Cover photo: A Rohingya girl sits with her caregiver moments before venous blood collection © Jasbir Kaur

POLICY BRIEF

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Anaemia in the First Two Years of Life in Cox's Bazar: Why Early, Integrated Intervention Matters for Rohingya Children

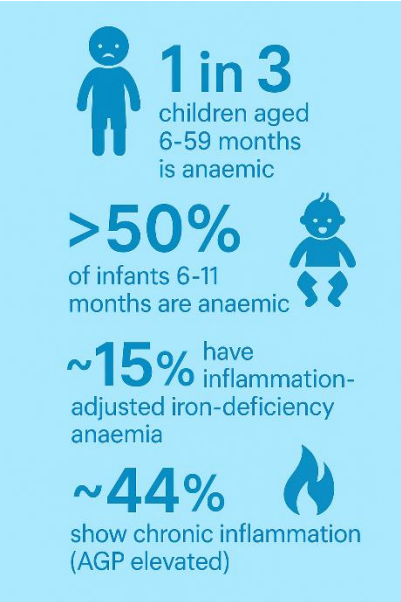
Anaemia remains one of the most persistent yet under-recognised threats to child survival and development in humanitarian settings. Among Rohingya children living in the FDMN camps in Cox's Bazar, anaemia is widespread, strongly age-patterned, and driven by overlapping nutritional and non-nutritional mechanisms that reinforce one another during early life. While severe anaemia is rare, the high prevalence of mild and moderate forms constitutes a silent public health emergency, increasing vulnerability to infection while impairing cognitive development, immune function, and physical growth during the most sensitive period of childhood..

> Anaemia is not the result of a single deficiency, but the biological imprint of cumulative early-life stress—nutritional, infectious, and environmental

This policy brief synthesises findings from a detailed causal analysis of anaemia among children aged 6–59 months and translates complex biological and epidemiological evidence into actionable priorities for UNICEF and partners. The analysis demonstrates that nutrition-specific interventions alone are insufficient to address the current burden. Meaningful and sustained reductions in childhood anaemia require **early, integrated strategies** that simultaneously address iron intake, recurrent infection, chronic inflammation, and the broader environmental and care-related conditions shaping child health in protracted displacement..

Key evidence at a glance

Approximately 32% of children aged 6–59 months are anaemic, with the burden heavily concentrated in early life. More than half of infants aged 6–11 months are affected, and children under two face more than a fourfold higher risk of anaemia compared to older children, even after accounting for household composition, sanitation, water access, and programme exposure. This pronounced age gradient is consistent across descriptive, bivariate, and multivariate analyses and confirms infancy and early toddlerhood as the critical window for effective prevention.



Heat map (below) shows the prevalence (%) of haemoglobin deficiency and anaemia severity among children aged 6–59 months in the Rohingya FDMN camps by age group. Darker (red) shading indicates higher prevalence. The figure highlights a strong age gradient, with

the highest burden of any anaemia, mild anaemia, and moderate anaemia concentrated among infants and young children (6–23 months), particularly those aged 6–11 months. Severe anaemia remains rare across all subgroups (green cells).

Anaemia	Overall	6-11 months	12-23 months	6-23 months	24-59 months
Haemoglobin deficiency (any anaemia)	32.0	55.0	42.0	45.0	27.0
Mild anaemia	22.2	37.6	25.9	28.5	19.9
Moderate anaemia	9.9	17.7	16.2	16.5	7.4
Severe anaemia	0.1	0.0	0.0	0.0	0.1
CRP	19.0	29.0	26.0	27.0	17.0
AGP	44.0	58.0	61.0	60.0	38.0

Iron deficiency contributes substantially but incompletely to the observed anaemia burden. After accounting for inflammation, approximately 15% of children meet criteria for iron-deficiency anaemia, rising to nearly one in four children aged 6–23 months. However, most anaemic children do not meet iron deficiency thresholds alone, indicating the presence of additional biological pathways. This distinction is crucial: anaemia in this setting cannot be understood, or addressed, solely as a micronutrient deficiency problem.

Markers of infection and inflammation are widespread and strongly age-patterned. Nearly half of all children have elevated AGP, with prevalence exceeding 60% among children under two. Red blood cell indices further show patterns consistent with inflammation-mediated suppression of erythropoiesis, including high rates of microcytosis, hypochromia, and elevated red cell distribution width in younger children. Multivariate models confirm that biological markers—low serum iron availability, abnormal red cell morphology, and reticulocyte response—are far stronger predictors of anaemia than household WASH access, food consumption scores, or programme participation once age is controlled for.

Why current approaches are not enough

Humanitarian anaemia programming has appropriately prioritised iron supplementation, micronutrient powders, fortified foods, and infant and young child feeding counselling. These interventions remain essential. However, the evidence clearly shows they address only part of the underlying problem. High burdens of infection and inflammation in early childhood reduce iron absorption, block iron utilisation through hepcidin-mediated pathways, and suppress red blood cell production. Inflammatory processes also elevate ferritin levels, masking true iron deficiency and complicating routine programme monitoring.

As a result, children may remain anaemic despite food assistance, supplementation, or

> Anaemia in Cox's Bazar is not a failure of feeding alone, it reflects the cumulative effects of early malnutrition, infection, and inflammation.

apparently adequate household food consumption. This explains why bivariate analyses show limited independent associations between anaemia and indicators such as WASH access, deworming coverage, or vaccination status once age is taken into account. These interventions are not ineffective; rather, their impact is indirect, cumulative, and biologically mediated over time. The multivariate findings make clear that isolated, input-driven approaches are insufficient in the face of early-life biological vulnerability.

Strategic implications for UNICEF and partners

The findings strongly reinforce and extend the UNICEF Nutrition Strategy's emphasis on the **first** 1,000 days, multi-sectoral programming, and action on the underlying determinants of undernutrition. Reducing childhood anaemia in Cox's Bazar is fundamentally a question of timing, integration, and biological realism.

Effective responses must begin earlier—during infancy and early complementary feeding—and must operate across nutrition, health, WASH, and infection prevention simultaneously. Anaemia should be understood not only as a nutrition outcome, but as a sentinel indicator of cumulative exposure to infection, inflammation, and constrained caregiving environments in early life. Programmes that fail to address this interplay risk limited impact, even when coverage targets are met.

What needs to change?

Humanitarian nutrition responses must evolve from predominantly input-focused delivery toward outcome-oriented, biologically informed strategies. This includes strengthening infant and young child feeding with specific attention to iron bioavailability; improving the continuity, dosage, and predictability of micronutrient provision; and intensifying infection prevention, case management, and environmental health interventions during the first two years of life.

Anaemia indicators should be interpreted alongside inflammation markers rather than in isolation, to avoid underestimating iron deficiency and misdirecting programme responses. Greater investment in routine biomarker-informed surveillance, integrated child health platforms, and analytic capacity is essential to guide adaptive, evidence-responsive programming in complex humanitarian settings.

Conclusion: an opportunity for impact

Anaemia among Rohingya children is both preventable and consequential. The evidence clearly shows that the heaviest burden falls within the very window where intervention yields the greatest lifetime returns. By aligning nutrition, health, and WASH programming around early childhood, and by grounding action in biological evidence rather than single-sector solutions, UNICEF and its partners have a powerful opportunity to reduce anaemia, strengthen child resilience, and protect long-term developmental potential in one of the world's most protracted humanitarian crises.