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CAUSAL LINKAGES AMONG URBANISATION, CARBON EMISSIONS AND INFECTIOUS DISEASES IN HEAVILY INDEBTED POOR AFRICAN COUNTRIES

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ABSTRACT

This paper examined the direction of causality among urbanisation, carbon emissions and infectious diseases in heavily indebted poor countries in Africa. The analysis was motivated by the rising health burden associated with HIV prevalence, tuberculosis incidence and malaria incidence in countries where rapid urban growth, weak infrastructure, environmental degradation and limited fiscal capacity continue to shape public health outcomes. The article was derived from a panel of 28 heavily indebted poor African countries over the period 1990 to 2023. Infectious diseases were captured through three indicators: prevalence of HIV among the population aged 15-49, incidence of tuberculosis per 100,000 people and incidence of malaria per 1,000 people at risk. Urbanisation was measured as urban population as a percentage of total population, while carbon emissions were measured as CO_2 emissions in metric tonnes per capita. The study was anchored on the epidemiological transition theory and the environmental Kuznets curve hypothesis, with additional insights from environmental determinants and urban health perspectives. The Dumitrescu-Hurlin panel Granger non-causality test was employed to determine the direction of causality across the panel. The findings revealed bidirectional causality between HIV prevalence and carbon emissions, as well as between HIV prevalence and urbanisation. The results further showed that carbon emissions Granger-caused tuberculosis incidence, while tuberculosis and urbanisation exhibited bidirectional causality. For malaria, the findings showed feedback relationships between malaria incidence and carbon emissions and between malaria incidence and urbanisation. These results indicate that infectious diseases, urban development and environmental degradation reinforce one another in heavily indebted poor African countries. The article concludes that disease control in these countries should not be treated as a purely health-sector issue but as a combined public health, urban planning and environmental governance challenge. It recommends integrated disease surveillance, sustainable urban planning, improved sanitation, cleaner energy use and health-sensitive environmental policy as coordinated strategies for reducing infectious disease vulnerability.

KEYWORDS: Urbanisation; Carbon emissions; Infectious diseases; Heavily indebted poor countries; Dumitrescu-Hurlin causality.

INTRODUCTION

Infectious diseases remain a major global health and development problem, particularly in countries where health systems are weak, infrastructure is underdeveloped, and public resources are constrained. Diseases such as tuberculosis, HIV/AIDS, and malaria continue to impose heavy mortality, morbidity, and economic

costs on vulnerable populations. The burden is especially severe in heavily indebted poor countries in Africa, where fiscal constraints limit governments' ability to invest sufficiently in healthcare delivery, urban infrastructure, sanitation, and environmental management. The public health relevance of infectious diseases is also reflected in Sustainable Development Goal 3, which emphasizes the need to combat communicable diseases and strengthen health systems (World Health Organization, 2024).

The contemporary disease burden in Africa is increasingly shaped by structural and environmental changes. Rapid urbanization, accompanied by overcrowding, informal settlements, poor sanitation, and pressure on health facilities, can intensify disease transmission. Urban expansion is not inherently harmful, but when it occurs without adequate housing, drainage, waste management, and health planning, it creates conditions that favor the spread of communicable diseases. This is particularly important in heavily indebted poor countries because urban growth often outpaces public investment capacity. Urbanization in these countries is driven by rural-to-urban migration, population growth, and the search for economic opportunities, yet many cities lack the infrastructure required to manage the resulting demographic pressure (Grinin & Korotayev, 2023; Turok et al., 2023).

Carbon emissions add another dimension to the health challenge. Increasing emissions from fossil fuel use, transportation, industrial activity, deforestation, and urban energy demand contribute to environmental degradation and climate-related health risks. In the context of infectious diseases, carbon emissions may influence disease vulnerability through air pollution, weakened respiratory health, climate variability, and ecological shifts that affect vectors such as mosquitoes. Urbanization and emissions are closely connected in African HIPCs because unplanned urban expansion increases demand for transport, energy, and construction, while limited access to clean energy encourages reliance on polluting energy sources. This suggests that environmental degradation and urban growth may jointly shape patterns of infectious disease.

Although several studies have examined urbanization, carbon emissions, and public health, the direction of causality among these variables remains an important empirical question. A relationship between urbanization and disease does not necessarily indicate whether urbanization causes disease prevalence, whether disease prevalence influences urbanization, or whether both processes reinforce each other. Similarly, carbon emissions may affect disease outcomes, but disease burden may also feed back into emissions through pressure on health systems, migration, and changes in economic activity. Establishing the direction of causality, therefore, has important policy value because it clarifies whether interventions should primarily focus on urban planning, environmental controls, disease management, or integrated strategies.

This article focuses specifically on determining the direction of causality between urbanization, carbon emissions, and infectious diseases in heavily indebted poor countries in Africa. The article contributes to the literature by presenting a disease-specific causality analysis for HIV prevalence, tuberculosis incidence, and malaria incidence across 28 HIPCs in Africa from 1990 to 2023. By using the Dumitrescu-

Hurlin panel Granger causality test, the article accounts for the possibility that causal directions may differ across countries while still producing panel-level evidence. The central argument is that infectious diseases in fiscally constrained African economies should be analyzed within a broader urban-environmental system rather than as isolated medical outcomes.

Literature Review

Infectious diseases and public health vulnerability

Infectious diseases are caused by pathogenic microorganisms such as bacteria, viruses, fungi and parasites and may spread directly or indirectly across individuals and communities. Infectious diseases can be transmitted through respiratory droplets, contaminated food or water, sexual contact, insect bites or contact with infected bodily fluids. These diseases may be mild or severe, acute or chronic, and can produce wide-ranging social and economic consequences (Hanson et al., 2024; Stockmaier et al., 2021). In heavily indebted poor countries in Africa, infectious diseases remain difficult to control because exposure to disease is reinforced by poverty, weak healthcare systems, inadequate sanitation, and limited disease surveillance capacity.

The three infectious disease indicators examined in this article capture different transmission pathways and public health challenges. HIV prevalence reflects social, behavioural and health-system conditions, particularly access to prevention, testing and treatment services. Tuberculosis incidence reflects respiratory disease vulnerability and is associated with overcrowding, weak immunity, poverty and poor housing conditions. Malaria incidence reflects vector-borne disease risks, climate conditions, environmental management and access to preventive tools. Analysing these diseases together provides a broader picture of infectious disease vulnerability in heavily indebted poor countries in Africa.

Urbanisation and infectious diseases

Urbanisation refers to the increasing concentration of people, economic activities and infrastructure in urban areas. It is driven by rural-to-urban migration, economic opportunities, industrialisation, and population growth (Zafar et al., 2024; Voukkali et al., 2023). In principle, urbanisation can improve access to education, healthcare and employment. However, rapid and unplanned urbanisation can also increase exposure to infectious disease risks through overcrowded housing, poor drainage, inadequate sanitation, weak waste management and limited healthcare access. These conditions are particularly severe in heavily indebted poor countries in Africa, where fiscal constraints limit investment in basic urban services.

Urbanisation in Africa has been driven by population increases, rural-to-urban migration and the pursuit of economic opportunities. While this process can create economic benefits, it also produces pressure on housing, infrastructure and public health systems. In urban settlements where sanitation is poor and healthcare access is limited, the spread of tuberculosis, malaria and HIV may be intensified. For example, overcrowded living spaces increase the risk of respiratory disease transmission, while urban poverty and inadequate health education may reduce access to prevention

services. Urban land-use changes, poor drainage and stagnant water may also support mosquito breeding, thereby affecting malaria transmission.

Carbon emissions and infectious disease risks

Carbon emissions refer mainly to the release of carbon dioxide into the atmosphere through human activities such as fossil fuel combustion, industrial production, transportation, deforestation and agricultural practices. Carbon emissions are a major driver of environmental degradation, air pollution and adverse health outcomes. In urbanising African economies, emissions are often linked to increased energy consumption, transport demand, industrial growth, biomass burning and weak environmental regulation.

The pathway from carbon emissions to infectious disease is both direct and indirect. Air pollution may weaken respiratory health and increase susceptibility to diseases such as tuberculosis. Carbon emissions also contribute to climate change, which may alter rainfall, temperature, and ecological conditions, thereby affecting vector-borne diseases such as malaria. In addition, environmental degradation can intensify poverty and displacement, thereby increasing population vulnerability to infectious disease. In highly indebted poor countries, the limited capacity to invest in clean energy, environmental protection and climate adaptation further heightens this vulnerability.

Heavily indebted poor African countries and disease vulnerability

Heavily Indebted Poor Countries are low-income countries with unsustainable external debt burdens and limited fiscal space for development spending. These countries are often characterised by weak institutions, political instability, limited infrastructure, fragile healthcare systems and high poverty levels (Henri, 2019; Adewumi, 2021). Such conditions reduce governments' ability to provide adequate healthcare, sanitation, housing, and environmental management. As a result, infectious disease risks may persist despite national and international health interventions.

The 28 countries covered in the study were selected because they share debt-related constraints and structural development challenges. These countries include Benin, Burkina Faso, Cameroon, Central African Republic, Chad, Comoros, Republic of the Congo, Democratic Republic of the Congo, Ethiopia, Gambia, Ghana, Guinea, Guinea-Bissau, Liberia, Madagascar, Malawi, Mali, Mauritania, Mozambique, Niger, Rwanda, São Tomé and Príncipe, Sierra Leone, Senegal, Tanzania, Togo, Uganda and Zambia. The shared vulnerability of these countries makes them analytically important for understanding how urbanisation and carbon emissions interact with infectious diseases under conditions of financial and institutional constraint.

Theoretical Framework

The article is anchored primarily on the epidemiological transition theory and the environmental Kuznets curve hypothesis, with additional insights from environmental determinants and urban health perspectives. Epidemiological Transition Theory explains how disease patterns change as societies undergo social, economic, and demographic transformation (Omran, 1971). In the context of heavily indebted poor African countries, this theory is particularly relevant because many countries face a dual disease burden. They continue to experience high levels of infectious diseases while also confronting environmental and lifestyle-related health risks associated with urbanization and development.

The Environmental Kuznets Curve hypothesis explains the relationship between economic development and environmental degradation. It suggests that environmental degradation may initially rise as development and industrial activity increase but may later decline when societies reach higher income levels and adopt cleaner technologies and stronger environmental regulation (Grossman & Krueger, 1995). Within the context of this article, the hypothesis provides a useful lens for understanding why carbon emissions may rise with urban and industrial expansion in heavily indebted poor countries before adequate environmental controls are established.

Environmental determinants' perspectives further explain that external environmental conditions such as air quality, water quality, sanitation, housing, and climate conditions shape health outcomes. Urban health perspectives also show that health risks are distributed unevenly within cities, with poorer urban residents often exposed to overcrowding, pollution, and inadequate services. Together, these theoretical perspectives support the expectation that urbanization and carbon emissions may not only influence infectious disease prevalence but may also maintain feedback relationships with disease burden over time.

Methodology

Research design and data scope

The article adopted an ex post facto panel research design because it relied on already existing secondary data across countries and years. The panel covered 28 heavily indebted poor countries in Africa from 1990 to 2023. The period captured more

than three decades of urban growth, environmental change, public health transitions, and debt-related development constraints. The use of panel data enabled the study to combine cross-country variation with time-series dynamics, thereby improving the analytical capacity to detect causal relationships among the variables.

The main variables were infectious diseases, urbanization, and carbon emissions. Infectious diseases were measured using HIV prevalence among people aged 15-49, tuberculosis incidence per 100,000 people, and malaria incidence per 1,000 people at risk. Urbanization was measured as the percentage of the total population living in urban areas, while carbon emissions were measured as CO_2 emissions in metric tons per capita. Although the full thesis included additional control variables such as health expenditure, GDP per capita, and sanitation, this article focused on the first objective and therefore emphasized the causal directions among urbanization, carbon emissions, and infectious disease indicators.

Table 1: Variables and Measurement

Variable	Symbol	Measurement
HIV prevalence	PHIV	Prevalence of HIV, total (% of population ages 15-49)
Tuberculosis incidence	IOT	Incidence of tuberculosis per 100,000 people
Malaria incidence	IOM	Incidence of malaria per 1,000 population at risk
Urbanisation	URB	Urban population (% of total population)
Carbon emissions	CE	CO2 emissions (metric tons per capita)

Source: Author's compilation, 2025.

Model and estimation technique

To determine the direction of causality, the article employed the Dumitrescu-Hurlin panel Granger non-causality test. This test is appropriate for heterogeneous panel data because it allows causal relationships to differ across cross-sectional units while producing panel-level inference (Dumitrescu & Hurlin, 2012). The test is particularly useful in multi-country studies where economic, demographic, environmental and health-system conditions vary across countries.

The null hypothesis of the test is that there is no Granger causality between variables across the panel. Rejection of the null hypothesis implies that past values of one variable contain useful information for predicting another variable. In this article, the test was applied to examine whether urbanisation and carbon emissions Granger-

cause infectious disease indicators and whether infectious diseases also Granger-cause urbanisation or carbon emissions. This approach enabled distinguishing unidirectional causality from bidirectional feedback relationships.

Results and Discussion

Descriptive profile of the variables

The descriptive statistics show substantial variation in infectious diseases, urbanisation and carbon emissions across the selected countries. HIV prevalence was 3.184% among the population aged 15-49, while tuberculosis incidence averaged 235.072 cases per 100,000 people. Malaria incidence averaged 304.536 cases per 1,000 population at risk, indicating a considerable disease burden across the countries. Urbanisation averaged 35.378%, while carbon emissions averaged 3.068 metric tons per capita. These results show that the countries experienced both infectious disease vulnerability and uneven urban-environmental development.

The descriptive evidence also suggests that the sample countries were heterogeneous. HIV prevalence ranged from 0.100% to 15.200%, tuberculosis incidence ranged from 24.200 to 1,083.600 cases per 100,000 people, and malaria incidence ranged from 1.966 to 1,139.133 cases per 1,000 population at risk. Urbanisation also varied widely, ranging from 5.416% to 76.401%. These differences justify the use of a heterogeneous panel causality technique, as the countries do not share identical demographic, health, and environmental structures.

Table 2: Descriptive Statistics of Core Variables

Statistic	PHIV	IOT	IOM	URB	CE
Mean	3.184	235.072	304.536	35.378	3.068
Median	1.700	207.500	334.166	36.298	1.907
Maximum	15.200	1083.600	1139.133	76.401	26.006
Minimum	0.100	24.200	1.966	5.416	0.039
Std. Dev.	3.482	157.460	163.723	13.940	3.733

Source: Researcher's computation, 2025.

Direction of causality among HIV prevalence, urbanisation and carbon emissions

The Dumitrescu-Hurlin panel causality results indicate a bidirectional causal relationship between HIV prevalence and carbon emissions. The null hypothesis that HIV prevalence does not Granger-cause carbon emissions was rejected, with a W-statistic of 19.021 and probability value of 0.000. Similarly, the null hypothesis that carbon emissions do not Granger-cause HIV prevalence was rejected, with a W-statistic of 5.542 and probability value of 0.000. This feedback relationship suggests

that HIV prevalence and carbon emissions are dynamically connected in heavily indebted poor African countries.

The result may be interpreted in two ways. First, rising carbon emissions may worsen public health vulnerability through environmental degradation, poor air quality and broader climate-related health risks. These factors can increase health-system pressure and weaken population resilience. Second, a high infectious disease burden may also influence emissions indirectly through increased use of health infrastructure, energy-consuming healthcare activities, migration to urban centres for treatment and changes in social and economic activity. Although the causality result alone does not establish structural mechanisms, it provides strong evidence that HIV prevalence and emissions are not independent over time.

The results also show bidirectional causality between HIV prevalence and urbanisation. HIV prevalence Granger-caused urbanisation with a W-statistic of 6.372 and probability value of 0.000, while urbanisation Granger-caused HIV prevalence with a W-statistic of 5.457 and probability value of 0.000. This finding supports the argument that urbanisation can shape HIV-related vulnerability through population concentration, social interaction patterns, informal settlements and uneven access to prevention services. At the same time, HIV prevalence may influence urbanisation through migration decisions, especially where people move toward cities in search of employment, treatment and support services.

Direction of causality among tuberculosis incidence, urbanisation and carbon emissions

The causality results for tuberculosis show a more specific pattern. There was no evidence that tuberculosis incidence Granger-caused carbon emissions, as the W-statistic of 1.161 yielded a p-value of 0.547. However, the reverse relationship was statistically significant. Carbon emissions Granger-caused tuberculosis incidence, with a W-statistic of 2.564 and probability value of 0.000. This result indicates that carbon emissions are important for predicting tuberculosis incidence in the panel, while tuberculosis incidence did not significantly predict carbon emissions.

This unidirectional causality is consistent with the health logic that environmental pollution and poor air quality can worsen respiratory health conditions. Since tuberculosis is a respiratory infectious disease, emissions-related air pollution may contribute to vulnerability by weakening respiratory defence mechanisms and intensifying exposure risks in crowded urban environments. The finding implies that tuberculosis control in heavily indebted poor African countries should include environmental health considerations, particularly pollution control, housing quality and urban air management.

The results further reveal bidirectional causality between tuberculosis incidence and urbanisation. Tuberculosis incidence Granger-caused urbanisation with a W-statistic of 5.242 and probability value of 0.000, while urbanisation Granger-caused tuberculosis incidence with a W-statistic of 3.681 and probability value of 0.000. This feedback relationship indicates that urbanisation and tuberculosis may reinforce each other. Urban overcrowding, poor ventilation, informal housing and inadequate

healthcare access may increase tuberculosis transmission, while the disease burden may influence migration and settlement patterns through health-seeking behaviour and economic displacement.

Direction of causality among malaria incidence, urbanisation and carbon emissions

For malaria incidence, the results reveal significant causal relationships with both carbon emissions and urbanisation. The test showed that malaria incidence Granger-caused carbon emissions, with a W-statistic of 1.899 and probability value of 0.001. Carbon emissions also Granger-caused malaria incidence, with a W-statistic of 3.383 and probability value of 0.000. This indicates a feedback relationship between malaria and carbon emissions. The result suggests that changes in environmental conditions associated with emissions may influence malaria transmission, while malaria burden may also be linked to changes in energy use, land use, healthcare demand and population behaviour.

The emissions-malaria feedback can be explained through climate-sensitive pathways. Carbon emissions contribute to climate variability, which can affect temperature, rainfall and ecological conditions associated with mosquito breeding and malaria transmission. At the same time, malaria outbreaks can influence economic activities, health expenditure and migration patterns, thereby indirectly affecting emissions-related processes. For heavily indebted poor countries, this result is important because these countries often have limited capacity to invest simultaneously in climate adaptation, malaria prevention and environmental governance.

The results also show bidirectional causality between malaria incidence and urbanisation. Malaria incidence Granger-caused urbanisation with a W-statistic of 12.405 and probability value of 0.000, while urbanisation Granger-caused malaria incidence with a W-statistic of 3.159 and probability value of 0.000. This finding suggests that malaria and urbanisation are dynamically linked. Urban expansion can alter land use, drainage systems and settlement patterns in ways that create mosquito breeding sites. Conversely, malaria prevalence may influence migration and urban settlement decisions, especially when people move in search of healthcare or economic opportunities.

Table 3: Dumitrescu-Hurlin Panel Causality Results

Causality Path	W-Stat	Z bar-Stat	Prob.	Decision
PHIV does not Granger cause CE	19.021	67.428***	0.000	Reject Ho
CE does not Granger cause PHIV	5.542	16.995***	0.000	Reject Ho

PHIV does not Granger cause URB	6.372	20.102***	0.000	Reject Ho
URB does not Granger cause PHIV	5.457	16.678***	0.000	Reject Ho
IOT does not Granger cause CE	1.161	0.602	0.547	Do not reject Ho
CE does not Granger cause IOT	2.564	5.851***	0.000	Reject Ho
IOT does not Granger cause URB	5.242	15.872***	0.000	Reject Ho
URB does not Granger cause IOT	3.681	10.034***	0.000	Reject Ho
IOM does not Granger cause CE	1.899	3.365***	0.001	Reject Ho
CE does not Granger cause IOM	3.383	8.917***	0.000	Reject Ho
IOM does not Granger cause URB	12.405	42.672***	0.000	Reject Ho
URB does not Granger cause IOM	3.159	8.077***	0.000	Reject Ho

Note: *** denotes statistical significance at the 1% level. PHIV = HIV prevalence; IOT = incidence of tuberculosis; IOM = incidence of malaria; CE = carbon emissions; URB = urbanisation. Source: Researcher's computation, 2025.

Discussion of findings

The overall causality evidence shows that urbanisation, carbon emissions and infectious diseases form an interdependent system in heavily indebted poor African countries. HIV prevalence showed feedback relationships with both urbanisation and carbon emissions, indicating that social, environmental and health-system conditions

may operate together. Tuberculosis showed unidirectional causality from carbon emissions and bidirectional causality with urbanisation, suggesting that environmental degradation and urban crowding are particularly relevant for respiratory disease vulnerability. Malaria showed feedback relationships with both emissions and urbanisation, confirming its sensitivity to ecological and settlement conditions.

The findings are consistent with the Environmental Kuznets Curve perspective, in that development processes associated with urban expansion and emissions may initially intensify environmental and health risks where environmental regulation and infrastructure are weak. They also align with Epidemiological Transition Theory because the selected countries are still facing a high infectious disease burden while simultaneously experiencing urban and environmental transitions. The coexistence of communicable disease burden with urban pollution pressures indicates that African HIPCs are experiencing a complex health transition rather than a simple shift from infectious to non-communicable diseases.

The strongest policy message is that infectious disease control cannot be separated from urban and environmental policy. Urbanisation that occurs without adequate housing, sanitation, waste management, clean water, and access to healthcare is likely to sustain disease vulnerability. Similarly, carbon emissions and environmental degradation can increase climate-sensitive and respiratory health risks. The feedback relationships found in the study suggest that public health outcomes can also shape urban and environmental processes through migration, healthcare demand and economic adjustment. Therefore, policy must be multidirectional rather than narrowly focused on isolated disease programmes.

The findings are particularly important because heavily indebted poor countries have limited fiscal and institutional capacity. These countries may find it difficult to finance large-scale health interventions, urban infrastructure, and environmental regulation simultaneously. However, the causality results indicate that such interventions are interconnected. Investing in sustainable urban planning can reduce exposure to disease, while environmental controls can reduce health vulnerability. Similarly, stronger disease control can reduce pressure on urban systems and improve development outcomes. This makes integrated policy design more efficient than fragmented interventions.

Conclusion and Policy Implications

This article examined the direction of causality among urbanisation, carbon emissions, and infectious diseases in 28 heavily indebted poor countries in Africa between 1990 and 2023. The evidence from the Dumitrescu-Hurlin panel causality test showed that the relationship among these variables is predominantly feedback-based. HIV prevalence and carbon emissions were bidirectionally related, and HIV prevalence also showed bidirectional causality with urbanisation. Carbon emissions Granger-caused tuberculosis incidence, while tuberculosis and urbanisation exhibited bidirectional causality. Malaria incidence showed bidirectional relationships with both carbon emissions and urbanisation.

The conclusion drawn from these findings is that infectious disease vulnerability in heavily indebted poor countries in Africa is not only a medical problem. It is also a function of urban development, environmental degradation, infrastructure gaps and fiscal limitations. Urbanisation and emissions appear to influence disease dynamics, while infectious disease burden can also influence urban and environmental processes. This interdependence requires a public health strategy that integrates disease control with urban planning and environmental management.

The policy implication is that governments in heavily indebted poor countries in Africa should strengthen sustainable urban planning by expanding access to clean water, sanitation, drainage systems, affordable housing, and waste management services. These interventions can reduce urban overcrowding and environmental exposure that increase infectious disease risks. Public health planning should also be incorporated into urban development projects so that disease surveillance, healthcare facilities and sanitation systems are designed alongside housing and transport infrastructure.

Environmental policy should be treated as part of infectious disease control. Cleaner energy alternatives, emission control measures, afforestation programmes and improved regulation of urban pollution can reduce the health risks associated with carbon emissions. Since carbon emissions were linked to tuberculosis and malaria dynamics, environmental interventions can support health outcomes beyond their climate benefits. Health ministries, environment ministries, and urban planning agencies should therefore coordinate their policies rather than work separately.

Finally, disease surveillance should be strengthened in urban areas and environmentally vulnerable communities. Early warning systems for malaria, tuberculosis and HIV should be linked to indicators of urban growth, population density, sanitation coverage and environmental pollution. Given the debt constraints faced by the countries, development partners and regional institutions should support integrated programmes that address health, urbanisation and environmental risks together. Such coordination can improve policy efficiency and help reduce the long-term burden of infectious diseases in heavily indebted poor countries in Africa.

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