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Household wealth inequalities in childhood anaemia and the role of malaria parasitaemia: a repeated cross-sectional analysis of Nigerian surveys, 2010–2021

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Abstract

Background: Socioeconomic differences in childhood anaemia may reflect malaria exposure alongside nutritional, infectious and social mechanisms. We estimated the component of the wealth-associated anaemia difference compatible with mediation through microscopy-confirmed parasitaemia in Nigeria.

Methods: We analysed de facto children aged 6–59 months from the 2010, 2015 and 2021 Nigeria Malaria Indicator Surveys. Within each round, the poorest household wealth quintile was compared with the richer and richest quintiles combined. Survey-weighted logistic models and interventional g-computation estimated total, direct and parasitaemia-mediated risk differences, adjusting for prespecified demographic and contextual variables. The primary analysis used 20 chained-equation imputations and 1,000 survey-bootstrap replicates distributed across imputations.

Results: The imputed analysis included 3,307 children in 2010, 3,643 in 2015 and 6,572 in 2021. The total anaemia risk difference was 11.0 percentage points (95% CI 4.1–17.9) in 2015 and 12.5 (8.4–16.6) in 2021; parasitaemia-mediated indirect differences were 3.4 (1.4–5.4) and 2.0 (1.1–2.9), respectively. The 2010 total-effect interval included zero. Proportion-mediated estimates were withheld in 2010 and 2015 because the total-effect denominators were unstable.

Conclusions: In 2015 and 2021, measured parasitaemia accounted for a smaller modelled component of the wealth-associated difference in childhood anaemia. These cross-sectional estimates depend on unverified temporal and confounding assumptions and cannot quantify the anaemia burden preventable through malaria control. Integrated approaches to child anaemia remain relevant.

Keywords: malaria; anaemia; socioeconomic inequalities; mediation analysis; microscopy; Nigeria; Demographic and Health Surveys; child health

Background

Malaria and anaemia commonly co-occur among young children in Nigeria, with substantial geographic and socioeconomic variation. Earlier Nigerian work has modelled their joint spatial distribution, but it did not estimate an exposure-specific pathway from household wealth through laboratory-confirmed parasitaemia to anaemia [1]. A recent systematic review found that formal mediation analyses of socioeconomic disparities in malaria are uncommon and often limited by cross-sectional measurement, incomplete confounding control, and inadequate handling of exposure-mediator interaction [2].

Household wealth may influence malaria risk through housing, prevention, access to care, and other pathways. Malaria parasitaemia can in turn contribute to anaemia, but nutritional deficiencies, helminths, inherited haemoglobin disorders, and other causes mean that malaria cannot account for all anaemia. The study objective was to quantify standardized interventional total, direct, and parasitaemia-mediated effects of the poorest household wealth category on child anaemia, separately in each NMIS round.

The disease-control relevance is whether the observed social gradient in anaemia can be understood solely as a malaria-linked disparity or whether the measured parasite pathway represents only one component. This distinction may help frame integrated malaria and child-health research, although the present analysis does not estimate an intervention effect.

Recent Nigerian analyses continue to show marked heterogeneity in childhood malaria risk by place, household circumstances, prevention coverage, and season [3]. National trend work found that reductions in infection were not uniform across states and zones, and recent analyses of the 2021

survey have likewise identified geographically patterned risk [4,5,6]. Studies of care seeking, antimalarial use, malaria knowledge, seasonal chemoprevention, malaria communication, vaccine readiness, parasite clearance, and programme delivery also illustrate that the social conditions surrounding exposure and response do not reduce to a single household asset score [7-19]. The present study therefore does not treat wealth as a manipulable intervention. It treats wealth as a summary exposure whose association with anaemia may operate through several pathways.

A mediation analysis adds information only if the causal question and the resulting assumptions are explicit. We chose microscopy-confirmed parasitaemia as a biologically proximal mediator, rather than using reported fever or treatment. That choice is supported by contemporary malaria research in Nigeria and elsewhere, but it also narrows the interpretation: a negative microscopy result does not exclude recent infection, and a positive test is not a complete measure of cumulative malaria burden [5,6,20-24]. We estimate an interventional indirect effect through the measured parasite status, not the proportion of anaemia biologically caused by malaria.

The primary objective was to estimate, within each survey round, the difference in anaemia risk had the observed study population experienced the mediator distribution expected under the poorest versus the comparison wealth categories. Secondary objectives were to compare the resulting estimates with complete-case analyses and to describe temporal changes in the total, direct, and indirect components. We report the work as an observational, design-based analysis in accordance with STROBE and current guidance for reporting mediation analyses [25,26].

We estimated the interventional contrasts separately in each survey rather than pooling survey years: wealth quintiles and the prevalence of malaria, anaemia and measured covariates differ across rounds. These are repeated cross-sectional comparisons, not within-child trajectories or a direct evaluation of malaria programmes.

Methods

Study design, data sources and participants

This repeated cross-sectional study used the nationally representative 2010, 2015 and 2021 Nigeria Malaria Indicator Surveys [27]. The source records were de facto household members aged 6–59 months in the household-member (PR) recode. Sampling weights (HV005/1,000,000), primary

sampling units (HV021) and strata (HV023) were retained; geopolitical zones in 2021 used SHREGION rather than the state variable HV024. The survey years were analysed separately. The analysis flow is in Additional file 1, Table S1. The authors accessed the DHS datasets for research purposes on 15 September 2026 and did not have access to direct identifiers.

Exposure, mediator, outcome and covariates

The exposure contrasted the poorest wealth quintile with the richer and richest quintiles combined, excluding the poorer and middle quintiles from this predefined analytic contrast. This maximised separation between the low-wealth group and two relatively advantaged categories but does not describe the full five-quintile gradient. Anaemia was defined using altitude-adjusted HC57 categories 1–3; the mediator was microscopy-confirmed parasitaemia (HML32=1). Diagnostic recodes were restricted to valid values, and unavailable or unknown responses were not treated as negative results. Covariates included child age (natural cubic spline), sex, maternal education, urban residence, six geopolitical zones, household size, rainy-season interview and altitude (natural cubic spline). ITN use was treated as a potential post-wealth pathway variable, not as a baseline confounder for the primary single-mediator contrast.

Causal structure and target estimands

The conceptual directed acyclic graph (Additional file 1, Figure S1) records hypothesised relationships among geographic and seasonal context, wealth, parasitaemia and anaemia. The primary outcome model includes the wealth-by-parasitaemia interaction. Standardised risks were averaged across the analytic covariate distribution. On the risk-difference scale, the reported total contrast is partitioned into interventional direct and indirect contrasts using the modelled mediator distribution under each wealth level. These are formal stochastic-mediator contrasts, not effects of a well-defined intervention that assigns household wealth. Identification would require consistency, positivity, correct model specification and sufficient control of exposure–outcome, exposure–mediator and mediator–outcome confounding. The simultaneous measurement of microscopy and haemoglobin limits temporal interpretation.

Statistical analyses, missing data and uncertainty

Weighted logistic models were fitted separately by survey round for parasitaemia and anaemia. Interventional g-computation yielded standardised total, direct and indirect risk differences (percentage points) and risk ratios. The primary analysis used 20 survey-specific chained-equation imputations; microscopy, rapid diagnostic testing, altitude-adjusted haemoglobin, ITN use and household net ownership were included in the imputation procedure, with anaemia passively derived from haemoglobin. Age, sex, education, residence, zone, household composition, season, altitude, housing and available cluster-level biomarker summaries informed the imputation models. The primary analysis was compared with complete-case estimates. The complete-case and imputed analytic sample denominators are distinct (Additional file 1, Tables S1–S5).

Uncertainty was calculated from 1,000 resamples of primary sampling units within strata, allocated as 50 survey-bootstrap replicates per imputation, with within- and between-imputation variances combined using Rubin's rules [31–33]. The fitted models and standardised quantities were recalculated in each replicate rather than resampling the final estimates. Ninety-five percent confidence intervals are reported without null-hypothesis p-values for mediation contrasts. The proportion mediated was withheld where its total-effect denominator failed the stability checks (2010 and 2015). Raw-code checks, imputation diagnostics and complete-case comparisons are available in Additional file 1. No prospective sample-size calculation was performed because all eligible survey records were analysed.

Results

The primary imputed analysis included 3,307, 3,643 and 6,572 eligible children in 2010, 2015 and 2021, respectively. Corresponding complete-case g-computation sample sizes were 2,965, 3,299 and 6,275 (Additional file 1, Table S1). Table 1 describes the complete-case distribution across the displayed wealth groups, whereas primary mediation estimates use the imputed sample. In 2021, weighted anaemia prevalence was 79.9% in the poorest quintile and 54.0% in the richest; microscopy positivity was 31.3% and 5.3%, respectively (Table 1).

In 2010, the standardised total anaemia risk difference was 1.5 percentage points (95% CI −6.3 to 9.3), and the parasitaemia-mediated indirect difference was 1.9 (0.5–3.4) (Table 2; Fig. 1). The total-effect interval crossed zero, and the proportion-mediated ratio was not reported.

In 2015, the total risk difference was 11.0 percentage points (95% CI 4.1–17.9), with direct and indirect differences of 7.6 (0.6–14.7) and 3.4 (1.4–5.4), respectively. In 2021, corresponding estimates were 12.5 (8.4–16.6), 10.5 (6.4–14.6) and 2.0 (1.1–2.9) percentage points (Table 2; Fig. 1). The 2021 proportion mediated was 16.2% (95% CI 7.0–25.3%; Fig. 2). The 2015 proportion-mediated ratio was withheld because one of the 1,000 bootstrap replicates yielded a near-zero total-effect denominator with sign reversal.

Complete-case estimates were broadly consistent with the primary estimates for 2015 and 2021: total risk differences were 10.8 versus 11.0 percentage points in 2015 and 12.4 versus 12.5 in 2021 (complete case versus imputation). Indirect differences were 3.1 versus 3.4 and approximately 2.0 under both approaches (Additional file 1, Table S5 and Figure S2). Microscopy gradients between the poorest and richest groups were 49.4% versus 18.6% in 2010, 43.2% versus 4.3% in 2015, and 31.3% versus 5.3% in 2021 (Table 1; Additional file 1, Table S6).

The secondary joint ITN–parasitaemia contrasts are shown in Additional file 1, Table S8 and Figure S4. These contrasts do not identify a unique sequential ITN-to-parasitaemia pathway. Supplementary Tables S2, S3 and S7 and Figure S3 report missingness, imputation and survey-recode diagnostics.

Discussion

In two of three survey rounds, the poorest-to-richer/richest household wealth contrast was associated with higher standardised anaemia risk, and the measured microscopy pathway represented a smaller positive component. The 2010 overall difference was imprecise. These findings provide a descriptive decomposition of an important socioeconomic association rather than evidence that a particular fraction of anaemia could be prevented by reducing malaria infection.

The distinction is relevant for malaria programmes because anaemia in childhood is multifactorial. Household wealth can affect infection risk through housing, prevention and access to treatment, while nutritional deficiencies, helminth infections, maternal anaemia and inherited haemoglobin disorders

can contribute through other pathways. The direct component in the fitted mediation decomposition encompasses mechanisms outside the single measured mediator and should not be equated with a single biological or policy pathway [2,20]. This supports evaluating malaria control in conjunction with broader child-health and nutrition strategies rather than inferring that malaria interventions alone can close the observed anaemia gradient.

The analyses complement earlier work on geographically heterogeneous malaria transmission in Nigeria [4–6], but cannot directly evaluate national programme effectiveness, intervention coverage or changes since the 2021 survey. Reported malaria positivity is a snapshot of patent infection, not cumulative exposure or a complete measure of malaria-related haemolysis. Comparisons across survey years are descriptive because transmission, survey implementation and socioeconomic group composition differ; the larger total contrast in 2021 should not be interpreted as a causal increase in the effect of poverty.

The primary limitations are temporal ambiguity and potential unmeasured confounding. Parasitaemia and haemoglobin were measured contemporaneously. Dietary intake, recent infection and treatment, helminth burden, inherited haemoglobinopathy, and fine-scale mosquito exposure were not harmonised as pre-mediator covariates. Exposure-induced confounders of the mediator–outcome relationship are plausible, and the wealth contrast itself is not a well-defined manipulation. Selection into biomarker testing and the missing-at-random assumptions of the imputation model could affect estimates. Bootstrap intervals capture specified sampling and imputation variability but cannot quantify uncertainty about these causal assumptions.

Strengths include three nationally representative surveys, a laboratory-based malaria measure, explicit allowance for wealth–mediator interaction, survey-aware uncertainty procedures, a prespecified covariate set and publicly documented missing-data diagnostics. Suppression of the unstable 2010 and 2015 ratio-based mediation summaries prevents a misleading interpretation of extreme bootstrap values. The analysis remains hypothesis-generating rather than a substitute for longitudinal infection, haemoglobin, nutrition and treatment data.

Conclusions

Microscopy-confirmed parasitaemia was compatible with a modest component of the wealth-associated childhood anaemia difference in the 2015 and 2021 Nigerian surveys. The findings should guide further investigation of malaria-linked and non-malaria pathways, not predictions of effects from changing household wealth or malaria-control policy. Reducing childhood anaemia inequities likely requires attention to both malaria and its nutritional, infectious and social co-determinants.

List of abbreviations

DAG, directed acyclic graph; DHS, Demographic and Health Surveys; Hb, haemoglobin; HC57, DHS anaemia category variable; ITN, insecticide-treated net; MI, multiple imputation; NMIS, Nigeria Malaria Indicator Survey; PR, household-member recode; PSU, primary sampling unit; RDT, rapid diagnostic test; RR, risk ratio.

Declarations

Ethics approval and consent to participate

The study analysed de-identified secondary data from the 2010, 2015 and 2021 Nigeria Malaria Indicator Surveys (NMIS). The 2010 survey protocol was reviewed by the National Health Research Ethics Committee of Nigeria; the 2015 and 2021 survey protocols were reviewed by the Nigerian National Health Research Ethics Committee and the ICF Institutional Review Board, as documented in the survey materials. Parents or guardians provided informed consent for children's participation and biomarker testing during original data collection. The investigators obtained authorised access to anonymised DHS survey datasets; no participants were contacted for this secondary analysis and no direct identifiers were provided to the investigators.

Consent for publication

Not applicable; no individual participant details are reported.

Availability of data and materials

The datasets analysed are the 2010, 2015 and 2021 Nigeria Malaria Indicator Surveys, which are made available to registered researchers by the DHS Program after an approved data-access request (https://dhsprogram.com/data/available-datasets.cfm). The participant-level data cannot be redistributed by the authors under the DHS Program access conditions. Eligible researchers may

request access to the same survey recodes directly from the DHS Program. Study-specific derived estimates, participant-flow information, variable definitions, missingness and imputation diagnostics, and sensitivity analyses are presented in the manuscript and Additional file 1.

Competing interests

The authors declare that they have no competing interests.

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The authors received no specific funding for this work.

Authors' contributions

KRF conceptualised the study, developed the study logic and analytical plan, designed the study and performed the data analysis. KRF, AA and TL contributed to manuscript preparation and discussion of the findings. AA contributed to the literature review. KRF, AA and TL read and approved the final manuscript.

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Additional files

Additional file 1 (.docx). Supplementary methods, Tables S1–S8 and Figures S1–S4, including the conceptual directed acyclic graph, participant flow, missingness and imputation diagnostics, complete-case sensitivity analyses, raw-code checks and exploratory joint-mediator contrasts.

Tables

Table 1. Descriptive anaemia, microscopy and net-use prevalence by household wealth and survey round.

Unweighted n is the complete-case analytic denominator for the displayed wealth group. Weighted prevalence estimates (%) are not the primary multiple-imputation standardised contrasts. The poorer and middle wealth categories are excluded from the prespecified comparison.

Year	Wealth group	n	Anaemia %	Microscopy %	ITN use %
2010	Poorest	917	71.7	49.4	33.4

Year	Wealth group	n	Anaemia %	Microscopy %	ITN use %
2010	Richer	1116	73.4	36.5	22.8
2010	Richest	932	65.0	18.6	17.6
2015	Poorest	1044	80.2	43.2	59.2
2015	Richer	1186	60.2	16.8	32.2
2015	Richest	1069	48.4	4.3	26.1
2021	Poorest	2178	79.9	31.3	44.7
2021	Richer	2175	63.2	14.6	37.1
2021	Richest	1922	54.0	5.3	28.1

Table 2. Standardised interventional risk contrasts through microscopy-confirmed parasitaemia by survey year.

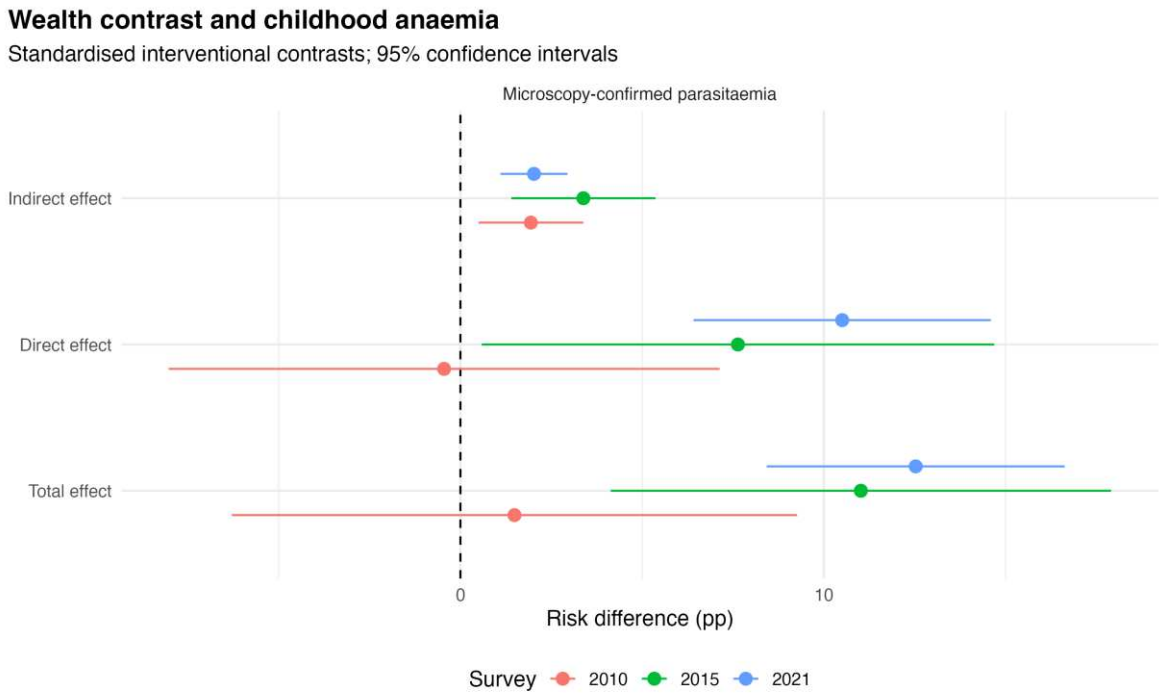
Primary multiple-imputation estimates; risk differences are percentage points (pp), risk ratios unitless, and the 2021 proportion mediated is a percentage. TE, total effect; IDE, interventional direct effect; IIE, interventional indirect effect; PM, proportion mediated. PM in 2010 and 2015 was deliberately withheld because the denominator-stability criterion failed.

Year	Quantity	Estimate	95% CI
2010	TE_rd	1.49	-6.29 to 9.26
2010	IDE_rd	-0.45	-8.03 to 7.13
2010	IIE_rd	1.94	0.50 to 3.38
2010	PM	Withheld	—
2010	TE_rr	1.02	0.91 to 1.13
2010	IIE_rr	1.03	1.01 to 1.05
2015	TE_rd	11.02	4.13 to 17.90
2015	IDE_rd	7.64	0.58 to 14.69
2015	IIE_rd	3.38	1.40 to 5.37
2015	PM	Withheld	—
2015	TE_rr	1.18	1.06 to 1.30
2015	IIE_rr	1.05	1.02 to 1.08
2021	TE_rd	12.53	8.43 to 16.63
2021	IDE_rd	10.50	6.41 to 14.59
2021	IIE_rd	2.02	1.10 to 2.95
2021	PM	16.17	7.00 to 25.34
2021	TE_rr	1.20	1.13 to 1.27

Year	Quantity	Estimate	95% CI
2021	IIE_rr	1.03	1.01 to 1.04

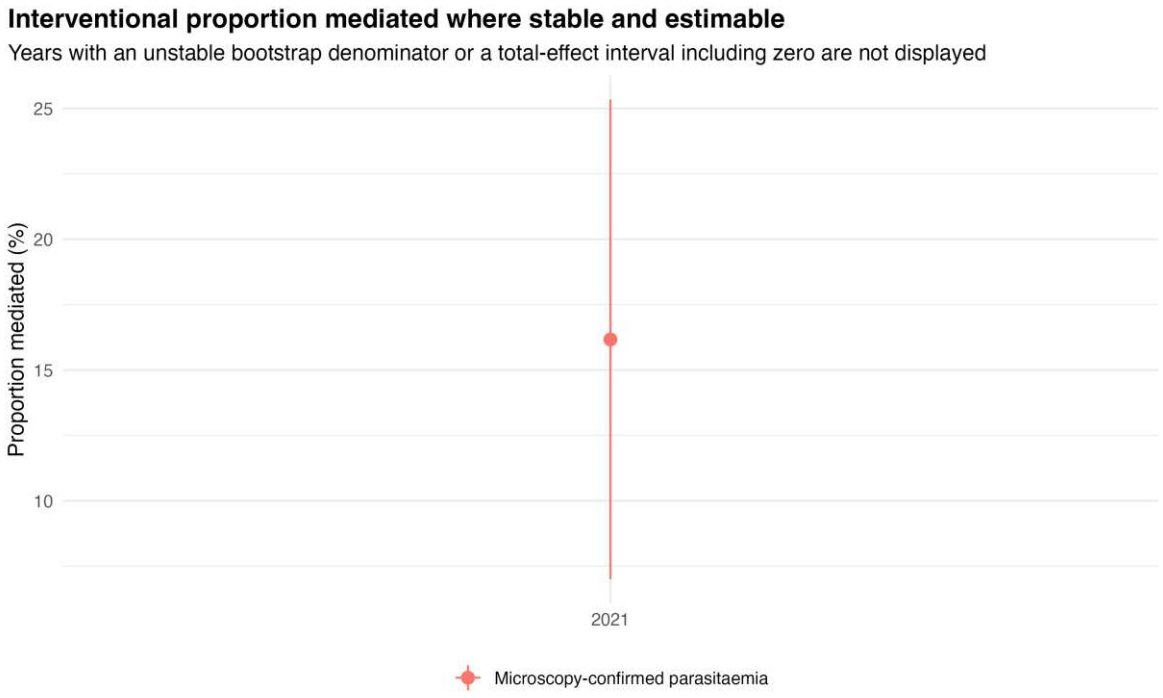
Figure legends

Fig. 1. Standardised total, direct and parasitaemia-mediated anaemia risk differences by survey year.



Point estimates and 95% confidence intervals from the primary multiple-imputation analysis; risk differences are percentage points. The graph is descriptive and does not identify a causal intervention on wealth.

Fig. 2. Proportion mediated through microscopy-confirmed parasitaemia in 2021.



Multiple-imputation estimate with 95% confidence interval. Ratios for 2010 and 2015 are withheld because unstable total-effect denominators make them non-interpretable.

References

1. Adebayo SB, Gayawan E, Heumann C, Seiler C. Joint modeling of anaemia and malaria in children under five in Nigeria. Spat Spatiotemporal Epidemiol. 2016;17:105-15. doi:10.1016/j.sste.2016.04.011.

2. Wafula ST, Habermann T, Franke MA, May J, Puradiredja DI, Lorenz E, et al. What are the pathways between poverty and malaria in sub-Saharan Africa? A systematic review of mediation studies. Infect Dis Poverty. 2023;12:58. doi:10.1186/s40249-023-01110-2.

3. World Health Organization. World malaria report 2025. Geneva: World Health Organization; 2025.

4. Oyibo W, Ntadom G, Uhomoibhi P, Oresanya O, Ogbulafor N, Ajumobi O, et al. Geographical and temporal variation in reduction of malaria infection among children under 5 years of age throughout Nigeria. BMJ Glob Health. 2021;6:e004250. doi:10.1136/bmjgh-2020-004250.

5. Adedun OA, et al. Geography and drivers of pediatric malaria in Nigeria. J Parasit Dis. 2026. doi:10.1007/s12639-025-01864-6.

6. Takramah W, et al. Geostatistical modelling and web-based mapping of malaria risks among children under five years in Nigeria: evidence from the 2021 Nigeria Malaria Indicator Survey. Health Sci Rep. 2026;9:e73006. doi:10.1002/hsr2.73006.

7. Olisakwe SC, Thwing J, Dionne JA, Irvin R, Kachur PS, Bruxvoort KJ. Receipt of antimalarials among children aged 6-59 months in Nigeria from 2010 to 2021. Malar J. 2024;23:249. doi:10.1186/s12936-024-05075-x.

8. Boulton C, et al. Understanding barriers to effective malaria diagnosis and treatment: a mixed-methods study in Uganda, Nigeria, and Ivory Coast. PLOS Glob Public Health. 2026;6:e0006921. doi:10.1371/journal.pgph.0006921.

9. Adeleke MA, et al. Patterns and determinants of antimalarial drug use and treatment adherence in Osun State, Southwestern Nigeria: a cross-sectional study. BMJ Glob Health. 2026;11. doi:10.1136/bmjgh-2025-023280.

10. Aremu O, et al. Predictors of optimal uptake of intermittent preventive treatment in pregnancy in Nigeria: survey-weighted regression and exploratory machine-learning analysis of the 2021 Nigeria Malaria Indicator Survey. Niger Med J. 2026;67:1801-18. doi:10.71480/nmj.v67i5.1537.

11. Oga EA, et al. Epidemiology of Plasmodium infection among acute febrile illness cases in two Nigerian regions. Clin Infect Dis. 2025. doi:10.1093/cid/ciaf468.

12. Nnaji CA, et al. Plausibility evaluation of seasonal malaria chemoprevention in Nigeria. Malar J. 2025;24. doi:10.1186/s12936-025-05604-2.

13. Ajayi IO, et al. Care-seeking behaviour of caregivers of children with suspected malaria in southwest Nigeria. Malar J. 2025;24. doi:10.1186/s12936-025-05433-3.

14. Esan DT, et al. Malaria knowledge and prevention practices among caregivers in Nigeria. Sci Rep. 2025;15. doi:10.1038/s41598-025-22713-9.

15. Tibenderana JK, et al. Acceptability of seasonal malaria chemoprevention in Osun State, Nigeria. Malar J. 2025;24. doi:10.1186/s12936-025-05734-7.

16. Ajaero CK, et al. Access to malaria messaging and mosquito nets in West Africa. BMC Public Health. 2025;25. doi:10.1186/s12889-025-25270-y.

17. Bazant ES, et al. Cost analysis of seasonal malaria chemoprevention with and without vitamin A supplementation for children under five years in Nigeria. PLoS One. 2025;20:e0315655. doi:10.1371/journal.pone.0315655.

18. Awotunde TA, et al. Readiness for RTS,S and R21 malaria vaccine rollout in Nigeria: a national survey. BMC Infect Dis. 2025;25. doi:10.1186/s12879-025-11760-2.

19. Akande EO, et al. Parasite clearance after artemisinin-based combination therapy among Nigerian children: a randomized trial. BMC Infect Dis. 2025;25. doi:10.1186/s12879-025-11704-w.

20. Owusu Sarfo J, Amoadu M, Kordorwu PY, Adams AK, Gyan TB, Osman AG, et al. Malaria amongst children under five in sub-Saharan Africa: a scoping review of prevalence, risk factors and preventive interventions. Eur J Med Res. 2023;28:111. doi:10.1186/s40001-023-01046-1.

21. Khan S, Nwafor EC, Obehi IF, et al. Sociodemographic factors associated with malaria infection among children under five years in Nigeria. Niger Med J. 2025;66. doi:10.71480/nmj.v66i3.796.

22. Isah MB, et al. Asymptomatic malaria in Nigeria: a systematic review. Malar J. 2025;24. doi:10.1186/s12936-025-05671-5.

23. Yeom J, et al. Age-specific and common risk factors for Plasmodium infection among children under five in Nigeria. PLOS Glob Public Health. 2026;6. doi:10.1371/journal.pgph.0006693.

24. Addo IY, et al. Weather and malaria among children under five years in sub-Saharan Africa. PLoS One. 2025;20:e0335031. doi:10.1371/journal.pone.0335031.

25. Lee H, Cashin AG, Lamb SE, Hopewell S, Vansteelandt S, VanderWeele TJ, et al. A guideline for reporting mediation analyses of randomized trials and observational studies: the AGReMA statement. JAMA. 2021;326:1045-56. doi:10.1001/jama.2021.14075.

26. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP. The Strengthening the Reporting of Observational Studies in Epidemiology statement: guidelines for reporting observational studies. PLoS Med. 2007;4:e296. doi:10.1371/journal.pmed.0040296.

27. National Population Commission, National Malaria Elimination Programme, National Bureau of Statistics, and ICF. Nigeria Malaria Indicator Survey 2010, 2015, and 2021 final reports. Abuja, Nigeria

and Rockville, Maryland: NPC, NMEP, NBS, and ICF. DHS Program data access portal: https://dhsprogram.com/data/available-datasets.cfm. Accessed 13 November 2026.

28. Williamson EJ, Aitken Z, Lawrie J, Dharmage SC, Burgess JA, Forbes AB. Introduction to causal diagrams for confounder selection. Respirology. 2021;26:886-92. doi:10.1111/resp.14101.

29. VanderWeele TJ, Vansteelandt S. Mediation analysis with multiple mediators. Epidemiol Methods. 2014;2:95-115. doi:10.1515/em-2012-0010.

30. Valeri L, VanderWeele TJ. Mediation analysis allowing for exposure-mediator interactions and causal interpretation: theoretical assumptions and implementation with SAS and SPSS macros. Psychol Methods. 2013;18:137-50. doi:10.1037/a0031034.

31. Schomaker M, Heumann C. Bootstrap inference when using multiple imputation. Stat Med. 2018;37:2252-66. doi:10.1002/sim.7654.

32. White IR, Royston P, Wood AM. Multiple imputation using chained equations: issues and guidance for practice. Stat Med. 2011;30:377-99. doi:10.1002/sim.4067.

33. Sterne JAC, White IR, Carlin JB, Spratt M, Royston P, Kenward MG, et al. Multiple imputation for missing data in epidemiological and clinical research: potential and pitfalls. BMJ. 2009;338:b2393. doi:10.1136/bmj.b2393.

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