

# Geospatial distribution and determinants of child mortality in rural western Kenya 2002–2005

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## Summary

**OBJECTIVE** To describe local geospatial variation and geospatial risk factors for child mortality in rural western Kenya.

**METHODS** We calculated under-5 mortality rates (U5MR) in 217 villages in a Health and Demographic Surveillance System (HDSS) area in western Kenya from 1 May 2002 through 31 December 2005. U5MRs by village were mapped. Geographical positioning system coordinates of residences at the time of death and distances to nearby locations were calculated. Multivariable Poisson regression accounting for clustering at the compound level was used to evaluate the association of geospatial factors and mortality for infants and children aged 1–4 years.

**RESULTS** Among 54 057 children, the overall U5MR was 56.5 per 1000 person-years and varied by village from 21 to 177 per 1000 person-years. High mortality villages occurred in clusters by location and remained in the highest mortality quintile over several years. In multivariable analysis, controlling for maternal age and education as well as household crowding, higher infant mortality was associated with living closer to streams and further from public transport roads. For children 1–4 years, living at middle elevations (1280–1332 metres), living within lower population densities areas, and living in the northern section of the HDSS were associated with higher mortality.

**CONCLUSIONS** Childhood mortality was significantly higher in some villages. Several geospatial factors were associated with mortality, which might indicate variability in access to health care or exposure and transmission of infectious diseases. These results are useful in prioritising areas for further study and implementing directed public health interventions.

**keywords** child mortality, risk factors, geospatial positioning, demographic surveillance system

## Introduction

Achieving the 4th Millennium Development Goal of lowering childhood mortality by two-thirds by 2015 depends on advances in sub-Saharan Africa where the highest mortality rates exist (Black *et al.* 2003). Success in lowering child mortality through wide-scale implementation of proven interventions, such as vaccines and insecticide-treated bednets, has been achieved in recent years in

parts of some African countries, such as Tanzania (Black *et al.* 2003; Jones *et al.* 2003; Masanja *et al.* 2008).

Despite these successes, challenges remain in lowering child mortality. Child mortality is a multifactorial phenomenon requiring progress on multiple fronts beyond effective biomedical interventions (Mosley & Chen 1984). Vaccines will not save lives if children are not vaccinated; insecticide-treated bednets will not save lives if children at the highest risk are not sleeping under them; effective treatment for infectious diseases will not save lives if sick children are not brought to health facilities. Much of the

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effectiveness of interventions depends on individual, household, and community-level variables, such as parental norms and attitudes, socio-economic resources, and the political economy, respectively (Mosley & Chen 1984).

Another factor that can influence effectiveness of interventions in several ways, which is often overlooked, is local geospatial variability. Physical location can affect child mortality through several mechanisms. Macro-geospatial differences in mortality rates are well described between African countries, as well as within regions within the same country (Central Bureau of Statistics (CBS) [Kenya] *et al.* 2004; Mturi & Curtis 1995; Snow *et al.* 2004; Terra de Souza *et al.* 1999; Root 1999). These macro-level geospatial differences in child mortality are attributed to national and regional differences in prevalence of diseases, such as malaria and HIV, as well as socio-economic development. Geospatial variation in child mortality has also been shown, albeit less frequently, within smaller geographical areas (Binka *et al.* 1998; Sankoh *et al.* 2001; Becher *et al.* 2004; Kazembe *et al.* 2006). Local geospatial differences in child mortality can be instrumental in charting out multipronged, customised approaches to reducing child mortality for Ministries of Health. We examine geospatial determinants of child mortality within the western Kenya Health and Demographic Surveillance System (HDSS) of the Kenya Medical Research Institute (KEMRI) and the Centers for Disease Control and Prevention (CDC) from 2002–2005.

## Methods

### Study site and population

The HDSS in western Kenya is located in Bondo and Siaya districts, rural parts of Nyanza Province, in an area of 450 km<sup>2</sup>. During the period of study, the HDSS was divided into two areas. Asembo is in the south bordering Lake Victoria with 75 villages and Gem in the north with 142 villages. The study area consists of gentle hills drained by small seasonal streams terminating in Lake Victoria. Gem has one major permanent river, Asembo has none. Gem for the most part is hillier than Asembo. Few paved or public transport roads exist in the area, with walking being the main means of transport. Rainfall generally occurs year round, with the heaviest rains usually falling in March–May, and a second rainy period in October–November. Total annual rainfall averages approximately 1400 mm/year. The residents are subsistence farmers growing mainly maize, sorghum, and groundnuts. The inhabitants are poor with over 66% of individuals living below the poverty line in 1999 (Krishna *et al.* 2004).

The area has high child mortality (Mcelroy *et al.* 2001; Adazu *et al.* 2005). Malaria is holoendemic and the leading cause of childhood death in the area (Phillips-Howard *et al.* 2003). The primary malaria vectors in this region are *Anopheles gambiae* Giles and *An. funestus* Giles, while *Plasmodium falciparum* is the principal species of human malaria in the region. After an ITN trial in the late 1990s and ending in 2002, >70% community ITN coverage has been maintained in the HDSS (Phillips-Howard *et al.* 2003; Adazu *et al.* 2005; CDC unpublished data). Malaria parasite prevalence averaged 75% in children younger than 5 years in the late 1990s, decreasing to 30%–40% in 2006–7 despite high ITN coverage. [(Bloland *et al.* 1999) and CDC unpublished data]. HIV prevalence is among the highest in Kenya (NASCO 2009). In 2003–2004, HIV prevalence in Asembo was highest in women of child-bearing age 25–29 years (36.5%) (Amornkul *et al.* 2009). Prevention of Mother-to-Child Transmission (PMTCT) services were mostly unavailable until 2003 and have been increasing since then (75% uptake in antenatal clinics in 2007, CDC unpublished data). Respiratory illness and diarrhoea are also prevalent causes of childhood death (Mcelroy *et al.* 2001).

### Demographic surveillance system

The HDSS has been described in detail elsewhere (Adazu *et al.* 2005). In brief, a baseline census of the population of the HDSS area was conducted from September–December 2001 for Asembo and from May–August 2002 in Gem. All members of the 23,326 participating compounds were enumerated. All participating compounds are visited in a 4-month cycle to update the current status of each resident. All pregnancies, births, deaths, in-migration and out-migration, and any new compounds that have been built since the last visit are documented. To capture all births and deaths, village reporters are recruited from the community, trained and paid to record all births and deaths in their village.

A trained team attempts to do verbal autopsies on all deaths among HDSS residents. The verbal autopsy interviews are conducted at least one month after death using a standardised questionnaire from the network organisation of DSSs, INDEPTH, (Alonso *et al.* 2002) and adapted from the WHO model (Anker *et al.* 1999). The individual who took care of the deceased is asked about circumstances, signs, and symptoms that preceded death. Two trained clinical officers independently review each verbal autopsy questionnaire and assign a single most likely cause of death according to the standards of interpretation of verbal autopsies.

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Approval for the HDSS protocol was obtained from the ethical review boards of KEMRI (SSC # 647) and CDC (IRB #3308). All participating heads of household gave written informed consent.

### GPS mapping of the HDSS

In 1996, all positions of existing compounds in the HDSS area were mapped with an estimated accuracy of  $\pm 1$  metres using hand-held, differential global positioning system (GPS; Trimble Navigation Ltd, California, USA) (Hightower *et al.* 1998). Mapping was updated at the start of the HDSS and annually thereafter to account for newly constructed houses. All health facilities, markets, schools, public transport roads (defined as roads which a public service vehicle uses daily), lakeshores, and streams within the HDSS area were also mapped. Data were downloaded with differential correction into a GPS database (GPS pathfinder office 2.8; Trimble Navigation Ltd, California, USA).

### Data analysis

The under-5 mortality rates (U5MR) for each village in the HDSS were calculated by dividing the observed number of deaths among children in a village by the cumulative

person-years of HDSS resident children in that village. A child's contribution to the denominator began at the beginning of the study period on 1 May 2002, when he/she in-migrated to the HDSS and became a resident, or when he/she was born. A child's contribution to the denominator ceased when he/she died, out-migrated, reached his/her 5th birthday or reached the end of the study period (31 December 2005). A child could contribute person-time to more than one village if he/she moved within the HDSS area during the study period. The U5MRs were divided into equal quintiles and plotted by village. The under-5 mortality ratio was calculated using the synthetic cohort life table approach, then expressing the probability of a child dying before his/her 5th birthday as the number of deaths per 1000 live births (Central Bureau of Statistics (CBS) [Kenya] *et al.* 2004).

The straight-line distances from each compound to the nearest streams, public transport roads, markets, and health facilities were calculated using Arcview 3.2 software. All distance-related variables were initially grouped into 20% quintiles. Child mortality was graphed using these distance quintiles. Visual inspection of the graphs led to categorisation for some variables into two or three categories based on obvious groupings of the quintile-level data (Table 1). The furthest distance category was used as the referent for all geospatial variables. Several

**Table 1** Selected non-geospatial risk factors and verbal autopsy cause of death by village mortality quintile based on under-5 mortality rates (U5MR) in deaths per 1000 person-years, western Kenya Health and Demographic Surveillance System (HDSS), 1 May 2002–31 December 2005

Village mortality quintiles (Under-5 mortality rate as deaths per 1000 person-years)	<45	45–54	55–62	63–81	≥82	<i>P</i> value, trend test	<i>P</i> value, vs. lowest 3 quintiles
Number of Villages	44	43	40	47	43		
Individual risk factors, <i>n</i> (%)							
Mother with no or primary school education only	12,230 (94.7)	15,320 (94.9)	13,478 (93.5)	15,269 (93.7)	12,483 (94.4)	<b>0.001</b>	<b>0.02</b>
<5 household members	6561 (42.4)	7808 (41.2)	6788 (41.3)	7844 (42.4)	6700 (46.6)	<b>&lt;0.0001</b>	<b>&lt;0.0001</b>
Maternal age <26.14 years	8304 (58.7)	10,265 (58.9)	8864 (58.8)	9930 (57.9)	7738 (57.2)	<b>0.0002</b>	<b>0.0008</b>
Crude birth rate (births per 1000 residents)	221.3	229.0	222.4	232.5	241.5	–	<b>0.001</b>
Verbal autopsy cause of death, <i>n</i> (%)							
Malaria	97 (27.6)	154 (26.9)	126 (22.7)	159 (22.3)	201 (26.1)	0.14	0.44
Pneumonia	57 (16.2)	102 (17.8)	92 (16.6)	113 (15.9)	141 (18.2)	0.77	0.95
Gastro enteritis/Dehydration	41 (11.7)	81 (14.1)	73 (13.2)	96 (13.5)	121 (15.7)	0.42	0.25
Anaemia	38 (10.8)	64 (11.2)	60 (10.8)	77 (10.8)	90 (11.7)	0.98	0.79
Malnutrition	17 (4.8)	25 (4.4)	40 (7.2)	60 (8.4)	56 (7.3)	<b>0.03</b>	<b>0.01</b>
HIV	22 (6.3)	35 (6.1)	29 (5.2)	42 (5.9)	47 (6.1)	0.96	0.83
Meningitis	27 (7.7)	37 (6.5)	37 (6.7)	58 (8.2)	44 (5.6)	0.35	0.98
Others	44 (12.5)	67 (11.7)	84 (15.2)	94 (13.2)	65 (8.4)	<b>0.05</b>	<b>0.04</b>
Undetermined	8 (2.3)	9 (1.6)	13 (2.4)	13 (1.8)	8 (1)	0.18	0.20

Bold values are statistically significant at *P* value <0.05.

non-geospatial variables known to be associated with child mortality were included in the model where appropriate. These variables were maternal age at the child's birth, maternal education, household crowding (number of people per house), and socio-economic status of the household categorised as low and high, based on principal component analysis of eight indicator variables (Vyas & Kumaranayake 2006). Because of potential differences in risk factors for mortality among infants and older children, we ran separate models for children aged 0–11 and 12–59 months. The latter model included a variable that dichotomised age into 12–23 and 24–59 months because of possible confounding by age. Individual children could contribute person-time to multiple age categories.

All variables were evaluated using Poisson regression at the level of the individual (Proc Genmod, SAS 9.1). The dependent variable was the vital status of the child. The log of the number of person-years contributed by the child was included in the model as an offset. Clustering at the level of the compound was controlled for using generalised estimating equations with an exchangeable working correlation matrix. Variables found to have a *P* value >0.1 in univariate analysis were included in an initial multivariable model. Variables were removed using a backwards elimination procedure. The final model only retained variables significant at the level of *P* < 0.05. When comparing characteristics of village mortality quintiles, proportions were compared using the Cochran-Armitage trend test and chi-square test and crude birth rates by a *Z* test.

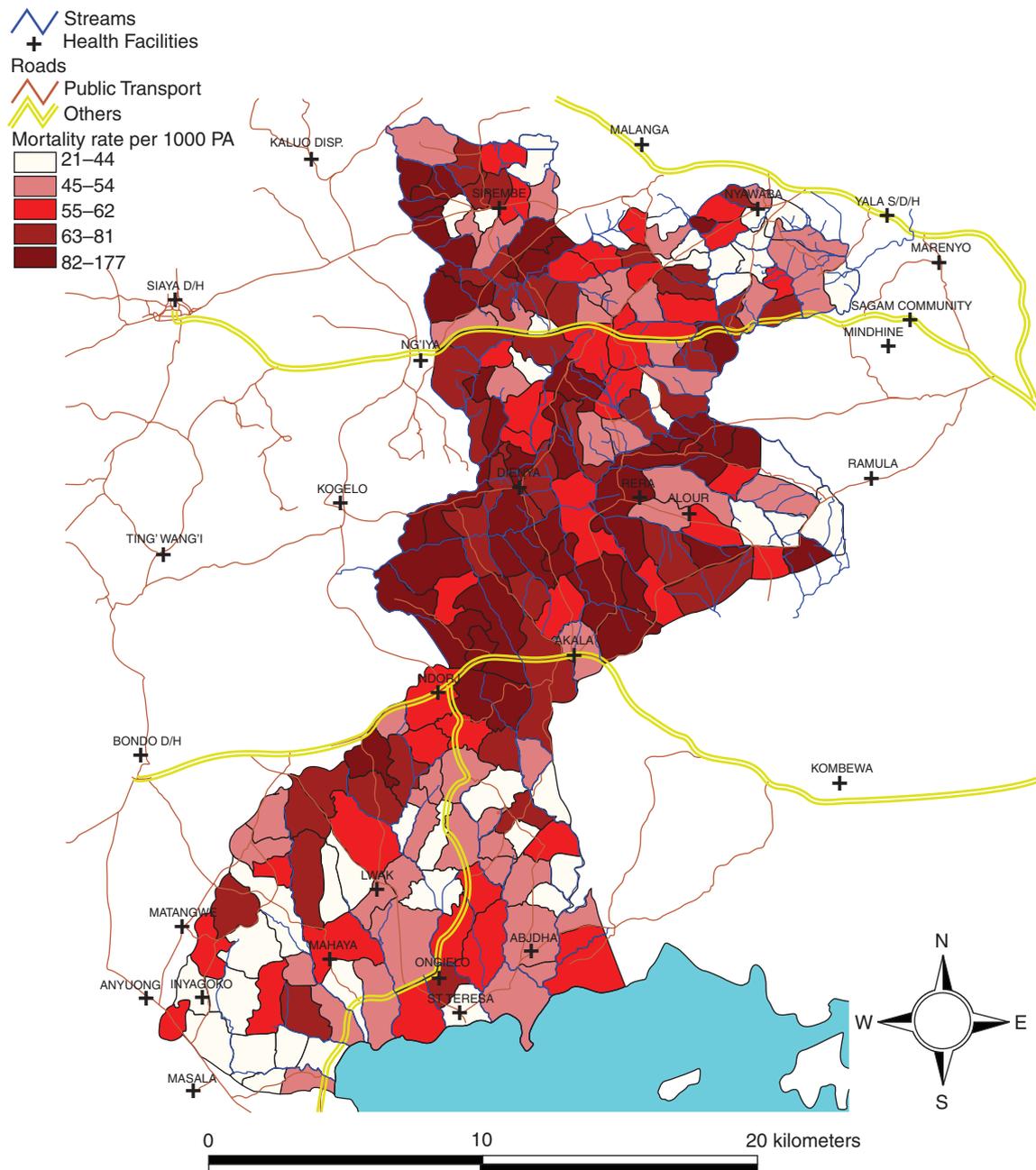
## Results

From 1 May 2002 to 31 December 2005, 4194 deaths occurred among 54 057 children under 5 years of age who contributed 74 233 person-years of follow-up. The overall U5MR was 56.5 deaths per 1000 person-years, and the under-5 mortality ratio was 218 deaths per 1000 live births. The infant mortality rate (IMR) was 138.9 deaths per 1000 person-years. Of infant deaths, 19% were among neonates (i.e. first 28 days of life). The U5MR varied significantly by village from a low of 21 to a high of 177 per 1000 person-years (Figure 1). High and low mortality villages tended to occur in clusters. Among 43 villages in the highest quintile (U5MR  $\geq$  82 per 1000 person-years) for all years combined, 39 (91%) were found in the highest quintile every year from 2003–2005. Of 44 villages in the lowest quintile (U5MR  $\leq$  44 per 1000 person-years), 26 (59%) were in the lowest quintile every year from 2003–2005. Of the 43 villages in the highest mortality quintile, 40 (93%) were in Gem and of the 44 villages in the lowest mortality quintile, 25 (60%) were in Asembo. Compared with the lower three village mortality quintiles, the highest

two village mortality quintiles were more likely to have fewer than five persons in the household, have fewer mothers older than 26 years, and have higher crude birth rates (Table 1).

In univariate analysis for both infants and older children, higher mortality was associated with several geospatial variables – living closer to a stream, further from a public transport road, in areas of lower population densities, and living in the northern area of the HDSS (Tables 2, 3). For older children, but not infants, living at an intermediate elevation (1280–1332 metres) was associated with higher mortality. Of note, proximity of residence to a health facility was not associated with mortality. In multivariable analysis, the models for infants and older children showed different geospatial risk factors for mortality (Tables 2, 3). For infants, living closer to a stream and farther from a public transport road were associated with higher mortality (Table 2). Whereas for older children, living at intermediate elevation (1280–1332 metres), living in areas of lower population density, living in the northern half of the HDSS, and living 768–1269 metres from a public market were associated with higher mortality (Table 3). For non-geospatial variables, for both infants and older children, living in households with fewer than five individuals was associated with higher mortality (Tables 2, 3). No other non-geospatial risk factors were associated with mortality for infants. For older children, higher mortality was associated with children aged 12–23 months (versus 24–59 months), no maternal education, and younger maternal age.

Among the 4194 deaths, 3018 (72%) verbal autopsies were performed, of which 2954 (98%) had linked GIS information available at the time of death. The leading causes of death were malaria (25%), pneumonia (17%), gastroenteritis/dehydration (14%), and anaemia (11%), (Table 4). Among infant deaths, specific neonatal causes, such as neonatal sepsis and pregnancy complications (e.g. prematurity, birth trauma/asphyxia), accounted for 10% of deaths (Table 4). The only verbal autopsy cause of death more likely in the higher mortality quintile villages was malnutrition (Table 1). Malaria as the cause of death on verbal autopsy was higher in the most sparsely populated quintile (29% compared with 24% in the other four quintiles, *P* = 0.01); however, malaria deaths were not associated with other geospatial factors. The percentage of deaths from gastroenteritis/dehydration was higher among children living closer to streams (15% among those living <436 metres *vs.* 12% among those living  $\geq$ 436 metres, *P* = 0.01) and in Gem (15% *vs.* 12% in Asembo, *P* = 0.003) and was lower among those living in the most densely populated areas (11% in areas with  $\geq$ 716 inhabitants per km<sup>2</sup> *vs.* 14% in areas with <716 inhabitants per



**Figure 1** Under-5 mortality rate (densities per 1000 person year) by village ( $n = 217$ ) divided into quintiles, western Kenya Health and Demographic Surveillance System (HDSS), 1 May 2002–31 December 2005.

$\text{km}^2$ ,  $P = 0.048$ ). Among children who died, those who lived closer to streams tended to be less likely to have a protected drinking water source (i.e. borehole, protected spring, and rainfall [27.6% *vs.* 30.4%,  $P = 0.062$ ]). Pneumonia deaths were more common among children living at

lower elevations (20% among those living  $\leq 1211$  metres *vs.* 16% among those living  $> 1211$  metres,  $P = 0.049$ ) and in Asembo (19% *vs.* 16% in Gem,  $P = 0.008$ ). Anaemia deaths were not associated with any geospatial factors. Although distance to a health facility was not associated

M. Ombok *et al.* **Geospatial distribution and determinants of child mortality in rural western Kenya****Table 2** Association between geospatial and other variables and mortality rate in infants <1 year old in western Kenya Health and Demographic Surveillance System (HDSS), 1 May 2002–31 December 2005

Variable	Categories	Univariate rate ratio (95% Confidence intervals)	Multivariable rate ratio (95% Confidence intervals)
<b>Geospatial-related variables</b>			
Distance to stream (metres)	0–435	<b>2.01 (1.21–3.36)</b>	<b>2.32 (1.08–4.97)</b>
	436–648	<b>1.89 (1.10–3.26)</b>	<b>2.29 (1.03–5.07)</b>
	649–2451	Ref	Ref
Distance to public transport road (metres)	11–757	<b>0.59 (0.37–0.93)</b>	<b>0.58 (0.34–0.99)</b>
	758–4286	<b>0.70 (0.55–0.91)</b>	<b>0.71 (0.53–0.94)</b>
	4287–8828	Ref	Ref
Distance to health facility (metres)	7–2205	0.95 (0.74–1.20)	NS*
	2206–6602	Ref	
Distance to market (metres)	11–757	0.75 (0.49–1.16)	NS*
	768–1269	0.84 (0.57–1.24)	
	1280–1703	0.99 (0.71–1.39)	
	1704–2238	1.11 (0.81–1.54)	
	2239–4954	Ref	
Elevation (metres)	1123–1211	0.99 (0.64–1.52)	NS*
	1212–1279	1.24 (0.86–1.81)	
	1280–1332	1.16 (0.76–1.78)	
	1333–1386	1.34 (0.93–1.94)	
Study area	1387–1474	Ref	
	Asembo	<b>0.71 (0.56–0.91)</b>	NS*
Population density (person per sq kilometres)	Gem	Ref	
	46–361	<b>1.82 (1.16–2.83)</b>	NS*
	362–456	1.55 (0.96–2.49)	
	457–564	1.48 (0.93–2.35)	
	565–715	1.48 (0.94–2.33)	
	716–2969	Ref	
<b>Non-geospatial variables</b>			
Education	None	0.54 (0.002–166.72)	NS*
	Primary	1.18 (0.74–1.87)	
	Secondary and Post secondary	Ref	
Household size	<5 household members	<b>4.75 (2.88–7.83)</b>	<b>3.29 (2.42–4.45)</b>
	≥5 household members	Ref	Ref
Maternal age (years)	11.55–19.33	0.99 (0.68–1.44)	NS*
	19.34–22.29	0.15 (0.82–1.61)	
	22.3–26.13	1.11 (0.78–1.58)	
	26.14–31.76	0.92 (0.64–1.30)	
	≥31.77	Ref	
Socio-economic status	Low	1.15 (0.66–1.98)	NS*
	High	Ref	

\*Not significant.

Bold values are statistically significant at  $P$  value <0.05.

with U5MR in the multivariable model, among children who died, those living closer to a health facility were more likely to visit it before death (69% of those living <2192 metres *vs.* 61% of those living >2192 metres,  $P < 0.001$ ).

## Discussion

Despite the apparent cultural and environmental homogeneity of the western Kenya HDSS area, we demonstrated

variation in child mortality rates by village, as well as by several geospatial factors related to a child's residence, namely, proximity to streams and public transport roads, elevation, and population density of the resident area. Clustering of child mortality was previously shown in a HDSS in Burkina Faso; however, elevated mortality did not persist in the same villages year after year, and a true cluster of persistently elevated mortality, as determined using spatial statistics, was found in only one village

**Table 3** Association between geospatial and other variables and mortality rate of children aged 1–4 years in western Kenya Health and Demographic Surveillance System (HDSS), 1 May 2002–31 December 2005

Variable	Categories	Univariate rate ratio (95% CI)	Multivariable rate ratio (95% CI)
<b>Geospatial-related variables</b>			
Distance to stream (metres)	0–435	<b>1.20 (1.06–1.36)</b>	NS*
	436–648	1.12 (0.97–1.31)	
	649–2451	Ref	
Distance to public transport road (metres)	11–757	0.89 (0.77–1.03)	NS*
	758–4286	<b>0.88 (0.79–0.99)</b>	
	4287–8828	Ref	
Distance to health facility (metres)	7–2205	0.92 (0.84–1.01)	NS*
	2206–6602	Ref	
Distance to market (metres)	11–757	<b>0.85 (0.73–0.99)</b>	0.99 (0.84–1.17)
	768–1269	1.08 (0.94–1.25)	<b>1.18 (1.03–1.36)</b>
	1280–1703	0.94 (0.82–1.09)	1.04 (0.90–1.21)
	1704–2238	0.94 (0.82–1.09)	0.99 (0.86–1.14)
	2239–4954	Ref	Ref
Elevation (metres)	1123–1211	<b>0.80 (0.69–0.94)</b>	1.00 (0.81–1.24)
	1212–1279	1.06 (0.92–1.23)	1.17 (0.98–1.40)
	1280–1332	<b>1.20 (1.04–1.38)</b>	<b>1.28 (1.09–1.49)</b>
	1333–1386	1.07 (0.92–1.23)	1.07 (0.92–1.24)
	1387–1474	Ref	Ref
Study area	Asembo	<b>0.80 (0.73–0.88)</b>	<b>0.81 (0.70–0.94)</b>
	Gem	Ref	Ref
Population density (person per sq kilometres)	46–361	<b>1.39 (1.20–1.62)</b>	<b>1.28 (1.07–1.52)</b>
	362–456	<b>1.28 (1.10–1.49)</b>	<b>1.20 (1.01–1.42)</b>
	457–564	1.18 (1.00–1.37)	1.09 (0.92–1.29)
	565–715	1.17 (1.00–1.37)	1.10 (0.94–1.30)
	716–2969	Ref	Ref
<b>Non-geospatial variables</b>			
Age category (months)	12–23	<b>3.93 (3.59–4.30)</b>	<b>3.87 (3.54–4.23)</b>
	24–59	Ref	
Education	None	1.70 (1.00–2.88)	<b>1.73 (1.03–2.91)</b>
	Primary	1.19 (0.97–1.45)	1.20 (0.97–1.46)
	Secondary and Post secondary	Ref	Ref
Household size	<5 household members	<b>1.46 (1.32–1.61)</b>	<b>1.42 (1.28–1.56)</b>
	≥5 household members	Ref	Ref
Maternal age (years)	11.55–19.33	<b>1.43 (1.23–1.66)</b>	<b>1.36 (1.17–1.58)</b>
	19.34–22.29	<b>1.32 (1.13–1.54)</b>	<b>1.32 (1.14–1.53)</b>
	22.3–26.13	<b>1.41 (1.22–1.63)</b>	<b>1.47 (1.28–1.70)</b>
	26.14–31.76	<b>1.25 (1.08–1.45)</b>	<b>1.32 (1.14–1.52)</b>
	≥31.77	Ref	Ref
Socio-economic status	Low	<b>1.20 (1.01–1.43)</b>	NS*
	High	Ref	

\*NS, Not significant.

Bold values are statistically significant at *P* value <0.05.

(Sankoh *et al.* 2001). Our study, in contrast, found that most high mortality villages tended to have consistently high mortality rates over a several year period.

The association between geospatial factors and child mortality can reflect several mechanisms of association. One such mechanism is geospatial constraints in access to health care. Access to health care is a cumulative effect of

factors including distance and cost, all of which can be influenced by geospatial factors (Hayes 1990). While we did not find differences in mortality related to proximity of residence to health facilities, we did find higher mortality among infants who lived further from public transport roads, which likely reflects their ability to reach healthcare services in a timelier manner. For older

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Cause of Death	Infant ( <i>n</i> = 1460)	Children 1–4 years ( <i>n</i> = 1494)	Overall ( <i>n</i> = 2954)
Malaria	334 (22.9%)	416 (26.7%)	750 (24.9%)
Pneumonia	305 (20.9%)	203 (13.3%)	508 (16.8%)
Gastroenteritis/Dehydration	233 (16.0%)	195 (12.5%)	428 (14.2%)
Anaemia	125 (8.6%)	210 (13.5%)	335 (11.1%)
HIV	82 (5.6%)	97 (6.2%)	179 (5.9%)
Sepsis	78 (5.3%)	2 (0.1%)	80 (2.7%)
Meningitis	77 (5.3%)	126 (8%)	203 (6.7%)
Pregnancy complications	68 (4.7%)	0 (0%)	68 (2.3%)
Malnutrition	38 (2.6%)	165 (10.6%)	203 (6.7%)
Tuberculosis	20 (1.4%)	46 (3%)	66 (2.2%)
Measles	17 (1.2%)	14 (0.9%)	31 (1%)
Others/Undetermined	83 (5.7%)	75 (5.0%)	158 (5.3%)

**Table 4** Cause of death from verbal autopsy among 2954 childhood deaths, western Kenya Health and Demographic Surveillance System (HDSS), 1 May 2002–31 December 2005

children, we found higher mortality in the northern part of the HDSS, which generally has rougher terrain and fewer roads, and in more sparsely populated areas, which might also reflect a more remote location with worse access to health care. Increasing distance to health facility is associated with higher child mortality (Binka *et al.* 1998; Becher *et al.* 2004; Kazembe *et al.* 2006). Besides this association with mortality, the so-called distance decay effect in clinic attendance has been well-established, whereby attendance at health facilities in rural developing country settings falls with increasing distance of residence from the facility (Feikin *et al.* 2008).

Besides variability in access to care, geospatial factors can determine exposure and disease transmission dynamics, leading to variability in disease burden. The most studied example of this type of mechanism is the association between geospatial factors and malaria prevalence (Thomson *et al.* 1999; Snow *et al.* 2004; Kazembe *et al.* 2006; Silué *et al.* 2008). In the Gambia, malaria prevalence was higher in locations with denser vegetation, suggestive of moisture environments more likely to support larval habitats (Thomson *et al.* 1999). Other studies have also demonstrated variability in local larval habitat prevalence based on drainage patterns and type of aquatic vegetation (Mwangangia *et al.* 2007; Shililu *et al.* 2007). The geospatial distribution of malaria infection in turn has been shown to be correlated with child mortality (Binka *et al.* 1998; Root 1999; Gemperli *et al.* 2004; Snow *et al.* 2004; Kazembe *et al.* 2007). In our study, we found increased mortality related to distance to streams. Proximity to streams is associated with larval habitat, particularly in the dry season when residual pools might serve as reservoirs for larvae (Shililu *et al.* 2007). We showed among infants that malaria deaths were more likely among those living closer to streams. This finding might have been limited by the low specificity of verbal autopsies for malaria deaths in

children, particularly where malaria and pneumonia have significant clinical overlap (English *et al.* 1996). The association between higher mortality and living at certain elevations and in the northern part of the HDSS in Gem might also be related to spatial variability in mosquito larval habitat formation. (Gemperli *et al.* 2004). More entomological work needs to be carried out to confirm this relationship.

Some of the village variability in mortality could be attributed to differences in ITN coverage by village. However, it is unlikely that ITN coverage varied by village as free ITNs were distributed through the entire KEMRI/CDC DSS throughout this time period as part of the DSS project. This ended in 2007 when ITNs became readily available through other sources in the area. After the scale-up of ITNs with the ITN efficacy trial in the late 1990s, entomologic inoculation rate (EIR) fell from 300 to 7 infective bites per year in 2002, and infant mortality declined by 25% (Phillips-Howard *et al.* 2003; Adazu *et al.* 2005). Despite the considerable impact of ITNs, malaria remains a major cause of morbidity and mortality in this area. This is not unexpected, as ITNs alone are unlikely to reduce EIR to a level required to substantially reduce malaria prevalence, which has been estimated at <1 infective bite per year (Beier *et al.* 1999). In the DSS in 2003, 60% of children <5 years of age included in a random community sample had malaria parasites in their blood, despite high ITN coverage in the area (KEMRI/CDC unpublished data).

The burden of other diseases besides malaria might also be influenced by geospatial factors. Diarrhoea prevalence, for example, is affected by local sources of drinking water, particularly in areas where few households treat their drinking water, such as our study area (Adazu *et al.* 2005). We found that deaths from gastroenteritis occurred more frequently in children living closer to streams. We found

that more children living closer to streams used unprotected water sources, mostly the stream itself. Unprotected water sources have been associated with increased child mortality in other studies (Binka *et al.* 1995; Hoque *et al.* 1999). Another disease that might vary geographically is HIV infection, which has been shown to be more prevalent near paved roads and local trading centres (Arroyo *et al.* 2006). We did not find that HIV diagnosis on verbal autopsy varied by village mortality quintile, although verbal autopsies have limitations in diagnosing paediatric HIV infection (Dowell *et al.* 1993). Although we did not have HIV prevalence for every village in our study area, in one area of Asembo where home-based HIV testing took place in 33 villages in 2008, the village prevalence ranged from 9% to 26% among adults  $\geq 18$  years, suggesting that variability in paediatric HIV prevalence might have influenced village-level mortality (KEMRI/CDC unpublished data). Further work needs to be carried out to explore the association between village HIV prevalence and child mortality rates.

Several studies investigated the effect of an area's population density on child mortality. One study showed that an increased population density in rural Zimbabwe led to higher rates of child mortality (Root 1997). The author postulated that this might be attributed to greater transmissibility of infectious diseases between people. The transmissibility hypothesis might also be supported by the finding of higher mortality in urban than rural Tanzania (Mturi & Curtis 1995). In contrast, other studies in Africa have shown excess mortality in less densely populated rural settings (Gemperli *et al.* 2004; Kazembe *et al.* 2007). While all of our HDSS area is considered rural, we found an increase in child mortality in more sparsely populated areas. The reason for this is unclear, but it might be attributed to issues related to poorer access to care and fewer health facilities not captured by straight-line distance variables, less infrastructure, and abundant larval habitats for *Anopheles* mosquitoes or other disease exposure differentials that we were unable to measure.

Our study had several limitations. We did not employ specific statistical methods to evaluate spatial clustering (Sankoh *et al.* 2001). Therefore, our demonstration of variability in mortality by village could have been attributed to random variation rather than true mortality differences. Specific spatial statistical modelling methods, such as Bayesian geostatistical models, might have been more appropriate to evaluate geospatial risk factors than Poisson regression (Gosoni *et al.* 2008). We did, however, control for clustering at the compound level in our model. Second, using villages as the unit of measure might be an artificial spatial demarcation in itself because many villages are large and spatially heterogeneous. Third, we used straight-line

distances in our analysis; straight-line distances might be less reflective of true travel time than would be an assessment of walking time or transport fare. Fourth, we included neonatal deaths in our infant analysis, as the distinct causes of neonatal mortality might have different geospatial risk factors than deaths among infants 1–11 months old; however, 81% of infant deaths were not neonatal, so the analysis of geospatial risk factors for infants is likely not biased by a preponderance of neonatal deaths.

Despite these limitations, our data provide evidence that a significant association between certain geospatial factors and child mortality exists at a local level. While such geospatial variability in mortality will likely not affect the implementation of large-scale interventions, such as introduction of new childhood vaccines, it might affect how local public health interventions, such as community-level initiatives by community health workers and mosquito larval habitat reduction, are employed (Jones *et al.* 2003). Directed interventions at higher risk populations in the context of limited financial and human resources have been suggested before as a legitimate strategy (Benzler & Sauerborn 1998; Sankoh *et al.* 2001). Evaluation of local geospatial risk factors for child mortality may be an important method for identifying the final barriers to reaching the 4th Millennium Development Goal.

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