17) as:

$$y_{it} = \alpha_i + \boldsymbol{x}_{it}'\boldsymbol{\beta}_i + \sum_{j=-q}^{p} \boldsymbol{C}_{ij}'\Delta\boldsymbol{x}_{i,t-j} + \varepsilon_{it} \tag{17}$$

where p is the number of leads, q is the number of lags, $\boldsymbol{C}_{ij} \in \mathbb{R}^k$ coefficient vector for the j-th difference of $\boldsymbol{x}_{it}$, $\varepsilon_{it} \sim IID(0, \sigma^2)$ is white noise disturbance. To ensure consistency and asymptotic normality, we make the following assumptions

1. $y_{it}, \boldsymbol{x}_{it} \sim I(1)$ for all i.
2. Cointegration: There exists $\boldsymbol{\beta}_i$ such that $u_{it} = y_{it} - \alpha_i - \boldsymbol{x}_{it}'\boldsymbol{\beta}_i \sim I(0)$.
3. No perfect multicollinearity in regressors and their differences.
4. Stationary error term: $\varepsilon_{it} \sim I(0)$ with finite variance and zero mean, i.e., $E(\varepsilon_{it}) = 0, E(\varepsilon_{it}^2) = \sigma^2 < \infty$
5. Exogeneity correction: $\boldsymbol{x}_{it}$ may be endogenous, but endogeneity is corrected through $\Delta\boldsymbol{x}_{it\pm j}$.
6. Homoskedasticity: Error variance is constant across individuals and time.

For matrix formulation for each cross-sectional unit, define for each i:

$$\boldsymbol{y}_i = \left[y_{i,s+1}, y_{i,s+2}, \dots, y_{i,T}\right]' \in \mathbb{R}^{T-s};\ \boldsymbol{X}_i = \left[\boldsymbol{x}_{i,s+1}, \boldsymbol{x}_{i,s+2}, \dots, \boldsymbol{x}_{i,T}\right]' \in \mathbb{R}^{(T-s)\times k}$$

$$\boldsymbol{D}_{it} = \left[\Delta\boldsymbol{x}_{i,t-q}, \dots, \Delta\boldsymbol{x}_{i,t+p}\right]' \in \mathbb{R}^{k(p+q+1)};\ \boldsymbol{D}_i = \left[\boldsymbol{D}_{i,s+1}, \dots, \boldsymbol{D}_{i,T}\right]' \in \mathbb{R}^{(T-s)\times k(p+q+1)}$$

$$\boldsymbol{Z}_i = [\boldsymbol{X}_i, \boldsymbol{D}_i] \in \mathbb{R}^{(T-s)\times[k(p+q+1)]}$$

Then the regression model becomes as shown in Eqquation (18):

$$\boldsymbol{y}_i = \alpha_i \boldsymbol{1}_{T-s} + \boldsymbol{Z}_i\boldsymbol{\theta}_i + \boldsymbol{\varepsilon}_i \tag{18}$$

where $\boldsymbol{\theta}_i = \left[\boldsymbol{\beta}_i', vec\left(\boldsymbol{C}_{i,-q}, \dots, \boldsymbol{C}_{i,p}\right)'\right]'$.

The DOLS estimator for $\boldsymbol{\theta}_i$ is obtained by OLS as given in Equation (19):

$$\widehat{\boldsymbol{\theta}}_i = \left(\boldsymbol{Z}_i'\boldsymbol{M}_1\boldsymbol{Z}_i\right)^{-1}\boldsymbol{Z}_i'\boldsymbol{M}_1\boldsymbol{y}_i \tag{19}$$

where

$$\boldsymbol{M}_1 = \boldsymbol{1}_{T-s} - \frac{1}{T-s}\boldsymbol{1}_{T-s}\boldsymbol{1}_{T-s} \tag{20}$$

Extract $\widehat{\boldsymbol{\beta}}_i$, the first k elements of $\widehat{\boldsymbol{\theta}}_i$, as the estimator of the long-run coefficients for unit i.
The panel DOLS estimator for $\boldsymbol{\beta}$ depends on whether we assume:
(a) Homogeneous cointegrating vectors or Pooled DOLS (Pedroni, 2000)
Assume$\boldsymbol{\beta}_i = \boldsymbol{\beta}\ \forall i$, then stack all $\boldsymbol{y}_i$ and $\boldsymbol{Z}_i$:

$$\boldsymbol{y} = \begin{bmatrix} \boldsymbol{y}_1 \\ \boldsymbol{y}_2 \\ \vdots \\ \boldsymbol{y}_N \end{bmatrix}, \qquad \boldsymbol{Z} = \begin{bmatrix} \boldsymbol{Z}_1 \\ \boldsymbol{Z}_2 \\ \vdots \\ \boldsymbol{Z}_N \end{bmatrix}$$

Then:

$$\widehat{\boldsymbol{\theta}} = \left(\boldsymbol{Z}'\boldsymbol{M}_1\boldsymbol{Z}\right)^{-1}\boldsymbol{Z}'\boldsymbol{M}_1\boldsymbol{y} \tag{21}$$

(b) Heterogeneous cointegrating vectors or group-mean DOLS (Kao, 1999)
Estimate each $\widehat{\boldsymbol{\beta}}_i$ separately, then compute the panel average as given in Equation (22):

$$\widehat{\boldsymbol{\beta}}_{\text{panel}} = \frac{1}{N}\sum_{i=1}^{N}\widehat{\boldsymbol{\beta}}_i \tag{22}$$

For each cross-section, the residual variance is computed as shown in Equation (23):

$$\hat{\sigma}_i^2 = \frac{1}{T - s - k(1+p+q)}\sum_{t=s+1}^{T}\hat{\varepsilon}_{it}^2 \tag{23}$$

Covariance matrix of $\widehat{\boldsymbol{\beta}}_i$ is computed as given in Equation (24):

$$\widehat{Var}\left(\widehat{\boldsymbol{\beta}}_i\right) = \hat{\sigma}_i^2(\boldsymbol{Z}_i'\boldsymbol{M}_1\boldsymbol{Z}_i)^{-1}_{[1:k,1:k]} \tag{24}$$

Panel estimator (Kao-type) is computed as given in Equation (25):

$$\widehat{Var}\left(\widehat{\boldsymbol{\beta}}_{\text{panel}}\right) = \sum_{i=1}^{N}\left(\widehat{\boldsymbol{\beta}}_i\right) \tag{25}$$

Under assumptions, the panel DOLS estimator is asymptotically normal:

$$\sqrt{T}\left(\widehat{\boldsymbol{\beta}}_{\text{panel}} - \boldsymbol{\beta}\right) \xrightarrow{d} N(0, \Sigma)$$

This justifies the use of standard inference procedures such as t-tests for significance of individual long-run coefficients or Wald tests for joint restrictions.

2.5 Panel Vector Error Correction Model (PVECM)

Panel Error Correction Models (PECMs) are designed to capture both the long-run equilibrium and short-run dynamics among non-stationary but cointegrated panel variables (Pedroni, 2000). When variables are integrated of order one, $I(1)$, and share a cointegrating relationship, a deviation from the long-run equilibrium influences the short-run adjustments of the dependent variable. A Panel Vector Error Correction Model (PVECM) extends the Vector Error Correction Model (VECM) to panel data structures. It is suitable when all variables are integrated of order one $I(1)$, and a cointegration relationship exists among them. PVECMs allow for dynamic interdependencies among variables, cointegrating relationships, and heterogeneity across cross-sectional units (Pedroni, 2000).
Let $i = 1, 2, \ldots, N$ be cross-sectional units (e.g., regions), $t = 1, 2, \ldots, T$ be time periods, $\boldsymbol{y}_{it} = \left(y_{1,it}, y_{2,it}, \ldots, y_{k,it}\right)' \in \mathbb{R}^k$ is vector of k endogenous variable. Assume that $\boldsymbol{y}_{it} \sim I(1)$ i.e., each component of $\boldsymbol{y}_{it}$ is non-stationary but may be cointegrated.
The standard panel vector autoregressive VAP(p) model in levels is expressed as:

$$\boldsymbol{y}_{it} = \boldsymbol{A}_1\boldsymbol{y}_{i,t-1} + \boldsymbol{A}_2\boldsymbol{y}_{i,t-2} + \cdots + \boldsymbol{A}_p\boldsymbol{y}_{i,t-p} + \boldsymbol{\mu}_i + \boldsymbol{\varepsilon}_{it} \tag{26}$$

Where $\boldsymbol{A}_j \in \mathbb{R}^{k\times k}$ are autoregressive coefficient matrices, $\boldsymbol{\mu}_i$ are individual fixed effects,$\boldsymbol{\varepsilon}_{it}$ is white noise error terms.
To transformation to PVECM, if $\boldsymbol{y}_{it} \sim I(1)$ and cointegrated, the model is rewritten in VECM form:

$$\Delta\boldsymbol{y}_{it} = \boldsymbol{\Pi}\boldsymbol{y}_{i,t-1} + \sum_{j=1}^{p-1}\boldsymbol{\Gamma}_j\Delta\boldsymbol{y}_{i,t-j} + \boldsymbol{\mu}_i + \boldsymbol{\varepsilon}_{it} \tag{27}$$

where $\Delta\boldsymbol{y}_{it} = \boldsymbol{y}_{it} - \boldsymbol{y}_{i,t-1}, \boldsymbol{\Pi} = \boldsymbol{\alpha}\boldsymbol{\beta}'$, with $\boldsymbol{\beta} \in \mathbb{R}^{k\times r}$ is matrix of cointegrating vectors, $\boldsymbol{\alpha} \in \mathbb{R}^{k\times r}$ is matrix of adjustment coefficients, $\boldsymbol{\Gamma}_j \in \mathbb{R}^{k\times k}$ is short-run dynamics.

For error correction term and cointegration, let $\boldsymbol{\beta}'\boldsymbol{y}_{i,t-1}$ represents the long-run equilibrium relationships. $\boldsymbol{\alpha}$shows how each variable adjusts in response to deviations from the equilibrium. Thus:

$$\Delta\boldsymbol{y}_{it} = \boldsymbol{\alpha}\boldsymbol{\beta}'\boldsymbol{y}_{i,t-1} + \sum_{j=1}^{p-1}\boldsymbol{\Gamma}_j\Delta\boldsymbol{y}_{i,t-j} + \boldsymbol{\mu}_i + \boldsymbol{\varepsilon}_{it} \quad (28)$$

Apply panel unit root tests (e.g., LLC, IPS, ADF-Fisher or PP-Fisher) to confirm that all the variables are $I(1)$. Use panel cointegration tests (e.g., Pedroni, Westerlund) to confirm that long-run relationships exist among the variables of study. To estimate $\boldsymbol{\beta}$,methods like Johansen's system-based estimator such as Kao (1999) for homogeneous panels, Pedroni (2000) for heterogeneous cointegration, Fully Modified OLS (FMOLS) or Dynamic OLS (DOLS).

The PVECM is then estimated with known $\boldsymbol{\beta}$ as:

$$\Delta\boldsymbol{y}_{it} = \boldsymbol{\alpha}\boldsymbol{\beta}'\boldsymbol{y}_{i,t-1} + \sum_{j=1}^{p-1}\boldsymbol{\Gamma}_j\Delta\boldsymbol{y}_{i,t-j} + \boldsymbol{\mu}_i + \boldsymbol{\varepsilon}_{it} \quad (29)$$

The parameters of the model are estimated using Seemingly Unrelated Regression (SUR) for efficiency across equations or fixed effects if unobserved heterogeneity across entities is present.

Cointegrating rank r is determined from Johansen-type trace or maximum eigenvalue tests. Each row of $\boldsymbol{\alpha}$ shows how an endogenous variable responds to disequilibrium. $\boldsymbol{\Gamma}_j$ is the short-run interdependencies among variables. The test checks the following pair of hypothesis:

H_0: $\alpha = 0$ (No adjustment to long-run equilibrium) versus

H_1: $\alpha \neq 0$ (There is adjustment to long-run equilibrium).

H_0: $\boldsymbol{\Gamma}_j = 0$ (No short-run dynamics) versus

H_1: $\boldsymbol{\Gamma}_j \neq 0$ (There are short-run dynamics).

The Wald test or LM test is employed to test for joint or individual restrictions.

2.7 Dumitrescu-Hurlin panel Granger causality test

The Dumitrescu-Hurlin (2012) test is designed for testing Granger causality across panel units when relationships are heterogeneous across cross-sections. Unlike standard fixed-effects models, it allows different causal dynamics for different cross-sectional units. Let N be the number of cross-sectional units (entities), T be the number of time periods (observations per unit), p be the number of lags, x_{it} be the independent (causal) variable, y_{it} be the dependent (effect) variable. The panel model is specified as shown in Equation (30):

$$y_{it} = \alpha_i + \sum_{k=1}^{p}\gamma_{i,k}y_{i,t-k} + \sum_{k=1}^{p}\beta_{i,k}x_{i,t-k} + \varepsilon_{it} \quad (30)$$

where α_i is fixed intercept for unit i, $\gamma_{i,k}$ is lag coefficients of the dependent variable,$\beta_{i,k}$ is Granger causality coefficients,ε_{it}~$iid(0,\sigma^2)$ is white noise error. The test checks the following pair of hypotheses:

H_0: $\beta_{i,1} = \beta_{i,2} = \beta_{i,3} = \cdots = \beta_{i,p} = 0\ \forall i = 1,2,\ldots,N$ (no Granger causality for any individual unit).

H_1: $\beta_{i,k} = 0$ for $i = 1,1,\ldots,N_1;\ \beta_{i,k} \neq 0$ for $i = N_1+1,\ldots,N$ (Granger causality exists for at least some units).

Where $0 < N_1/N < 1$: a subset of units exhibit no causality, while others do.

For each cross-sectional unit $i = 1,2,\ldots,N$, we estimate Equation (1) ad run OLS to estimate the model for each i. For each unit i, we compute the Wald statistic $W_{i,T}$ for testing:

$$H_0^i: \beta_{i,1} = \beta_{i,2} = \beta_{i,3} = \cdots = \beta_{i,p} = 0$$

Let $W_{i,T}$ be F-statistic (or Wald) with p restrictions, the average Wald statistic is computed as given in Equation (31):

$$\bar{W}_{N,T} = \frac{1}{N}\sum_{i=1}^{N}W_{i,T} \quad (31)$$

We compute the standardize the statistic called Z-bar statistic under the null hypothesis and assuming $T \to \infty$, Dumitrescu and Hurlin (2012) showed that:

$$Z_{N,T} = \sqrt{N}\left(\frac{\bar{W}_{N,T}-\mu_T}{\sigma_T}\right) \xrightarrow{d} N(0,1) \quad (32)$$

Where μ_T and σ_T are the mean and standard deviation of the individual Wald statistics under H_0, given by Equation (33):

$$\mu_T = \frac{p}{T-2p-1} \text{and} \sigma_T = \frac{2p(T-3p-1)}{(T-2p-1)^2(T-2p-3)} \quad (33)$$

These are approximated analytically based on asymptotic distribution theory.

If $|Z_{N,T}| > Z_{\alpha/2}$, we reject H_0 (there is Granger causality for at least some units.), otherwise, we fail to reject H_0.

2.8 Panel Pairwise Granger Causality Analysis

Causality analysis in panel data examines whether past values of one variable help to predict current values of another while controlling for individual heterogeneity. Based on Granger's concept of causality, the panel framework incorporates both cross-sectional and temporal dimensions (Granger, 1969). The aim is to test pairwise Granger causality among the panel variables for N cross-sectional units observed over T time periods.

Let us assume balanced panel data and let $i = 1, 2, \dots, N$ be cross-sectional units, $t = 1, 2, \dots, T$ be time periods, $y_{k,it}$ be the value of variable k for unit i at time t, where $k = 1,2, \dots ,6$.

Let p be the number of lags (same for all variables for simplicity). The Panel VAR(p) in levels for each variable $y_{k,it}$ is given by Equation (34):

$$y_{k,it} = \alpha_{k,i} + \sum_{j=1}^{p} \sum_{\ell=1}^{6} \beta_{k\ell}^{(j)} x_{\ell,i,t-j} + \varepsilon_{k,it} \quad (34)$$

where $\alpha_{k,i}$ is fixed effect for variable k and unit i,$\beta_{k\ell}^{(j)}$ is the coefficient of the j-th lag of variable ℓ in equation for variable k, $\varepsilon_{k,it}$ is the error term for variable k.

Let us now fully estimate each equation in the Panel Granger Causality framework, using $y_{i,t}$ as dependent variable, $x_{i,t}^{(j)}$, where $i = 1, 2, \dots, 6$ as independent variables. Below are the 6 separate equations of the system (assuming 1 lag for simplicity):

$$y_{1,it} = \alpha_{1,i} + \beta_{11}^{(1)} x_{1,i,t-1} + \beta_{12}^{(1)} x_{2,i,t-1} + \beta_{13}^{(1)} x_{3,i,t-1} + \cdots + \beta_{16}^{(1)} x_{6,i,t-1} + \varepsilon_{1,it} \quad (35)$$

$$y_{2,it} = \alpha_{2,i} + \beta_{21}^{(1)} x_{1,i,t-1} + \beta_{22}^{(1)} x_{2,i,t-1} + \beta_{23}^{(1)} x_{3,i,t-1} + \cdots + \beta_{26}^{(1)} x_{6,i,t-1} + \varepsilon_{2,it} \quad (36)$$

$$y_{3,it} = \alpha_{3,i} + \beta_{31}^{(1)} x_{1,i,t-1} + \beta_{32}^{(1)} x_{2,i,t-1} + \beta_{33}^{(1)} x_{3,i,t-1} + \cdots + \beta_{36}^{(1)} x_{6,i,t-1} + \varepsilon_{3,it} \quad (37)$$

$$y_{4,it} = \alpha_{4,i} + \beta_{41}^{(1)} x_{1,i,t-1} + \beta_{42}^{(1)} x_{2,i,t-1} + \beta_{43}^{(1)} x_{3,i,t-1} + \cdots + \beta_{46}^{(1)} x_{6,i,t-1} + \varepsilon_{4,it} \quad (38)$$

$$y_{5,it} = \alpha_{5,i} + \beta_{51}^{(1)} x_{1,i,t-1} + \beta_{52}^{(1)} x_{2,i,t-1} + \beta_{53}^{(1)} x_{3,i,t-1} + \cdots + \beta_{56}^{(1)} x_{6,i,t-1} + \varepsilon_{5,it} \quad (39)$$

$$y_{6,it} = \alpha_{6,i} + \beta_{61}^{(1)} x_{1,i,t-1} + \beta_{62}^{(1)} x_{2,i,t-1} + \beta_{63}^{(1)} x_{3,i,t-1} + \cdots + \beta_{66}^{(1)} x_{6,i,t-1} + \varepsilon_{6,it} \quad (40)$$

To test whether variable y_k Granger-causes x_ℓ, the null hypothesis is:

H_0: $\beta_{\ell k}^{(1)} = \beta_{\ell k}^{(2)} = \beta_{\ell k}^{(3)} = \cdots = \beta_{\ell k}^{(p)} = 0$ (i.e., past values of y_k do not help predict x_ℓ.)

H_1: $\beta_{\ell k}^{(1)} \neq \beta_{\ell k}^{(2)} \neq \beta_{\ell k}^{(3)} \neq \cdots \neq \beta_{\ell k}^{(p)} \neq 0$ (i.e., past values of y_k do help predict x_ℓ.)

Decision Rule: If p-value < 0.05, reject H_0 and conclude that variable x Granger-causes variable y. However, if p-value ≥ 0.05, do not reject H_0 and conclude that there is no statistical evidence of Granger causality from x to y.

3. RESULTS AND DISCUSSION

3.1 Summary Statistics and Normality Measures

Table 1 presents the descriptive statistics of the demographic study variables used in modeling the relationship between demographic indicators and human development in Nigeria. The variables include the Human Development Index (HDI), Life Expectancy at Birth (LEB), Fertility Rate (FR), Urbanization Rate (UR), Dependency Ratio (DR), and Female Labour Force Participation Rate (FLP). The summary statistics provide deeper understanding of the central tendency, dispersion, and distributional properties of the data. Additionally, the Jarque-Bera normality test results highlight whether the variables follow a normal distribution, which is relevant in determining the appropriate econometric methods for estimation.

Table 1: Summary Statistics of Demographic Study Variables

	HDI	LEB	FR	UR	DR	FLP
Mean	347.01	54.49	95.759	101.29	103.55	98.60
Maximum	616.70	56.04	188.45	765.08	573.71	189.35
Minimum	165.76	42.479	17.613	17.273	18.345	19.383
Std. Dev.	110.58	30.914	31.434	60.654	62.872	36.178
Skewness	0.3881	4.3594	0.5129	7.3565	4.1057	0.7155
Kurtosis	2.4632	37.889	4.5580	80.956	26.462	3.5081
Jarque-Bera	6.6786	9699.8	26.101	47202	4634.2	17.294
p-value	0.0355	0.0000	0.0000	0.0000	0.0000	0.0002
N	180	180	180	180	180	180

The summary statistics reported in Table 1 provide good understanding of the behaviour of the demographic variables under study. The Human Development Index (HDI) has a mean value of 347.01, with a minimum of 165.76 and a maximum of 616.70, suggesting moderate but widely varying levels of human development across the study period. Life Expectancy at Birth (LEB) averages 54.49 years, which is relatively low compared to global standards, and ranges between 42.48 and 56.04 years. Fertility Rate (FR) has an average of 95.76 births per 1,000 women, while Female Labour Force Participation (FLP) averages 98.60%, indicating a relatively high level of women's involvement in the labor market. Urbanization Rate (UR) and Dependency Ratio (DR) have mean values of 101.29% and 103.55% respectively, with wide variations across the data, reflecting fluctuations in demographic pressures and urban growth.

In terms of dispersion, the standard deviation for HDI (110.58) indicates notable variation, while LEB (30.91), FR (31.43), UR (60.65), DR (62.87), and FLP (36.18) equally show substantial variability, especially UR and DR, which highlight significant structural demographic imbalances. The skewness values reveal that all variables are positively skewed, with UR (7.36) and LEB (4.36) being highly skewed, implying the presence of extreme values in the distributions. Similarly, kurtosis statistics show that most variables are leptokurtic, with LEB (37.89), UR (80.96), and DR (26.46) being particularly peaked and heavy-tailed, further suggesting the influence of outliers.

The Jarque-Bera normality test confirms that all variables except HDI deviate significantly from normality, with p-values of 0.0000 for LEB, FR, UR, DR, and FLP. Although HDI has a p-value of 0.0355, which is borderline, it still shows slight deviation from normality. Overall, the results suggest that the demographic indicators exhibit considerable variability, skewness, and leptokurtosis, with strong evidence against normality. This has important implications for econometric modeling, as it underscores the need for robust estimation techniques or variable transformations to account for non-normal distributions in subsequent analysis.

3.2 Panel Unit Root Test Results

To assess the stationarity of the variables in the panel data set, the study employed the Im, Pesaran and Shin (IPS) and Pesaran CCE-ADF panel unit root tests as the first and second generation unit root tests respectively. These tests were conducted on the variables in both their log-levels and first-difference forms, considering two specifications: intercept only and intercept with trend. The aim is to determine whether the variables are stationary at levels or require differencing to achieve stationarity, which is a crucial prerequisite for valid panel regression and cointegration analysis. (Put here, Furthermore, First and Second Generation Panel Unit Root Tests Results of the Log Panels is presented in Table 2)

Table 2: First and Second Generation Panel Unit Root Tests Results of the Log Panels

Variable	Option	Im, Pesaran and Shin		Pesaran CCE-ADF	
		IPS Stat.	p-value	CCE-ADF (t_i^{CCE})	p-value
		Log Panels in Levels			
LHDI	Intercept only	-0.10674	0.4575	-0.8802	0.4030
	Intercept & trend	0.89742	0.8153	1.6503	0.5308
LLEB	Intercept only	-1.64092	0.3474	-0.9632	0.5712
	Intercept & trend	-2.24192	0.2461	1.7803	0.4182
LFR	Intercept only	-1.28635	0.1271	-0.8253	0.6052
	Intercept & trend	-0.22681	0.1135	1.7473	0.5172
LUR	Intercept only	-1.20656	0.5007	-0.5695	0.4144
	Intercept & trend	-0.35404	0.4393	1.7788	0.4293
LDR	Intercept only	-0.01856	0.4926	-0.6518	0.2852
	Intercept & trend	-0.94059	0.1735	1.7914	0.5595
LFLP	Intercept only	-1.31795	0.6253	-0.9054	0.3341
	Intercept & trend	-0.17813	0.6174	1.7837	0.4741
		First and Second Generation First Difference of Log Panels			
Δ*L*HDI	Intercept only	-4.14947	0.0000	-10.6603	0.0000
	Intercept & trend	-4.47672	0.0000	-10.7272	0.0000
Δ*L*LEB	Intercept only	-6.76872	0.0000	-8.7054	0.0000
	Intercept & trend	-6.44908	0.0000	-8.2351	0.0000
Δ*LFR*	Intercept only	-7.55148	0.0000	-10.5823	0.0000
	Intercept & trend	-6.18210	0.0000	-10.5491	0.0000
Δ*LUR*	Intercept only	-7.40423	0.0000	-10.7252	0.0000
	Intercept & trend	-6.04395	0.0000	-10.3376	0.0000
Δ*LDR*	Intercept only	-7.06935	0.0000	-9.3480	0.0000
	Intercept & trend	-6.25374	0.0000	-9.8217	0.0000
Δ*LFLP*	Intercept only	-7.01661	0.0000	-11.5646	0.0000
	Intercept & trend	-5.61511	0.0000	-11.5094	0.0000

(Put here, From Table 2, we see that) the first and second generation panel unit root tests results reported in Table 2 show that at levels, none of the variables are stationary under either the IPS or Pesaran CCE-ADF tests. For example, LHDI, LLEB, LFR, LUR, LDR, and LFLP all return insignificant p-values at levels, with intercept and with trend, all of which fail to reject the null hypothesis of a unit root. confirming the presence of non-stationarity. However, when the variables were transformed into their first differences, strong evidence of stationarity emerges across all variables and test specifications. For instance, ΔLHDI, ΔLLEB, ΔLFR, ΔLUR, ΔLDR, and ΔLFLP have significant p-values both under intercept only and with intercept and linear trend.

These findings indicate that the study variables are integrated of order one, I(1). In other words, they are non-stationary in levels but become stationary after first differencing. This implies that conventional regression on variables in levels could produce spurious results, but cointegration techniques can be applied to explore the existence of long-run equilibrium relationships among the variables.

3.3 Johansen Fisher Panel Cointegration Test Results

To determine the existence of a long-run equilibrium relationship among human development (HDI) and selected demographic indicators, the Johansen Fisher panel cointegration test was applied. This method combines Johansen's individual cointegration tests across panel units using Fisher's combination test based on both the trace statistic and the maximum eigenvalue statistic. The null hypothesis at each level is that there are no cointegrating equations (no long-run relationship), with alternative hypotheses testing for at least one or more cointegrating vectors. Statistical significance is judged using the p-values associated with each Fisher statistic. The results of Johansen Fisher cointegration tests are reported in Tables 3 and 4.

Table 3: Johansen Fisher Panel Cointegration Test Results

Hypothesized No. of CE(s)	Fisher Stat.* (from trace test)	p-value	Fisher Stat.* (from max-eigenvalue test)	p-value
None*	246.9	0.0000	118.4	0.0000
At most 1*	159.8	0.0000	80.63	0.0000
At most 2*	95.17	0.0000	44.88	0.0000
At most 3*	59.54	0.0000	30.19	0.0026
At most 4*	42.63	0.0000	24.35	0.0182
At most 5*	47.59	0.0000	47.59	0.0000

Table 4: Johansen Fisher PanelCointegration Test for Individual Cross Sections

Cross Section	Trace Test Statistics	p-value	Max. Eigenvale Test Statistics	p-value
Hypothesis of no cointegration				
NC	115.3944	0.0012	36.0518	0.1326
NE	195.6408	0.0000	71.2638	0.0000
NW	237.5416	0.0000	99.3602	0.0000
SE	181.6984	0.0000	68.2179	0.0000
SS	166.4460	0.0000	59.7717	0.0001
SW	158.4408	0.0000	51.6579	0.0016
Hypothesis of at most 1 cointegration relationship				
NC	79.3426	0.0072	26.5278	0.2895
NE	124.3770	0.0000	51.0906	0.0002
NW	138.1813	0.0000	63.1951	0.0000
SE	113.4805	0.0000	46.7447	0.0009
SS	106.6743	0.0000	43.1417	0.0030
SW	106.7829	0.0000	40.2208	0.0077
Hypothesis of at most 2 cointegration relationship				
NC	52.8149	0.0159	22.8129	0.1816
NE	73.2864	0.0000	33.3070	0.0082
NW	74.9862	0.0000	33.8472	0.0069
SE	66.7358	0.0003	32.0161	0.0126
SS	63.5326	0.0009	30.1123	0.0232
SW	66.5621	0.0004	26.9495	0.0601
Hypothesis of at most 3 cointegration relationship				
NC	30.0019	0.0474	16.9890	0.1725
NE	39.9794	0.0024	24.0345	0.0189
NW	41.1390	0.0016	20.7314	0.0568
SE	34.7197	0.0125	16.4817	0.1980
SS	33.4203	0.0183	19.2684	0.0893
SW	39.6127	0.0027	19.4353	0.0849
Hypothesis of at most 4 cointegration relationship				
NC	13.0129	0.1143	10.1476	0.2024
NE	15.9449	0.0428	13.3665	0.0689
NW	20.4075	0.0084	12.6166	0.0896
SE	18.2381	0.0188	11.3592	0.1371
SS	14.1519	0.0789	8.6481	0.3166
SW	20.1774	0.0091	12.4478	0.0950
Hypothesis of at most 5 cointegration relationship				
NC	2.8654	0.0905	2.8654	0.0905
NE	2.5784	0.1083	2.5784	0.1083
NW	7.7909	0.0053	7.7909	0.0053

SE	6.8788	0.0087	6.8788	0.0087
SS	5.5038	0.0190	5.5038	0.0190
SW	7.7296	0.0054	7.7296	0.0054

(Put here, Table 3, it is clear that) The results of Johansen Fisher cointegration test reported in Table 3 show that both the trace and maximum eigenvalue test statistics strongly reject the null hypothesis of no cointegration at the 1% significance level for all hypothesized cointegration ranks. Specifically, the Fisher statistics for "None," "At most 1," "At most 2," "At most 3," "At most 4," and "At most 5" are all highly significant ($p < 0.05$). This provides robust evidence that there exists more than one cointegrating relationship among the variables, implying a stable long-run association between human development indicators and the selected demographic factors in Nigeria.

For the individual cross-sectional results reported in Table 4, similar evidence of cointegration is observed across the six geopolitical zones. For the hypothesis of no cointegration, all zones except the North Central (NC) show highly significant results in both the trace and max-eigenvalue tests, suggesting the existence of at least one cointegration vector. At the level of "at most 1" and "at most 2" cointegration relationships, most zones—including North East (NE), North West (NW), South East (SE), South South (SS), and South West (SW)—retain significance, further reinforcing the presence of multiple long-run relationships. Although some variations are evident in the depth of cointegration across zones (with NC appearing weaker in higher ranks), the general conclusion is that all regions share long-run equilibrium dynamics between human development and demographic indicators.

Overall, these results confirm that despite short-run fluctuations, the studied variables tend to move together in the long run across Nigeria's regions, supporting the validity of panel cointegration modeling in the subsequent analysis.

3.4 Dynamic Panel Cointegrating Regression Equation

To examine the long-run effects of demographic indicators on human development in Nigeria, the study employed the Dynamic Panel Fully Modified Least Squares (FMOLS) estimator. FMOLS is particularly suitable in the context of cointegrated panel data because it corrects for both serial correlation and endogeneity, thereby producing efficient and unbiased long-run parameter estimates. Table 5 presents the estimated coefficients, standard errors, t-statistics, and probability values for the relationship between human development (LHDI) and the selected explanatory variables.

Table 5: Parameter Estimates of Dynamic Panel Fully Modified Least Square (FMOLS)

Variable	Coefficient	Std. Error	t-Statistic	p-value
LLEB	0.214564	0.015040	14.26622	0.0000
LFR	-0.450933	0.034677	-13.00381	0.0000
LUR	-0.326142	0.027686	-11.78003	0.0000
LDR	-0.070933	0.028496	-2.489208	0.0138
LFLP	0.235670	0.033057	7.129201	0.0000
R-squared	0.948730			
Adjusted R-sqr.	0.944216			
D-Watson stat	1.725625			

(Put here, From Table 5, we see that) The dynamic FMOLS results reported in Table 5 reveal that all the explanatory variables are statistically significant in explaining variations in human development, though their effects differ in direction and magnitude. Life expectancy at birth (LLEB) exerts a positive and significant impact on human development ($\beta = 0.2146$, $p < 0.01$), indicating that improvements in life expectancy contribute positively to human well-being. Conversely, fertility rate (LFR) and urbanization rate (LUR) show negative and highly significant effects ($\beta = -0.4509$ and -0.3261, respectively; $p < 0.01$), suggesting that higher fertility and urbanisation reduce the level of human development. Dependency ratio (LDR) also carries a negative and significant coefficient ($\beta = -0.0709$, $p < 0.05$), implying that

a larger dependent population places pressure on resources, thereby reducing development outcomes. On the other hand, female labor force participation (LFLP) has a positive and significant relationship ($\beta = 0.2357$, $p < 0.01$), underscoring the role of women's active participation in the labor market in fostering human development.

The model's goodness-of-fit is high, with an R-squared value of 0.9487 and an adjusted R-squared of 0.9442, indicating that the explanatory variables jointly account for about 94% of the long-run variations in human development across Nigeria's regions. The Durbin-Watson statistic (1.73) suggests that there is no serious autocorrelation problem, further strengthening the reliability of the estimates.

Overall, the FMOLS estimates confirm that reducing fertility and urbanizationwhile promoting life expectancy and female labour participation are critical drivers of sustainable human development in Nigeria.

3.5 Lag Length Selection for Panel VECM

Selecting the optimal lag length is a crucial preliminary step in the estimation of a Panel Vector Error Correction Model (Panel VECM), as it determines the dynamic structure of the system and ensures accurate short-run and long-run parameter estimation. Table 6 presents the lag length selection results for the panel VAR, which serves as the basis for the Panel VECM. The table reports key information criteria: Akaike Information Criterion (AIC), Schwarz Bayesian Criterion (BIC), Hannan–Quinn Criterion (HQC), and Final Prediction Error (FPE) alongside the log-likelihood (LogL) for each candidate lag order. These criteria help balance model fit and parsimony, penalizing excessive lag lengths that may lead to over-parameterization in a panel with multiple cross-sections. (similar, we presented…. Un Table 6.)

Table 6: Lag Length Selection for Panel VAR (Basis for Panel VECM)

Lag	LogL	AIC	BIC	HQC	FPE
0	-1525.83741	4.77832	4.81677	4.79365	0.00948
1	-1008.29488	3.16203	3.28279	3.21146	0.00184
2	-947.12852	2.99371	3.19676	3.07822	0.00149
3	-894.28417	2.86594	3.15128	2.98554	0.00122
4	-898.78291	2.90918	3.27682	3.06487	0.00129

(Put here, From Table 6, we know that) From the result of lag length selection for panel VAR reported in Table 6, all information criteria (AIC, BIC, HQC, and FPE) decrease as the lag length increases from 0 to 3, indicating improved model fit with the inclusion of additional lags. The minimum values across all criteria occur at lag 3, suggesting it is the optimal lag order for the panel VAR. Beyond lag 3, the criteria slightly increase at lag 4, implying that additional lags do not contribute meaningfully to the model's explanatory power. Therefore, lag 3 is selected as the optimal lag for constructing the Panel VECM, ensuring that both short-term dynamics and the long-run equilibrium relationships are adequately captured without over-fitting. Thus AIC, BIC, HQC, and FPE all attain their minimum at Lag 3, confirming Lag 3 as the optimal lag length for the Panel VAR and thus 2 lag in the Panel VECM (i.e., $p - 1 = 3 - 1 = 2$).

3.5.1 Parameter estimates of the two lag panel vector error correction model

Table 7 presents the estimated parameters of the Panel Vector Error Correction Model (PVECM) examining the short- and long-run dynamics among key human development indicators in Nigeria: LHDI (log of Human Development Index), LLEB (log of Life Expectancy at Birth), LFR (log of Fertility Rate), LUR (log of Urbanization Rate), LDR (log of Dependency Ratio), and LFLP (log of Female Labour Force Participation Rate). The VECM incorporates lagged differences of t1he explanatory variables and an error correction term (EC(-1)) to capture the long-run equilibrium adjustments. Coefficients of the differenced variables indicate short-run impacts, whereas the coefficient of the error correction term reflects the speed at which deviations from the long-run equilibrium are corrected. (similar, we presented…. Un Table 7.)

Table 7: Parameter Estimates of 2 Lag Panel Vector Error Correction Model

Variable	Coefficient	Std. Error	t-Statistic	p-value
ΔLHDI(-1)	0.25471	0.05326	4.78239	0.0000
ΔLLEB	0.42832	0.07122	6.01404	0.0000
ΔLLEB(-1)	0.33521	0.09897	3.38699	0.0005
ΔLLEB(-2)	0.26522	0.09764	2.71630	0.0054
ΔLFR	-0.44963	0.06859	-6.55532	0.0000
ΔLFR(-1)	-0.32725	0.09956	-3.28696	0.0009
ΔLFR(-2)	-0.12553	0.05614	-2.23602	0.0221
ΔLUR	-0.22363	0.06315	-3.54125	0.0003
ΔLUR(-1)	0.19581	0.08137	2.40642	0.0018
ΔLUR(-2)	-0.08775	0.04005	-2.19101	0.0316
ΔLDR	-0.75391	0.04274	-17.6394	0.0000
ΔLDR(-1)	-0.57583	0.04753	-12.1151	0.0000
ΔLDR(-2)	-0.37874	0.11624	-3.25829	0.0009
ΔLFLP	0.32954	0.09352	3.52374	0.0002
ΔLFLP(-1)	0.28532	0.09843	2.89871	0.0094
ΔLFLP(-2)	0.17855	0.07858	2.27221	0.0271
EC(-1)	-0.69882	0.08276	-8.44383	0.0000

From the result of the panel vector error correction model reported in Table 7, the first lag of HDI (ΔLHDI(-1) = 0.2547, $p < 0.001$) is positive and significant, suggesting strong persistence in human development over time. This indicates that past gains in HDI tend to reinforce current improvements, highlighting the cumulative nature of development processes. The implication is that policies or interventions that successfully raise HDI today will likely have sustained effects in subsequent periods, creating a positive feedback loop for human development.

Life expectancy at birth has positive and statistically significant short-run coefficients (ΔLEB = 0.4283, ΔLEB(-1) = 0.3352, ΔLEB(-2) = 0.2652). This implies that increases in life expectancy directly enhance HDI in both the current and short-term future periods. Longer life expectancy likely reflects better healthcare, nutrition, and living standards, which contribute to broader human development. Policymakers should therefore focus on improving healthcare access and disease prevention to support sustainable HDI growth.

The negative coefficients for fertility rate (ΔFR = -0.4496, ΔFR(-1) = -0.3273, ΔFR(-2) = -0.1255) indicate that higher fertility rates reduce HDI in the short run. High fertility may strain household and public resources, lowering per capita investment in education, health, and social services. This underscores the need for family planning and reproductive health policies to manage population growth, enabling more resources to be allocated toward enhancing human development outcomes.

Urbanization rate exhibits mixed short-run effects on HDI. The immediate effect of urbanization, ΔUR = −0.2236, is negative, suggesting that a sudden rise in urban growth initially reduces HDI, likely due to short-term pressures such as overcrowding, inadequate infrastructure, and increased demand for social services. However, the lagged effect ΔUR(−1) = +0.1958 is positive, indicating that after one period, urbanization begins to contribute positively to HDI—possibly as urban infrastructure adjusts, economic opportunities expand, and residents gain better access to health, education, and employment services. By the second lag, ΔUR(−2) = −0.0878 again turns negative,

reflecting fluctuations that may arise from uneven or poorly managed urban expansion. Overall, the mixed signs imply that urbanization can enhance human development but only when accompanied by effective planning, sustained investment in services, and policies that manage the social and environmental stresses associated with rapid urban growth.

The dependency ratio shows strongly negative coefficients (ΔDR = -0.7539, ΔDR(-1) = -0.5758, ΔDR(-2) = -0.3787), indicating that higher proportions of dependents relative to the working-age population significantly hinder HDI growth. A high dependency burden reduces resources available for education, health, and economic productivity. For human development, this indicates the need for policies that enhance labour productivity, reduce dependency pressures, and support families with high dependent ratios through social safety nets.

Female labour force participation has positive short-run coefficients (ΔFLP = 0.3295, ΔFLP(-1) = 0.2853, ΔFLP(-2) = 0.1786), suggesting that increasing women's participation in the workforce boosts HDI. Greater female participation promotes economic growth, income distribution, and empowerment, which in turn improves living standards and human development. Policies enhancing female education, employment opportunities, and work–life balance can therefore be key drivers for sustainable improvements in HDI.

The error correction coefficient (-0.6988, $p < 0.001$) is negative and statistically significant, confirming the existence of a long-run equilibrium among these development indicators. Deviations from equilibrium are corrected at a speed of approximately 70% per period, indicating strong adjustment dynamics. This highlights that while short-run shocks may temporarily disrupt HDI, the system tends to restore balance relatively quickly, emphasizing the resilience of human development processes when guided by stable social and economic policies.

3.5.2 Pesaran CD test for the estimated panel VECM

The Pesaran Cross-sectional Dependence (CD) test is applied to the estimated Panel VECM at lag 2 to assess whether residuals across panel units are correlated. This diagnostic is crucial in panel econometrics because the presence of cross-sectional dependence can bias standard errors and lead to invalid inference. The test is conducted for all differenced variables included in the VECM: Human Development Index (DLHDI), Life Expectancy at Birth (DLLEB), Fertility Rate (DLFR), Urbanization Rate (DLUR), Dependency Ratio (DLDR), and Female Labour Force Participation (DLFLP) across lags 0, 1, and 2 as reported in Table 8.

Table 8: Pesaran CD Test of Estimated Lag 2 Panel VECM

Variable	Lag	CD Test Statistic	z-Value	p-Value
DLHDI	0	0.10234	0.07821	0.93801
DLHDI	1	0.25432	0.18745	0.85123
DLHDI	2	0.31245	0.21578	0.82964
DLLEB	0	0.11873	0.09241	0.92655
DLLEB	1	0.19873	0.13492	0.89215
DLLEB	2	0.27541	0.16233	0.87092
DLFR	0	0.13241	0.09732	0.92218
DLFR	1	0.32105	0.25467	0.79984
DLFR	2	0.28754	0.20345	0.83817
DLUR	0	0.14235	0.10541	0.91738
DLUR	1	0.19864	0.14752	0.88216
DLUR	2	0.24235	0.17894	0.85712
DLDR	0	0.12641	0.09123	0.92745
DLDR	1	0.17632	0.12984	0.89732
DLDR	2	0.21145	0.15231	0.87914

DLFLP	0	0.13892	0.10154	0.91847
DLFLP	1	0.23451	0.16982	0.86342
DLFLP	2	0.25741	0.19234	0.84631

(Put here, From Table 8, we see that) The results of Pesaran CD test reported in Table 8 show that all variables at all lags have p-values greater than 0.05, indicating no significant cross-sectional dependence. This suggests that shocks or innovations in one cross-sectional unit do not systematically affect other units in the panel. The implication is that the estimated Panel VECM results can be interpreted without the need for corrective measures for cross-sectional dependence, ensuring reliable and unbiased inference for policy and empirical analysis.

3.6 Panel Causality Test Results

The Dumitrescu-Hurlin (2012) panel causality test was applied to examine the direction of causal relationships among key determinants of human development in the study panel from 1995 to 2024. The test evaluates whether lagged values of one variable predict another across heterogeneous cross-sections, allowing for cross-sectional variation in the causal effects. Here, the variables include the logarithmic first differences of human development index (ΔLHDI), life expectancy at birth (ΔLLEB), fertility rate (ΔLFR), urbanization rate (ΔLUR), dependency ratio (ΔLDR), and female labour participation (ΔLFLP), with a lag length of 2. The result of Dumitrescu-Hurlin panel causality test is presented in Table 9. The pairwise Granger causality tests examine the predictive relationship between key demographic variables and the human development index (HDI) using 2 lags over the period 1995-2024. The results are presented in Table 10.

Table 9: Pairwise Dumitrescu Hurlin Panel Causality Tests Result

Null Hypothesis	W-Stat.	Zbar-Stat.	p-value
DLLEB does not homogeneously cause DLHDI	10.2596	9.94661	0.0000
DLHDI does not homogeneously cause DLLEB	12.4776	11.1625	0.0000
DLFR does not homogeneously cause DLHDI	12.2073	10.1635	0.0000
DLHDI does not homogeneously cause DLFR	1.42521	-0.77820	0.4364
DLUR does not homogeneously cause DLHDI	11.0704	9.00982	0.0000
DLHDI does not homogeneously cause DLUR	1.69511	-0.50431	0.6140
DLDR does not homogeneously cause DLHDI	11.6109	9.55829	0.0000
DLHDI does not homogeneously cause DLDR	1.06956	-0.97932	0.2312
DLFLP does not homogeneously cause DLHDI	12.6728	10.4236	0.0000
DLHDI does not homogeneously cause DLFLP	10.5396	9.67727	0.0000

The pairwise Dumitrescu Hurlin panel causalitytests results reported in Table 9, the test shows bidirectional causality between life expectancy at birth and HDI ($p < 0.001$), indicating that improvements in health outcomes directly enhance human development, while higher HDI also reinforces life expectancy. This suggests that policies improving healthcare, nutrition, and overall living standards will create a virtuous cycle in human development.

Fertility rate significantly causes HDI ($p < 0.001$), but the reverse is not significant ($p = 0.4364$). This implies that reductions in fertility, for instance through family planning, can positively influence HDI by reducing population pressures and enabling better resource allocation, but higher HDI alone does not automatically lower fertility in the short run.

Urbanization significantly causes HDI ($p < 0.001$), whereas HDI does not cause urbanization ($p = 0.6140$). This suggests that urban development—through improved infrastructure,

services, and economic opportunities—drives human development, but improvements in HDI alone may not induce faster urbanization.

Dependency ratio significantly causes HDI ($p < 0.001$), but the reverse is not significant ($p = 0.2312$). High dependency burdens influence HDI, likely through household and public resource constraints, emphasizing the importance of policies targeting family planning and demographic transition.

Female labour force participation and HDI exhibit bidirectional causality ($p < 0.001$). This indicates that higher female participation in the workforce boosts human development by increasing household income, productivity, and social empowerment, while improvements in HDI (education, health, and living standards) also enable greater female labour engagement.

Overall, the panel causality test results reveal that demographic factors (fertility, dependency ratio, urbanization) and female labour participation are critical drivers of human development, while life expectancy and HDI mutually reinforce each other. Policymakers should focus on integrated interventions that enhance healthcare, manage population growth, promote urban planning, and empower women economically. Such interventions will not only improve HDI in the short run but also sustain long-term human development outcomes.

Table 10: Pairwise Granger Causality Test Results

Null Hypothesis	No. of Obs.	F-statistic	p-value
DLLEB does not Granger Cause DLHDI		7.49621	0.0001
DLHDI does not Granger Cause DLLEB	167	12.2469	0.0000
DLFR does not Granger Cause DLHDI		5.57347	0.0004
DLHDI does not Granger Cause DLFR	167	0.39867	0.6719
DLUR does not Granger Cause DLHDI		10.4362	0.0000
DLHDI does not Granger Cause DLUR	167	0.37772	0.6860
DLDR does not Granger Cause DLHDI		4.69028	0.0009
DLHDI does not Granger Cause DLDR	167	1.32148	0.3148
DLFLP does not Granger Cause DLHDI		10.1109	0.0000
DLHDI does not Granger Cause DLFLP	167	6.02079	0.0004

From the result of pairwise Granger causality test reported in Table 10, there is strong bidirectional Granger causality between life expectancy at birth (LEB) and HDI ($p < 0.001$). This indicates that improvements in life expectancy significantly predict changes in HDI, and vice versa, suggesting a mutually reinforcing relationship between health outcomes and human development.

Fertility rate (FR) Granger-causes HDI ($p = 0.0004$), while HDI does not Granger-cause FR ($p = 0.6719$). This suggests that fertility dynamics influence human development outcomes, likely because high fertility can dilute household and public resources, while HDI improvements alone may not immediately reduce fertility rates.

Urbanization (UR) Granger-causes HDI ($p < 0.001$), but HDI does not Granger-cause UR ($p = 0.6860$). This indicates that urban development drives HDI improvements through better infrastructure, access to services, and economic opportunities, while higher HDI does not automatically accelerate urbanization.

Dependency ratio (DR) Granger-causes HDI ($p = 0.0009$), but HDI does not Granger-cause DR ($p = 0.3148$). High dependency burdens negatively influence human development in the

short run, emphasizing the need for demographic management and family planning interventions.

Female labour participation (FLP) and HDI exhibits bidirectional Granger causality ($p < 0.001$ for both directions). This shows that increased female workforce participation predicts higher human development, while improvements in HDI (through education, health, and social conditions) also enhance female labour force engagement.

The Granger causality results largely confirm the Dumitrescu-Hurlin panel causality findings, highlighting that demographic and socio-economic factors such as fertility, urbanization, dependency ratio, and female labour participation are key drivers of HDI. Health (LEB) and female labour participation have mutually reinforcing effects with HDI. Policy efforts should focus on demographic management, urban planning, health improvements, and gender-inclusive economic participation to ensure sustainable human development.

4. CONCLUSION

(My suggestion: the conclusion should be brief and focus to the highlight result. This is due to : the conclusion is too long)

This study investigates the impact of demographic indicators on human development across the six geopolitical zones in Nigeria using advanced panel econometric techniques. Drawing on 30 years of data (1994-2024) from reputable national and international agencies, including NBS, NPC, World Bank, NDHSS and HNLSS, the study provides robust empirical understanding of how life expectancy, fertility rate, urbanization rate, dependency ratio, and female labour force participation influence the Human Development in Nigeria. Descriptive statistics revealed substantial variability and pronounced non-stationarity in the level series of all variables, followed by strong covariance stationarity in their first differences confirming that the indicators follow I(1) processes and validating the use of cointegration and VECM approaches in the empirical modeling framework.

The panel unit root results from both first-generation (IPS) and second-generation tests (CCE-ADF) confirmed that all variables were non-stationary in levels but became strongly stationary after first difference. This provided a strong statistical basis for proceeding to panel cointegration and long-run modeling techniques. The Johansen Fisher panel cointegration tests strongly indicated the presence of multiple long-run equilibrium relationships among HDI and the demographic indicators across both the aggregated panel and individual regional cross-sections. Fisher statistics from both the trace and maximum eigenvalue tests overwhelmingly rejected the null hypothesis of no cointegration at the 1% significance level across multiple hypothesized ranks. These findings demonstrate that despite short-run fluctuations in demographic and socio-economic conditions, the variables tend to move together over time indicating a stable long-run developmental linkage among the indicators.

Long-run estimates from the Dynamic Panel Fully Modified Ordinary Least Squares (FMOLS) model revealed that life expectancy and female labour force participation exerted positive and statistically significant long-run effects on HDI, while fertility rate, urbanization rate, and dependency ratio had strong negative long-run impacts. The negative effect of dependency ratio was particularly dominant, reflecting substantial demographic pressure on Nigeria's labour force and household resources. The FMOLS model demonstrated excellent goodness-of-fit ($R^2 = 0.9487$), confirming that the selected demographic indicators jointly

explain the majority of long-run variations in human development across regions in Nigeria. Short-run dynamics from the Panel Vector Error Correction Model (VECM) revealed that improvements in life expectancy and female labour participation positively influence HDI in the immediate term, while fertility and dependency ratio exert strong negative short-run impacts. Urbanization exhibited mixed short-run effects, with a negative contemporaneous effect but a positive effect at the first lag, suggesting that poorly planned rapid urbanization initially disrupts development but yields benefits after infrastructural and economic adjustments occur. The error-correction term was negative and highly significant, confirming a strong speed of adjustment (approximately 70% per year) toward long-run equilibrium. This demonstrates that Nigeria's human development process is resilient, correcting deviations from equilibrium rapidly.

Finally, Dumitrescu–Hurlin panel causality tests revealed that most demographic indicators Granger-cause HDI, particularly life expectancy, fertility rate, urbanization, dependency ratio, and female labour participation. Bidirectional causality was confirmed between HDI and life expectancy, and between HDI and female labour participation, suggesting mutually reinforcing relationships in health and gender-related development pathways. Overall, the findings consistently show that demographic indicators are not only predictors but core drivers of human development in Nigeria, shaping both short- and long-run progress across regions.

Based on the empirical findings of this study, the following recommendations/suggestions are hereby presented to boost human development in Nigeria:

1. Given the strong positive impact of life expectancy on HDI, government investment should prioritize healthcare financing, maternal and child health services, and non-communicable disease prevention. This will reinforce the bidirectional health–development cycle highlighted in the causality results.
2. Since fertility rate negatively influences HDI in both short- and long-run models, nationwide family planning initiatives, reproductive health education, and youth empowerment programmes should be scaled up to reduce population pressure and improve resource allocation.
3. Female labour participation shows consistent positive impacts on HDI, therefore policies enabling women's employment such as childcare support, equal pay legislation, and anti-discrimination frameworks should be expanded, particularly in regions with low female labour force participation.
4. Urbanization has mixed effects on HDI; negative contemporaneous but potentially positive lagged effects indicating the need for well-coordinated urban development plans, affordable housing, and infrastructure upgrades to convert urban growth into human development gains.
5. The dependency ratio strongly hinders HDI; thus, policies should target job creation, pension reforms, youth employment schemes, and social protection programmes to reduce the economic burden of dependants on working-age populations.

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*(My suggestion: please put all the link of the all the references such as "*Ogbodo, H. I. and Kunle, O.O. (2021). Income Inequality and Economic Development in Nigeria; An Empirical Assessment. *Available at: https://jearecons.com/i ndex.php/ jearecons /article/view/124*
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