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Economic Development and Chronic Kidney Disease Prevalence: Evidence of Nonlinearity from a Global Panel, 1995–2023

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Abstract

Chronic kidney disease (CKD) affects millions worldwide, with substantial cross-country disparities in prevalence and sociodemographic development. This study examines whether the association between CKD prevalence and economic development is nonlinear and consistent with an inverted U-shaped pattern. An unbalanced country-level panel for 1995–2023 combines age-standardized CKD prevalence estimates from the Global Burden of Disease Study 2023 with economic and health indicators from the World Development Indicators. Economic development is measured by GDP per capita at purchasing power parity in constant 2021 international dollars. The analysis uses two-way fixed effects, semiparametric estimation, and supplementary two-step System GMM. Fixed-effects estimates generally support an inverted U-shaped relationship after controlling for country and year fixed effects and environmental, metabolic, and health-system covariates. High systolic blood pressure exposure is positively and significantly associated with CKD prevalence. In the preferred broad-control model, the implied turning point is approximately 34,685 international dollars, although sensitive to specification and sample composition. Semiparametric estimates show a similar pattern with a flat peak and gradual decline at higher incomes. System GMM indicates substantial persistence and specification-dependent evidence of nonlinearity. Overall, economic development alone may be insufficient to reduce CKD prevalence, highlighting the importance of health investment and risk-factor management.

JEL classification numbers: I15, C23, O11.
Keywords: Chronic Kidney Disease, Economic Development, Nonlinearity, Two-Way Fixed Effects, System GMM.

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1. Introduction

Chronic kidney disease (CKD)[3], a long-term condition characterized by impaired kidney function or structural kidney damage, has become a major global public health concern and economic challenge. According to The Global Burden of Diseases, Injuries, and Risk Factors Study 2023, approximately 788 million adults aged 20 years and older were living with CKD in 2023, more than double the estimated 378 million cases in 1990. The global age-standardized prevalence of CKD among adults was approximately 14.2%, representing a relative increase of 3.5% since 1990 (GBD 2023 Chronic Kidney Disease Collaborators, 2025). The increasing prevalence of CKD underscores its importance as a population-health concern beyond its clinical implications. At the population level, CKD prevalence may also be associated with broader health-system conditions that vary across countries and income levels (Bello et al., 2024). Economic development may therefore be relevant because countries at different development levels have different resources and health-system capacities for disease prevention and healthcare provision. This raises the question of whether CKD prevalence varies systematically across different stages of economic development.

1.1 Global prevalence of CKD

The distribution of CKD prevalence across levels of sociodemographic development suggests that the disease prevalence may vary across stages of development. The age-standardized prevalence of CKD in 2023 rose from 15.2% in low-SDI countries to 16.1% in low-middle-SDI countries, reaching a peak of 16.3% in middle-SDI countries. It then declined to 15.1% in high-middle-SDI countries and further to 12.2% in high-SDI countries (GBD 2023 Chronic Kidney Disease Collaborators, 2025). This pattern suggests that CKD prevalence does not decline steadily as sociodemographic development advances. Rather, it increases from low- to middle-SDI levels before decreasing at higher levels of development. The World Health Organization also reports that most people living with CKD reside in low- and middle-income countries, indicating that a substantial share of CKD cases is concentrated in less-developed economies (World Health Organization, 2026). Overall, this evidence highlights the uneven distribution of CKD prevalence across different stages of sociodemographic development.

The rise in CKD prevalence from low- to middle-SDI countries, followed by its decline in high-middle- and high-SDI countries, is broadly suggestive of an inverted U-shaped pattern. Such a pattern would imply that CKD prevalence increases during the early and intermediate stages of development before declining at higher levels of development. However, SDI is a composite index incorporating lag-distributed income per capita, education attainment, and fertility, rather than a direct measure of GDP per capita. Hence, these descriptive differences alone are

[3] CKD is identified either by a reduction in estimated glomerular filtration rate (eGFR) or by increased urinary albumin or protein excretion (GBD 2023 Chronic Kidney Disease Collaborators, 2025).

insufficient to establish an inverted U-shaped relationship between GDP per capita and CKD prevalence. Nevertheless, they provide a clear empirical rationale for formally examining this hypothesis using country-level panel data.

1.2 Conceptual framework

The Environmental Kuznets Curve framework provides a useful theoretical basis for examining whether the relationship between economic development and CKD prevalence is nonlinear. Grossman and Krueger (1995) found that many indicators of environmental degradation initially worsened and subsequently improved as income increased. At higher income levels, such improvements may be associated with structural transformation, changes in production techniques, and greater demand for environmental protection.

Subsequent studies have applied similar Kuznets-type reasoning to population-health outcomes. Grecu and Rotthoff (2015) found evidence of an Obesity Kuznets Curve among White women, while Windarti et al. (2019) identified a nonlinear relationship between economic development and obesity using international panel data. Spiteri and von Brockdorff (2019) likewise found an inverted U-shaped association between GDP per capita and cardiovascular disease mortality in European countries. Moreover, Atamari-Anahui et al. (2020) showed that regional GDP per capita was negatively associated with CKD prevalence in Peru, suggesting that socioeconomic conditions may be related to the distribution of CKD burden. Together, these studies suggest that chronic-disease risks may initially rise during economic development but later decline as healthcare capacity, prevention, technology, and regulation improve. This reasoning provides a conceptual basis for examining whether a comparable nonlinear relationship exists between economic development and CKD prevalence.

Applied to CKD, economic development may initially increase disease prevalence through industrialization, urbanization, particulate-matter exposure, behavioral changes, and the rising prevalence of diabetes and hypertension. Improvements in diagnostic capacity may also increase measured prevalence by identifying previously undiagnosed cases. At higher income levels, stronger health systems, earlier detection, more effective management of metabolic risk factors, wider access to kidney-protective treatment, and improved environmental regulation may begin to offset these adverse effects. These competing mechanisms suggest that the relationship between GDP per capita and CKD prevalence may follow an inverted U-shaped pattern.

1.3 Study aim and contributions

Despite the growing research on CKD and its relationship with broader socioeconomic conditions, important gaps remain in the empirical literature on the relationship between economic development and CKD prevalence. First, cross-country econometric research on the development–health relationship has examined a range of health outcomes, including mortality, life expectancy, and obesity. Although international studies have documented variation in CKD prevalence

across countries and levels of socioeconomic development, CKD prevalence appears to have been examined less frequently as the primary outcome in cross-country econometric studies of economic development. Second, the functional form of the GDP–CKD relationship remains relatively underexplored. Available evidence provides limited insight into whether CKD prevalence changes in a consistent direction with economic development or follows a nonlinear pattern across different income levels. Third, the robustness of this relationship appears not to have been extensively examined in a global longitudinal setting. Further evidence is needed to assess whether the estimated association persists after accounting for unobserved country-specific heterogeneity, common time shocks, and relevant environmental, metabolic, and health-system factors. Accordingly, both the shape and robustness of the relationship between economic development and CKD prevalence remain important empirical questions.

This study aims to address these issues by examining whether economic development, measured by GDP per capita, is nonlinearly associated with age-standardized CKD prevalence, including whether the relationship is consistent with an inverted U shape across income levels. Using country-level panel data covering 1995–2023, the analysis applies two-way fixed-effects models to account for time-invariant country characteristics and common year-specific shocks. The shape of the relationship is evaluated using a quadratic income specification, the implied turning point and its location within the observed income range, and semiparametric estimation. A two-step System GMM estimator is additionally employed as a supplementary analysis to account for the persistence in CKD prevalence. By focusing on CKD prevalence, the study extends the application of a Kuznets-type framework to population health and, more specifically, to a major chronic disease that has received comparatively limited attention in cross-country economic research.

Theoretically, economic development may be associated with CKD prevalence through competing pathways. Industrialization, urbanization, metabolic risks, and lifestyle changes may be associated with higher CKD prevalence at lower and middle income levels, whereas stronger healthcare systems, improved prevention, cleaner technologies, and more effective environmental regulation may partly offset these risks at higher income levels.

2. Literature Review

2.1 Economic development and health outcomes

The relationship between economic development and population health may vary across income levels and could be nonlinear. Cross-country research has long documented that population health tends to improve as national income rises, partly because economic development is generally accompanied by improvements in nutrition, sanitation, housing, public infrastructure, and access to medical care. However, the magnitude of these health gains may differ substantially across income levels and over time.

Preston (1975) documented a positive but nonlinear cross-country association between national income per capita and life expectancy, with gains in life expectancy diminishing at higher income levels. The study also showed that the relationship between income and life expectancy shifted upward over time, indicating that improvements in health cannot be attributed to income growth alone. Deaton (2003) similarly reviewed multiple pathways linking income, poverty, inequality, and broader socioeconomic conditions to population health, while emphasizing the difficulty of identifying the underlying causal relationships. Economic development may therefore be associated with improvements in population health while also being accompanied by changes in environmental, behavioral, and social risks. Because the relative importance of these beneficial and adverse pathways may change as countries develop, the relationship between economic development and chronic-disease outcomes may not be linear.

2.2 Development-related pathways to CKD

Several mechanisms may link economic development to chronic kidney disease. During the early and intermediate stages of development, industrialization and urbanization may increase exposure to environmental pollution, alter dietary patterns, reduce physical activity, and increase the prevalence of metabolic risk factors. These changes may contribute to a higher prevalence of kidney disease. At the same time, urban development may expand access to healthcare, sanitation, transportation, and other public services. These competing pathways therefore suggest that the relationship between economic development and CKD may vary across stages of development.

In a cross-sectional study of Chinese adults, Inoue et al. (2017) found that greater urbanization was associated with reduced renal function. Jagannathan and Patzer (2017) likewise highlighted the potential kidney-health consequences of rapid urbanization in low- and middle-income countries, where changes in lifestyle and environmental exposure may occur more rapidly than improvements in healthcare capacity. However, urbanization encompasses multiple dimensions, including health infrastructure, sanitation, communication, and other public services, and some of these components were inversely associated with reduced renal function. The association between urbanization and renal function may therefore reflect competing pathways, with potentially beneficial components partly offsetting adverse components (Inoue et al., 2017).

Air pollution represents a potential pathway linking economic development to CKD. Exposure to particulate matter has been associated with adverse kidney outcomes. Chen et al. (2024) found that short-term exposure to ambient PM2.5 and PM10 was associated with increased CKD hospital admissions in China, while Wathanavasin et al. (2024), in a systematic review and meta-analysis, found that long-term PM2.5 exposure was associated with higher risks of both CKD incidence and prevalence. The relationship between economic development and environmental pollution may itself be nonlinear. Grossman and Krueger (1995) showed that several environmental indicators initially deteriorated as income increased but improved

beyond certain income levels. Combined with the evidence linking particulate-matter exposure to CKD, this pattern suggests that changes in air pollution may constitute one pathway through which the association between economic development and CKD prevalence may be nonlinear.

Economic integration may also influence CKD indirectly through changes in food systems and dietary behavior. Thow and Hawkes (2009) showed that trade liberalization can alter food availability, and consumption patterns, including through greater availability of processed foods. Lin et al. (2018) found that imports of sugar and processed foods were associated with higher average body mass index, particularly in countries with already high levels of overweight and obesity. Cuevas García-Dorado et al. (2019) similarly showed that economic globalization may affect nutrition and health through trade, foreign investment, and changes in food markets. These dietary transformations may increase obesity, diabetes, and hypertension, all of which are major CKD risk factors (Klag et al., 1996; Tuttle et al., 2014; Alicic et al., 2017).

2.3 Metabolic and lifestyle pathways

Diabetes and hypertension are among the most important clinical and metabolic determinants of CKD. Coresh et al. (2007) found that rising CKD prevalence in the United States was partly associated with increases in diabetes and hypertension. Tuttle et al. (2014) and Alicic et al. (2017) identify diabetic kidney disease as a common complication of diabetes and a leading cause of kidney failure. Klag et al. (1996) also found that the risk of end-stage renal disease increased progressively as blood pressure increased among men.

Lifestyle factors may further shape CKD risk. CKD is influenced by a broad range of behavioral and metabolic risk factors. Kelly et al. (2021), for example, showed that dietary patterns, physical activity, and tobacco smoking are associated with the incidence of CKD. Xia et al. (2017) found that cigarette smoking was associated with an increased risk of incident CKD in the general adult population. Evidence regarding alcohol consumption is more complex. Although chronic heavy alcohol consumption may contribute to kidney injury through direct and indirect mechanisms, epidemiological findings remain inconsistent and the causal relationship is not firmly established (Pati and Rathore, 2024).

2.4 Health-system capacity

Economic development may be associated with greater healthcare capacity, preventive services, early detection, and disease management, which may in turn improve CKD care. Allen et al. (2011) found that primary care recognition of CKD and nephrology co-management were associated with better care quality, including improved disease monitoring and medication safety. Ricardo et al. (2016) similarly reported that prior nephrology contact was associated with selected aspects of CKD management, although not with significantly lower risks of CKD progression, cardiovascular disease, or mortality.

Access to kidney care nevertheless remains highly unequal. Osman et al. (2018) documented substantial international disparities and shortages in the nephrology workforce, while Bello et al. (2024) identified persistent cross-country differences in kidney-disease burden and care. Yeung et al. (2024) also reported wide variation in kidney-care financing, infrastructure, and oversight. These disparities may help explain why countries at similar income levels experience different CKD outcomes.

3. Methodology and Data Source

This study constructs an annual country-level panel covering the period 1995–2023 by merging data from the Global Burden of Disease Study 2023 and the World Development Indicators by country and year. The starting year is set in 1995 to ensure the availability of most of the data used in the analysis.

3.1 Data sources, variables and sample construction

Table 1 shows the definitions and descriptive statistics of the variables used in empirical analysis. Sample sizes vary across variables because of differences in data availability. The dependent variable is the age-standardized prevalence rate of chronic kidney disease (CKD) per 100,000 population, obtained from the GBD 2023 CKD estimates (Global Burden of Disease Collaborative Network, 2025a). Age is an important factor associated with CKD prevalence, which increases substantially at older ages (Flaherty et al., 2024). The use of age-standardized prevalence therefore facilitates comparisons across countries and over time by minimizing the influence of differences in population age structures.

GDP per capita and the remaining control variables are obtained from the World Development Indicators (World Bank, 2026). Economic development is measured by GDP per capita based on purchasing power parity in constant 2021 international dollars. The PPP adjustment accounts for differences in price levels across countries, while constant prices remove the effects of inflation.

Both CKD prevalence and GDP per capita are expressed in natural logarithms. A squared term for log GDP per capita is included to allow for a nonlinear association between economic development and CKD prevalence.

The control variables include diabetes mellitus prevalence, physicians per 1,000 people, current health expenditure as a percentage of GDP, mean annual PM2.5 exposure, urban population as a percentage of the total population, trade as a percentage of GDP, total alcohol consumption per capita among individuals aged 15 years and older, and the prevalence of current tobacco use among adults.

The age-standardized diabetes mellitus prevalence rate per 100,000 population is obtained from the GBD 2023 diabetes mellitus estimates (Global Burden of Disease Collaborative Network, 2025b). Diabetes mellitus prevalence is included as a control variable because it is an important risk factor for CKD, allowing the analysis to assess whether the nonlinear relationship between GDP per capita and CKD prevalence persists after accounting for cross-country differences in diabetes mellitus prevalence.

Table 1: Variable definitions and descriptive statistics

Variable	Definitions	N	Mean	SD	Min	Max
CKD Prevalence	Age-standardized chronic kidney disease prevalence rate (per 100,000)	4,814	9,280.53	2,361.07	4,563.00	15,106.74
GDP Per Capita	GDP per capita, PPP (constant 2021 international $)	4,583	22,006.57	23,876.41	525.42	145,590.76
Urbanization	Urbanization rate (%)	4,756	57.87	22.96	7.17	100.00
Trade	Trade openness (% GDP)	4,291	83.40	50.78	0.00	437.33
Health Expenditure	Health expenditure (% GDP)	3,809	6.17	2.62	1.22	23.09
Physician Density	Physician density (per 1,000)	3,051	2.06	1.50	0.01	9.34
Tobacco Use	Prevalence of current tobacco use (% of adults)	1,168	23.72	10.87	3.20	65.50
Alcohol Consumption	Per capita alcohol consumption (liters, age 15+)	3,303	5.65	4.37	0.00	19.40
Diabetes Mellitus	Age-standardized diabetes mellitus prevalence rate (per 100,000)	4,611	5,105.28	2,656.02	1,599.66	23,138.32
SEV	Age-standardized summary exposure value for high systolic blood pressure (0–100)	4,379	33.31	7.67	11.42	51.33
PM2.5	Ambient PM2.5 (μg/m³)	4,756	26.81	15.16	1.39	124.66

Notes: Table 1 reports descriptive statistics for country-year observations. *N* denotes the number of observations, and SD denotes the standard deviation. CKD and diabetes mellitus prevalence are age-standardized rates per 100,000 population. GDP per capita is expressed in constant 2021 international dollars at purchasing power parity (PPP). SEV denotes the age-standardized summary exposure value for high systolic blood pressure and is measured on a 0–100 scale, with higher values indicating greater risk-weighted exposure. Sample sizes vary across variables because of data availability. *Source abbreviations:* GBD denotes the Global Burden of Disease Study 2023, and WDI denotes the World Development Indicators.

The Summary Exposure Value (SEV) for high systolic blood pressure is obtained from the GBD 2023 risk-exposure estimates (Global Burden of Disease Collaborative Network, 2025c). SEV is a risk-weighted measure of population exposure, with higher values indicating greater risk-weighted exposure to high systolic blood pressure. High systolic blood pressure is included as a control variable because it is also an important risk factor for CKD, allowing the analysis to assess whether the nonlinear relationship between GDP per capita and CKD

prevalence persists after accounting for cross-country differences in exposure to high systolic blood pressure.

Physician density and health expenditure capture healthcare resources that may affect CKD detection, prevention and management (Ricardo et al., 2016; Osman et al., 2018; Yeung et al., 2024). PM2.5 exposure is included because long-term particulate-matter exposure has been associated with reduced renal function and adverse CKD outcomes (Chen et al., 2024; Wathanavasin et al., 2024). Urbanization captures structural and demographic transformation and may affect kidney health through changes in healthcare access, pollution exposure, physical activity and metabolic risks (Inoue et al., 2017; Jagannathan and Patzer, 2017).

Trade openness may influence health indirectly through changes in production structures, food markets and dietary environments (Thow and Hawkes, 2009; Cuevas García-Dorado et al., 2019). Tobacco use is associated with an increased risk of CKD, while the relationship between alcohol consumption and kidney health may vary according to the amount and pattern of consumption (Xia et al., 2017; Pati and Rathore, 2024; White et al., 2009).

The initial merged dataset contains 166 countries and territories. Because data availability differs across variables, countries and years, the dataset forms an unbalanced panel. The least restrictive specifications contain usable observations for 159 countries. As additional control variables are introduced, the number of countries declines because CKD prevalence, GDP per capita, and all other controls must be jointly observed in any given specification. The most comprehensive specification therefore contains 128 countries, with the reduction in sample size reflecting differences in data availability.

3.2 Two-way fixed-effects model

The principal empirical specification is a two-way fixed-effects model: Equation (1)

$$lnCKD_{it} = \beta_1 lnGDPpc_{it} + \beta_2 (lnGDPpc_{it})^2 + \gamma' X_{it} + \mu_i + \sum_t \lambda_t D_t + \varepsilon_{it} \quad (1)$$

In Equation (1), i denotes country and t denotes year. ln CKD_{it} is the natural logarithm of the age-standardized CKD prevalence rate for country i in year t, while $GDPpc_{it}$ is the natural logarithm of GDP per capita, measured in constant 2021 international dollars at purchasing power parity, for country i in year t, and $\mathbf{X}_{it}$ is the vector of time-varying control variables. The terms μ_i represents country fixed effects, which control for time-invariant differences across countries. Year fixed effects are represented by $\sum_t \lambda_t D_t$, where D_t denotes the year dummy variables and λ_t represents the corresponding coefficients. Finally, ε_{it} is the idiosyncratic error term.

Country fixed effects control for unobserved national characteristics that remain relatively stable over time, including geography, long-standing institutions, cultural conditions and persistent features of national health systems. These factors may be correlated with both GDP per capita and CKD prevalence. The use of fixed effects

is therefore consistent with Mundlak's (1978) argument that unit-specific heterogeneity may be correlated with the explanatory variables. Hausman's (1978) specification-testing framework provides the conventional basis for evaluating the fixed-effects and random-effects assumptions. Year fixed effects account for time shocks and trends shared across countries. Standard errors are clustered at the country level to account for heteroskedasticity and serial correlation within countries over time.

3.3 Testing the inverted U-shaped relationship

An inverted U-shaped relationship requires the coefficient on log GDP per capita to be positive and the coefficient on its squared term to be negative. Under this coefficient pattern, CKD prevalence initially increases with economic development but begins to decline after GDP per capita reaches a particular threshold. Because GDP per capita is included in logarithmic form, the estimated turning point in constant 2021 international dollars is calculated as:
Equation (2)

$$\ln GDPpc^{*} = \exp\left(-\frac{\beta_1}{2\beta_2}\right) \tag{2}$$

The turning point obtained from Equation (2) represents the level of GDP per capita at which the estimated quadratic relationship reaches its maximum.

3.4 Semiparametric estimation

The quadratic model restricts a specific functional form on the relationship. To assess whether the estimated pattern depends on this restriction, the study also employs the semiparametric fixed-effects estimator implemented by Libois and Verardi (2013):

$$\ln CKD_{it} = m(lnGDPpc_{it}) + \gamma' X_{it} + \mu_i + \sum_t \lambda_t D_t + \varepsilon_{it} \tag{3}$$

In Equation (3), $m(\ln\ GDPpc_{it})$ is an unspecified smooth function estimated from the data. This approach allows the GDP–CKD relationship to take a flexible form rather than requiring it to follow a predetermined quadratic curve. The estimated function is presented graphically to determine whether CKD prevalence rises at lower- and middle-income levels and declines at higher income levels after the functional-form restriction is relaxed.

3.5 Two-step GMM robustness analysis

CKD prevalence is likely to exhibit substantial persistence because CKD develops and progresses gradually. Prevalence in a particular year may therefore be strongly related to prevalence in the preceding year. A supplementary dynamic panel specification includes the lagged dependent variable:

The Two-step GMM specification is given in Equation (4)

$$lnCKD_{it} = \rho_1 lnCKD_{i,t-1} + \rho_2 lnCKD_{i,t-2} + \beta_1 lnGDPpc_{it} + \beta_2 (lnGDPpc_{it})^2 + \gamma' X_{it} + \mu_i + \lambda t + \varepsilon_{it} \quad (4)$$

The coefficients ρ_1 and ρ_2 capture the persistence and dynamic adjustment of CKD prevalence. Estimating this dynamic specification using a conventional fixed-effects estimator may produce dynamic panel bias because the lagged dependent variables are correlated with the within-transformed error term. Therefore, as a supplementary robustness analysis, Equation (4) is estimated using two-step System GMM with Windmeijer-corrected robust standard errors and a small-sample correction, following the System GMM framework developed by Arellano and Bover (1995) and Blundell and Bond (1998), and the implementation described by Roodman (2009).

System GMM jointly estimates equations in first differences and in levels. In the differenced equation, appropriately lagged levels of CKD prevalence are used as internal instruments, while lagged differences provide additional instruments for the level equation. In the reported specifications, the second and third lags of CKD prevalence are used as collapsed GMM-style instruments, whereas GDP per capita, its squared term, and the control variables are treated as IV-style instruments. Year indicators are included to account for common time shocks. To limit instrument proliferation, the lag range is restricted and the instrument matrix is collapsed. Model adequacy is assessed using the Arellano–Bond serial-correlation tests and the Hansen test of overidentifying restrictions.

4. Empirical Results

As a first step, the choice between fixed-effects and random-effects estimation is assessed using the Hausman specification test. Both specifications include year fixed effects to account for common global shocks and secular trends affecting all countries.

As reported in Table 2, the test statistics are 42.35 with six degrees of freedom for the baseline specification and 101.31 with 15 degrees of freedom for the full specification. In both cases, the null hypothesis that the fixed-effects and random-effects estimates do not differ systematically is rejected at the 1% significance level. These results suggest that the random-effects assumption that unobserved country-specific effects are uncorrelated with the explanatory variables are unlikely to hold. Hence, the country fixed-effects estimator is preferred. The main specification therefore takes the form of a two-way fixed-effects model incorporating both country and year fixed effects.

The resulting estimates are based on within-country changes over time while controlling for common year-specific shocks.

Table 2: Hausman specification test results

Specification	Hausman χ^2	df	p-value	Preferred estimator
Baseline specification	42.35	6	<0.001	Fixed effects
Full specification	101.31	15	<0.001	Fixed effects

Notes: The Hausman tests compare the fixed-effects and random-effects estimators. Both specifications include year fixed effects to account for common year-specific shocks. The null hypothesis is that the random-effects estimator is consistent; rejection of the null therefore favors the fixed-effects estimator.

4.1 Two-Way Fixed-Effects Results

Table 3 reports the two-way fixed-effects estimates. All specifications include country and year fixed effects, with the control variables introduced sequentially. Model 1 includes log GDP per capita and its squared term, while Models 2–8 progressively control for diabetes mellitus prevalence, PM2.5 exposure, physician density, current health expenditure, urbanization, high systolic blood pressure exposure, and trade openness. Models 9 and 10 additionally include tobacco use and alcohol consumption, respectively.

The baseline results support a concave relationship between economic development and CKD prevalence. In Model 1, the coefficient on log GDP per capita is positive (0.0996) and statistically significant at the 1% level, whereas the coefficient on its squared term is negative (−0.0055) and statistically significant at the 1% level. This coefficient pattern is consistent with an inverted U-shaped relationship, under which CKD prevalence initially increases with income but begins to decline after GDP per capita reaches a sufficiently high level.

This pattern remains stable across Models 2–6 after the sequential inclusion of the principal health, environmental, and health-system controls. The linear GDP coefficient remains positive and statistically significant in all specifications, while the squared term remains negative and statistically significant. The persistence of this nonlinear pattern after controlling for these covariates suggests that the estimated GDP–CKD relationship is not driven solely by differences in diabetes mellitus prevalence, PM2.5 exposure, physician density, health expenditure, or urbanization.

The results also remain robust when high systolic blood pressure exposure and trade openness are included. In Model 7, the coefficients on log GDP per capita and its squared term are 0.0803 and −0.0038, respectively, with the linear term statistically significant at the 1% level and the squared term at the 5% level. In Model 8, which includes economic, environmental, health-system, and metabolic controls, the corresponding coefficients are 0.0817 and −0.0039. The linear term is significant at the 5% level and the squared term at the 10% level. Model 8 may therefore be treated as the preferred broad-control specification because it provides extensive covariate adjustment while retaining 2,274 observations from 144 countries.

Table 3: Two-way fixed-effects estimates of the GDP–CKD relationship

Variables	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Log GDP per capita	0.099583*** (0.034341)	0.097229*** (0.034634)	0.110086*** (0.036599)	0.133224*** (0.041444)	0.109117*** (0.040210)	0.113273*** (0.041192)	0.080317*** (0.030507)	0.081709** (0.039426)	0.090422** (0.042698)	0.061463 (0.040040)
(Log GDP per capita)²	-0.005509*** (0.001941)	-0.005256*** (0.001941)	-0.005990*** (0.002047)	-0.007178*** (0.002324)	-0.005603** (0.002203)	-0.005832** (0.002258)	-0.003816** (0.001699)	-0.003908* (0.002121)	-0.004374* (0.002299)	-0.002791 (0.002138)
Diabetes mellitus prevalence		0.000003 (0.000002)	0.000003 (0.000002)	0.000004 (0.000002)	0.000002 (0.000002)	0.000002 (0.000002)	0.000001 (0.000002)	0.000001 (0.000002)	0.000001 (0.000002)	0.000001 (0.000002)
PM2.5 exposure			-0.000429** (0.000210)	-0.000565** (0.000236)	-0.000405** (0.000180)	-0.000391** (0.000178)	-0.000252* (0.000133)	-0.000300** (0.000131)	-0.000203 (0.000173)	-0.000124 (0.000152)
Physicians per 1,000 people				-0.001737 (0.001749)	0.000078 (0.001527)	-0.000043 (0.001591)	0.001263 (0.001602)	0.001659 (0.001653)	0.001183 (0.001873)	-0.000126 (0.002879)
Health expenditure (% of GDP)					-0.000711 (0.000567)	-0.000837 (0.000559)	-0.000107 (0.000489)	-0.000510 (0.000524)	0.000062 (0.000617)	-0.000210 (0.000667)
Urban population (%)						-0.000268 (0.000309)	-0.000106 (0.000315)	-0.000151 (0.000333)	0.000057 (0.000409)	-0.000481 (0.000445)
High systolic blood pressure (age-standardized SEV)							0.002563*** (0.000408)	0.002494*** (0.000443)	0.002800*** (0.000555)	0.002563*** (0.000554)
Trade openness (% of GDP)								-0.000068 (0.000058)	-0.000087 (0.000064)	-0.000116 (0.000071)
Current tobacco use (% of adults)									-0.000372 (0.000390)	-0.000378 (0.000407)
Alcohol consumption per capita (liters, age 15+)										0.000945 (0.000809)
Constant	8.650858*** (0.150814)	8.642636*** (0.155294)	8.596820*** (0.162846)	8.445856*** (0.183837)	8.540725*** (0.182982)	8.537845*** (0.183171)	8.556128*** (0.135728)	8.556234*** (0.185703)	8.501428*** (0.199975)	8.662658*** (0.199802)
Year fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Country fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	4,583	4,525	4,496	2,960	2,519	2,519	2,406	2,274	693	545
Within R-squared	0.193	0.212	0.221	0.196	0.163	0.166	0.346	0.322	0.314	0.338
Countries	159	157	156	156	156	156	148	144	132	128

Notes: The dependent variable is the natural logarithm of the age-standardized CKD prevalence rate per 100,000 population. GDP per capita is expressed in constant 2021 international dollars at purchasing power parity (PPP). Controls are added sequentially across specifications. All models include country and year fixed effects. Country-clustered robust standard errors are reported in parentheses. Sample sizes vary because of data availability. SEV denotes the age-standardized summary exposure value for high systolic blood pressure. R^2 is the within R-squared. ***, **, and * denote significance at the 1%, 5%, and 10% levels, respectively.

The estimated relationship becomes less precisely estimated after the inclusion of the behavioral variables. In Model 9, the GDP coefficients retain the expected signs, with the linear term (0.0904) statistically significant at the 5% level and the squared term (−0.0044) significant at the 10% level. In Model 10, the corresponding coefficients remain positive and negative, respectively, but neither is statistically significant. These results coincide with a substantial reduction in sample size following the inclusion of tobacco use and alcohol consumption, with the number of observations declining from 2,274 in Model 8 to 693 and 545 in Models 9 and 10, respectively. The loss of statistical significance in the most restrictive specification may therefore reflect reduced statistical power and changes in sample composition, rather than necessarily indicating the absence of a nonlinear relationship.

Among the control variables, PM2.5 exposure has a negative and statistically significant coefficient in Models 3–8 but becomes statistically insignificant in Models 9 and 10. Diabetes mellitus prevalence is positively associated with CKD prevalence across Models 2–10, although its coefficient is not statistically significant in any specification. High systolic blood pressure exposure is positive and statistically significant at the 1% level in Models 7–10. Its estimated coefficients range from approximately 0.0025 to 0.0028, implying that a one-point increase in the age-standardized Summary Exposure Value is associated with an approximately 0.25–0.28% increase in CKD prevalence, holding the other variables constant.

4.2 Estimated GDP per capita turning points

Using the coefficients reported in Table 3, the estimated turning points vary across specifications. Models 1–6 imply thresholds ranging from approximately 8,419 to 16,939 international dollars, suggesting that CKD prevalence increases at lower and intermediate levels of economic development but may begin to decline once income reaches a sufficiently high level. After high systolic blood pressure exposure is introduced, the estimated threshold rises substantially: the turning points are approximately 37,187 international dollars in Model 7, 34,685 in Model 8, and 30,832 in Model 9. These estimated turning points fall within the range of GDP per capita observed among countries in the estimation sample.

Based on the preferred broad-control specification in Model 8, the estimated GDP–CKD relationship reaches its maximum at approximately 34,685 international dollars. Below this threshold, GDP per capita is positively associated with CKD prevalence, whereas above it, the estimated association becomes negative. Because the squared GDP term is significant only at the 10% level, however, this threshold should be regarded as suggestive rather than as a precise or universally applicable income level. The turning point in Model 10 is not discussed further because neither the linear nor the quadratic GDP term is statistically significant. Overall, the variation across specifications suggests that the estimated threshold is sensitive to the included controls and the composition of the estimation sample.

4.3 Semiparametric evidence

Figure 1 presents the baseline semiparametric fixed-effects estimate of the relationship between log GDP per capita and log age-standardized CKD prevalence. Unlike quadratic specifications, the semiparametric approach allows the shape of the relationship to be determined flexibly by the data rather than imposing a predetermined functional form. The estimated curve exhibits a shallow inverted U-shaped pattern: CKD prevalence rises at lower income levels, remains relatively stable with a modest upward tendency across much of the middle-income range, and declines at higher income levels. Although the confidence bands widen near both tails, where observations are relatively sparse, the overall pattern provides additional support for a nonlinear GDP-CKD relationship.

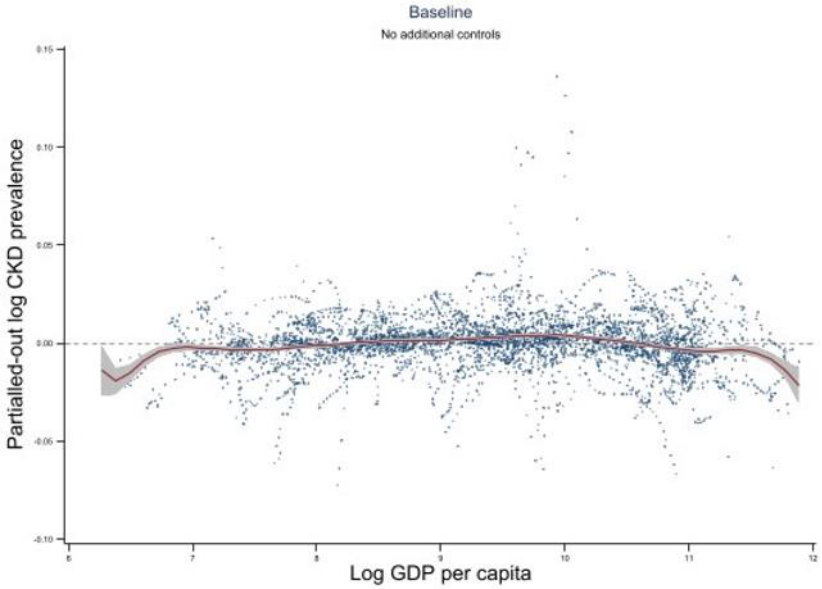

Figure 1: Baseline semiparametric relationship between log GDP per capita and log CKD prevalence.

Note: The dots represent partialled-out observations. The solid line shows the fourth-degree local-polynomial estimate, and the shaded area denotes the 95% confidence interval. The baseline specification includes country and year fixed effects and no additional covariates.

The corresponding semiparametric estimates obtained after the sequential inclusion of control variables are reported in Figure A1 in the Appendix, while the coefficients on the parametrically specified controls are presented in Table A1 in the Appendix. Across the controlled specifications, the estimated curves display a consistent nonlinear pattern, rising or remaining relatively flat over the lower-and middle-income ranges before declining in the upper part of the income distribution. Although the magnitude and location of the decline vary across specifications, the inverted U-shaped pattern remains evident. In the later specifications, including

Models 6 and 7, the decline at higher income levels persists, although it becomes more gradual. Overall, the semiparametric estimates reinforce the nonlinear relationship identified by the quadratic fixed-effects models.

The differences across specifications may reflect both changes in sample composition and the conditioning effects of the additional covariates. As controls are introduced, the number and composition of available country-year observations change, so part of the variation in the estimated curves may be attributable to differences in the estimation sample. In addition, healthcare capacity, pollution exposure, urbanization, and high systolic blood pressure exposure may account for variation associated with potential pathways linking economic development to CKD prevalence. Recorded CKD prevalence may also remain comparatively high in wealthier countries because improved diagnostic capacity increases case detection, while more effective treatment prolongs survival among individuals with CKD. These mechanisms may attenuate the observed decline in prevalence even when CKD prevention and disease management improve. The semiparametric estimates indicate that the decline in CKD prevalence emerges primarily in the upper part of the income distribution. This pattern provides a useful qualitative benchmark for assessing the turning points implied by the quadratic specifications and indicates that the peak occurs relatively late in the income distribution. Given the relatively flat peak and wider confidence bands toward the upper tail, the semiparametric estimates are used to assess the overall shape and approximate location of the turning region rather than to identify a precise income threshold.

4.4 Two-step system GMM supplementary robustness analysis

Table 4 reports two-step System GMM estimates for Models 1-8 as a supplementary robustness analysis. For brevity, the coefficients on the control variables are not reported in the table, while the complete coefficient estimates are provided in Table A2 in the Appendix. Each specification uses its maximum available estimation sample, with the number of observations declining from 4,273 in the baseline model to 2,274 in the fully controlled specification, and the number of countries ranging from 159 to 144. The first two lags of log CKD prevalence are included as regressors to account for persistence in CKD prevalence. The second and third lags of the dependent variable are used as collapsed GMM-style instruments, whereas GDP per capita, its squared term, and the control variables are treated as IV-style instruments. Year fixed effects are included in all specifications to account for common time shocks.

The lagged dependent-variable coefficients indicate substantial dynamic dependence in CKD prevalence. The coefficient on the first lag is positive in all specifications and statistically significant throughout, whereas the coefficient on the second lag is negative in all models and statistically significant in all but Model 3. The sums of the first- and second-lag coefficients range from 0.653 to 0.934 across specifications. The positive sums indicate substantial dynamic dependence in CKD prevalence across specifications.

Table 4: Two-step system GMM estimates of the relationship between economic development and CKD prevalence

Variables	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Specification	**Baseline**	**+ PM2.5**	**+ Diabetes mellitus**	**+ Physicians**	**+ Health exp.**	**+ Urbanization**	**+ SEV**	**+ Trade**
Panel A. Coefficient estimates								
Lagged ln(CKD prevalence), t−1	3.271201*** (1.035061)	3.173116*** (0.931943)	2.841296** (1.273056)	2.294899*** (0.261105)	2.190121*** (0.150335)	2.190836*** (0.151918)	2.143860*** (0.154333)	2.062772*** (0.141242)
Lagged ln(CKD prevalence), t−2	-2.617975** (1.110675)	-2.491099** (1.020704)	-2.120960 (1.520940)	-1.397864*** (0.248025)	-1.262059*** (0.145309)	-1.262373*** (0.146480)	-1.220451*** (0.146468)	-1.128424*** (0.136121)
Log GDP per capita	0.271581* (0.161815)	0.264350* (0.152323)	0.180781 (0.195407)	0.065179** (0.026257)	0.036960* (0.020752)	0.035904* (0.021047)	0.035130 (0.022986)	0.031420 (0.020760)
Log GDP per capita squared	-0.016295* (0.009510)	-0.015464* (0.008802)	-0.011306 (0.012096)	-0.003936*** (0.001493)	-0.002269* (0.001186)	-0.002235* (0.001191)	-0.002229* (0.001312)	-0.002116* (0.001200)
Panel B. Model diagnostics								
Year fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	4,273	4,246	4,192	2,781	2,519	2,519	2,406	2,274
Countries	159	158	156	156	156	156	148	144
Instruments	32	33	34	35	33	34	35	36
AR(1) p-value	0.040	0.550	0.052	0.347	0.359	0.359	0.378	0.367
AR(2) p-value	0.612	0.530	0.967	0.708	0.914	0.890	0.700	0.898
AR(3) p-value	0.462	0.257	0.376	0.913	0.269	0.279	0.252	0.149
Hansen test p-value	0.829	0.731	0.218	0.883	0.908	0.970	0.813	0.613
Joint GDP terms p-value	0.180	0.188	0.634	0.009	0.056	0.057	0.071	0.024
Sum of lag coefficients (L1 + L2)	0.653	0.682	0.720	0.897	0.928	0.928	0.923	0.934

Notes: The dependent variable is ln(CKD prevalence). Windmeijer-corrected robust standard errors are reported in parentheses. Year fixed effects are included in all specifications to account for common time shocks.

The System GMM estimates retain the positive linear and negative quadratic GDP coefficients across all eight specifications, indicating a coefficient pattern consistent with an inverted U-shaped relationship. Statistical support, however, varies across specifications. In Models 1 and 2, both GDP terms are individually significant at the 10% level, although the joint tests are not statistically significant. Model 3 provides no statistically significant evidence for the GDP terms. In Model 4, both the linear and quadratic GDP terms are statistically significant, and the joint test strongly rejects their joint insignificance ($p = 0.009$). In Models 5–7, the coefficients continue to exhibit the expected positive-linear and negative-quadratic signs, with joint-test p-values of 0.056, 0.057, and 0.071, respectively, providing marginal evidence of nonlinearity. In the fully controlled Model 8, the linear GDP coefficient remains positive and the squared term negative; although the linear term is not individually significant at conventional levels and the quadratic term is significant only at the 10% level, the two GDP terms are jointly significant ($p = 0.024$). Overall, the dynamic-panel estimates therefore provide supplementary evidence that the nonlinear GDP–CKD relationship is not eliminated after accounting for lagged CKD prevalence and common year effects, although the strength of the evidence varies across specifications.

The specification diagnostics are generally supportive of the System GMM estimates. The Arellano–Bond tests show no evidence of second- or third-order serial correlation in the first-differenced residuals across any of the eight specifications, with all AR(2) and AR(3) p-values exceeding 0.10. The Hansen test likewise fails to reject the null hypothesis of instrument validity in every model, with p-values ranging from 0.218 to 0.970. The number of instruments remains between 32 and 36, substantially below the corresponding number of countries. Collectively, these results support the use of the System GMM estimates as a supplementary dynamic robustness analysis.

5. Discussion

The findings suggest that the association between economic development and CKD prevalence becomes more favorable at higher income levels, but the estimated turning point should not be interpreted as a universal income target or policy threshold. The turning point of approximately 34,685 international dollars in the preferred Model 8 specification is therefore best viewed as a conditional empirical reference rather than as a level of income beyond which CKD prevalence will necessarily decline. The semiparametric estimates are broadly consistent with the inverted U-shaped pattern but indicate a relatively flat peak and a more gradual decline at higher income levels, suggesting that the turning region is not sharply defined.

These findings complement the descriptive evidence reported by Morton and Shah (2021), who documented increases in CKD prevalence across high-, middle-, and low-income countries between 1990 and 2017. Although the study did not formally examine the functional form of the GDP–CKD relationship, its descriptive evidence

suggests that economic growth alone does not necessarily lead to declining CKD prevalence. This study extends this evidence by showing that the association between GDP per capita and CKD prevalence is nonlinear, becoming more favorable only at higher income levels.

The results also highlight the importance of how the resources generated by economic development are translated into health outcomes. In the two-way fixed-effects specifications, the SEV for high systolic blood pressure is positively and significantly associated with CKD prevalence in the relevant models, underscoring the importance of hypertension as a major CKD risk factor. By contrast, the negative coefficient on PM2.5 exposure in Models 3–8 does not necessarily imply a protective effect, given the existing evidence linking PM2.5 exposure to adverse CKD outcomes (Chen et al., 2024; Wathanavasin et al., 2024). Table A3 in the Appendix shows that increases in GDP per capita are associated with lower PM2.5 exposure within countries, indicating that environmental conditions may improve as economies develop. Together with existing evidence linking PM2.5 exposure to adverse CKD outcomes (Chen et al., 2024; Wathanavasin et al., 2024), this pattern suggests that environmental improvement may be one possible explanation for the more favorable GDP–CKD association at higher income levels. However, the role of this pathway remains uncertain because PM2.5 exposure is negatively associated with CKD prevalence in the two-way fixed-effects specifications.

The findings further suggest that the health benefits associated with economic development may depend partly on the efficiency with which additional resources are allocated toward risk-factor prevention and management. Economic growth alone may therefore be insufficient to generate improvements in CKD outcomes if increases in available resources are not accompanied by effective health investment and risk-factor control.

6. Conclusion

Overall, the two-way fixed-effects estimates provide consistent evidence of a nonlinear relationship between GDP per capita and CKD prevalence. Across Models 1–9, the linear GDP term is positive and the squared term is negative, indicating an inverted U-shaped relationship under the quadratic specification. The semiparametric estimates reinforce this finding: the estimated curves generally rise over the lower- and middle-income ranges, reach a relatively flat peak, and decline in the upper part of the income distribution. This decline persists across the controlled specifications, although it is more gradual than that implied by the quadratic specification. Taken together, these findings provide consistent evidence of an inverted U-shaped relationship between GDP per capita and CKD prevalence, while showing that the peak is relatively flat and the turning region becomes less sharply defined once the quadratic functional-form restriction is relaxed.

The two-step System GMM estimates provide supplementary, although specification-dependent, support for the nonlinear relationship between economic development and CKD prevalence. Across all dynamic specifications, the linear

GDP coefficient remains positive and the squared term negative, consistent with an inverted U-shaped pattern, although the degree of statistical support varies across specifications. The lag structure also indicates substantial dynamic dependence in CKD prevalence. Overall, the System GMM results suggest that the nonlinear GDP–CKD relationship is not eliminated after accounting for lagged CKD prevalence and common year effects, but these estimates are interpreted as supplementary robustness evidence rather than as the primary basis for inference.

Economic development may expand fiscal, institutional, and technological capacity for prevention and disease management, but the potential health benefits may depend on whether these resources are translated into stronger primary care, earlier detection, effective disease management, and sustained access to kidney care (Bello et al., 2024; Allen et al., 2011; Ricardo et al., 2016). The conclusion is that the GDP–CKD relationship is nonlinear, although the magnitude of the decline at higher income levels and the estimated turning point remain sensitive to functional form, sample composition, and model specification. The supplementary dynamic estimates further indicate substantial persistence in CKD prevalence, while the positive linear and negative quadratic GDP coefficients remain broadly consistent with an inverted U-shaped relationship, although their statistical support varies across specifications.

Several limitations should be acknowledged. First, although the fixed-effects specifications control for persistent country-specific heterogeneity and common time shocks, causal interpretation remains limited because reverse causality and unobserved time-varying confounders cannot be fully ruled out. Second, the estimated turning points vary across specifications and with changes in sample composition, limiting their interpretation as precise or universal income thresholds. Future research could examine whether economic development is differently associated with CKD incidence, mortality, disease progression, and kidney failure. Re-estimating alternative specifications on a common estimation sample would also help distinguish the effects of additional controls from those arising from changes in sample composition. Further work could explore heterogeneity across income groups, regions, healthcare-system characteristics, and stages of demographic transition, while considering alternative treatments of the potential endogeneity of GDP and other covariates.

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Appendix

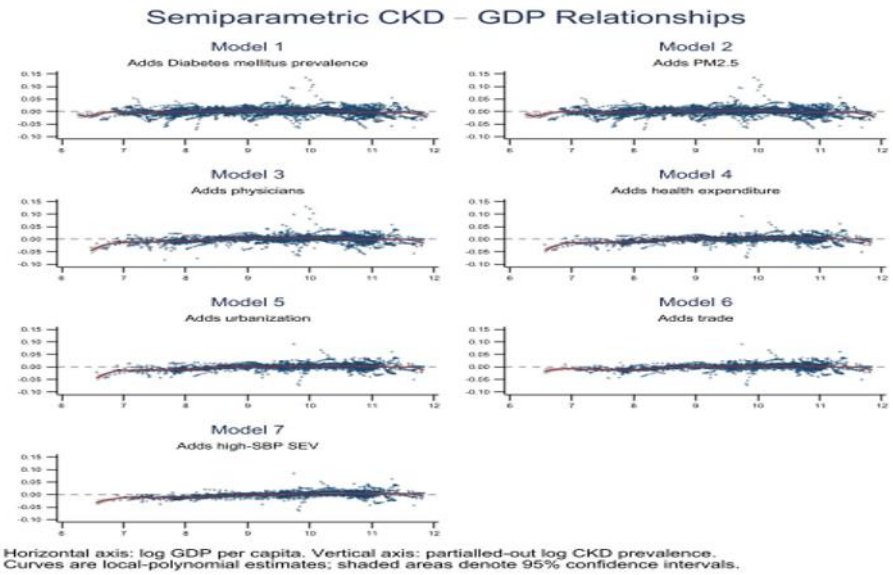

Figure A1: Semiparametric relationships between log GDP per capita and log CKD prevalence across alternative specifications.

Note: The dots represent partialled-out observations. The solid curves show the fourth-degree local-polynomial estimates, and the shaded areas denote the 95% confidence intervals. All specifications include country and year fixed effects. Model 1 additionally controls for diabetes mellitus prevalence; Model 2 adds PM2.5 exposure; Model 3 adds physician density; Model 4 adds health expenditure; Model 5 adds urbanization; Model 6 adds trade openness; and Model 7 adds high-SBP SEV.

Table A1: Parametric component estimates from semiparametric fixed-effects models

	Baseline	**(1)**	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**	**(7)**
Specification	**No controls**	**Diabetes mellitus**	**+ PM2.5**	**+ Physicians**	**+ Health exp.**	**+ Urbanization**	**+ Trade**	**+ High-SBP SEV**
Diabetes mellitus prevalence		0.00000144 (0.00000176)	0.00000147 (0.00000176)	-0.00000031 (0.00000183)	-0.00000070 (0.00000190)	-0.00000064 (0.00000189)	-0.00000061 (0.00000192)	-0.00000171 (0.00000185)
PM2.5 exposure			0.000007 (0.000012)	-0.000007 (0.000012)	-0.000012 (0.000011)	-0.000013 (0.000011)	-0.000014 (0.000011)	-0.000015 (0.000010)
Physicians per 1,000 people				-0.000078 (0.000184)	-0.000047 (0.000198)	-0.000036 (0.000196)	-0.000029 (0.000201)	0.000047 (0.000189)
Health expenditure (% of GDP)					-0.000146 (0.000126)	-0.000135 (0.000126)	-0.000165 (0.000132)	-0.000056 (0.000119)
Urban population (%)						0.000248 (0.000378)	0.000250 (0.000396)	0.000246 (0.000392)
Trade openness (% of GDP)							-0.000013 (0.000008)	-0.000009 (0.000007)
High-SBP SEV								0.002001*** (0.000344)
Country fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Nonparametric ln(GDP per capita)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Local-polynomial degree	4	4	4	4	4	4	4	4
Bandwidth	0.759	0.759	0.759	0.759	0.759	0.759	0.759	0.759
Observations	4424	4368	4340	2407	2071	2071	1976	1885
Within R^2	0.0436	0.0478	0.0469	0.0607	0.0738	0.0753	0.0705	0.1421
Root MSE	0.0036	0.0035	0.0036	0.0027	0.0026	0.0026	0.0027	0.0026

Notes: The dependent variable is the natural logarithm of age-standardized CKD prevalence. Log GDP per capita is estimated nonparametrically and therefore does not have a single reported coefficient. The nonparametric component is estimated using a fourth-degree local polynomial with an Epanechnikov kernel and a bandwidth of 0.759. All specifications include country and year fixed effects. Standard errors clustered at the country level are reported in parentheses. $p < 0.10$, $p < 0.05$, $p < 0.01$.

Table A2: Two-step system GMM estimates of CKD prevalence

Variables	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Specification	**Baseline**	**+ PM2.5**	**+ Diabetes mellitus**	**+ Physicians**	**+ Health exp.**	**+ Urbanization**	**+ SEV**	**+ Trade**
Panel A. Coefficient estimates								
Lagged ln(CKD prevalence), t−1	3.271201*** (1.035061)	3.173116*** (0.931943)	2.841296** (1.273056)	2.294899*** (0.261105)	2.190121*** (0.150335)	2.190836*** (0.151918)	2.143860*** (0.154333)	2.062772*** (0.141242)
Lagged ln(CKD prevalence), t−2	-2.617975** (1.110675)	-2.491099** (1.020704)	-2.120960 (1.520940)	-1.397864*** (0.248025)	-1.262059*** (0.145309)	-1.262373*** (0.146480)	-1.220451*** (0.146468)	-1.128424*** (0.136121)
Log GDP per capita	0.271581* (0.161815)	0.264350* (0.152323)	0.180781 (0.195407)	0.065179** (0.026257)	0.036960* (0.020752)	0.035904* (0.021047)	0.035130 (0.022986)	0.031420 (0.020760)
Log GDP per capita squared	-0.016295* (0.009510)	-0.015464* (0.008802)	-0.011306 (0.012096)	-0.003936*** (0.001493)	-0.002269* (0.001186)	-0.002235* (0.001191)	-0.002229* (0.001312)	-0.002116* (0.001200)
PM2.5		0.001574* (0.000906)	0.000079 (0.000347)	-0.000066 (0.000128)	-0.000182* (0.000108)	-0.000184* (0.000108)	-0.000179 (0.000112)	-0.000194* (0.000099)
Diabetes mellitus prevalence			0.000014 (0.000015)	0.000005*** (0.000001)	0.000003*** (0.000001)	0.000003*** (0.000001)	0.000003** (0.000001)	0.000003*** (0.000001)
Physician density				-0.003769 (0.002311)	-0.001323 (0.001398)	-0.001331 (0.001396)	-0.001055 (0.001401)	-0.000104 (0.001121)
Health expenditure (% GDP)					-0.002537** (0.001085)	-0.002541** (0.001084)	-0.002827** (0.001202)	-0.002361** (0.000940)
Urbanization						0.000031 (0.000087)	0.000029 (0.000091)	0.000064 (0.000080)
SEV							0.000019 (0.000203)	-0.000099 (0.000161)
Trade openness								0.000056** (0.000028)
Panel B. Model diagnostics								
Year fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	4,273	4,246	4,192	2,781	2,519	2,519	2,406	2,274
Countries	159	158	156	156	156	156	148	144
Instruments	32	33	34	35	33	34	35	36
AR(1) p-value	0.040	0.550	0.052	0.347	0.359	0.359	0.378	0.367
AR(2) p-value	0.612	0.530	0.967	0.708	0.914	0.890	0.700	0.898
AR(3) p-value	0.462	0.257	0.376	0.913	0.269	0.279	0.252	0.149
Hansen test p-value	0.829	0.731	0.218	0.883	0.908	0.970	0.813	0.613
Joint GDP terms p-value	0.180	0.188	0.634	0.009	0.056	0.057	0.071	0.024
Sum of lag coefficients (L1 + L2)	0.653	0.682	0.720	0.897	0.928	0.928	0.923	0.934

Notes: The dependent variable is ln(CKD prevalence). Windmeijer-corrected robust standard errors are reported in parentheses. Each specification uses its maximum available estimation sample. The first two lags of ln(CKD prevalence) are included as regressors, with lags 2–3 used as collapsed GMM-style instruments. GDP per capita, its squared term, and the control variables are treated as IV-style instruments. Year fixed effects are included in all specifications.

Table A3: GDP per capita and PM2.5 exposure country fixed-effects estimates

Variables	(1)	(2)
Log GDP per capita	-5.288*** (0.955)	-1.770* (1.012)
Constant	76.22*** (8.961)	44.53*** (9.195)
Year fixed effects	No	Yes
Country fixed effects	Yes	Yes
Observations	4,554	4554
Countries	158	158

Notes: The dependent variable is PM2.5 exposure. Both specifications include country fixed effects, while year fixed effects are included only in Model (2). Standard errors clustered at the country level are reported in parentheses. Year fixed-effect coefficients are not reported. $p < 0.10$, $p < 0.05$, $p < 0.01$.