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School meals and non-communicable disease prevention across the life course

Evidence, mechanisms, and the conditions for impact

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Executive Summary

Non-communicable diseases (NCDs) are the leading cause of death worldwide, accounting for approximately 74% of all deaths globally—over 43 million annually. This reality is no longer confined to high-income countries: over 80% of premature NCD deaths (before age 70) now occur in low- and middle-income countries, where NCD mortality is rising faster than anywhere else in the world and where resources are the lowest to manage it. Diet is among the most powerful modifiable drivers of NCD risk, yet population diets have proven remarkably resistant to improvements. The central challenge for policymakers is identifying interventions that can reach the populations most at risk, early enough in the life course to alter long-term dietary patterns and health trajectories, through systems that already exist, are supported by public opinion and have the political backing to be sustained.

School meal programmes meet this challenge in ways that few other interventions can. They already reach 466 million children, supported by approximately US\$84 billion in annual investment. They operate at the intersection of nutrition, education, agriculture and food systems, social protection, and health—and reach children during a developmental window when dietary behaviours are being established and biological risk trajectories, which originate from conception, pregnancy and the first two years of life, are shaped further. Yet until now, the evidence on whether and how school meal programmes contribute to long-term NCD prevention has not been comprehensively synthesised.

This White Paper addresses that gap. Drawing on over 700 data sources and contributions from 115 experts in the field from 71 organisations and 46 countries, it presents the most comprehensive integrated assessment to date of the evidence linking school meal programmes to NCD prevention across the life course. The evidence is organised across four chapters, each examining one link in the causal chain connecting school meals to long-term NCD prevention.

The first establishes what school meals can deliver in the short term—improvements in dietary intake, micronutrient status, school participation, and household food security—the foundation without which any longer-term health benefit cannot occur. The second asks whether those immediate gains, where they occur, also ripple outward, shaping what children eat at home, among peers, and in their communities in ways that could establish lasting dietary habits. The third examines whether those behavioural changes translate into measurable improvements in the biological markers that predict adult disease—anthropometric outcomes, metabolic markers, mental wellbeing, and, where the evidence exists, NCD outcomes in adulthood. The fourth identifies the programme design, governance, and food environment conditions under which the benefits are most likely to be realised in practice. Following on these four evidence-based chapters, conclusions are offered to guide future research, funding, implementing, and policy agendas.

Where the evidence is most consistent, school meal programmes can improve dietary intake, diet quality and micronutrient status, with the largest gains in low- and middle-income settings where nutritional deficits are greatest. School meals also act as a reliable incentive for school participation, particularly in food-insecure regions with low participation at baseline.

Where the evidence is suggestive but more limited, school meal exposure may influence children's eating behaviour beyond the school meal itself. In a Danish trial, school meals providing about half of daily intake improved children's overall weekly diet, although behaviour outside school was not measured separately. Observational and qualitative studies suggest effects on what children request at home and on how families organise food provisioning.

The evidence on health outcomes is also more limited. Improvements in growth and body measurements (anthropometric outcomes), mainly height-for-age (stunting) and weight-for-age, are positive and reasonably consistent in middle-income settings, though much of the evidence is observational. These outcomes matter for NCD prevention in different ways: stunting at school age largely reflects undernutrition earlier in life, itself a risk factor for later NCDs, while overweight and obesity are risks that school meals, and the dietary patterns they shape, can still influence. Improvements in metabolic markers have been observed in only a small number of studies, mainly in Denmark, Thailand and Brazil, where meals met high nutritional standards. Evidence on mental wellbeing, inflammation and the gut microbiota is sparser still, and too inconsistent to draw firm conclusions.

Direct evidence on medium- and long-term NCD outcomes remains limited and covers only high- and upper-middle-income settings. Findings are mixed for body mass index (BMI), and are more consistently positive for self-reported health, but there is almost no empirical evidence on NCD mortality or morbidity, which may reflect the time over which chronic disease develops rather than an absence of effect. To explore the long-term question further, a comparative-risk scenario analysis was commissioned for this White Paper (Chapter 4, Box 4.3). It suggests that school meals that follow healthy diet principles and are delivered consistently could reduce diet-linked NCD mortality over the long run, but that the benefit would be modest, would emerge only after several decades, and would depend on how far the improvement in children's diets persists into adulthood and on the nutritional quality of the meals. In the model, menus high in sodium or red meat could offset these gains, which is an argument for binding nutritional standards rather than against provision.

One finding cuts across all four evidence chapters: meal quality matters for the size and direction of effects, not only whether meals are provided. Where meal composition is reported, programmes with binding nutritional standards or improved meal composition show stronger effects on dietary and anthropometric outcomes than provision without them. The independent contribution of meal quality cannot, however, be conclusively

established, because the studies reviewed often do not report meal composition in sufficient detail.

The evidence base has further limitations. It spans all country income groups, but much of it comes from high-income countries; studies address different aspects of meal quality and different outcomes; and the evidence is likely to over-represent programmes that were evaluated because an effect was expected. Addressing these gaps, and systematically documenting meal composition against defined nutritional standards and children's baseline dietary quality across programme contexts, are foundational priorities for the field.

This White Paper concludes that well-designed school meals, delivered at scale and supported by appropriate governance, quality standards and adequate budgets, are a meaningful and achievable upstream strategy for NCD prevention. Their benefits for children's diets and nutrition are evidenced in the short and intermediate term; their contribution to preventing NCDs in adulthood is plausible and supported by modelling, but not yet demonstrated directly. Investment in coverage without parallel investment in quality is unlikely to realise this potential. School meals are not only an education intervention — they are a support to agriculture, a health intervention, a social protection mechanism, an equity strategy. The evidence assembled provides a principled basis for treating them as such, and for acting on that basis without waiting for empirical confirmation that the timescales of chronic disease may never provide in full.

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1. Introduction

Non-communicable diseases (NCDs) now kill more people than all other causes combined, accounting for approximately 74% of all deaths globally—over 43 million deaths annually [1,2]. They also impose the greatest economic burden on health systems worldwide, with estimated costs exceeding US\$30 trillion between 2011 and 2030, representing 48% of global GDP in 2010 [3,4]. This reality is no longer confined to high-income countries: over 80% of premature NCD deaths (before age 70) now occur in low- and middle-income countries, where NCD mortality is rising faster than anywhere else in the world and where health systems are least equipped to manage the growing demands of chronic disease [1,2]. Despite overwhelming evidence linking diet to NCD risk [5,6], population diets globally have proven remarkably resistant to change in a healthier direction over recent decades, with suboptimal intake of fruit, vegetables, whole grains, and legumes remaining among the leading risk factors for NCD mortality worldwide [6]. The question facing policymakers is therefore not only what could be done, but how to implement the changes to food environments and food systems that are needed, and where to start.

The unequal global distribution of NCD risk sharpens this question. While NCDs are now the leading cause of death in nearly all world regions, the burden is concentrated among populations and countries with the fewest resources to manage it. Low- and middle-income countries, home to most of the world's population, bear over three-quarters of the global NCD burden despite accounting for a much smaller share of global health expenditure, and within these countries the risk falls most heavily on socioeconomically disadvantaged populations [7,8]. The main modifiable risk factors that drive NCDs are behavioural, including unhealthy diets, low physical activity, tobacco use and harmful use of alcohol, and metabolic, including overweight and obesity, elevated blood pressure, abnormal blood lipid levels that accelerate cardiovascular disease, and chronically elevated blood sugar (the defining feature of diabetes) [1]. These risk factors are themselves shaped by structural conditions that vary systematically by country income, by socioeconomic position within countries, and by geography [6,9]. Each of the

metabolic risk factors reflects a disruption in normal metabolic function that, without intervention, compounds over time: persistently high blood pressure strains the cardiovascular system; abnormal blood lipid levels drive arterial damage; and chronic high blood sugar underlies systemic organ deterioration. Unhealthy diets are among the primary drivers of all three, making early dietary intervention a key leverage point for NCD prevention across the life course.

Children in many low- and middle-income contexts arrive at school already affected by poverty, food insecurity, limited access to safe water and sanitation, inadequate housing, and early-life malnutrition [37]. These conditions do not simply co-exist with NCD risk—they actively contribute to it, by shaping the diets children eat, the environments they grow up in, alongside biological trajectories set in early life. Healthcare systems in these settings are also less equipped to manage the growing demands of chronic disease, making preventive strategies — and particularly those operating earlier in the life course — disproportionately important [10].

The NCD crisis is therefore not only a public health problem but a problem of equity. In many contexts, the behavioural risk factors that drive NCDs, such as unhealthy diets, are not freely chosen but are the predictable consequences of constrained circumstances. Preventive strategies that rely only on individual access to healthcare, specialised foods, or specific consumer goods risk bypassing precisely the populations most affected. Effective prevention must therefore also operate early and at the level of food environments. School meals constitute a strategic and controllable food environment for early intervention, providing repeated exposure to appropriate foods during childhood, a critical period for the development of dietary behaviours and preferences. Schools are also one of the few settings where the food environment can be shaped directly through policy, through nutritional standards, procurement rules and restrictions on the sale and marketing of less healthy foods [38,39].

1.1 The case for early intervention

A growing body of evidence suggests that dietary behaviours are shaped early in life and persist into adulthood (11–17). Longitudinal studies tracking dietary patterns across the life course have provided particularly clear evidence of this persistence. The Cardiovascular Risk in Young Finns Study, following participants from childhood into adulthood over 21 years, found that dietary patterns established in childhood remained largely consistent into adult life, with low fruit and vegetable consumption and high intake of energy-dense foods tracking across decades [15]. The Saskatchewan Pediatric Bone Mineral Accrual Study, following Canadian children over 20 years, similarly documented tracking of dietary patterns from childhood through adolescence into young adulthood [14]. Evidence from low- and middle-income contexts points in the same direction: the Birth to Twenty cohort study, following urban adolescents in Soweto,

South Africa, found that poor dietary habits including high consumption of energy-dense, micronutrient-poor foods—were well established by 13 years of age and persisted or worsened through adolescence [40].

The biological consequences of these dietary trajectories are observable from school age onwards. Poor diet quality in childhood contributes to excess adiposity typically, but not necessarily, showing as overweight and obesity, which in turn disrupts metabolic function by elevating blood lipids, impairing insulin sensitivity and raising blood pressure, which can set a biological trajectory towards NCDs in motion well before adulthood. Rapid BMI acceleration during the preschool years has been shown to substantially increase the risk of sustained obesity through adolescence [41]. Childhood adiposity and elevated blood pressure also appear to track into adulthood: the Cardiovascular Risk in Young Finns Study found that overweight or obese children were three to five times more likely to be obese as adults, and were at a substantially elevated risk of carotid intima-media thickening, hypertension, type 2 diabetes, and metabolic syndrome by their thirties [18]. Childhood blood pressure tracks into adulthood and childhood blood fat profiles and insulin resistance follow similar patterns [19,20]. Underweight, obesity, and the dietary risk factors that drive them are increasingly recognised as conditions associated with adverse health outcomes throughout the life course rather than at a single point in time [21]. This suggests that intervening during childhood, before unhealthy patterns become entrenched, may offer the greatest potential to alter long-term health trajectories. Intervention evidence is consistent with this: in the Finnish STRIP trial, dietary counselling from infancy to early adulthood was associated with lower saturated fat intake and more favourable cholesterol and glucose levels six years after the intervention ended [42]. Tracking and early-intervention evidence of this kind does not, however, show that changing school meals will change adult NCD outcomes; that link is examined in Chapter 4, where direct evidence remains limited.

Children in many parts of the world now face a dual burden of malnutrition, where undernutrition and overweight coexist within the same populations and increasingly within the same households [22]. In many settings this is now a triple burden, with micronutrient deficiencies alongside undernutrition and overweight: among adolescents in Dar es Salaam, for example, 16% were overweight, 39% anaemic, 23% stunted and 12% thin [43]. The global prevalence of obesity among children and adolescents aged 5–19 years rose from around 2% in 1990 to around 8% in 2022, with the burden of underweight remaining substantial in much of South Asia and parts of Africa [21]. These two forms of malnutrition are not simply parallel problems — early-life undernutrition, including low birthweight, stunting, and micronutrient deficiencies, has been shown to increase susceptibility to NCDs in later life, through mechanisms including altered metabolic programming, increased adiposity relative to lean mass, and heightened insulin

resistance [21]. Without effective intervention, the children carrying this dual burden today represent a future cohort of adults at elevated risk of NCDs.

This dual burden of malnutrition reflects a broader transition in the global food system, driven by rising incomes, urbanisation, the relative affordability of different foods, marketing and changing social norms as well as by availability [23]. In many low-income settings, diets remain dominated by staples because diverse, healthy diets are unaffordable for many households; healthy diets remain unaffordable for around a third of the world's population [44]. As incomes rise, foods and beverages high in fat, sugar or salt, such as sugar-sweetened beverages (SSBs) and energy-dense, nutrient-poor snacks, often become cheap, convenient and heavily marketed, and their growing availability and consumption has reshaped what children eat both at home and during the school day [23,24]. Schools sit at the centre of this transition: they represent critical potential platforms for shaping lifelong dietary habits, yet in practice they are frequently surrounded by food environments that systematically undermine healthy eating behaviours [25,26]. This occurs both through exposure to these foods from formal and informal vendors, and through the consequent displacement of whole grains, fruit, and vegetables from children's diets. Children's diets are also shaped by household resources, family practices, peers, marketing and social norms, which school meals can influence only in part.

Childhood represents a critical window of opportunity for dietary intervention as this is a period before eating behaviours become entrenched and increasingly resistant to change. That intervention needs to be operable: it must reach children at scale, be financially and politically feasible, and integrate with existing systems rather than require entirely new infrastructure. The challenge is not abstract but rather one of identifying interventions that can reach the children most at risk, at the developmental stages when intervention is most likely to translate into lifelong protection, through systems that already exist and have the political backing to be sustained over decades. While earlier intervention — from pregnancy, exclusive breastfeeding and complementary feeding onwards — would likely offer greater impact, reaching all children at risk is less practical at scale. This does not make the first 1,000 days a lower priority: interventions from pregnancy to age two remain essential, and school meals complement them by extending support across the next stages of childhood, starting from pre-school where programmes reach early childhood education. Schools provide an existing, institutionalised point of contact with nearly all children across this early, critical window, and where school meal programmes operate universally, which is not yet the case in many low-income countries, they offer a rare combination of population-wide reach and targeted benefit — serving all children while particularly improving outcomes for those facing the greatest nutritional risk. School meal programmes may therefore be one of the most operationally realistic platforms for improving the diets of school-age children at scale, alongside wider food environment policies.

1.2 School meals as part of the solution

School meals programmes are already firmly established on the policy agenda in about three quarters of countries. They reach at least 466 million children globally [27,28], supported by approximately US\$84 billion in annual investment in 2024 [28]. Because schooling is compulsory in most countries, schools reach most children for many hours each week during the years when food habits are forming. The issue may therefore not be whether to create entirely new systems, but rather how to strengthen and better align those already in place.

Their scale and reach are matched by their potential breadth of effect. Beyond delivering food during school hours, school meal programmes are increasingly recognised for their potential to drive local food system transformation through local, public food procurement that supports smallholder farmers' livelihoods, and to function as child-focused social protection mechanisms that reduce household food insecurity (29–31). This multi-sectoral potential is visible in programmes operating at very different scales and in very different contexts: Brazil's National School Feeding Programme (PNAE), for example, is constitutionally enshrined and legally requires at least 45% of food procurement to come from local family farmers, integrating nutrition, agriculture, and social protection within a single framework [32,33]; India's PM POSHAN, the world's largest school meal programme, serves hot meals to approximately 120 million children daily [34]; Japan's school lunch system is centrally regulated under a national food education framework that institutionally treats school mealtimes as educational environments [36]; and Finland has provided free school meals to all pupils since 1948, now required by law for every school day [45]. These examples illustrate that school meal programmes can be institutionally embedded at national scale across different income levels, governance structures, and cultural contexts — and that the political and financial commitment to sustain them over decades is achievable. They describe policy commitment and reach rather than demonstrated effects; the evidence on effects is reviewed in Chapters 2 to 5.

The case for school meals as a population-level NCD prevention strategy rests on their ability to reach large numbers of children on school days during the developmental window in which dietary behaviours and biological risk trajectories are being established. Yet the evidence on whether and how school meal programmes contribute to long-term NCD prevention has not been comprehensively synthesised. The question of whether school meals deliver immediate nutritional and educational benefits across income settings, whether these ripple into lasting behavioural change, translate into measurable improvements in biological markers, also into adulthood, and ultimately reduce the burden of diet-related NCDs across the life course — and the conditions under which they would do so — has remained largely unexamined as an integrated body of evidence. Because school feeding programmes continue to be implemented mainly as social

assistance and school retention policies, monitoring efforts tend to prioritise participation and educational indicators, with limited assessment of nutritional and health impacts, leaving important evidence gaps regarding their longer-term contribution to preventing diet-related NCDs. Synthesising this evidence across the full causal chain is the contribution this White Paper seeks to make.

1.3 The structure of this White Paper

Following this introductory chapter (Chapter 1), the White Paper builds its argument across four substantive evidence chapters that each assess one component of the causal logic through which school meals might contribute to NCD prevention (Figure 1).

Chapter 2 — Do school meal programmes deliver immediate impacts on nutrition, education, agriculture, and social protection? If school meals are to influence long-term health and reduce NCD risk, they must first deliver tangible short-term benefits: improved dietary intake during school hours, increased school participation and learning, support for local economies through procurement linkages, and meaningful contributions to household food security. Education and household food security matter for NCD risk indirectly, because educational attainment and socioeconomic position are strong predictors of health across the life course [9]. These immediate effects form the foundation upon which any longer-term benefits would rest.

Chapter 3 — Do school meals have behavioural ripple effects relevant to NCD risk? For school meals to influence adult health status, their effects must extend beyond what children eat at school. The chapter asks whether regular exposure to school meals shapes children's food preferences and eating habits outside school and later in life, whether these effects ripple into households and peer networks, and whether the behavioural pathways through which school meals operate can be characterised in ways that inform programme design. Few studies follow children beyond school age, so the chapter can say little about whether these effects persist into adulthood.

Chapter 4 — Do school meals improve anthropometric measures, metabolic and inflammatory markers, mental wellbeing, and long-term NCD outcomes across the life course? If school meals influence both children's diets and their eating behaviour beyond school, measurable effects should be observable on the biological markers that predict adult disease, and ultimately on long-term health outcomes themselves. This chapter examines the available evidence at each step of this biological pathway.

Chapter 5 — Under what conditions do school meal programmes achieve their potential? School meals are not uniform interventions. Their ability to deliver immediate benefits, shape lasting behaviours, and improve health outcomes depends fundamentally on the quality and stability of what is served, how programmes are governed, how food is sourced, whether they are linked to food and nutrition education

in the curriculum and to health systems, and the broader food environments in which children live and learn. This chapter examines the design, governance, and food environment conditions under which the benefits identified in the preceding chapters are most likely to be realised.

The logic for the structure of this White Paper is sequential (Figure 1). Long-term empirical evidence on whether school meals prevent adult NCDs is scarce and is likely to remain so for some time, given the time horizons over which NCD risk develops and the rarity of cohorts followed for the decades required to observe such outcomes directly. If, however, the evidence shows that school meals produce positive immediate impacts on children's diets and education (Chapter 2), influence what children eat outside school and into adolescence and adulthood (Chapter 3), and are associated with favourable anthropometric and metabolic changes during childhood (Chapter 4) — and that all of this occurs under identifiable programme design and implementation conditions, including meal composition, consistent delivery and supportive food environments (Chapter 5) — then there is a reasonable basis to expect long-term benefits for NCD prevention, even though the full chain from school meals to adult NCD outcomes cannot yet be demonstrated directly. On that basis, school meal programmes could be recognised as a public health measure with the potential to contribute to the prevention of diet-related NCDs at scale.

Figure 1 presents this logic as a sequence, but school meals could influence NCD risk through more than one pathway. The first is the meal itself: what is served, and how consistently, shapes children's dietary intake and nutritional status during school age. The second runs through the food supply chain and food environment in which programmes operate, including local procurement and the regulation of foods sold in and around schools. The third is the dietary patterns established at school and whether they persist beyond school age, which depends on the wider food environment and on families' ability to access and afford nutritious foods. The evidence reviewed in this White Paper bears mainly on the first pathway. Evidence that school-age dietary patterns persist into adulthood, or that school meals reduce adult NCD risk directly, is limited, and the chapters identify these as critical gaps.

The urgency of the NCD crisis demands that we work with the tools we have, strengthen the systems already in place, and build the evidence base needed to guide future action. School meal programmes represent a rare alignment of political feasibility, existing infrastructure, and biological plausibility. Whether they can deliver on their potential to prevent diet-related NCDs at scale is the question this White Paper seeks to address.

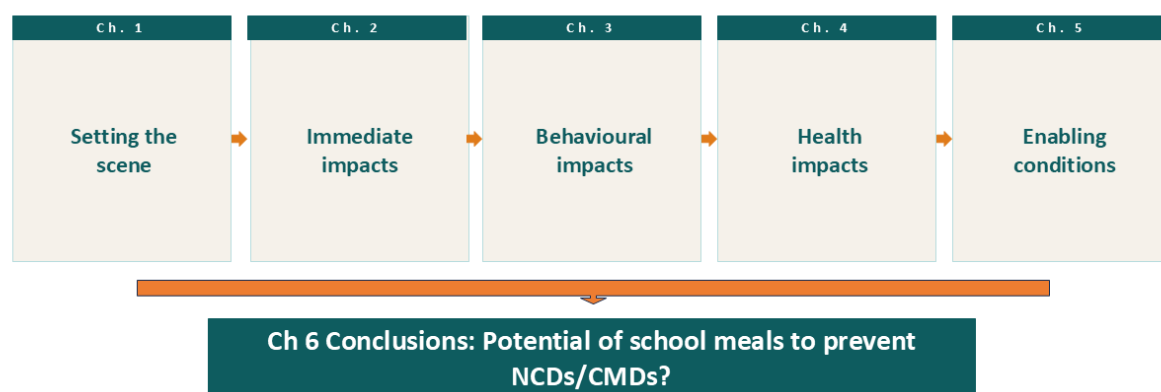


Figure 1. Causal logic through which school meals might contribute to NCD prevention discussed in this White Paper.

1.4 Methodology and scope

The White Paper draws on scoping reviews conducted by four international research teams each examining the evidence for one component of the causal pathway described above (Figure 1). Beyond these four core teams, a large pool of contributing authors provided essential input through critical review of draft chapters, thematic writing contributions, and the provision of data and resources that informed the synthesis.

Methodological details are provided in Annex 1. The synthesis covers high-, upper-middle-, lower-middle-, and low-income country settings, and includes both peer-reviewed evidence and, where relevant, grey literature from international organisations operating at scale in school feeding (WFP, GCNF, World Bank, UNICEF). The concluding chapter (Chapter 6) synthesises this evidence in three sections: conclusions that can be drawn from the evidence at varying levels of confidence; implications that follow from this evidence profile for policy, research, implementers, and research funders; and closing positional statements, which go beyond the direct evidence and are marked as such.

This White Paper is not a systematic review. The four reviews mapped the breadth and nature of the evidence; screening was mostly done by single reviewers, with uncertainties resolved within each team, and no formal risk-of-bias or certainty assessment was carried out (Annex 1). Study designs range from randomised and quasi-experimental studies to cohort, cross-sectional, qualitative and modelling studies, and the mix differs by chapter and outcome. Because exposures, comparators and outcome measures varied widely, results were not pooled; the chapters summarise the direction and consistency of findings and report effect sizes for individual studies where these help interpretation. To make the strength of each finding visible, Chapter 6 grades findings as consistent (supported by many studies, including experimental and quasi-experimental designs, across multiple contexts), suggestive (supported by fewer studies, narrower geographical bases or designs that limit causal inference) or limited (a critical gap in the evidence). Findings reported by country income group often rest on a small number of studies from a few countries and should not be generalised to all countries in that group. The evidence is also likely to over-represent programmes that were evaluated because an effect was expected, so it may not reflect the typical school meal programme.

Many included studies evaluated school meal programmes as delivered, which can include components such as nutritional standards, fortification, nutrition education or school gardens, and some examined related school food environment interventions. In these studies, effects cannot be attributed to the provision of food alone. Throughout this White Paper, “school meal exposure” therefore refers to participation in a school meal programme as delivered, including any accompanying components.

2. Immediate impacts of school meal programmes on nutrition, education, agriculture and social protection

2.1 Introduction to the chapter

This chapter examines the immediate impacts of SMPs on four interconnected domains: nutrition, education, agriculture, and social protection. It opens the White Paper's sequential argument by establishing what school meals deliver in the short term — what children eat at school, how this affects their learning, how local food systems respond to school feeding demand, how households benefit through food security and social support, and how programmes can promote greater equity in access to nutrition and education. Without these immediate effects, the longer-term behavioural changes (Chapter 3) and health outcomes (Chapter 4) cannot be realised; Chapter 5 then examines the design conditions under which these effects are most likely.

The chapter argues that long-term health improvements are predicated on achieving these immediate outcomes. "SMPs have evolved beyond their traditional role as hunger, nutrition and education interventions (including school enrolment) into multi-sectoral platforms capable of influencing agriculture and social protection through local food sourcing, smallholder farmer engagement, and household food security [1,2,3] (Table 1). Each of the four domains can potentially contribute to long-term NCD prevention through distinct but overlapping pathways. Nutrition outcomes — dietary intake, diet quality, diet diversity, and micronutrient status — are the most direct route, given the well-established links between childhood diet and the biological development of NCD risk factors in adulthood [4]. Education outcomes matter through the well-documented association between educational attainment and better health outcomes across the life course, including lower NCD risk in adulthood [5, 6]. These immediate outcomes may form part of the pathway to longer-term health, but they are not sufficient on their own: the pathway also depends on factors largely outside the programme, including access to health services, water and sanitation, infectious disease burden, poverty and the wider food environment.

Agriculture outcomes are relevant because procurement arrangements, together with nutrition standards, menu design and budgets, shape what is on children's plates during the school day, and because school feeding can influence food environments: where meals include diverse, nutrient-dense foods, and where there is supply capacity and purchasing power in the wider community, institutional demand may support smallholder farmer livelihoods and the availability of healthy foods where children live

[7]. Social protection outcomes¹ matter because food insecurity and household economic stress are themselves independently associated with elevated NCD risk over the life course [8] and because programmes that reduce these pressures create structural conditions for healthier diets. Understanding what SMPs can deliver across these four domains in the short term is therefore essential to understanding whether and how they can contribute to NCD prevention over the life course.

The chapter proceeds in three sections: an overview of the evidence base, a synthesis of the evidence by outcome domain (nutrition, education, agriculture, and social protection), and a summary of the cross-cutting findings and key evidence gaps. Methodological details on study selection, search strategy, and data analysis are provided in Annex 1.

Table 1. Domains and outcomes*

Domains	Outcomes
Nutrition	a) Dietary intake (energy intake, protein intake) b) Diet quality (SSB, snacks, unprocessed, ultra-processed, saturated fat, sodium, HEI) c) Diet diversity (food groups, fruits and vegetable consumption, DDS) d) Micronutrients intake, status and outcomes (vitamin A and iron status - haemoglobin or anaemia)
Education	a) School participation (enrolment and attendance) b) Cognitive Performance c) Academic performance and attainment (test scores and completion)
Agriculture	a) Smallholder farmer market participation b) Access to local and diverse foods for school children c) Food system linkages between schools and local agricultural supply chains
Social protection	a) Household/child food security b) Social equity - family finances, social support, and wellbeing

*Outcome categories reflect the dominant themes in the included studies. Some overlap: for example, dietary diversity is often treated as a dimension of diet quality, and micronutrient intake and status are different levels of outcome. Anthropometric outcomes are covered in Chapter 4.

2.2 Overview of the evidence base

The evidence synthesised in this chapter draws on a systematic search across nine databases covering nutrition, education, agriculture, and economics literature. Following deduplication, title and abstract screening, and full-text review, 364 studies were included in the narrative synthesis: 248 examining nutrition outcomes, 98 examining education outcomes, 6 examining agriculture outcomes, and 12 examining social protection outcomes (see Annex 2 for PRISMA diagram and Annex 3 for extraction table).

¹ In this White Paper Social protection is focussed only on food security and measures of economic stability.

The search across nine databases (PubMed, Scopus, Google Scholar, ERIC, PsycINFO, CAB Abstracts, EconLit, AgEcon and Web of Science) identified 36,027 articles. After removing duplicates, 22,432 articles remained. Title and abstract screening excluded 20,234 records, and full-text screening of the remaining 2,198 publications excluded 1,834. The main reasons for exclusion were intervention not meeting the eligibility criteria (n=441), outcomes not meeting the eligibility criteria (n=465), population not meeting the eligibility criteria (n=283), policy analyses or statements (n=228), opinion pieces, reports or books (n=205), descriptive designs that did not report outcome data (n=115), study settings not in schools (n=81), language (n=6), retractions (n=4), study protocols (n=2), and duplicates (n=4).

The included evidence spans multiple regions and income settings, with research design ranging from randomised controlled trials and quasi-experimental studies through to observational studies and reviews. However, the distribution of evidence is uneven: at least 60% of included studies were conducted in high-income countries, while low-income countries are severely underrepresented across all four domains (only 9% of included studies, Table 2). Nutrition and education are the most heavily evidenced domains, both methodologically and geographically, with some studies employing strong causal designs. By contrast, agriculture and social protection together account for fewer than 5% of included studies and are concentrated in a small number of country contexts. The evidence base is also dominated by observational designs, particularly cross-sectional surveys, which limits causal inference for several outcomes.

Given the heterogeneity of study designs and outcome measures, the data could not be combined for meta-analysis. Instead, a qualitative narrative synthesis was used to summarise findings across the four domains. This approach is well suited to characterising the existence and nature of effects across diverse contexts but is less precise in quantifying effect sizes than statistical pooling would be.

Table 2. Number of studies by outcome and income setting*

Outcome	LIC	ULMIC	HIC	Total
Nutrition**				
Dietary intake (total energy and protein)	4	39	108	151
Dietary diversity	1	9	26	36
Diet quality (HEI)	0	2	25	27
Micronutrients intake and status	3	26	5	34
Education***				
School participation (enrolment and attendance)	13	18	18	49
Cognitive performance	2	3	1	6
Academic performance and attainment	11	25	22	58
Agriculture***				
Smallholder farmer market participation	0	2	4	6

Access to local and diverse foods for school children	0	2	4	6
Food system linkages between schools and local agricultural supply chains	0	2	4	6
Social protection***				
Household/child food security	0	3	9	12
Social equity - family finances, social support, and wellbeing	0	3	9	12

* LIC = low-income countries; ULMIC = upper and lower-middle-income countries; HIC = high-income countries. Income classifications follow World Bank definitions

**Studies examining more than one outcome are counted once per outcome category

***Unique studies are reported on multiple outcomes

2.3 Evidence by outcome

2.3.1 Nutrition

The evidence base spans multiple regions (Africa, Asia, North America & Europe, South America), income settings (LIC, LMIC/UMIC and HIC) and includes RCTs, quasi-experimental studies, and cross-sectional analyses, with a predominance of observational designs. Anthropometric outcomes are covered in Chapter 4.

Dietary intake

Dietary intake (total energy and protein intake) has the largest body of evidence, with 151 studies from five continents. HIC contribute 108 studies, largely examining the National School Lunch Program (NSLP) and school food standards in the UK. LMIC and UMIC follow with 39 studies mainly focused on India's Mid-Day Meal scheme, programmes adding fortified rice to school meals, and studies from countries in Africa (Ghana, Kenya, and Nigeria). In LIC, the few available studies (n=4) consistently show that school meals increase energy and protein intake and help reduce dietary deficits [9]. In LMIC, findings are generally positive but heterogeneous, with improvements often insufficient to meet recommended intake levels (30% of daily nutrient requirements), as shown in Nigeria [10] and Ghana [11]. In HIC, the evidence is mixed; while school meals significantly improve intake during school hours [12], total daily intake often remains unchanged, possibly because of compensation at other meals — meaning that improvements in dietary quality at school may not translate into lower NCD risk without wider food environment regulation. Overall, school meals contribute to dietary intake the most in contexts with baseline nutritional deficits.

Dietary diversity

The evidence base comprised 36 studies, with HIC (North America and Europe) accounting for the largest share (26 studies), focusing on fruit and vegetable consumption, salad bars, chef-enhanced meals, and school gardens. Asia accounts for eight studies from Lebanon, India, Nepal, and the Philippines, while Africa accounts for

three studies from Ethiopia and Ghana. Several of these studies evaluated interventions delivered alongside or within school meals, such as choice architecture, food presentation or school gardens, rather than meal provision itself. School meals generally improve dietary diversity during school hours. Limited evidence from one LIC study suggests positive effects in contexts where school meals introduce food groups — such as animal-source foods, legumes, or fresh vegetables — that are otherwise largely absent from children's diets due to cost or availability constraints [13]. Evidence from HIC suggests that "nudging" school children through choice architecture can shift consumption, improving diet diversity [14]. Similarly, modifying food presentation, such as slicing apples [15] or introducing fruit smoothies [16], increased fruit consumption compared with offering whole fruit. However, gains in dietary diversity are often confined to the school setting.

Diet quality

Diet quality is reported in 27 studies, with the vast majority concentrated in HIC (North America and Europe), evaluating the Healthy, Hunger-Free Kids Act and nutrient-based standards on Healthy Eating Index (HEI) scores, sodium, and saturated fat. Asia, Africa, and South America each contribute only 2 studies, highlighting a significant evidence gap in lower-income regions. The quality of meals from SMPs varies widely globally. The available studies, mostly from HIC, suggest that school meal participation improves diet quality, as reflected in higher HEI scores in Canada [17] and lower sodium and ultra-processed food intake compared with packed lunches in the UK [18]. These results underscore the importance of valuing and optimising the specific nutritional content of the meals served. Evidence from Brazil's national programme, which operates under binding nutritional guidelines, is discussed in Chapter 5.

Micronutrient intake and status

The evidence base included 34 studies, with Asia leading at approximately 20 studies from India, Bangladesh, and Thailand, focusing on fortified rice, double-fortified salt, and deworming to address haemoglobin, anaemia, and iron status. Several of these studies evaluated fortification or deworming delivered through schools, which act through different mechanisms from meal provision. Africa contributed about 8 studies on biofortified cassava, red palm oil, and fortified biscuits. Unlike other domains, micronutrient research is driven by LMIC, where deficiencies are most prevalent. In UMIC, effects are moderate and programme dependent. In HIC, evidence is limited due to lower baseline deficiency levels. The effectiveness of these interventions is enhanced when combined with complementary health measures, such as deworming [20] and Water, Sanitation, and Hygiene (WASH) interventions [21]. However, gains may be attenuated by infection and are often not sustained during school breaks (e.g. summer break); in Burundi [22] and Cambodia [23], children with febrile illness showed significantly smaller increases in haemoglobin despite receiving fortified foods.

2.3.2 Education

The impact of SMPs on education outcomes is supported by various research designs including randomised controlled trials, quasi-experimental and econometric studies, observational studies (cohort and cross-sectional) and reviews. We report the three outcomes along the impact pathway of SMP and education outcomes. Although our synthesis identified 98 studies, the majority of these reported multiple education outcomes. Most education outcomes reported were from HIC [24] and LMIC/UMIC [25]. Only 26 outcomes were reported from studies conducted in LIC.

School participation (enrolment and attendance)

In LIC, studies (n=13) consistently report positive effects on school participation, with most reporting improvements in attendance, participation, enrolment and sometimes specifically girls' enrolment (26-29). In LMIC, the evidence remains positive but less consistent, with stronger associations on attendance than enrolment (30-33). However, one study in UMIC reported no significant effect on annual class attendance across regions over a period of 10 years [34]. In HIC, evidence is less consistent and generally weaker, with improvements in participation primarily driven by increased meal uptake. For example, studies in the United States, found that universal free breakfast increased participation in school breakfast by 20-25%, with only modest gains in attendance (35-37), yet other studies in HIC found no statistically significant changes in attendance (38-40).

Cognitive performance

In LIC, the few available studies (n=2) report improvements in cognitive outcomes associated with increased nutritional intake. For example, in Ghana, adding milk powder to school meals led to significant improvements in cognitive test scores [41], while studies in Rwanda also reported consistent gains in cognitive performance among programme participants [42]. In ULMIC, the evidence remains positive but somewhat variable, with effects depending on nutrient composition and programme design. Two studies from China reported significant improvements in cognitive test scores, particularly among poorer school children [43,44]. In Cambodia, multi-micronutrient-fortified rice was found to improve specific cognitive domains, although effects varied by formulation [45]. Evidence from HIC, on cognition is limited and inconclusive, with few studies reporting direct quantitative effects. Available evidence suggests improvements in concentration and classroom behaviour, but without statistically significant gains in cognitive test scores [46].

Academic performance and attainment

Evidence from HIC suggests that SMPs have small and often limited improvements in academic performance, with effects concentrated among vulnerable populations and low-income families (47-50). Some studies report modest statistically significant gains in

math scores and other academic outcomes following expansion of SMP [51,52]. Other studies reported non-significant findings, for example a study of a "Breakfast After the Bell" (BATB) programme, in which breakfast is served after the start of the school day, found no statistically significant changes in math or reading test scores over a two-year period, despite higher school children attendance rates [53]. Studies from Norway and New Zealand also found no statistically significant effects on academic performance [38,46]. In ULMIC, many studies have reported consistent improvements in school performance as an outcome of SMP interventions. Free school meals significantly increased mathematics test scores even though the effect was not significant in verbal improvement, especially in disadvantaged children in China [43,44]; while they contributed significantly to better scores in core subjects (English, Mathematics, and Integrated Science) in Ghana [23]. A few studies in LMIC also reported non-significant effects of SMP on learning and education outcomes [54]. In LIC most studies (11 studies), report improvements in academic performance [27,29,45,46]. In Kenya, adding animal-source foods to a staple-based school snack led to statistically significant gains in test scores [55].

2.3.3 Agriculture

The evidence base for agriculture outcomes is small and methodologically diverse. The review draws on six studies across Ghana, Brazil, and the United States that collectively provide evidence for the three outcomes; two of these are reviews (a systematic review and a scoping review) that synthesise further studies rather than reporting primary data. Market participation is primarily informed by a cluster-RCT from Ghana, a qualitative case study from Brazil, and a cross-sectional survey from the United States. Access to local and diverse foods is largely examined through a systematic review and a scoping review drawing from high-income contexts. Food system linkages are explored through both the Ghana trial and the Brazil qualitative study, alongside system-level insights from high-income settings. The evidence base is methodologically diverse, including experimental, observational, qualitative, and economic modelling approaches, with the Ghana cluster-RCT providing the only experimental evidence (on smallholder market participation) and the remaining studies offering contextual and system-level insights (56-61).

Across the three outcomes, the studies were guided by three central research questions. First, under market participation, whether SMPs that procure from smallholder farmers can create reliable and economically beneficial markets for them (local procurement does not in itself imply procurement from smallholders). Second, under access to local and diverse foods, whether local procurement, compared with other procurement arrangements, improves children's access to fresh, diverse, and nutritious foods and influences dietary intake and behaviour. Third, under food system linkages, what structural, institutional, and supply chain factors enable or constrain effective connections between schools and local agricultural systems. These questions are critical

for understanding whether school feeding can simultaneously deliver nutrition and agricultural development outcomes, and whether improvements in local food systems stimulated by school procurement can contribute to the dietary conditions that reduce NCD risk over the life course [57,59].

The evidence shows mixed and context-dependent results. Under market participation, there is some increase in procurement from smallholders, such as in Ghana where procurement rose from 5–12% under home-grown school feeding programmes (HGSFP), but this was not statistically significant and no improvements were observed in farmer income or production. In contrast, Brazil demonstrates stronger participation due to policy mandates, while in high-income settings participation depends on farmer motivations and institutional demand [26,61]. Under access to local and diverse foods, the most consistent finding is a modest improvement in children's vegetable intake, particularly among those with low baseline consumption and in high-intensity programmes, while fruit intake shows no significant change across studies in the US [57]. All of these studies evaluated farm-to-school programmes that included other activities alongside local procurement, so the change in vegetable intake cannot be attributed to local procurement alone. Under food system linkages, weak coordination, delayed payments, and reliance on intermediaries limits linkages in low-income contexts, whereas stronger institutional arrangements and intermediary organisations in Brazil enable more effective and stable linkages. Economic modelling evidence suggests that local procurement generates positive economic multiplier effects, although these do not necessarily translate into improved farmer livelihoods [58].

Taken together, the limited evidence across the three sub-outcomes suggests that SMPs are more effective in achieving immediate and proximal outcomes — such as increased school-level demand for local produce and short-term improvements in vegetable intake — than in driving the structural transformation of local food systems that would be needed to produce sustained changes in food availability and dietary quality at population level. Under market participation, increased demand alone is insufficient to improve farmer livelihoods due to supply-side constraints such as limited capacity, weak infrastructure, and financing delays, limiting the potential for procurement-led agricultural development to translate into the broader food environment changes that could support healthier diets and, over time, reduced NCD risk. Under access to local and diverse foods, improvements are observed mainly in vegetable intake, suggesting that dietary benefits are outcome-specific and depend on programme intensity and on what is procured and served. Under food system linkages, success depends heavily on institutional coordination, trust, and the presence of intermediary organisations, as seen in Brazil, while in the low-income contexts studied, their absence weakened programme effectiveness. The strength of evidence varies; the only experimental study shows no significant impact on farmer outcomes (income and production), while other studies provide important contextual insights [56,61]. The intermediate steps from procurement to the availability and affordability of nutritious foods, to children's diets and ultimately

to NCD risk have not been demonstrated by the studies reviewed and remain hypothesised.

2.3.4 Social protection

Evidence on the effects of school meals on social protection is limited and drawn predominantly from HIC settings. The evidence suggests beneficial effects of school meals on food security, for both the child and household more broadly, with more limited evidence examining other social protection outcomes (e.g., family finances, social support, and wellbeing). We included in the synthesis an international systematic review conducted among HIC to examine the impact of universal free school meal policies (UFSM) [62]. Among the 47 publications included in this systematic review, 5 examined associations between UFSM policies and food security, and the findings were mixed. UFSM was associated with lower parent-reported food insecurity, higher odds of not reporting hunger problems, and reduced child self-reported hunger. On other, closely related measures, however, no statistically significant differences were observed — including for parent-reported, child, and household food security. Taken together, the evidence points to some beneficial effects of UFSM on specific dimensions of food insecurity, though effects are not uniform across all measures [63,64]. Additionally, an increase in students' food security (3.7%) and households with children that were previously food insecure (3.2%) was also reported from simulation modelling with a US national dataset that estimated the impact of Community Eligibility Provision (CEP), which is a policy that enables higher poverty schools or districts to provide free meals to all school children within the school district [65]. Students attending schools in districts that opted not to participate in CEP had increased odds of being in a food-insecure household compared with students attending CEP schools using a parent-reported food security survey [66]. Other studies examining food security in HIC, found that access to free school meals policy was associated with a 5% decline in food insecurity [67] and enhanced household food security [68]. The decline in food insecurity was potentially in part due to the reported improvements in household incomes. Similarly two studies examining a national dataset found access to the NSLP in the U.S. was associated with reductions in the risk of household food insecurity and food insufficiency compared with summer months when schools are closed; the result indicated that NSLP participation was associated with a reduction of food insufficiency risk by approximately 14% (based on parent-report using a food security survey) [69,70].

Studies from other HIC also suggest positive SMPs impacts on child and household food security and other social protection outcomes. For example, a qualitative study in New Zealand, using focus groups with parents and school children, found that participants perceived improvements in food security, wellbeing, and reduced financial hardship following the introduction of a government-funded healthy school lunch programme [71]. Lastly, an international scoping review examining the association between global

SMP on children's wellbeing among 13 countries (n=5 studies of the 44 publications included in the review examined social health impacts) found that SMP were associated with better social engagement (e.g., social interactions with peers at lunch, reduced stigma and narrowing perceived inequalities), and in particular, universal free school meal programmes were associated with perceived improved social support [72].

In upper-middle-income countries, evidence tentatively suggests beneficial effects of school meals on social protection outcomes, though the evidence base is more limited. A study in India examining the mid-day meal scheme found that it was associated with improved social equity [73]. A study in South Africa examined training in sustainable agricultural practices and growing food as part of a school feeding programme and found improvements in overall community development [74]. A further study in South Africa examined spillover effects of a school meal programme on household health spending, finding that parents of children who benefitted from school feeding recorded lower out-of-pocket health expenditure compared with parents of children who did not — suggesting that improvements in children's nutritional status and health resulting from programme participation may reduce the need for curative healthcare, with direct financial benefits for households [75].

2.4 Summary

This chapter has examined the immediate impacts of SMPs across four interconnected domains — nutrition, education, agriculture, and social protection. The synthesis draws on 364 studies, with the evidence heavily concentrated in nutrition (n=248) and education (n=98), and at least 60% of all included studies conducted in high-income settings.

Across nutrition outcomes, most included studies report improvements in dietary intake, diversity, quality, and micronutrient status, with the most pronounced gains in LIC and LMIC where baseline deficits are greatest. However, total daily nutrient requirements are often still not met, as improvements frequently do not extend beyond school hours or through holiday breaks. In HIC, total daily intake often remains unchanged, possibly because of compensation at other meals. The included studies rarely describe the composition or cost of the meals evaluated, and they may over-represent programmes designed and evaluated with these outcomes in mind.

Education outcomes follow a similar pattern. School enrolment and attendance are the most consistently documented benefits, particularly in food-insecure regions where SMPs act as a direct incentive for parents to enrol children. Cognitive performance and academic attainment are also positively associated with school meal exposure, with the largest effects observed in LIC and LMIC and more modest effects in HIC. Programme design matters: enhanced modalities such as take-home rations and fortified meals

show stronger effects on participation and cognitive outcomes than standard provision. However, it is important to recognise that the observed changes in academic performance cannot be attributed solely to SMPs. Educational outcomes are shaped by a range of interconnected factors, including household socioeconomic conditions, the quality of teaching, the school environment, students' health and well-being, and other concurrent social and educational interventions. Therefore, the improvements reported in the literature may reflect the combined influence of these factors rather than the direct effect of school meals alone. Further context-specific analyses and rigorous studies are needed to better understand the pathways through which school meal programmes contribute to learning outcomes and to disentangle their effects from those of other determinants of educational achievement.

The agriculture and social protection evidence bases are much smaller (n=6 and n=12 respectively) and geographically skewed, meaning that findings from these domains should be interpreted as illustrative rather than as a basis for firm conclusions. For agriculture, the evidence suggests modest improvements in children's access to local and diverse foods, particularly vegetable intake in the US, but limited causal evidence of impact on farmer income or livelihoods. Effective food system linkages depend heavily on institutional coordination, as illustrated by Brazil's HGSP; in the LIC contexts studied, their absence weakened programme effectiveness. For social protection, the available evidence, concentrated in HICs, shows reductions in household food insecurity of around 5%, with some additional evidence of social support benefits for children. Evidence from UMIC and LMIC is sparse and methodologically heterogeneous, and no LIC studies were identified.

Three cross-cutting gaps emerge. First, the scarcity of high-quality evidence from LIC and LMIC across all four domains limits what can be concluded about contexts where school meal programmes may have the greatest potential to benefit children. Second, comprehensive multi-domain studies are largely absent across all income settings, making it difficult to assess how immediate impacts in one domain (e.g. nutrition) interact with those in another (e.g. social protection) over time. Third, the composition, cost and delivery of the meals evaluated are rarely reported, and the evidence may over-represent programmes that were evaluated because an effect was expected, so it may not reflect school meal programmes in general.

3. Behavioural impacts of school meals beyond the school gate

3.1 Introduction to the chapter

This chapter examines the behavioural impacts of school meal programmes — whether and how school meal exposure (participation in a school meal programme as delivered, including any accompanying components such as nutrition education; see Methodology and scope in Chapter 1) shapes children's eating behaviour in ways that persist beyond the moment of school meal consumption itself. It sits between Chapter 2's evidence on immediate dietary intake during school hours and Chapter 4's evidence on biological and longer-term health outcomes, and asks whether the dietary benefits documented at the point of consumption ripple outward into households, peer networks, and communities in ways that could shape long-term non-communicable disease (NCD) risk.

Behavioural pathways matter for long-term NCD prevention because the dietary patterns that shape adult disease risk are formed through cumulative daily food choices across many settings, not just during school hours, and through the social, economic and environmental conditions that shape those choices [38]. If school meals shift only what children eat at school, their contribution to long-term prevention depends on how much of the daily diet they provide, which can be substantial where children receive both breakfast and lunch at school. If, however, school meal exposure shapes what children eat at home, how they respond to peer norms, and how families make food decisions, then the potential contribution of school meals to NCD prevention extends well beyond the meal itself. Influence can also run in the other direction: household and peer norms can shape whether children accept and eat the school meal (Section 3.3.1). Understanding these behavioural spillover effects is therefore essential to understanding whether school meals can shape dietary patterns over the life course [39].

The chapter focuses on three inter-related behavioural dimensions: whether school meal exposure produces spillover effects into non-school hours and home settings; how peer dynamics and social eating environments within the school shape children's food selection and consumption; and how school meal participation generates ripple effects through families and communities. These behavioural effects operate across several intersecting domains — the home food environment (what children eat at home and how meals are structured), household food purchasing and preparation decisions, peer and social eating norms, and the broader community food culture and commercial food environment, including food marketing, in which children and their families are embedded. Within the school, children may also eat foods brought from home or bought from canteens and vendors, and these other sources are considered where studies

address them. The three domains are used to organise a heterogeneous evidence base rather than as defined outcome measures. The outcomes reported range from behaviours such as dietary intake outside school, food requests and household food practices, to behavioural determinants, such as food preferences, social norms and the acceptability of school meals, and they are measured mostly at the level of the child or the household. The chapter did not apply a formal conceptual framework; the summary (Section 3.4) indicates which pathways have direct empirical support and which remain largely hypothesised. Throughout, the chapter examines whether and how these behavioural effects vary across high-income (HIC), upper-middle-income (UMIC), lower-middle-income (LMIC), and low-income (LIC) settings. The dietary intake and nutritional outcomes that result from school meal participation are addressed in Chapter 2; the programme design conditions and food environments that moderate behavioural impacts are addressed in Chapter 5.

The chapter proceeds in three sections: an overview of the evidence base; a synthesis of evidence by outcome domain; and a summary of the cross-cutting findings and gaps.

3.2 Overview of the evidence base

The evidence synthesised in this chapter draws on four contributing scoping reviews, combining peer-reviewed studies that examine the behavioural dimensions of school meal exposure with grey literature sources — including reports from the World Food Programme [35], Global Child Nutrition Foundation [37], World Bank [36], UNICEF [31], and Nielsen [8] — that capture programme-level insights at scale, particularly from LMIC and LIC settings where peer-reviewed evidence is sparse. Grey literature sources are evaluated with appropriate caution regarding methodological rigour and are explicitly distinguished from peer-reviewed evidence throughout the synthesis.

Across the three retained behavioural outcome domains, the combined evidence base comprises 40 primary peer-reviewed study-domain entries (some studies are classified under more than one domain and are therefore counted once per domain; the unique study count across the three domains is a subset of 40) supplemented by 24 grey literature sources. The geographic distribution is highly skewed: approximately 58% of primary study-domain entries originate from HICs (predominantly the USA and the UK), 15% from UMICs (largely Brazil and China), 25% from LMICs, and 2% from LICs. LIC representation is particularly limited, identifying a fundamental gap in evidence for low-resource settings where school meals may have the greatest potential for behavioural impact. This gap is unlikely to reflect limited policy or action on school meals in LICs. Rather, the case for investing in school meals in these settings has been framed mainly around education, food security, social protection and human capital [35,36], so evaluations have tended to measure enrolment, attendance, learning and nutritional status rather than eating behaviour. Behavioural outcomes are therefore seldom part of

the theory of change against which programmes in LICs are assessed, even though they matter for the NCD-prevention case made in this White Paper. A related problem is the low visibility of research on large LMIC programmes in the global databases searched. For PM POSHAN, India's mid-day meal scheme, which serves about 120 million children, the India-specific studies included in this synthesis were identified only through supplementary India-targeted searches. Table 3 summarises the distribution of primary study-domain entries by outcome domain and income setting.

Table 3. Distribution of primary study-domain entries by behavioural domain and World Bank income setting

Behavioural domain	LIC	LMIC	UMIC	HIC	Total
Behavioural spillover into non-school hours and home settings	0	3	4	12	19
Peer-mediated and social influences on eating	0	2	2	6	10
Family and community ripple effects	1	5	0	5	11
Total study-domain entries*	1	10	6	23	40

* Studies appearing in more than one domain are counted once per domain. LIC = Low Income Country; LMIC = Lower-Middle Income Country; UMIC = Upper-Middle Income Country; HIC = High Income Country (World Bank 2024–25 classification). Geographic distribution: HIC 58%, UMIC 15%, LMIC 25%, LIC 2%.

3.3 Evidence by outcome

3.3.1 Behavioural spillover into non-school hours and home settings

This section addresses whether school meal exposure changes what and how children eat outside the school setting — at home, in the community, and during non-school hours. Such effects are likely to depend on the home food environment: several of the studies below suggest that pre-existing household norms and food practices shape whether, and in which direction, school food exposure carries over into the home.

Experimental evidence comes from the OPUS School Meal Study, a cluster-randomised cross-over trial in Denmark in which 834 children aged 8–11 years received New Nordic Diet (NND) school meals or their usual packed lunches during two three-month periods [1]. Dietary intake was recorded over seven consecutive days. During the NND period,

children's intake across the whole week was higher for fish, potatoes, vegetables, cheese and eggs and lower for bread and fats, with no difference in energy intake. Fruit intake did not differ between periods, and neither did sugar intake; the authors suggested that changes during school hours might have been offset by eating outside school hours. The trial shows that a substantial school meal can improve children's overall weekly diet, but it did not measure eating behaviour outside school separately, so it does not show directly that behaviour changed beyond the school setting.

Dijkstra et al. (2025) [2], in a six-month Dutch pre-post study with comparison schools, found that a free healthy school lunch, consisting mainly of whole-grain bread sandwiches with healthy fillings, 50g ready-to-eat vegetables coupled with at least one additional item like soup, eggs, wrap or veg meat balls, increased vegetable consumption at school; children were still allowed to bring food from home. A parent-reported questionnaire did not indicate compensatory unhealthy eating outside school hours, although dietary intake at home was not measured directly. Ohri-Vachaspati et al. (2018) [3], in a cross-sectional study of fourth-grade students in low-income schools in Phoenix, USA, found that school children taking part in the USDA Fresh Fruit and Vegetable Program were more likely to request fruits and vegetables at the grocery store and at home than peers in non-participating schools. This programme provides fresh fruit and vegetable snacks separately from school meals and the design does not allow causal inference. Therefore, the study indicates a possible school-to-home pathway rather than an effect of school meals.

Eating patterns at home and at school also appear to run in parallel. Baghlaf et al. (2020) [4], using three-day diet diaries and systematic lunch observation in UK primary schools, found that children classified as high free-sugar consumers at home made systematically less healthy food choices at school lunch: 73% of high-sugar-consuming children selected high-sugar desserts at school lunch, while 69% of low-sugar consumers chose lower-sugar options. In a study from Finland, where free school meals are provided to all pupils, fewer than half of 531 schoolchildren aged 11–16 years regularly ate a balanced school lunch, and those who did also had more regular meals and healthier eating patterns at home [45]. This parallel pattern suggests that school food programmes cannot be designed in isolation from the home food environment.

Not all evidence is positive. Beck et al. (2019) [5], in a qualitative study of low-income Latino adolescents in San Francisco, found that when school meals were perceived as unpalatable, students rejected them and ate chips, soda and fast food instead. This reflects low uptake of the meal and substitution with other foods rather than spillover into the home, but it shows that poor acceptability can undermine the intended dietary effect of a programme. Reagan et al. (2022) [6], in a Canadian RCT of a school snack-only programme, found no significant effect on home fruit and vegetable availability or parental dietary modelling, suggesting that full-meal programmes may be necessary to

activate household dietary change. Most evidence is from HIC settings and may not be directly generalisable to LIC/LMIC contexts, underscoring the need for more context-specific evidence. However, Fernandes et al. (2017) [7] found that school meal provision in Ghana who received free school meals were less likely to go home for lunch and had reduced the likelihood of children leaving campus to buy food, suggesting that meal provision itself can change how children use the surrounding food environment.

From grey literature, the Akshaya Patra Foundation impact assessment in India [8] reports that 80% of parents consciously attempted to improve fruit and vegetable inclusion at home following their child's exposure to healthy school meals. The report does not show whether household diets changed as a result. The finding suggests that child-mediated parental influence may operate at scale in LMIC settings, but this remains to be confirmed through rigorous study designs.

3.3.2 Peer and social eating dynamics

Much of the evidence in this section comes from studies of social eating, peer norms, choice architecture and school food environments in general, rather than from evaluations of school meal programmes. It is used here to indicate pathways through which school meals could influence eating behaviour, not as direct evidence of programme effects. The school meal setting creates structured peer eating contexts that can influence both what and how much children eat. The studies below suggest that peer dynamics do not in themselves promote healthy or unhealthy eating, but may reinforce whichever dietary norms are most socially salient in a given school food environment.

Social facilitation of eating — the tendency for individuals to consume greater quantities of food in the presence of others — is well-documented among children and adolescents. Salvy et al. (2012) [9] Salvy et al. (2011) [9] found that young children ate more energy from unhealthy snacks when eating with a friend than with their mother, whereas adolescent girls ate more healthily with friends, and Lumeng and Hillman (2007) [10] found that preschool children ate about 30% more during longer snacks when eating in a group of nine rather than three. Peer presence in the school lunchroom may inadvertently amplify energy intake, particularly where energy-dense or competitive foods are available.

Peer norms exert substantial influence on food selection. Wouters et al. (2010) [11] found that adolescents' snack and soft drink consumption was associated with that of their friends, particularly where canteens and vending machines offered more snacks. In an experiment in Dutch primary schools, Bevelander et al. (2012) [12] found that a peer's snack intake set a norm for how much children ate, which persisted when they later ate alone, and teenage girls shopping with a peer bought higher-calorie products when the peer did so [13]. Andersen et al. (2015) [14], in a cross-sectional study of 702 New Orleans adolescents, found that salad bar use was significantly more likely among students with

higher healthy food preferences and those who encouraged peers to consume fruits and vegetables, indicating that social norm endorsement can function as a positive multiplier for healthy dietary behaviour.

Positive peer modelling can be deliberately cultivated as a dietary intervention. Hendy (2002) [15] found that trained peer models increased preschool children's acceptance of new foods at meals, although the effect did not persist one month later.

School meal acceptability — shaped by taste, cultural resonance, meal presentation, and perceived quality — is a critical moderating factor, but one that operates conditionally: acceptability determines whether children consume what is offered, while nutritional quality determines whether what is offered is worth consuming. School meals are not inherently healthy, and evidence on meal quality varies considerably across programme contexts, as discussed in Chapter 4. In a randomised trial in 14 US schools, Cohen et al. (2015) [16] found that chef-enhanced meals increased fruit and vegetable selection and, after seven months of exposure, consumption, whereas choice architecture alone increased selection but not consumption. Ensaff et al. (2015) [17] demonstrated that repositioning and presenting plant-based foods more prominently in a school canteen (choice architecture) significantly increased their selection, underscoring that the physical and social presentation of school food may directly shape what children choose to eat. Puhl and Latner (2007) [18] identified stigma around food and body weight as a driver of compensatory eating behaviours among youth. Agaronov et al. (2018) [19] found that school-home food culture mismatch drove compensatory eating outside school. Chortatos et al. (2018) [20], in a Norwegian mixed-methods study, found that students who did not use school canteens (on-site outlets where students buy food, rather than a school meal programme) were more likely to purchase unhealthy foods from commercial outlets near the school.

Children's food preferences, and the norms that circulate among peers, are also shaped by food marketing, both before children reach the school canteen and within schools. In the United States, most students in 2012 attended schools with at least one form of food or beverage commercialism, such as exclusive beverage contracts or food coupons used as incentives [42]. Policies that set nutritional standards for foods served and sold in schools, restrict competitive foods and limit marketing are therefore complementary to behavioural approaches such as choice architecture. A systematic review found that school food environment policies, including direct provision of healthy foods and standards for competitive foods, improved some targeted dietary behaviours [43], and the 2026 WHO guideline recommends policies on school food provision, nutritional standards for foods served and sold in schools, and nudging [44]. Improving the acceptability of healthy school meals alone may not be sufficient where children can easily buy less healthy foods in or near the school, as the findings of Chortatos et al. suggest [20].

In LMIC settings, particularly India, peer-mediated effects take on additional dimensions shaped by socioeconomic context. Increased peer participation in mid-day meal programmes in India has been shown to improve individual meal consumption, and shared school meals promote social inclusion and programme uptake [21,22,23]. Peer acceptance also influences uptake of modified interventions such as introduction of millet-based dishes and fortified foods within school meal programmes [24].

Methodological caution warrants a note. Toporoff et al. (2002) [25] found that the food choices Native American schoolchildren said they intended to make agreed poorly with the actual choices observed (composite kappa = 0.09). Intended food choices are therefore weak proxies for actual eating behaviour. Self-reported intake, based on recall or records of foods already eaten, is a different method, but it is also subject to reporting error and much of the literature reviewed in this chapter relies on self-report of one kind or another.

3.3.3 Family and community ripple effects

Most of the evidence in this section concerns families and households; evidence on wider community effects comes mainly from grey literature. Evidence documents two primary pathways through which school meals influence family dietary behaviour: child-mediated household influence — children acting as agents of dietary change within their families — and parental burden relief — the reduction of daily cognitive, financial, and logistical demands of food provisioning.

The most direct evidence for the child-mediated pathway comes from Ohri-Vachaspati et al. (2018) [3], who found that students in schools taking part in the Fresh Fruit and Vegetable Program were more likely to request fruit and vegetable purchases at the grocery store than students in non-participating schools. The programme provides produce outside the school meal programme and the study was cross-sectional, so these findings indicate an association rather than an effect. The study also reported greater self-efficacy to choose vegetables at home, stronger vegetable preferences, and more frequent fruit consumption, suggesting that school-based food exposure may extend into household food choices. From grey literature, the Nielsen impact assessment of the Akshaya Patra Foundation [8] reports that 81% of parents observed good vegetable inclusion in school meals and that child food exposure at school influenced household dietary conversations.

Two studies identified outside the scoping searches point to a further pathway, through adults present at the school meal. In Sweden, “pedagogic meals” position teachers as social and educational models during lunch, with observed teacher roles ranging from sociable to explicitly educative interactions. Such adult engagement may reinforce food learning and acceptance, although the evidence also indicates that teachers’ preparedness and attitudes can influence how effectively this role is enacted [40].

Conversely, the Hungarian school-catering reform illustrates how inadequate teacher preparedness, lack of nutrition literacy and weak communication with children can undermine implementation and acceptance of healthier meals [41].

The parental burden relief pathway is consistently documented across HIC and LMIC settings. Michnik et al. (2024) [27], in a Canadian cross-sectional survey, found that a universal free lunch pilot substantially reduced the daily cognitive and financial burden of packing lunches, with 68% of parents expressing willingness to contribute a small fee to sustain provision. Shwed et al. (2023) [28] found that COVID-19-related cancellations of school food programmes forced major shifts in family provisioning, with parents reporting increased time burden, greater reliance on packaged and processed foods, and intensified food-related anxiety. Borger et al. (2021) [29], in a US longitudinal cohort study, found that pandemic-related school closures led families to shift toward food banks and ultra-processed, shelf-stable foods, with parental food stress driving less intentional dietary choices.

The temporal boundaries of school meal programmes create structured periods of dietary risk during school holidays. Shinwell and Defeyter (2021) [30], in a qualitative study of UK families, found that parents made significant dietary sacrifices during term breaks as the loss of free school meals created acute household food insecurity. These studies of programme disruption suggest what school meals help prevent: when the programme was absent, families reported poorer dietary quality, more parental stress and less capacity to provide fresh, nutritious food. In grey literature, UNICEF and WFP (2021) [31] reported global-scale disruptions during COVID-19, with 370 million children losing access to an average of 40% of their in-school meals at peak disruption.

In LIC settings, Bliznashka et al. (2024) [32], in a qualitative evaluation of an emergency school feeding programme in conflict-affected Yemen, found that families viewed school meal provision as a direct income transfer, enabling them to redirect food resources to other household members. This matters when assessing the dietary contribution of school meals: where households reallocate food away from children who eat at school, the net addition to a child's diet may be smaller than the meal itself provides (see also Section 2.3.5). In rural India, comparing 24-hour recalls on school and non-school days, Afridi (2010) [21] found that the mid-day meal raised children's daily nutrient intake by 49–100% of the nutrients it provided, implying partial substitution of food at home. In Andhra Pradesh, India, longitudinal data showed that the midday meal scheme offset the decline in height-for-age and weight-for-age among children whose households were hit by severe drought [23].

These peer-reviewed findings are complemented by grey literature sources that provide programme-level insight at scale, particularly from LMIC contexts where peer-reviewed evidence is sparse. The International Labour Organization (2021) [34] identifies shared

school meals as a major child-focused social protection mechanism that reduces socio-economic disparities. School meal participation has also been linked to structured eating routines and "time anchoring" of family meal patterns: data from the Akshaya Patra Foundation [8] indicate that 83% of children eat school meals six days a week and 91% rely on them as their primary nutrition source, which may create predictable daily eating patterns that extend into the home. The WFP (2024) [35] and World Bank (2024) [36] similarly report that institutionalised meal patterns shape household routines and food preparation expectations, particularly in LMIC contexts where school meals may be the most reliable daily food source.

3.4 Summary

The evidence reviewed in this chapter suggests that school meal exposure can influence eating behaviour beyond the school meal itself, through three pathways: carry-over of dietary behaviour into non-school hours and the home; peer and social eating dynamics within the school; and effects on families, through children's influence on household food choices and through the relief that school meals give to household food budgets and time. Direct evidence is limited, and its strength differs by pathway. The clearest experimental evidence comes from Denmark [1], where school meals providing a morning snack, lunch and an afternoon snack, roughly half of daily dietary intake, improved children's overall weekly diet. That trial did not measure behaviour outside school separately, and fruit and sugar intakes did not change. Studies from the Netherlands and the USA suggest that children may request, choose and eat more vegetables or fruit after exposure at school, but these studies used pre-post or cross-sectional designs and one evaluated a fruit and vegetable snack programme rather than school meals [2,3]. Evidence on peer dynamics comes mostly from studies of social eating and school food environments rather than of school meal programmes; it suggests that peer contexts can reinforce either healthy or unhealthy eating, depending on the food environment and social context [9-13,15,16]. Effects on families and communities are the least experimentally documented. The available studies, mainly qualitative or cross-sectional and largely at household level, suggest that school meals relieve the time, cost and stress of feeding children, and studies of programme disruption during school holidays and COVID-19 closures suggest that household food insecurity and stress increase when school meals are withdrawn [27,28,30], although one US cohort found largely stable diet quality where emergency food responses were in place [29]. Programme data from China's rural Nutrition Improvement Programme show how quickly provision can shift responsibility for children's daily meals from households to schools: by mid-2012, 64% of covered students were in the school-feeding mode, 32% received a food package and 4% family feeding [33]. A review of parents' perspectives across 11 countries likewise found that parents see both benefits and challenges in school food programmes, whatever the provision model [26]. Adults present at the

school meal, such as teachers, may also shape children's eating, but this rests on two studies [40,41].

Risk-amplifying behavioural pathways are also documented (although less so) and warrant attention. Social facilitation of eating in peer contexts may inadvertently amplify energy intake where energy-dense foods are available [9,10]. Low meal acceptability and school-home food culture mismatch may lead children to skip school meals and eat less healthy foods instead, increasing rather than reducing NCD-relevant dietary exposures [5,19], and weight stigma among young people may add to this risk [18]. These risk pathways are less studied than the protective ones but are equally important for understanding the behavioural mechanisms through which school meals shape NCD risk.

Several gaps constrain what the behavioural evidence base can support. The most fundamental is the relative scarcity of research that directly examines behavioural pathways: most studies in this area measure dietary intake or food selection rather than behavioural change as such, and few are designed to isolate the specifically behavioural contribution of school meal exposure. The evidence is also geographically skewed, with LIC representation limited to a single primary peer-reviewed study [32]. Large programmes in LMICs are also under-represented. India's PM POSHAN serves about 120 million children, yet the global databases searched yielded no primary peer-reviewed evidence on its effects on behaviour outside school, and the India-specific studies included here were found only through supplementary searches. It is not clear whether this reflects an absence of research or the low visibility of research in the databases searched. The same is likely to apply to other large LMIC programmes, which may bias the global evidence base. No longitudinal study in this review directly tracks dietary behaviour patterns from school age into adulthood as a function of school meal participation, representing the most significant gap for the long-term NCD-prevention case. Methodological limitations, including frequent reliance on self-reported measures, among them intended food choices [25], and the predominance of cross-sectional designs, substantially limit causal inference.

This synthesis did not examine whether behavioural effects differ by age, gender or rural and urban setting, or whether the included studies were guided by a theoretical framework. Because the studies reviewed include both primary-school children and adolescents in very different settings, findings from one group should not be assumed to apply to another. Future studies should measure dietary intake directly, at school and at home, compare children who do and do not receive school meals, report results by age, gender and setting, and draw on behavioural frameworks to examine the mechanisms involved, such as social norms, habit formation, caregiver influence, observational learning and children's own agency. This would help clarify how school meals might contribute to NCD prevention through behaviour.

Taken together, the evidence suggests that the pathways through which school meals may influence children's eating behaviour run through social and family systems, not through the delivery of food or nutrients alone. If so, the effects of school meals on diet cannot be predicted simply from what is served: they depend on whether children accept and eat what is offered, how meals are experienced socially, and how families respond to what children encounter at school. Direct empirical support is strongest for effects on children's diets across the week where meals provide a large share of intake [1]. Effects on what children request at home, on peer norms and on how families organise food provisioning are plausible, but rest mainly on cross-sectional, qualitative or self-reported evidence, often from interventions other than school meal programmes. The evidence also suggests that these pathways depend on aspects of programmes such as meal acceptability, cultural fit and the social eating context, which measures of food or nutrient provision do not capture. School meals also operate alongside competitive foods, canteens, vending, informal vendors and food marketing, which can reinforce or counteract their effects. A whole-school approach that covers what is sold, promoted and made available in and around schools, as well as what is served, is therefore likely to be needed for behavioural effects to be realised; Chapters 5 and 6 return to this point.

4. The health impacts of school meals across the life course

4.1 Introduction to the chapter

This chapter sits at the core of the White Paper's sequential argument, linking short-term nutritional gains to long-term health risk. Chapter 2 established that school meal programmes can deliver immediate benefits — with variation across income settings and outcomes — including improved dietary intake during school hours and support for nutrition, education, and social protection. Chapter 3 examined whether school meal exposure influences eating behaviour beyond the school meal itself. It found suggestive but limited evidence of effects in the home and through peers and families, and no study that tracked dietary behaviour from school age into adulthood. This chapter asks whether school meal exposure is accompanied by measurable changes in the biological markers that predict future NCD risk, and whether any such effects endure. If school meals improve children's dietary intake in the short term, and if those improvements last, we would expect to observe measurable effects on intermediate biological markers that lie on the causal pathway to adult chronic disease, as well as on longer-term health itself (Figure 1). The same logic applies in reverse: where school meals are of poor nutritional quality, they could add to rather than reduce NCD risk, and the chapter reports such findings where they arise.

To assess this, the chapter examines evidence across four intermediate outcome categories, each of which lies on the causal pathway between diet and NCD risk. Anthropometric measures—including high BMI, stunting, wasting, and underweight—are well-established markers of nutritional status (both over and undernutrition) and early growth trajectories that predict NCD risk in later life, particularly for cardiovascular disease and type 2 diabetes [1,2]. Metabolic markers, including blood pressure, lipid profiles, insulin resistance, and blood glucose and glycated haemoglobin (HbA1c), are direct biological precursors of cardiovascular disease, type 2 diabetes, and stroke, and are known to track from childhood into adulthood [3,4]. Mental health conditions are closely linked to NCDs and are now addressed alongside them in the global NCD agenda. Mental health is increasingly recognised as both influenced by diet and food security [5,6] and independently associated with NCD risk through behavioural and neuroendocrine pathways [7,8]; because the included studies measured wellbeing and symptoms rather than clinical diagnoses, this chapter refers to mental wellbeing. Inflammatory markers and gut microbiota composition represent emerging but biologically plausible intermediaries, given the role of chronic low-grade inflammation and gut dysbiosis in the development of metabolic disease [9]. Evidence linking diet-related changes in gut microbiota during childhood to later NCD risk is, however, still very limited. The strength

of these links differs. Blood pressure, lipids and glucose regulation are established risk factors that track from childhood into adulthood, whereas it is less clear whether undernutrition during the school years, as distinct from early life, predicts later NCD risk, or whether dietary change during school age can offset risks associated with early undernutrition. Chapter 1 sets out the pathways through which school meals could influence NCD risk. The chapter also examines the medium- and long-term evidence that most directly addresses whether school meal exposure translates into health benefits over time. The conditions under which these effects are most likely to occur are addressed in Chapter 5.

4.2 Overview of the evidence base

The studies included in this chapter were identified through a systematic search of the published literature. From an initial yield of 7,763 records, 4,240 remained after deduplication, of which 348 proceeded to full-text review and 132 (11–142) were ultimately included (see Annex 2 for PRISMA diagram and Annex 3 for extraction table). These 132 unique studies contribute 149 outcome assessments² across five main outcome categories. The evidence base is heavily dominated by anthropometric outcomes which account for the largest share of both assessments (n=108, over 72% of total assessments) and unique studies, followed by medium- and long-term outcomes (n=14 assessments, all unique studies), metabolic markers (n=14 assessments), mental wellbeing (n=7 assessments), and inflammation and gut microbiota (n=6 assessments) (Table 4). Study designs vary widely and include randomised/cluster randomised controlled trials (RCTs/cluster RCTs), quasi-experimental studies, longitudinal cohort studies, modelling studies, and cross-sectional analyses, with cross-sectional designs predominating overall. Intervention duration was not systematically extracted, but many trials of school meals, particularly in LMICs, evaluated interventions lasting two years or less, whereas programmes at scale typically run for many years; shorter studies may therefore understate the effects of long-running programmes, particularly on growth. Studies span a broad range of countries across all World Bank income groups, though the evidence base for metabolic markers, mental wellbeing, inflammation, gut microbiota, and medium- and long-term outcomes is concentrated almost exclusively in high- and middle-income settings. Low-income country representation is limited to anthropometric outcomes and a single trial on mental wellbeing.

² Some papers reported on multiple outcomes.

Table 4. Number of health outcome assessments by outcome category and 2024 World Bank income setting.

Outcome category	LIC	ULMIC	HIC	Total
Anthropometric	15	55	38	108
Metabolic markers	0	5	9	14
Mental wellbeing	1	2	4	7
Inflammation and gut microbiota	0	4	2	6
Medium- and long-term health outcomes	0	3	10	14**
Total	16	69	63	149

*Note: Figures represent the number of outcome assessments (i.e. rows in the extraction table) rather than unique studies. The 149 assessments come from 132 unique studies. Studies examining more than one outcome are counted once per outcome category. LIC = low-income country; ULMIC = upper and lower-middle-income countries; HIC = high-income country. Income classifications follow World Bank definitions. **One study (Springmann et al., 2025) is a global modelling study not assigned to a single income group and is included in the total but not in the income-group columns.

4.3 Evidence by outcome

4.3.1 Anthropometric outcomes

The anthropometric evidence base is the largest of any outcome category, but findings are highly heterogeneous and context-dependent, varying by income setting, study design, school meal design and the specific anthropometric indicator examined (Table 5).

In high-income countries (HICs), the evidence does not support a consistent directional effect of school meals on anthropometric outcomes, with the majority of studies assessing BMI/related adiposity measures observationally, and with findings roughly evenly distributed across positive, null, and negative directions (Table 5). Among the few experimental studies (RCTs or cluster RCTs) examining impacts on BMI or adiposity outcomes, none [38,39,113] found a significant effect on BMI of school meals rich in fish, vegetables and potatoes. Two of these studies [38,39], however, found that waist circumference, another adiposity measure, increased in children receiving school meals compared with those bringing packed lunches from home. As for the four quasi-experimental studies, findings on BMI are also mixed. For example, Holford & Rabe [58]

found significant reductions in obesity prevalence associated with universal free school meals in England, and Jones-Smith et al. 2025 [67] found small but significant reductions in BMI z-score associated with the Community Eligibility Provision (CEP) in the USA, which allows schools in high-poverty areas to offer free meals to all pupils. In contrast, a small quasi-experimental study in Norway [127], with 55 children in the intervention group, found that BMI z-scores increased significantly in the intervention group relative to controls (consuming home packed meals), with no significant effect on waist circumference. The observational evidence base is the largest, with 36 analysed measurements, of which 29 focused on BMI and adiposity for which findings are highly heterogeneous—10 positive, 8 negative, 7 null, and 4 mixed. Negative associations, where participants have higher BMI than non-participants, are concentrated in cross-sectional studies from the USA and likely reflect selection effects, whereby children at higher obesity risk, who in the USA are more often from low-income households, are more likely to participate in subsidised meal programmes³. Several studies report findings that vary by subgroup or meal type: Paxton-Aiken et al. 2012 [98] found that school meal participation was associated with BMI in opposite directions depending on meal type, with breakfast participation associated with higher BMI and lunch participation with lower BMI. Sudharsanan et al. 2016 [117] found no association between school breakfast receipt and obesity in the overall sample, but among children from families below the poverty line, receipt was associated with 2.3 times the odds of becoming obese between fifth and eighth grade. Conversely, Murayama & Nakamura 2025 [80] found significant obesity-reducing effects specifically among children from low socioeconomic backgrounds in Japan, with effects persisting beyond graduation.

³Selection can also run the other way: in universal but fee-based systems, where cost limits uptake, participants may be better off than non-participants, so associations with lower BMI may also reflect selection rather than programme effects. The relevant question is whether participation changes BMI, which cross-sectional comparisons cannot answer, and the content of the meals served also needs to be known before any association can be interpreted.

Table 5. Direction of results across anthropometric outcomes, study designs and 2024 World Bank income setting.

Outcome	HIC Experimental	HIC Quasi- experimental	HIC Observational	UMIC/LMIC Experimental	UMIC/LMIC Quasi- experimental	UMIC/LMIC Observational	LIC Experimental	LIC Quasi- experimental	LIC Observational
BMI / adiposity	Nul: 3	Pos: 2 Nul: 2 Neg: 1	Pos: 10 Nul: 7 Neg: 8 Mix: 4	Pos: 3 Nul: 3	Pos: 4 Nul: 6	Pos: 13 Nul: 6 Neg: 4	—	Pos: 1 Nul: 1	Pos: 7
HAZ/stunting	Pos: 1	—	—	Pos: 5 Nul: 2	Pos: 5 Nul: 3 Neg: 1	Pos: 7 Nul: 7 Neg: 2	—	Pos: 1 Nul: 1	Pos: 3 Nul: 3
WAZ	—	—	—	Pos: 1 Nul: 3	Pos: 3 Nul: 4	Pos: 4 Nul: 2 Neg: 1	—	Nul: 1	Pos: 3 Nul: 1
WHZ	—	—	—	Nul: 2	Pos: 1	Pos: 4	—	Nul: 1	Pos: 2 Nul: 2
Waist circumference	Neg: 2	Nul: 1	Nul: 1	—	—	Pos: 1	—	—	Pos: 1
Height	—	—	Nul: 2	Pos: 1 Nul: 2	—	Pos: 1 Nul: 2	Pos: 1	Nul: 1	—
Weight	—	—	Pos: 1 Nul: 1	Pos: 1 Nul: 2	Pos: 1	Pos: 2 Nul: 1	Pos: 1	Nul: 1	—
Bone area ratio	—	—	Pos: 1	—	—	—	—	—	—
MUAC	—	—	—	—	—	—	—	Pos: 1	—

Pos = Positive; Nul = Null; Neg = Negative; Mix = Mixed; — = not measured in this group; BMI = Body mass index; HAZ = height for age; WAZ = weight for age; WHZ = weight for height; MUAC = Mid-upper arm circumference. Experimental = randomised or cluster-randomised controlled trials; quasi-experimental = non-randomised designs with a comparison group or a before-and-after comparison, including natural experiments; observational = cohort, case-control and cross-sectional studies. The direction of each finding was judged by the author team based on how each study framed it. For example, an increase in BMI, weight-for-age or weight-for-height was recorded as positive where the study framed it as a reduction in undernutrition (usually in low-income settings) and as negative where it was framed as an increase in overweight or obesity (usually in high-income settings).

In upper- and lower-middle-income countries (ULMICs), the evidence across anthropometric outcome assessments spans a broader range of outcome measures than in HICs, with studies examining both overnutrition and undernutrition outcomes. Evidence generally points toward positive effects of school feeding on weight-for-age (WAZ) and height-for-age (HAZ) z-scores, interpreted in these studies as reduced undernutrition, though findings are heterogeneous across study designs. Because higher WAZ can also reflect rising overweight, the interpretation depends on the population's nutritional status at baseline. Among the seven experimental studies, findings lean in a positive direction across anthropometric outcomes. For HAZ, five studies report positive effects on HAZ/stunting [13, 20, 21, 50, 72], while two report null effects [60, 78]. For BMI, findings are more evenly split — three positive [13, 20, 21] and three null [60, 78, 136]. WAZ and WHZ outcomes are assessed in fewer studies and findings are mixed, though positive effects are more common than null for WAZ. Key examples include Anitha et al. [21] that found significant improvements in both HAZ and BMI following a millet-based midday meal intervention in India, and Gelli et al. [50] whose RCT of the Ghana home-grown school feeding (HGSE) programme found improvements in HAZ concentrated among younger children, girls, and the most deprived households. Among the 14 quasi-experimental studies, HAZ and BMI are the most frequently assessed outcomes. For HAZ, most studies report positive effects; for example, the studies by Wang et al. [131] and Ren et al. [104] both found improvements in HAZ after the implementation of school feeding programmes. For BMI and WAZ, findings are predominantly non-significant, with some positive examples. For example, De Silva Perera & Inder [41] showed that children with access to the midday meals had significant increases in weight-for-age. The observational evidence base is the largest, with assessments covering a wide range of outcomes. For BMI, a majority of studies report positive effects—in both Brazil [30] and Ecuador [134] school meal programmes have been associated with lower odds/prevalence of obesity. For HAZ/stunting, findings are more evenly split between positive and null, with a small number of negative findings. Positive HAZ findings include studies from Bangladesh [89] and China [133] observing significant reductions in stunting rates after SMP implementation. The negative HAZ findings from Benin [108] and Nepal [11], are likely attributable to selection bias, whereby children with worse baseline nutritional status are preferentially enrolled in feeding programmes, rather than adverse programme effects. WAZ shows a predominantly positive pattern, with evidence from Pakistan [61] and India [102] reporting significant reductions in underweight. WHZ also shows consistently positive findings across studies from Pakistan [61], India [102,123] and Nigeria [12], including reductions in wasting in Pakistan and India [61,102].

In low-income countries (LICs), all anthropometric outcome assessments come from observational or quasi-experimental studies and the evidence base is heavily concentrated in Ethiopia, with a small number of studies from Sudan, Malawi, and

Uganda. Among the two quasi-experimental studies, Nour et al. [93] found improvements in BMI in Sudan following a school-based daily breakfast intervention, with severe thinness falling from 29.3% to 0%; in this setting, higher BMI indicated reduced thinness. Furthermore, Nkhoma et al. [92] found that school feeding reduced stunting by 0.45 units (HAZ-score) in Malawi, though no significant effects were observed for WAZ or WHZ. The observational evidence, predominantly from Ethiopia, points toward protective effects of school feeding on BMI and undernutrition indicators. Multiple studies from Ethiopia [22,86,135] report significantly better BMI outcomes⁴ among HGSF beneficiaries compared to non-beneficiaries. For HAZ/stunting, findings are more mixed, with roughly equal numbers of positive and null results. Positive HAZ findings include evidence from Ethiopia [59,139] while studies from the same context [45,82] show no significant association⁵. WAZ and WHZ show predominantly positive patterns where assessed, though the number of studies is small. The near-total absence of experimental evidence in this income group represents a significant methodological gap.

Box 4.1 presents an unpublished randomised trial from a refugee settlement and its host community in western Uganda that begins to address this gap. Children who received a daily school porridge in which 25% of the maize was replaced with powdered yellow mealworm grew more in height and gained more fat-free mass over 14 weeks than children who received a standard maize porridge (Boit and Roos, unpublished data). As the findings have not yet been peer reviewed and the study was not identified through our systematic search, it is not included in Table 5.

⁴ In these studies, better BMI outcomes meant higher BMI.

⁵ With Desalegn et al. (2022) highlighting implementation challenges around meal quality and duration as likely explanatory factors.

Box 4.1. Yellow mealworm-enriched school porridge improves linear growth and body composition among children in Uganda.

Authors: Tele Boit, PhD, and Nanna Roos, Associate Professor, Head of research group, PhD from the Department of Nutrition, Exercise & Sports, University of Copenhagen.

The context

Edible insects present a novel and promising solution to address nutritional deficiencies among populations with limited intake of animal-sourced foods. Their nutrient profile is comparable to that of conventional animal-sourced foods, with a protein content ranging from 35% to 61% of dry matter, alongside essential micronutrients [1].

The Refugee Insect Production For Food and Feed Project (REFIPRO, 2022-2026)[2] explored the potential of introducing yellow mealworm as a locally produced, protein source in Kyaka II refugee settlement in Western Uganda. Children in and around Kyaka II face food insecurity and high rates of undernutrition. More than half of children under five years in the settlement are stunted, a sign of chronic undernutrition. In the surrounding host district of Kyegegwa, the prevalence of stunting is around 40% [3]. The dietary diversity is also low, with particularly limited intake of animal-source foods.

As part of REFIPRO, the school meal study evaluated whether yellow mealworm can be incorporated into school porridges as a source of high-quality protein to improve the nutritional status and growth of school children.

The school meal study

The school meal study enrolled in total 342 children, age 6–9 years from two primary schools. Bukere primary school was located in the Kyaka II settlement and Kako primary school in the nearby host community. All children received a daily 400 ml serving of porridge, either with or without yellow mealworm. Each child was randomly assigned to one of the two porridges: A standard maize porridge or porridge where 25% of the maize was substituted with powdered yellow mealworm. The school meal intervention lasted 14 weeks, from June to September 2024.

Both porridges were developed to provide at least 30% of a children's daily energy requirements, in line with World Food Programme guidelines [4]. The intervention porridges nutritional composition is shown in Table 1. The porridges were prepared using micronutrient-fortified maize flour in accordance with Uganda's national standards. Changes in children's height, body composition and micronutrient status were measured to evaluate the impact of including yellow mealworm in the school meals.

What the study found

Linear growth improved

Children who received the yellow mealworm porridge grew taller over the 14-week intervention, gaining 0.24 cm more in height than those who consumed the standard maize porridge, after accounting for differences in age, sex and school. Their height-

for-age Z-score (HAZ) also improved by 0.05 more, indicating a positive change in height relative to age and sex.

Signs of a growth response

Insulin-like growth factor 1 (IGF-1), a hormone involved in child growth, increased significantly more among children who consumed the yellow mealworm-enriched porridge. This provides a possible biological link to the observed improvement in linear growth.

Children gained more lean tissue

Overall, children in both groups gained a similar amount of weight, but distribution of the weight gained differed. Children who consumed the yellow mealworm-enriched porridge gained 0.35 kg more fat-free mass than those who received the standard maize porridge. Fat-free mass includes muscle, organs, bone and other lean tissues that are essential for healthy growth. Although the change in fat mass did not differ significantly between the groups, children in the yellow mealworm group gained a greater proportion of their weight as fat-free mass, while those in the maize porridge group gained a relatively greater proportion as fat mass.

Micronutrient status remained unchanged

At baseline, iron and vitamin A deficiencies were less prevalent than anticipated. Anaemia affected 9.3% of children, while iron deficiency affected 4.6% and vitamin A deficiency was present in fewer than 5% of the children. Neither the yellow mealworm-enriched porridge nor the standard maize porridge significantly changed haemoglobin, iron, or vitamin A status of the children over the intervention period. Given the relatively low prevalence of these deficiencies at baseline, the study could not determine whether the intervention would have a different effect among children with a greater burden of micronutrient deficiency.

High burden of infection

In this setting, infection was common. Approximately half of the children tested positive for malaria, while some had elevated acute-phase biomarkers, indicating infection. During illness and recovery, the body's need for nutrients, including protein, increases. This reinforces the importance of including high-quality protein in school meals for children with a high burden of infection.

The dietary context

Dietary surveys conducted through the study showed that the children's usual diets were low in energy and inadequate in protein relative to their requirements. Incorporating yellow mealworm into the diet provided a source of animal protein with a favourable amino acid profile. The yellow mealworm-enriched porridge therefore increased nutrient density and addressed an important gap in children's usual diets, rather than simply providing more energy.

What this means for school meal programmes

The observed growth and body composition effects in the REFIPRO school meal study highlights the importance of prioritising high-quality protein in school meals in refugee and resource-constrained settings. The findings further reinforce the importance of designing school meal programmes around the nutritional needs of the populations they serve. This is an important reminder that school meals cannot follow a one-size-fits-all approach, as nutritional gaps vary across populations.

Overall, the school meal study demonstrates the potential of incorporating edible insects like the yellow mealworm as a novel strategy in school feeding initiatives to address protein insufficiency and promote healthy growth among vulnerable populations. Providing unbalanced school meals that are insufficient to fill the nutritional gaps of protein and potentially other nutrients may inadvertently lead to unhealthy growth patterns and long-term health risks.

Table 1: Nutrient composition of school porridges per 400 ml serving size

Nutrient	Yellow mealworm porridge	Maize porridge
Energy, kcal	379.5	318.0
Protein, g	15.8	5.7
Fat, g	13.3	6.64
Carbohydrate, g	40.5	51.2
Dietary fiber, g	5.2	5.9
Polyunsaturated fatty acids, g	3.3	3.4
Vitamin A, µg	74.1	110.4
Carotene, mg	0.36	0.51
Vitamin E (eq.), mg	3.6	3.9
Vitamin B1, mg	0.18	0.26
Vitamin B2, mg	0.27	0.09
Vitamin B6, mg	0.18	0.26
Folic acid, µg	29.3	15.5
Vitamin C, mg	0.0	0.0
Sodium, mg	2.5	3.7
Potassium, mg	298.9	196.9
Calcium, mg	19.3	9.10
Magnesium, mg	108.0	71.6
Phosphorus, mg	262.5	152.6
Iron, mg	1.6	0.9
Zinc, mg	3.9	1.5

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4.3.2 Metabolic markers

Table 6 maps the metabolic marker outcome assessments by income setting, study design, and direction of effect (see Box 4.2 for a brief guide to each biomarker and its relevance to NCD risk). In high-income countries, findings vary by study design, with positive effects more apparent in experimental and longitudinal studies, and null findings more common among cross-sectional designs.

In HICs, experimental evidence broadly shows positive or null effects — where positive effects indicate an improvement in metabolic markers, or less deterioration than in the comparison group, and null effects indicate that no difference was detected. A null finding does not establish the absence of harm, as these studies were not designed to test for it. In Denmark, the OPUS School Meal study [38]—a cluster-randomised crossover trial in which children received Nordic school meals rich in fish, vegetables and potatoes for three months followed by their habitual lunch—found that school meals following the New Nordic Diet were associated with significant reductions in mean arterial pressure, total cholesterol, HDL-cholesterol, triglycerides, and insulin resistance (HOMA-IR) compared to home-brought meals (despite an increase in waist circumference, see section on anthropometrics). Similarly, an RCT from Slovenia [28] found a significant decrease in a urinary oxidative stress biomarker among children receiving antioxidant-rich school meals. In contrast, an exploratory secondary analysis from the OPUS School Meal Study [99] found no significant impact on

Box 4.2: Metabolic biomarkers and their relevance to NCD risk

The metabolic markers assessed in this chapter reflect different aspects of cardiometabolic health. For readers unfamiliar with these terms, the following provides a brief guide to what each marker measures and why it matters for NCD prevention.

Blood pressure: Chronically elevated blood pressure (hypertension) is a leading risk factor for cardiovascular disease and stroke, and one of the most prevalent modifiable risk factors globally.

Glucose / insulin / HOMA-IR: These markers reflect how effectively the body regulates blood sugar. Impaired glucose regulation is the hallmark of type 2 diabetes and a driver of cardiovascular disease. HOMA-IR (Homeostasis Model Assessment of Insulin Resistance) specifically captures insulin resistance, an early indicator of metabolic dysfunction that can precede the onset of type 2 diabetes by years or decades.

Metabolic syndrome: A cluster of conditions — including abdominal obesity, high blood pressure, elevated blood sugar, and abnormal blood lipid levels — that together substantially increase the risk of heart disease, stroke, and type 2 diabetes.

Total cholesterol, LDL, HDL, and triglycerides: Measures of blood lipid levels. Elevated LDL (low-density lipoprotein, or "bad" cholesterol) and triglycerides, and reduced HDL (high-density lipoprotein, or "good" cholesterol), are associated with increased cardiovascular disease risk.

Whole blood docosahexaenoic acid (DHA): An omega-3 fatty acid associated with cardiovascular and cognitive health. Lower levels have been linked to increased inflammation and cardiometabolic risk.

Osteocalcin (OC): A hormone produced by bone-forming cells that plays a role in regulating insulin sensitivity and glucose metabolism. It has been proposed as a marker linking bone health to cardiometabolic risk, with higher levels associated with improved metabolic function.

Insulin-like growth factor 1 (IGF-1): A hormone involved in cell growth and metabolism, with complex associations with cardiometabolic health depending on levels and life stage. Both very low and very high levels have been associated with adverse health outcomes.

Oxidative stress (F2-isoprostane): F2-isoprostanes are a marker of oxidative stress — the cellular damage caused by an imbalance between harmful free radicals and the body's antioxidant defences. Elevated levels are associated with increased risk of cardiovascular disease, type 2 diabetes, and other NCDs.

osteocalcin or insulin-like growth factor 1 (IGF-1). From the quasi-experimental evidence, Campos Pastor et al. [33], in a before-after study in Spain, found that metabolic syndrome prevalence reduced from 32% to 19.7% following a school meal intervention. Observational findings are predominantly non-significant or mixed. For example, findings from the USA are inconsistent: one study reported no association between school lunch consumption and lipid or glucose markers [110], while another [66] observed lower fasting triglycerides but no significant impacts on insulin, plasma glucose, LDL, HDL, or blood pressure. In contrast, another study in the same context [74] found that participation in the Community Eligibility Provision (CEP) was associated with a significant reduction in high blood pressure over five years.

In ULMICs, the evidence is limited to five studies (four from Brazil and one from Thailand, with 11 outcome measurements) but leans more consistently in a positive direction across study designs (Table 6). The only experimental study comes from Thailand [118] which reports significant reductions in total cholesterol, triglycerides, and LDL-cholesterol following an egg-supplemented school lunch RCT. From the quasi-experimental evidence, Menezes et al. [81] examined the impact of replacing animal-based and ultra-processed foods in school meals with plant-based options twice a week in Brazilian schoolchildren over one year. Using a pre-post design, they found a significant reduction in serum LDL-cholesterol, increased levels of triglycerides, but no significant effects on fasting blood glucose or total cholesterol. While the absence of a control group limits causal inference, this study suggests that dietary composition within school meals and specifically the inclusion of plant-based foods, may matter for NCD-relevant metabolic risk markers, not just participation per se. The most consistent observational findings come from Brazil with school feeding associated with beneficial impacts for both diabetes related markers [94] and hypertension [52,114]. However, the cross-sectional nature of these studies precludes causal inference. No studies from low-income countries examined metabolic outcomes.

Table 6. Direction of results across metabolic markers, study designs and 2024 World Bank income setting.

Outcome	HIC Experimental	HIC Quasi- experimental	HIC Observational	UMIC/LMIC Experimental	UMIC/LMIC Quasi- experimental	UMIC/LMIC Observational	LIC Experimental	LIC Quasi- experimental	LIC Observational
Blood pressure	Pos: 1 Nul: 1	—	Pos: 1 Nul: 1 Neg: 1	—	—	Pos: 2	—	—	—
Glucose / insulin / HOMA-IR	Pos: 1 Nul: 1	—	Nul: 2	—	Nul: 1	Pos: 1	—	—	—
Metabolic syndrome	Nul: 1	Pos: 1	—	—	—	—	—	—	—
Whole blood DHA	Nul: 1	—	—	—	—	—	—	—	—
Total cholesterol	Pos: 1	—	Nul: 1	Pos: 1	Nul: 1	—	—	—	—
Triglycerides	Pos: 1 Nul: 1	—	Pos: 1 Nul: 1	Pos: 1	Neg: 1	—	—	—	—
LDL	Nul: 1	—	Nul: 2	Pos: 1	Pos: 1	—	—	—	—

HDL	Pos: 1 Nul: 1	Pos: 1	Nul: 2	Pos: 1	—	—	—	—	—
Osteocalcin (OC)	Nul: 1	—	—	—	—	—	—	—	—
IGF-1	Nul: 1	—	—	—	—	—	—	—	—
Oxidative stress (F2-isoprostane)	Pos: 1	—	—	—	—	—	—	—	—

Pos = Positive; Nul = Null; Neg = Negative; Mix = Mixed; — = not measured in this group; HOMA-IR = Homeostasis Model Assessment of Insulin Resistance; DHA = Docosahexaenoic acid; LDL = low-density lipoprotein; HDL = high-density lipoprotein; IGF = Insulin-like growth factor. The direction of each finding was judged by the author team based on how each study framed it: positive indicates a change the study reported as beneficial for metabolic health.

4.3.3 Mental wellbeing

As set out in the chapter introduction, mental health is closely linked to NCDs and may influence NCD risk through behavioural and neuroendocrine pathways. Evidence on the relationship between school meals and mental wellbeing is sparse, with only seven studies (and seven assessments) identified (Table 7): four from HICs, two from UMICs/LMICs and one from a low-income country (Yemen).

In HICs, the only experimental evidence comes from Norway where an RCT [113] examining fish versus meat versus omega-3 supplemented school meals, found no significant differences in mental health scores between groups, though adolescents with higher baseline mental health problems in the fish group showed less improvement in emotional problems. From the quasi-experimental evidence, Bethmann & Cho [29] exploited a natural policy experiment in South Korea in which the free school lunch policy was first abolished and then reintroduced, finding that mental health worsened when the policy was abolished and improved when it was reinstated. Observational findings are mixed with significant positive associations between free school meal receipt and wellbeing among primary school children but no significant association in secondary school pupils in the UK [56], nor in the USA [141].

In UMICs/LMICs, the limited evidence suggests that nutritional quality of school meals may be relevant to mental wellbeing outcomes. An RCT conducted in rural China [73], found that a nutrition subsidy significantly reduced total anxiety scores across multiple dimensions. In contrast, a cross-sectional study from Pakistan [111], found that consumption of school canteen food was associated with 52% higher odds of moderate to severe anxiety compared to home food, which the authors attributed to the poorer nutritional quality of canteen offerings.

Only one study from a low-income country examined mental wellbeing outcomes: a cluster-randomised trial in Yemen [142], which examined the effect of adding milk to a fortified high-energy biscuit school feeding programme. The trial did not find impacts on child mental health, but did find reductions in severe anxiety in caregivers.

Table 7. Direction of results across mental wellbeing, inflammation and gut microbiota outcomes, study designs and 2024 World Bank income setting.

Outcome	HIC Experimental	HIC Quasi- experimental	HIC Observational	UMIC/LMIC Experimental	UMIC/LMIC Quasi- experimental	UMIC/LMIC Observational	LIC Experimental	LIC Quasi- experimental	LIC Observational
Mental health	Nul: 1	Pos: 1	Nul: 1 Mix: 1	Pos: 1	—	Neg: 1	—	—	—
Osteocalcin (OC)	Nul: 1	—	—	—	—	—	—	—	—
IGF-1	Nul: 1	—	—	—	—	—	—	—	—
Oxidative stress (F2-isoprostane)	Pos: 1	—	—	—	—	—	—	—	—
CRP	Nul: 1	—	—	Nul: 1	—	Nul: 1	—	—	—
IL-6	Nul: 1	—	—	—	—	—	—	—	—
Adiponectin	Nul: 1	—	—	—	—	—	—	—	—

AGP	—	—	—	Pos: 1	—	—	—	—	—
Gut microbiota	—	—	—	Pos: 1	Nul: 1	—	—	—	—

Pos = Positive; Nul = Null; Neg = Negative; Mix = Mixed; — = not measured in this group; IGF-1 = Insulin-like growth factor 1; CRP = C-reactive protein; IL-6 = Interleukin-6; AGP = Alpha-1-acid glycoprotein.

4.3.4 Inflammation and gut microbiota

Evidence on the effects of school meals on inflammatory markers and gut microbiota is very limited, with only 11 measurements across six studies identified (Table 7), all from HIC or UMIC/LMIC settings, and findings are largely inconclusive.

In HICs, both assessments come from experimental studies. For example, a cluster-randomised trial of the New Nordic Diet [38] found no significant changes in CRP, IL-6, or adiponectin. In UMICs/LMICs, findings are mixed. The one experimental study [118] found that adding whole eggs to the standard school lunch significantly increased the relative abundance of beneficial gut bacteria, including *Bifidobacterium* and *Prevotella*. A quasi-experimental pre-post study from Brazil examining a plant-based school meal intervention [81] found no significant impact on gut microbiota composition. Among the observational studies, neither the study from Ghana [10] nor that from India [70] showed significant differences in C-reactive protein between school meal participants and non-participants, though the latter observed a reduction in alpha-1-acid glycoprotein in the intervention group. No studies from low-income countries examined these outcomes.

Outside the school meals literature, systematic reviews of observational studies in children and adolescents have linked higher intake of ultra-processed foods, added sugars and saturated fats to higher levels of inflammatory markers such as C-reactive protein, interleukin-6 and tumour necrosis factor-alpha [144], and sustained ultra-processed food intake to greater adiposity [145]. These associations do not establish causation, but they are consistent with the WHO recommendation to reduce foods that do not contribute to a healthy diet in school food environments [146], and they suggest that the level of processing of foods served in schools is worth measuring in future studies.

4.3.5 Medium- and long-term outcomes

The medium- and long-term evidence base consists of 14 studies (Tables 8 and 9). Of these, eight [19,25,34,36,48,51,53,105] examine medium-term effects (follow-up under 20 years) on outcomes that are almost solely BMI-related, while six (35,57,76,100,115,132, 143⁶) examine longer-term impacts (follow-up of 20 years or more) on general or self-reported health, BMI, and linear growth. Of the six long-term studies, three [76,115,132] examine NCD mortality or morbidity directly of which only one [76] does so empirically (using real world data with longitudinal design and not modelling). This section predominantly includes longitudinal observational studies and quasi-experimental

⁶This result was not found during scoping as it is still under peer review. Their findings were brought to our attention by a White Paper contributor and was included here, although it is not included in any text or table summaries of the reviewed literature (132 publications).

designs, alongside three modelling studies. The overall picture is mixed, but a clearer signal emerges when studies are considered by whether they examine the effect of meal provision alone or the effect of introducing or improving nutritional standards for school meals.

Among studies examining the effect of meal provision, in high-income countries the medium-term evidence (<20 y follow up) is mixed. In a longitudinal difference-in-differences study from the USA [19], no significant overall effect of universal free school meals provided through the CEP was found on BMI z-score, overweight, or obesity, though in the low-income subgroup, CEP reduced the probability of overweight by 3.1 percentage points over five years of schooling. In contrast, Capogrossi & You [34], following children from 1st to 8th grade, found that long-term participation in both the School Breakfast Program and NSLP in the USA was associated with a statistically significant increase in the probability of being overweight, with effects more pronounced in rural and Southern populations. Miller [84], in a six-year prospective cohort study also from the USA, found that additional school lunches were associated with lower initial BMI but faster rates of BMI growth over time.

Table 8. Medium-term impacts.

Outcome	HIC Experimental	HIC Quasi- experimental	HIC Observational	HIC Modelling	UMIC/LMIC Exp...	UMIC/LMIC Quasi- exp...	UMIC/LMIC Obs...	UMIC/LMIC Modelling	LIC Exp...	LIC Quasi- exp...	LIC Obs...	LIC Mod...
Under-nourishment	—	—	—	—	—	—	—	—	—	—	—	—
Deaths/DALYs from NCDs	—	—	—	—	—	—	—	—	—	—	—	—
HAZ/Height	—	—	—	—	—	—	—	—	—	—	—	—
Self-reported health	—	—	—	—	—	Pos: 1	—	—	—	—	—	—
BMI	—	Pos: 3 Nul: 1 Neg: 2	—	Pos: 1	—	Nul: 1	Pos: 1	—	—	—	—	—
Mental health	—	—	—	—	—	Pos: 1	—	—	—	—	—	—

Pos = Positive; Nul = Null; Neg = Negative; Mix = Mixed; — = not measured in this group; BMI = Body mass index; HAZ = height for age; WAZ = weight for age; WHZ = weight for height; DALYS; Disability Adjusted Life Years; NCDs = Non-communicable diseases.

The longer-term HIC evidence is similarly inconclusive. Lundborg et al. [76], following children for up to 43 years after exposure to universal free school meals in Sweden, found that nine years of full exposure increased the likelihood of near-perfect health in early adulthood by 4 percentage points, but no significant long-term NCD effects were found in later adulthood, which the authors attribute partly to the study population being relatively healthy and non-disadvantaged, and partly to the follow-up period being too early to capture chronic conditions such as cardiovascular disease. Hinrichs [57], following participants for up to 30 years after NSLP exposure in the USA, found no statistically significant effects on height, BMI, or weight status, though a 10-percentage-point increase in exposure was associated with a lower probability of poor or fair self-reported health in men. As in Lundborg et al., the author cautions that health was measured at a relatively young age, likely too early to detect chronic disease outcomes. Lastly, Peterson [100], in a repeated cross-sectional ecological panel study linking adult overweight and obesity prevalence in 1984–2008 to estimated NSLP participation during schooling, with up to about five decades between exposure and outcome, found that a one percentage-point increase in estimated NSLP participation during schooling was significantly associated with a 0.29 percentage-point increase in adult overweight and obesity prevalence among the pooled 18–64 year cohort (schooling 1925–2007), and a comparable but smaller association (0.14 percentage points) among the oldest cohort (ages 50–64, schooling 1925–1975). Associations were not statistically significant among younger cohorts (ages 18–34 and 35–49), leading Peterson to conclude that the NSLP might have influenced population health historically, in this case towards higher adult overweight and obesity, and to call for longitudinal studies of more recent cohorts.

In upper- and lower-middle-income countries, the medium-term evidence is more encouraging. Gharge et al. [51], in a seven-year longitudinal cohort study in India, found that early and persistent mid-day meal exposure reduced the likelihood of low BMI z-scores in adolescence. In addition, Fang & Zhu [48], in a quasi-experimental study in China spanning approximately ten years, found that programme exposure increased the probability of reporting excellent health status by 7.2 percentage points and reduced negative emotion by 0.112 standard deviations, though no significant effect on BMI was observed. The long-term evidence from this income group, although scarce, is equally positive. Chakrabarti et al. [35], in a quasi-experimental intergenerational study in India with a 23-year follow-up period, found that children born to mothers with full Mid-Day Meal exposure had significantly higher HAZ (+0.40 SD), with the largest effects among the poorest households (+0.50 SD)—providing rare evidence of intergenerational nutritional benefits. No studies from low-income countries examined medium- or long-term health outcomes, representing a critical gap in the evidence base.

Taken together, the evidence on meal provision alone presents a mixed picture, particularly in high-income settings. A more consistent signal emerges from studies

examining what happens when nutritional quality is explicitly improved through the introduction of standards. Chandran et al. [36], in a cohort study with interrupted time-series analysis, found a significant decrease in BMI z-scores following full implementation of the Healthy, Hunger-Free Kids Act (HHFKA) in the USA (2010 legislation that strengthened nutritional standards for school meals and other foods sold in schools), with similar decreases in adolescents and lower-income households. Richardson et al. [105], in a complementary longitudinal cross-cohort analysis, found that before HHFKA, NSLP participants had significantly higher BMI by grade 5, but that this association was entirely eliminated after implementation — suggesting that improved nutritional standards attenuated a previously obesogenic effect of participation. Two modelling studies extend this finding to estimate population-level impact: Gortmaker et al. [53] estimated that the US nutrition standards for school meals, reaching about 28 million children, would prevent about 1.8 million cases of childhood obesity in 2025 at a cost of \$53 per unit of BMI reduced, while Wang et al. [132], in a modelling study with the longest projected time horizon of 50 years, estimated that aligning US school meal standards with the 2020–2025 Dietary Guidelines for Americans would prevent about 10,600 deaths a year from cardiovascular disease, diabetes and cancer in adulthood, under assumptions about full compliance by schools and the persistence of childhood dietary changes into adulthood. A national microsimulation of the 2025 Czech school meal standards reform⁷ which modelled changes in free sugars, sodium and whole grains in school lunches, estimated a mean BMI reduction of 0.11 kg/m² among children aged 7–16 years and about 6,500 cases of chronic disease prevented over ten years in adulthood, assuming that 35% of the dietary improvement persists [143]. Echoing these findings, a global modelling study [115] estimated that providing every school-age child with a healthy and sustainable school meal by 2030 could reduce undernourishment, here meaning inadequate intake of energy, vitamins or minerals, by an average of 24% in low- and middle-income countries, prevent approximately 120 million cases of undernourishment, and avoid 0.8–1.2 million deaths a year, predominantly from coronary heart disease and cancer. This estimate assumes that children retain, at least in part, their preference for healthy foods into adulthood. Modelling studies of this kind provide the most direct, if modelled, support for the White Paper's central argument that school meals, when designed well and delivered at scale, could contribute meaningfully to the prevention of diet-related NCDs, but their results depend on assumptions that have not yet been tested empirically.

⁷ This result was not found during scoping as it is still under peer review. Their findings were brought to our attention by a White Paper contributor and was included here, although it is not included in any text or table summaries of the reviewed literature (132 publications).

Table 9. Long term impacts.

Outcome	HIC Experimental	HIC Quasi- experimental	HIC Observational	HIC Modelling	UMIC/LMIC Exp...	UMIC/LMIC Quasi-exp...	UMIC/LMIC Obs...	UMIC/LMIC Mod...	LIC Exp...	LIC Quasi- exp...	LIC Obs...	LIC Mod...	Global Mod...
Under-nourishment	—	—	—	—	—	—	—	—	—	—	—	—	Pos: 1
Deaths/DALYs from NCDs	—	Nul: 2	—	Pos: 1	—	—	—	—	—	—	—	—	Pos: 1
HAZ/Height	—	Pos: 1 Nul: 1	—	—	—	Pos: 1	—	—	—	—	—	—	—
Self-reported health	—	Pos: 2	—	—	—	—	—	—	—	—	—	—	—
BMI	—	Nul: 2	Nul: 1	Pos: 1*	—	—	—	—	—	—	—	—	—
Mental health	—	—	—	—	—	—	—	—	—	—	—	—	—

Pos = Positive; Nul = Null; Neg = Negative; Mix = Mixed; — = not measured in this group; BMI = Body mass index; HAZ = height for age; WAZ = weight for age; WHZ = weight for height; NCDs = Non-communicable diseases; *Findings are still under peer review.

Building on this modelling evidence, a comparative-risk scenario analysis was commissioned specifically for this White Paper to explore the conditions under which a long-term mortality benefit from school meals might emerge (Box 4.3). The analysis confirmed that any mortality benefit is structurally delayed by decades, staying negligible before around 2060 and emerging more fully towards the end of the century. The single biggest driver of the modelled effect was the assumed persistence of childhood dietary gains into adulthood, that is, the share of the improvement in children's diets assumed to be retained in adult diets: moving from 25% to full persistence increased projected health gains threefold. Under reference scenarios that assumed hypothetical improvements in children's diets across ten countries, improvements that would require programmes to change children's overall diets rather than only provide a meal, diet-linked premature mortality fell by roughly 0.3–1.2 percentage points by 2095. When the analysis was grounded in actual school menus from five countries (India, Nepal, Cambodia, Guatemala, and Lesotho) projected effects were more modest, reducing diet-linked mortality by 0.02–0.04 percentage points by 2095, equivalent to 1–8% of the effect of moving adult diets fully to their ideal levels. Notably, not all meals were uniformly beneficial: sodium and red meat in some menus partly offset projected gains, and for the Cambodian menu analysed the modelled net effect was a small increase in diet-linked mortality. This is a modelled scenario about menu composition, not an observed effect of school meals, and it is an argument for binding nutritional standards for school meals rather than against provision. The analysis underscores that school meals are not a panacea for NCD prevention, but that well-designed programmes delivering meals of high nutritional quality could, if their dietary benefits persist beyond childhood, serve as a meaningful launch pad for broader efforts to shape dietary patterns and reduce long-term NCD risk.

Box 4.3. Modelling the long-run impact of school meals on chronic disease

Commissioned analysis for this White Paper (William Msemburi and David Watkins; technical details in Annex 4)

Background. Noncommunicable diseases (NCDs) are a growing threat in most countries, and mortality from NCDs is driven in large part by dietary risks. There is good reason to expect that early intervention to improve diets among children and adolescents via school feeding programmes could reduce NCD risk later in life. However, it is challenging to establish empirically whether school meals could reduce deaths from NCDs. Mortality reductions, if they exist, would emerge long after childhood exposure, and no trial can observe school feeding in childhood and NCD mortality in adulthood across the sort of time horizon required. Yet policymakers and advocates need credible estimates of the long-run health impact of school meals to assess their role in the national response to NCDs. To explore this question, a two-part comparative-risk scenario analysis was commissioned specifically for this White Paper:

a hypothetical analysis (Part 1), followed by an analysis anchored in real, food-composition-matched school menus from five countries (Part 2).

Analytic approach. The analysis asked a conditional question: if school meals improve children's diets, how much lower might premature mortality from diet-linked causes be over the long run, and when would those benefits emerge? Part 1 explored this using hypothetical dietary shifts due to implementation of school feeding programmes across ten countries that illustrate a range of dietary variation worldwide. Part 2 replaced those assumptions, where the data allowed, with the actual food content of school menus served in India, Nepal, Cambodia, Guatemala and Lesotho, and compared the resulting effect with benchmarks in which diets improved either 50% or 100% of the way to their optimal level, with optimal being defined by theoretical minimum (harmful) or maximum (protective) levels of different dietary components estimated in the Global Burden of Disease Study. Both parts linked an assumed or menu-derived dietary shift; a persistence assumption for how much of a childhood gain would carry into adulthood; a comparative-risk module translating adult dietary exposure into cause-specific mortality hazards; and a demographic projection module. The primary quantity of interest was the probability of dying between ages 30 and 70 from nine diet-linked causes (ischaemic heart disease, type 2 diabetes, stroke, several gastrointestinal cancers, and chronic kidney disease) among child cohorts exposed between 2025 and 2050. Children were assumed to be exposed at ages 5–15 years (up to 11 school years). In Part 2, the food served per meal was converted into an annual change in diet assuming 180 school days, 80% programme coverage, 90% uptake and 75% net addition to the diet; this change was applied to country-specific adult dietary exposure distributions from the Global Burden of Disease Study, which served as the baseline diet. The assumed dietary improvement therefore depends on meals being designed to close dietary gaps, through appropriate nutritional standards, and being consistently delivered and eaten; provision of a meal is not in itself equivalent to an improvement in dietary intake.

Findings. Part 1 confirmed that any mortality benefit is structurally delayed by decades, staying negligible before around 2060 and emerging more fully towards the end of the century. Further, whether a childhood dietary gain persists into adulthood is the single biggest driver of the modelled effect. For example, moving persistence from 25% to 100% of childhood diet would increase the health gains by a factor of 3. By contrast, changing the assumed dietary shift from a conservative to an ambitious shift would increase the health gains by a factor of 2.5. Under the Part 1 reference scenario, this would reduce diet-linked mortality by roughly 0.3–1.2 percentage points by 2095 across the ten countries. Country differences in mortality reduction would be driven by baseline mortality, diet, and demography.

Part 2 replaced the hypothetical scenarios above with real school menus from five countries, with each ingredient matched to food-composition tables to estimate its food-group and nutrient content. The potential impact would be much more modest than found in Part 1: school meals would reduce diet-linked mortality by only 0.02–0.04 percentage points by 2095, equivalent to 1–8% of the effect of moving adult diets all the way to their ideal levels. Of note, school meals are not uniformly protective on health; some meals contain ingredients such as added sodium and red meat. When these risks were incorporated into the analysis, they partly offset the benefits, and for the Cambodian menu the modelled net effect was a small increase in diet-linked mortality. This finding concerns menu composition and supports binding nutritional standards, not withdrawal of school meals.

Interpretation and caveats. The analysis asked “what-if” questions and, in the case of Part 2, was grounded in real school menu data, but it did not produce causal estimates of programme impact. It measured food served, not food eaten: fixed assumptions for coverage, uptake and net addition were applied, but consumption, plate waste and displacement of other foods were not observed. Part 2 also assumed full persistence of the dietary changes into adulthood, which might not be realistic: as Chapter 3 shows, no study has yet tracked school meal-related dietary changes into adulthood. On the other hand, the analysis focused on mortality from a narrow set of diet-related NCDs that affect older adults; the benefits of school meals on mortality among younger individuals and on nonfatal health outcomes were not considered. In these respects the analysis may have underestimated the overall health impact of school meals, while the assumption of full persistence may have overestimated it.

Conclusion. School feeding programmes could change childhood diets enough to lead to a reduction in later-life NCD risk, though the absolute effect would be modest. The size of this effect would depend critically on the degree to which childhood dietary changes persist into adulthood. The quality of the menu would also matter: in the model, menus high in harmful dietary components such as sodium and red meat could lead to increases, rather than decreases, in NCD mortality, which underlines the importance of nutritional standards. School meals are not a panacea for NCDs, but they could serve as a launch pad for broader public health programmes to shape dietary patterns among children and adolescents and achieve greater public health impact.

See technical details for this analysis in Annex 4.

4.4 Summary

The evidence reviewed in this chapter spans 132 unique studies and 149 outcome assessments, covering 44 countries, across five outcome categories, reflecting an important body of literature on the health effects of school meal programmes. In addition to this empirical evidence base, the chapter draws on findings from a comparative-risk scenario analysis commissioned specifically for this White Paper, which modelled the potential long-run impact of school meals on diet-linked NCD mortality under a range of assumptions.

Across outcome categories, the most consistent evidence of benefit comes from middle-income country settings, predominantly China and Brazil (UMICs) and Ghana and India (LMICs), where school feeding is associated with positive impacts particularly on BMI, WAZ and HAZ. In high-income settings, mainly from the USA, Japan and Scandinavia, the anthropometric evidence is largely null or mixed, though studies examining universal free school meals and improved nutritional standards point toward modest protective effects against overweight and obesity. Evidence on metabolic markers is limited, but findings from experimental and quasi-experimental studies, mainly from Denmark, Thailand and Brazil, suggest beneficial effects on blood pressure, cholesterol and insulin resistance when meals are of high nutritional quality. No formal quality or risk-of-bias assessment was carried out, so these findings should be read with caution. The nutritional composition of school meals, and the share of children's daily intake they provide, are rarely reported in enough detail to judge what children actually consumed. This limits how far differences in outcomes can be attributed to meal quality, and underscores the need for future studies to report meal content, the number of meals per day, the share of daily energy provided and the number of days served, ideally against defined nutritional standards.

Evidence on mental wellbeing is sparse and inconsistent, and no firm conclusion can be drawn. However, given that school meals seem to improve food security in HICs as per the evidence covered in Chapter 2, and that food security has been associated with better mental health outcomes [5,6], an indirect pathway can plausibly be drawn. Other mediating pathways, including social dimensions of shared mealtimes, are also plausible but rarely examined as mechanisms in the school meals literature specifically. Inflammation and gut microbiota also remain poorly studied overall.

The medium- and long-term evidence is mixed and, critically, the majority of empirical studies measure BMI outcomes over relatively short follow-up periods. A strong signal in this outcome category comes from studies examining nutritional standards rather than provision alone, and from modelling studies, which estimate substantial benefits under assumptions that have not yet been tested empirically [53,115,132]. The commissioned scenario analysis (Box 4.3) further suggests that well-designed school meals have the

potential to reduce diet-linked NCD mortality over the long run, with the size of the benefit dependent on the persistence of childhood dietary gains into adulthood and the nutritional quality of meals delivered, underscoring once more that what is served matters as much as whether meals are provided. In sum, the available evidence does not yet allow a definitive answer to whether school meals improve long-term NCD outcomes, but it does not support a conclusion of no effect either. Conditions for long-term benefit exist and are partially evidenced, but direct empirical confirmation remains elusive given the time horizons involved and the limitations of the current evidence base. Several questions remain open: how much a healthy school meal during the school years contributes to lower NCD risk independently of diet in the rest of life; how far school meal programmes change dietary practices beyond school age; and how far a healthy diet from school age can offset NCD risk associated with undernutrition in early life. The cost and affordability of healthy diets, for programmes providing meals and for families sustaining such diets throughout life, also bear on whether any benefit can be realised.

Several important gaps in the evidence base deserve attention. First, the dominance of cross-sectional study designs across all short-term outcome categories limits causal inference. Many trials also evaluated interventions lasting two years or less, which may understate the effects of programmes that run for many years. Second, low-income countries have very limited representation in the evidence base overall and are entirely absent from the evidence base for metabolic markers, inflammation, gut microbiota, and medium- and long-term outcomes, and are represented by a single study on mental wellbeing. Even within anthropometric outcomes, where LIC evidence does exist, studies are largely observational and concentrated in a small number of countries—predominantly Ethiopia. This represents a critical gap given the rising NCD burden in LICs, where health systems are often less equipped to manage the growing demands of chronic disease, making preventive strategies such as school meal programmes particularly important. Furthermore, the number of studies examining medium- and long-term health outcomes remains limited, and most empirical studies have follow-up periods of fewer than 20 years, which is insufficient to capture the time scales over which NCD risk typically develops. Importantly, of the six long-term studies (follow-up period 20+y), only one study—Lundborg et al. (2022)—examines NCD mortality or morbidity empirically, with the remaining evidence on these outcomes relying on modelling studies [115,132].

A further limitation is that the majority of medium- and long-term studies (11 of 14) focus on BMI as their primary outcome. While BMI is a useful and widely available measure, it does not necessarily fully capture underlying metabolic health; individuals with a healthy BMI may carry significant cardiometabolic risk, and conversely, BMI improvements may not necessarily translate into reduced risk of cardiovascular disease or type 2 diabetes. This considerably limits the conclusions that can be drawn about the lasting health

impacts of school meal exposure. However, the studies identified here largely follow relatively healthy populations over follow-up periods that may be too short for chronic NCD outcomes to have fully emerged. Evidence from other welfare programmes targeting more disadvantaged populations has shown positive long-term health impacts [147,148], suggesting that school meals could yield similar benefits, though this remains an open empirical question. Lastly, intervention heterogeneity in meal quality, frequency and programme design makes cross-study synthesis challenging. The studies also test different things: participation in, or universal provision of, school meals as delivered, and interventions that change meal composition or nutritional standards. Findings should be interpreted with this distinction in mind, which underscores the importance of Chapter 5's focus on enabling conditions.

5. Enabling conditions: school meals modalities and programme design

5.1 Introduction to the chapter

This chapter completes the White Paper's sequential argument. Chapter 2 established that school meal programmes can deliver immediate benefits to nutrition, education, agriculture and social protection, with variation across income settings and outcomes. Chapter 3 examined whether school meal exposure influences eating behaviour beyond the school meal itself, finding suggestive but limited evidence, and Chapter 4 explored the biological and longer-term health consequences of school meal provision across the life course. What remains to be addressed is: Under what programme design, governance, and food environment conditions are the preventive benefits for NCDs and CMDs identified in the preceding chapters most likely to be realised (Figure 2).

This question matters for NCD/CMD prevention because the effects documented in the earlier chapters are unlikely to be uniform. If school meal programmes were evenly interchangeable, the evidence in Chapters 2 to 4 could be applied to any programme in any country context. If, however, the same intervention produces different dietary, behavioural and health effects depending on what is served, who procures and prepares it, how the wider school food environment is regulated and what social determinants surround it (household poverty, food security, nutrition and health, food supply, governance capacity), then design choices become another central factor of how school meals may contribute to healthier dietary patterns.

There is also an open qualitative conceptual question: how might school meal programmes design also influence NCD/CMD risk and prevention? One pathway is through the meal itself, by improving nutritional status and health during school age, as discussed in Chapters 2, 3 and 4. A second is through the food supply chain and food environment in which the programme operates. These systems shape what is produced, procured and made available to children and their communities, potentially affecting health beyond the school meal itself. This is the point Chapter 5 addresses.

Finally, a third pathway is through dietary patterns established at school and whether these patterns persist into adulthood. Their persistence may depend on the wider food environment and on people's access to and ability to afford nutritious foods. These pathways are not mutually exclusive, but they point to different programme priorities. The evidence reviewed here rarely allows their relative contributions to be distinguished, and a full assessment of all these pathways is beyond the scope of this White Paper.

This chapter focuses on programme design at three levels of specificity: modality (the broad category of delivery, defined mainly by who supplies the food and how governance is organised), model (a specific way in which a modality is operationalised) and component (an individual operational building block, such as a procurement method or

a menu-setting process). It examines four design dimensions: who procures food and from where, including what type of food is provided; how meals are prepared and served; the food environments children experience within different programmes; and how programme design is supported by policy and legal structures.

The chapter draws on a scoping review, the same method used in the previous chapters. Studies that compare programme modalities or components directly, whether randomised trials or meta-analyses, are uncommon. Most evidence on programme design is descriptive, administrative or policy-oriented by nature, and much of it is qualitative. Importantly, in most cases, programme design is not explicit, and it must be inferred from the descriptions or by the legal framework of the implementing country and/or institution. No formal assessment of risk of bias could be carried out. Findings are labelled with the evidence grades used across this White Paper (see Chapter 6), so that terms such as “consistent” and “suggestive” carry the same meaning throughout.

Chapter 5 does not estimate the effects of different programme designs on NCD/CMD outcomes, nor does it establish that any particular design feature prevents NCDs or CMDs. Its contribution is to identify the programme design, governance and food environment conditions that shape the pathways through which school meals could influence these outcomes. By examining differences in procurement, food provision, preparation, food environments, and policy and legal frameworks, the chapter identifies which conditions are associated with the availability and consumption of healthier foods and with the development of healthier dietary patterns. It therefore provides the link between the health and dietary pathways identified in Chapters 2–4 and the enabling factors and the social and policy determinants that may enable or constrain programme features. Where evidence is consistent, Chapter 5 can identify conditions that warrant greater attention in programme design; where evidence is limited or indirect, it highlights the specific gaps that need more research.

The chapter proceeds in five sections: a set of key concepts that anchor the analysis (Section 5.1.1); an overview of the evidence base (5.2); a synthesis of evidence by outcome domain, with study design and income setting identified for each (5.3); a framework linking enabling conditions, programme design and outcomes (5.4); and a summary of what the evidence supports, its gaps and the resulting research priorities (5.5).

5.1.1 Key concepts of programme design: modality, model, and component

The evidence reviewed in this chapter is organised around three nested concepts—modality, model, and components, which together make up programme design. Table 10 sets out these concepts before the evidence synthesis to establish a common analytical vocabulary. In this framework, modality refers to a broad policy and governance category of programme organisation; a model refers to a specific technical configuration through which that modality is implemented; and components are the operational elements that constitute and deliver that configuration.

These three terms describe different levels of specificity, from broadest to most granular:

Table 10. Modality, model and component: definitions and examples

Term	What it captures	Example
Modality	The broad category of school feeding delivery — defined mainly by who supplies the food and how governance is structured. There are five modalities in this White Paper.	Brazil’s home-grown school feeding programme (HGSF), managed by a Federal agent within the ministry of Education, which has an intersectoral agenda with the ministries of Health (nutrition guidelines) and Agriculture (food supply chain)
Model	A specific way a modality is operationalised — either a generic design variant (e.g., centralised vs. decentralised) or a real, named programme in each part of the country.	Ghana’s centralised school meals programme, which is also piloting some HGSF modality components (for example, local purchase of fresh food as stated by national nutrition guidelines).
Component	An individual operational building block that, combined with others, makes up a model — procurement method, preparation and delivery arrangement, governance feature, menu-setting process.	United States’s public school meals programme, with different centralisation models presenting different components, such as targeting, type of food purchased locally, school menu composition, level of subsidy in different states.

Source: developed for this White Paper from the scoping review (2026), formalising the distinction implicit in Klotz-Silva et al. [32], Alvarenga et al. [40], and HLPE [36]; WFP [28] and GCNF [45].

The five modalities defined in Box 5.1 below are not exhaustive, and they overlap analytically: they are defined along different axes (sourcing for HGSF, preparation for catering, destination for transfers, regulation for the non-institutionalised environment), so one programme can fit more than one label and was classified by its predominant feature. The classification is intended, rather, to make visible a methodological point: programmes classified under the same modality label can differ substantially in governance (who sets menus), sourcing (local vs. centralised procurement), and delivery mechanism — which is exactly the kind of variation that forced inference from components rather than a stated modality label in many of the studies reviewed.

This distinction is useful because school meal programmes are rarely described in the literature through a single, standardised label. Rather, studies often report individual programme characteristics—such as where food is procured, who prepares meals, whether food is cooked on-site, or who is responsible for procurement and menu decisions—from which the underlying configuration must be reconstructed. This is consistent with the way global monitoring frameworks disaggregate school meal

programmes into their constituent features, including food sources, procurement arrangements, delivery practices and governance structures, as described in the annual school meals reports from the World Food Programme (WFP) [28] and the Global Child Nutrition Foundation (GCNF) [45], for example.

In the scoping review, programme delivery modalities were recorded as explicitly stated or inferred from the programme's documented components and underlying operational arrangements. Where the available study did not provide sufficient information to establish the modality, related publications and programme documentation were consulted to clarify the arrangement. This approach is particularly important because programme labels do not necessarily capture the way food is sourced, prepared, procured or governed. The WFP State of School Feeding Worldwide 2024 [28] and GCNF Global Survey [45], for example, treat food access as a multidimensional characteristic, distinguishing local food purchases from domestic and foreign in-kind contributions, rather than reducing programmes to a single procurement label.

It is important to state that globally, nearly 99% of the total cost of school meal programmes, estimated at USD 80+ billion annually, is financed directly through national government budgets, while international cooperation and external assistance account for only a residual share of around 1% or less. However, this pattern varies substantially by country income level: in low-income countries, national governments finance a smaller proportion of programme costs, around 42–45%, making these programmes more dependent on external support, whereas in middle- and high-income countries, financing is predominantly or almost entirely provided by national governments and their accredited institutions, which also manage the entirety of the school meals and the food purchase [28].

The distinction is important because financing is not merely a question of who pays; it can shape how programmes are designed, implemented and sustained. For NCD/CMD prevention, this matters because the effects documented in Chapters 2–4 are mediated by programme characteristics and context. The degree of institutionalisation and stable public financing may influence procurement, food standards, infrastructure and programme continuity. Moreover, school meals are inherently multisectoral, extending beyond Education to engage Health, Agriculture, and Social Protection, for example. These institutional and financing arrangements can therefore shape the foods served, the wider food environment and the programme's potential contribution to healthier dietary patterns and longer-term NCD/CMD risk [14,30,32].

Such differences can affect what foods are purchased and served, whether meals can incorporate local or minimally processed foods, the capacity to regulate the school food environment, and the programme's ability to operate consistently over time. The income-related financing distinction therefore reinforces the chapter's broader argument: school meal programmes should not be treated as interchangeable interventions, because their potential contribution to NCD/CMD prevention depends

partly on the institutional and resource conditions under which they are designed and delivered.

Each modality is therefore characterised by two features that are particularly useful for differentiating programme configurations: food supply—including where food originates, how it is procured, and how it moves through the supply chain—and governance—including who holds responsibility for procurement, menu design, implementation and the setting or enforcement of standards. This framing also recognises that school meals are embedded within the food systems that supply them, with procurement acting as an important mechanism linking programme design to local markets, producers and broader food-system outcomes.

There is no universally agreed glossary or common classification system for describing school meal programmes. Terms such as modality, model, hybrid, home-grown school feeding, and catering are used differently across countries and organisations, sometimes referring to distinct aspects of programme design. This White Paper therefore uses the following terminology as an analytical framework to distinguish how meals are sourced, procured, prepared and delivered. The definitions are specific to the framework used in this chapter and are intended to make comparisons across programmes more consistent, rather than to establish a universal taxonomy.

Box 5.1. The five programme modalities used in this White Paper

1. Home-Grown School Feeding (HGSF)

School meals procured from local or domestic smallholder farmers, linking school meals to local agricultural production. As used here, HGSF is defined by three features: the supplier (schools, local governments or a national agency), the place of production (local or domestic) and the producing entity (smallholder or family farmers). Programmes with a mandated or targeted share of procurement from local family farmers, as in Brazil's HGSF, are counted as HGSF even when they are combined with centrally supplied foods. HGSF is not a homogeneous group: programmes differ in who manages them, who procures, through which supply chain and supplier, and where the commodities originate.

Local sourcing under any modality also depends on the availability, affordability and seasonality of local produce, and HGSF is not by definition a healthier programme, since what is procured matters for health as much as where it is procured. HGSF can span three design models that differ mainly in where procurement decisions are made:

- Decentralised — schools or local governments purchase directly from smallholders within a defined radius. Food supply is tied to local harvest cycles: strong dietary diversity potential but exposed to seasonality and local supply shocks.
- Centralised — the national government purchases and distributes domestically sourced food to schools. Food supply depends on aggregation, warehousing, and transport logistics rather than any single local harvest, giving more standardisation but greater pipeline risk.
- Semi-centralised — a mix of centrally and locally sourced components depending on the produce and the location of schools, combining both supply logics and inheriting some risk from each.

This distinction between decentralised, centralised and semi-centralised procurement is not unique to HGSF and can also apply to other school meal modalities, including programmes in the United States and India, where procurement arrangements may combine different levels of central and local decision-making regardless of whether food is sourced from smallholder or local farmers.

2. Hybrid

Combines centralised procurement or menu guidelines with local sourcing, preparation, or a mix of different models. It can also combine centralised sourcing and storage with cooking in individual schools, without any local sourcing. Food supply typically runs on two parallel tracks — centrally distributed staples and locally sourced perishables — so reliability depends on how well the two are coordinated. This modality does not by definition source locally, and often lacks strong interministerial coordination between education, health, and agriculture.

3. Catering

Meals prepared at scale in central kitchens or by contracted catering companies and distributed to schools or cooked on school premises by school staff or community cooks (decentralised catering). Food supply relies on large-scale commercial or wholesale supply chains, decoupled from local production and largely insulated from seasonality, offering consistency but with weak links to local food systems. Catering usually includes manufactured and industrialised food and does not by definition source locally.

4. Food transfer / take-home rations

Food is distributed to households rather than consumed at school; common in humanitarian and social-protection contexts. Food supply follows conventional food-aid logistics — centrally procured, often fortified or long-shelf-life commodities distributed in bulk — and is not tied to local production or the school food environment. This modality does not by definition source locally.

5. Non-institutionalised food environment

Food obtained outside the formal programme — canteens, vendors, retail outlets, food brought from home — typically weakly regulated. There is no programme-level food supply chain: availability depends entirely on private vendors' or retailers' independent sourcing decisions, which sit outside any programme control. This modality does not purchase from home-grown produce and often carries the added risk of ultra-processed food availability on school premises.

5.2 Overview of the evidence base

Records were screened from four databases, including two based in the Global South, in two sequential stages⁸. At title and abstract level, 1,167 records were assessed against predefined eligibility criteria following the Population–Concept–Context (PCC) framework recommended by the Joanna Briggs Institute, with 338 excluded at this stage for wrong population (non-school aged children) or concept (non-school meals programmes). Of the 829 reports sought for retrieval, 35 could not be obtained, leaving 794 assessed for full-text eligibility. A further 678 were excluded at full-text stage — the leading reasons being wrong population or setting, wrong concept (not a school meals programme or a stand-alone/isolated intervention not considered a programme), and ineligible publication type (opinion pieces, policy statements without primary data) — yielding a final corpus of 116 unique studies (see Annex 2 for PRISMA diagram and Annex

⁸ This chapter's search included Portuguese- and Spanish-language databases, facilitated by the presence of native and fluent speakers of these languages within the Chapter 5 review team. This approach enabled the inclusion of evidence from Latin American countries and locally published studies that may otherwise have been missed by an English-focused search.

3 for extraction table). These were supplemented by 10 grey literature sources and reference tracking shared by Research Consortium on School Health and Nutrition partners. Screening was conducted using the Rayyan platform, with two reviewers independently assessing records and one mediator for any misalignment.

Data were charted using a structured extraction form capturing bibliographic information, study characteristics, population details, programme modalities, food environment components, governance and procurement arrangements, and reported outcomes across dietary, behavioural, health, and economic domains. Findings are presented through a descriptive and narrative synthesis organised around these domains, stratified where appropriate by country income level. No quantitative meta-analysis was conducted, consistent with the exploratory and mapping purpose of the review and the methodological heterogeneity of the included evidence base. The full protocol, PRISMA-ScR checklist, search strategies, and extraction table are available at the Open Science Framework (OSF) [1].

Studies were included if they examined school meals programme modalities or components in relation to dietary, behavioural, health, social or economic outcomes among school-going children and adolescents, or analysed programme design, policy or frameworks conceptually (the methodological/conceptual category described below). Evidence spans low-income countries (LICs), lower-middle-income countries (LMICs), upper-middle-income countries (UMICs), and high-income countries (HICs).

The distribution of outcome assessments across income settings is markedly uneven: HICs account for approximately 69% of outcome assessments (141 of 203), largely concentrated in the United States and the United Kingdom, with UMICs and LMICs together contributing around 20% (40 of 203), dominated by Brazil and China (UMICs) and by Ghana and Ethiopia (LMICs), and multi-country or global studies around 9% (18 of 203). LICs remain the least documented context in scientific repositories (2%, 4 of 203). The quantitative literature is heavily concentrated on dietary and behavioural outcomes, with health and NCD outcomes documented less often (34 of 203). By comparison, methodological and conceptual analyses, along with social science and public policy perspectives, critical for evidence-to-policy translation, remain underrepresented across all income settings.

The review was conducted in English, French, Portuguese, and Spanish. Each study's outcomes were classified into the five domains, and each country was assigned to a 2024 World Bank income group. The review identified 116 unique studies. However, because a single study can report outcomes across more than one domain and one country, the number of domain-specific outcome assessments exceeds the number of unique studies: the 116 studies generated 203 outcome assessments. Table 11 therefore reports outcome assessments, rather than unique studies, and the same study may contribute to more than one row. The methodological/conceptual category is not an outcome domain in the strict sense; it groups studies that analyse programme design, policy or

frameworks without reporting programme outcomes. Table 11 summarises these assessments by outcome domain and income setting.

Table 11. Number of outcome assessments by outcome domain

Outcome Domain	LIC	U & LMIC	HIC	Multi-country/Global	Total
Dietary outcomes	2	15	41	6	64
Behavioural outcomes	1	12	61	4	78
Health / NCD outcomes	1	6	24	3	34
Economic / systems outcomes	0	5	6	3	14
Methodological / conceptual	0	2	9	2	13
Total	4	40	141	18	203

Source: Scoping review (2026). * Studies appearing in more than one domain are counted once per domain. The methodological/conceptual category groups studies that analyse programme design, policy or frameworks without reporting programme outcomes. LIC = Low Income Country; LMIC = Lower-Middle Income Country; U & LMIC = Upper- & Lower-Middle Income Country; HIC = High Income Country (World Bank 2024–25 classification).

When the modality is explicitly reported in the reviewed studies, home-grown school feeding (HGSF) is often the most frequently described and cited approach. However, its prominence in the literature does not necessarily indicate greater effectiveness for health outcomes than other modalities. What is procured matters as much for health as where it is procured. This approach frames school meal programmes not only as mechanisms for improving child nutrition and health, but also as policy instruments capable of generating wider social, economic, and food system benefits. By linking school food procurement to local agricultural production, particularly smallholder farmers, the HGSF modality creates pathways through which school meals can influence dietary outcomes, rural livelihoods, local markets, community development, and sustainability. This broader understanding is reflected in the evidence base reviewed here, which encompasses not only health and dietary impacts but also social, economic, and conceptual dimensions of school meal programmes across different income settings.

HGSF is also the approach recognised in African Union policy commitments [56]. Two cautions apply. HGSF is not a homogeneous group of programmes (Box 5.1). And much of the experimental evidence on modality comes from WFP-supported programmes. These programmes reach only a small share of the children receiving school meals worldwide and are externally supported rather than run by governments, although in low-income countries this share is not negligible. Table 12 summarises the findings of these studies. Table 12 covers all modalities, not only HGSF; the modality behind each finding is named in the cells or implied by the country examples.

Table 12. Summary of scoping review findings on school meals programmes and outcome domains, by income setting

Outcome Domain	N (outcome assessments)	LICs / LMICs	UMICs	HICs	Country examples
Dietary outcomes	64	Largest reported effects of food transfers and vouchers. HGSF ↑ fruit/veg access and dietary diversity. Challenges remain in the stability of school food baskets from HGSF models.	SMPs associated with higher healthy food scores and lower unhealthy food scores; can increase stability of school food baskets. Dietary improvements attenuated by competitive foods.	Largest reported effects of catering programmes and non-institutionalised meals. Dietary improvements attenuated by competitive foods.	Brazil, Ghana, US, UK, China
Behavioural outcomes	78	Limited direct evidence. Communal eating habits reinforced behavioural and health outcomes. Vendor influence dominant near schools.	Vendor influence as a risk factor near public schools. Canteens/ in-school vendors influence dominant at private schools. Brazil: meal adherence associated with healthy dietary patterns, and national food and nutrition security system, policies and plans in place.	Canteens/ in-school vendors influence dominant at a significant number of schools, notably in North America. Japan/US: structured mealtimes + food education = largest reported norm-setting effects; peer dynamics critical.	Japan, Brazil, US, Ghana
Health / NCD outcomes	34	Sparse NCD data. Most data concentrate on: ↑ child growth, ↓ stunting	Mixed body-mass index (BMI) results. HGSF linked to ↑ height-for-age z-score in vulnerable adolescents.	Modest BMI reductions post-social protection school meals. Largest reported effects among food-insecure children.	India, Ethiopia, Tanzania, Brazil, US
Economic / systems outcomes	14	HGSF stimulates smallholders, ↑	HGSF meets a higher local/family farming	Centralised catering dominant: cost-	Brazil, Ethiopia,

		income stability, ↑ food freshness.	procurement target; supply-side food system benefits documented.	efficiency but ↓ dietary diversity concerns; Italy shows portion misalignment.	Ghana, Italy
Methodological / conceptual	13	Missing policy landscape documentation. Gaps in implementation assessment.	Underrepresented. Brazil shows monitoring gaps between policy intent and implementation.	Frameworks concentrated in HICs. Policy analysis tools developed (US, UK, Australia).	HIC and UMIC

Source: Scoping review (2026). N = number of outcome assessments, as in Table 11; studies may span multiple domains and income settings. LICs = low-income countries; LMICs = lower-middle-income countries; UMICs = upper-middle-income countries; HICs = high-income countries.

The narrative synthesis above draws on studies of varied design, but the underlying scoping-review protocol did not tabulate study design counts systematically — unlike the outcome and income-setting counts reported in Table 11. This is itself a methodological gap worth flagging explicitly (see Section 5.5). Where design is stated in the source studies, the pattern that emerges is consistent with a scoping review of this kind: a small number of experimental and quasi-experimental studies sit alongside a much larger body of observational, cross-sectional, and qualitative evidence, with grey literature contributing mostly to LIC and humanitarian settings. Table 13 summarises what can be established about the origin and design mix of the evidence, organised by outcome domain, to make this transparent rather than implicit.

Table 13. Evidence base by outcome domain, study design, and evidence source

Outcome domain	Predominant study designs	Illustrative experimental / quasi-experimental studies	Peer-reviewed vs. grey literature
Dietary outcomes	Predominantly observational (cross-sectional and cohort); a small number of RCTs from WFP-supported programmes.	Jordan randomised controlled trial (RCT) comparing standard vs. freshly prepared meals [19]; Burundi RCT comparing commodity-voucher vs. in-kind procurement [20].	Mainly peer-reviewed; WFP external programme evaluations included as grey literature and flagged as such.
Behavioural outcomes	Mostly observational and qualitative; latent class analysis used in Brazil.	None identified as randomised; the most rigorous designs are quasi-experimental (Brazil, Ghana).	Predominantly peer-reviewed.
Health / NCD outcomes	Mixed — observational cohort dominant in LIC/LMIC;	WFP Gambia Home-Grown School Meals Programme	Mainly peer-reviewed, with WFP grey literature contributing

	one phased cluster-RCT identified.	evaluation, a phased cluster-RCT [22].	the only cluster-RCT in a LIC.
Economic / systems outcomes	Observational and economic modelling; no experimental studies identified.	None identified as randomised; multiplier estimates derived from modelling [28,29].	Mainly peer-reviewed; WFP external programme evaluations included and GCNF report as grey literature and flagged as such.
Methodological / conceptual	Policy analysis and document review; not applicable to experimental design.	Not applicable.	Mainly peer-reviewed; WFP external programme evaluations included and GCNF report as grey literature and flagged as such.

Source: derived from the narrative synthesis in Section 5.3, cross-referencing study designs stated in the original sources. This table reports the design mix that could be established from the text of included studies; it is not a full risk-of-bias assessment, and a systematic count of designs was not part of the original review protocol — addressing this gap is a priority for any future update of this chapter (see Section 5.5).

The evidence base is strongest where programme characteristics can be linked to dietary outcomes, particularly when observational evidence is complemented by experimental or quasi-experimental studies. Evidence on behavioural outcomes is more limited and is predominantly observational or qualitative, while evidence on economic and systems outcomes relies mainly on observational analysis and modelling, with no experimental studies identified. Evidence directly connecting programme design to health and NCD/CMD outcomes is also limited, despite the importance of these pathways for the broader argument of this White Paper.

These limitations are particularly pronounced in low-income countries (LICs), where comparatively little peer-reviewed research was identified and a larger share of the available evidence comes from WFP and other United Nations programme evaluations and grey literature. These sources are important for documenting how programmes are actually designed, governed and implemented, especially where academic research is sparse, but they often provide less scope for causal inference and independent replication. The main research gap is therefore not simply a lack of studies, but a lack of comparative and longitudinal evidence that can identify how specific design, governance and food-environment conditions shape dietary, behavioural, economic and health pathways, particularly in LICs. Future research could give greater attention to these settings and to the rigorous evaluation of programme modalities and components, while also improving the reporting of programme design in both academic and programme-based evidence.

Across the outcome domains synthesised in Section 5.3, a further factor emerges that precedes programme design: the enabling environment within which a school meal programme operates. WFP [28], for example, refer to enabling environments as the overarching rules, laws, policies, and social behaviours that shape and govern a

programme. For school meal programmes, the World Health Organisation (WHO) [42] conceptualises health and well-being as shaped by enabling environments, in which biomedical and social determinants interact to influence living conditions, health outcomes and equity. Within the enabling environment, a distinction can be drawn between voluntary guidance and binding rules: mandatory nutritional standards, procurement requirements and regulation of competitive foods, vending and marketing apply to all schools, rather than relying on individual schools, caterers or children to make healthier choices. The 2026 WHO guideline on healthy school food environments recommends policies on school food provision, nutritional standards for foods served and sold in schools, and nudging [57].

Biomedical factors involve individual biology and genetic traits that interact with a child's and their family's external environment. Social determinants include both physical conditions—such as access to clean air, safe water and sanitation—and broader social and economic factors, including income, education, housing, employment and social inclusion [43]. Engaging with the enabling environment therefore requires recognising the complex determination of child health that shape the outcomes a SMP seeks to address [44].

A stronger or weaker enabling environment may help explain why the same modality can show different effect sizes across income settings, and — as Tables 11 and 13 indicate — why the evidence base itself is unevenly distributed, with experimental designs concentrated where external governance and monitoring capacity (e.g. WFP-supported programmes) is already in place. Section 5.4 examines food supply and governance as enabling conditions in more depth.

5.3 Evidence of school meal modalities and programme design by outcome domain

The sub-sections below report the study designs and income settings of the evidence on which each finding rests. Where a source study named the programme modality, it is reported as stated; where the modality had to be inferred from programme components (Section 5.1.1), the classification is a reconstruction and should be read with caution. Findings are labelled with the evidence grades used in Chapters 2 to 4 and Chapter 6: consistent (supported by multiple studies, including experimental and quasi-experimental designs, across multiple contexts), suggestive (supported by fewer studies, narrower geographical bases or designs that limit causal inference) and limited (a critical gap in the evidence). Nutritional status outcomes, such as growth and stunting, are reported with health and NCD outcomes in Section 5.3.3.

5.3.1 Dietary outcomes

Dietary outcomes are the second most frequently documented domain (64 of 203 outcome assessments; about 55% of the 116 unique studies). In LICs and LMICs, a small number of mainly observational studies associate HGSP with greater access to fruits,

vegetables, and traditional foods where menus are designed to include them, with effects most pronounced where programmes cover the majority of children's daily intake. As shown in the previous chapters, this is relevant to NCD/CMD prevention because school meals can influence the foods children regularly consume and, in turn, the quality and diversity of dietary patterns during a period when eating habits are being established.

Greater availability and consumption of minimally processed foods such as fruits, vegetables and traditional staple foods may increase exposure to dietary patterns consistent with lower NCD/CMD risk, while reducing reliance on less nutritious alternatives. However, the evidence reviewed here primarily concerns dietary availability and consumption rather than subsequent NCD/CMD outcomes, so the prevention pathway should be understood as plausible and indirect rather than as evidence that HGSF itself directly prevents NCDs or CMDs – it is rather its potential.

In Africa, Singh and Fernandes [13], in a descriptive analysis of 24 districts in Ghana, found that planned school menus corresponded with local agricultural production and consumption patterns. The analysis does not establish the direction of this association or compare procurement modalities; menus may equally have been adapted to existing local production. In UMICs, Brazil's national school feeding programme (Programa Nacional de Alimentação Escolar, PNAE), which includes HGSF components, provides the largest evidence base, although largely observational. These studies examine the level of school meal consumption or adherence rather than comparing procurement models, so they show associations between eating school meals and diet quality, not effects of HGSF as such: Boklis-Berer et al. [2] showed that, among Brazilian adolescents, high consumption of school meals was significantly associated with higher overall diet quality and healthy food scores, and lower unhealthy food scores. Froelich et al. [3] reported a similar pattern at national scale among Brazilian adolescents, finding high adherence to school meals positively associated with the co-occurrence of healthy dietary markers and inversely associated with unhealthy markers.

In HICs, Smith et al. [4] documented that pre-Healthy, Hunger-Free Kids Act (HHFKA) in the United States (2005–2008) school food had no impact on diet quality, but post-HHFKA (2013–2020) participation has been significantly associated with improved Healthy Eating Index-2020 scores, with the largest improvements among food-insecure children. These gains, however, are attenuated where ultra-processed competitive foods remain accessible within or near schools [18,34]. The post-HHFKA improvement is consistent with the content of the reform. Before the Healthy, Hunger-Free Kids Act (HHFKA) of 2010, USA school meal standards had not undergone major revisions in decades. Lunches relied on traditional menu planning, featured higher levels of sodium and saturated fats, and lacked the strict calorie caps, whole-grain, and separate fruit-and-vegetable mandates introduced later.

Further research is needed to disentangle the contribution of the HHFKA standards from other features of the US school food system. The decentralised organisation of school

food provision means that implementation can vary across states, districts and schools, while the continued availability of competitive foods on school premises and in the surrounding food environment may modify what children consume. These factors make it difficult to interpret changes in diet quality as attributable to meal standards alone and highlight the need for studies that examine school meals together with the wider food environment and implementation context. A recurring finding across the literature is that the exposure to and the availability and consumption of competitive foods may contribute to dietary patterns associated with increased NCD/CMD risks among children.

In the grey literature on programme evaluation provided by the WFP, the school meals supported by the WFP provide complementary experimental evidence on how modality shapes dietary outcomes. These programmes reach only a small share of the children receiving school meals worldwide and are externally supported rather than run by governments, so the findings describe externally supported delivery and may not generalise to national programmes. In Jordan, a randomised controlled trial (RCT) assessing the impact of replacing the standard centralised meal (date bars and high-protein biscuits) with freshly prepared, nutritionally diverse meals found that children in healthy meal schools had significantly more diverse diets than those in standard provision schools [19]. Because the intervention changed both the meal and how it was prepared and delivered, the improvement cannot be attributed to procurement or decentralisation alone.

In Burundi, an RCT comparing a decentralised commodity voucher procurement model — in which schools procure food from local cooperatives via a voucher transferred to provincial education authorities — against the traditional centralised WFP in-kind procurement model found that the commodity voucher model delivered significantly more meal days per month (13 vs. 7.4), primarily through increased supply of local rice, but at a cost to dietary diversity as measured by the Global Diet Quality Score for Meals (GDQS-Meal) [20]. In terms of the modality-model distinction introduced in Section 5.1.1, the Burundi trial compares a decentralised model of a food transfer / commodity modality and a centralised in-kind procurement model — illustrating that model-level choices within a modality can move dietary diversity in opposite directions from meal frequency. The contribution of these trials in LICs is not that a more nutritious ration improves diet, which is to be expected, but that the procurement source and the food actually served are separable design choices: local procurement raised meal frequency in Burundi at a cost to dietary diversity, whereas in Jordan it was the composition of the meal that drove the improvement. The Burundi trial therefore shows a trade-off between the frequency and reliability of meals and their dietary diversity, rather than a single effect of local procurement.

Taken together, the dietary evidence shows that menu composition, nutritional standards and the regulation of competing foods, which are themselves programme interventions, are the most proximal determinants of what children are offered and eat. Few studies compared procurement modalities directly; where they did, as in Burundi,

procurement affected the feasibility, cost, reliability and sourcing of meals rather than their nutritional quality as such. Across income settings, the relationship between programme modality and dietary quality may also be linked to the supply stability of school food baskets, particularly the consistent provision of nutrient-dense foods that are less exposed to competition from external food environments, including fruits, vegetables, cereals, minimally processed foods, and animal-source proteins. These outcomes are proximal dietary risk factors rather than NCD/CMD outcomes themselves; evidence on NCD/CMD-related outcomes is examined in Section 5.3.3. Overall, the evidence that the composition of the meal and the regulation of competing foods shape dietary outcomes is consistent (experimental and quasi-experimental studies in Jordan, Burundi and the United States); the evidence that any given modality improves dietary quality in itself is suggestive, and evidence from LICs is limited.

In LICs and LMICs, some studies associate HGSF with improved fruit and vegetable access and greater dietary diversity where menus are designed to include these foods, although supply-chain constraints, seasonality, and procurement challenges can undermine the consistency of foods provided. In contrast, evidence from UMICs suggests that more institutionalised HGSF arrangements can support both healthier food profiles and greater stability of school food baskets through stronger procurement systems and more predictable sourcing from local producers. In HICs, where local procurement initiatives are less frequently framed as HGSF, reforms promote healthier and locally sourced foods have improved dietary quality, although gains are often attenuated by the availability of competitive UPF foods in the school food environment. The interaction between programme design and the surrounding food environment therefore appears to be an important mechanism through which dietary outcomes and the stability of food provision are shaped, although this is an interpretation of the evidence rather than a finding tested directly. Yet even well-designed modalities operate within social conditions that precede and constrain their effects: in Ghana, Burundi, and Ethiopia, household poverty, food insecurity, and early-life malnutrition shape children's nutritional baselines before any meal is served, determining the marginal contribution a school meal can make to daily intake [6,8,14,20].

The evidence also indicates that not all school food modalities produce favourable dietary outcomes, particularly where food provision is mediated through commercial vendors, vending machines, school stores, or other competitive-food channels. In California, USA, Cisse-Egbunoye et al. [46] found that foods of minimal nutritional value were widely available through vending machines and school stores, with availability significantly associated with students' purchases and, for several food categories, with reported consumption. These findings illustrate how vendor-based provision can create an obesogenic food environment in which the availability of energy-dense, nutrient-poor foods shapes dietary behaviour independently of the nutritional quality of the formal school meal.

Similarly, in the Global North, Cullen et al. [47] showed that students' dietary improvements following the Texas Public School Nutrition Policy were partly driven by a shift away from snack bars and vending machines towards the regulated school meal, while Wojcicki and Heyman [48] found that removing less healthy foods from à la carte services and vending machines was accompanied by increased participation in the healthier school lunch programme. A systematic review by Driessen et al. [49] found that most interventions modifying school food environments—including changes to canteen menus and vending-machine contents—improved either dietary behaviours or body weight, although the methodological quality of the underlying evidence was generally limited. Together, these findings suggest that the dietary effects of a formal meal modality cannot be considered in isolation from the parallel food channels through which children can access food at school.

This is particularly relevant to mixed-provision environments (distinct from the hybrid modality defined in Box 5.1), in which a regulated school meal operates alongside externally supplied, commercially purchased, or independently managed foods. In such arrangements, the overall dietary exposure of children is determined not simply by the composition of the formal meal, but by the relative accessibility, attractiveness and regulation of competing sources. Some of the US experiences illustrate this interaction: strengthening the nutritional standards of the formal meal was associated with healthier consumption patterns when accompanied by restrictions on competitive foods, whereas access to snack bars, vending and à la carte foods provided alternative pathways for less healthy consumption [47,48]. Conversely, interventions that add healthier components to the existing food environment can shift this balance without necessarily replacing the wider system; the Let's Move Salad Bars to Schools initiative, for example, used a public-private partnership to introduce salad bars into schools as an additional source of fruits and vegetables [50]. However, the study on salad bars, like most studies examining school feeding modalities, is non-experimental and does not report nutritional outcomes attributable to the intervention. It therefore illustrates an approach to modifying the school food environment rather than providing evidence of its nutritional effectiveness.

The evidence therefore suggests that mixed-provision arrangements are not inherently beneficial or detrimental: their dietary effects depend on how the different components are governed, whether commercial and non-commercial food channels are subject to coherent nutritional standards, and which foods are most consistently available to children. In this sense, modality should be understood not only in terms of what the programme provides, but also in terms of what other forms of food provision it permits, regulates, or leaves untouched.

Beyond Brazil, evidence from other middle-income countries suggests that the relationship between school meal modality and dietary outcomes is more heterogeneous, extending beyond HGSP to include school milk programmes, canteens, vendors and mixed food environments. In Latin America and the Caribbean, Wineman et al. [38] found that school milk programmes were operational in five of seven countries

and often prioritised local procurement but also highlighted a persistent tension between nutritional objectives and palatability, particularly where sugar and fat were used to improve acceptability and where dairy production and refrigeration are unstable. This illustrates how the nutritional effect of a modality depends not only on its supply arrangement but also on the composition of the foods delivered. Similarly, Wijesinha-Bettoni et al. [17], examining 18 middle-income countries, documented substantial variation in school food standards, procurement practices, fruit and vegetable schemes, school gardens, tuck shops and external vendors. Only seven of 18 countries recommended vegetables daily and five recommended fruit daily, while cost, storage and procurement constraints limited implementation.

In India, Bandoni et al. [27] synthesised evidence from PM POSHAN (formerly the Mid-Day Meal Scheme) alongside Brazil's HGSF, reporting associations with improved nutritional status and growth. Taken together, these findings indicate that in the Global South, school food provision cannot be reduced to a single HGSF pathway: the same programme may combine formal meals with snacks, beverages, vendors and other food channels, and the resulting dietary exposure depends on the nutritional standards, procurement arrangements and governance applied across these components.

Importantly, some of these modalities may also introduce or reinforce dietary risk factors. In Malaysia, Mohammadi et al. [53] designed a school-based intervention specifically around canteen vendors, reflecting recognition that commercially operated school food outlets can be an important determinant of adolescents' food choices; although the study was a feasibility protocol and therefore provides no outcome evidence, its design targeted the availability and affordability of healthier foods through vendor training and subsidies.

In the Eastern Mediterranean Region, Al-Jawaldeh et al. [52] found that school meals were present in all 22 countries reviewed, but that canteen standards, vending-machine regulation and food marketing policies were also common components of the school food environment, with fewer programmes undergoing formal impact assessment. Without such assessment, the contribution of these components to children's diets remains unknown. In China, Liang et al. [54] provides empirical evidence, from a single-arm pre-post study and therefore suggestive, that the configuration of a school canteen can modify dietary behaviour: a digital-nudge intervention increased consumption of fruits, coarse grains, seafood and soy products while reducing sweets, sugary drinks and fast food.

The two findings should not be conflated: evidence that exposure to, availability of, and consumption of competitive foods may contribute to dietary patterns associated with increased NCD/CMD risk does not imply that canteens or vendor-based provision are inherently harmful modalities. Rather, their potential effects depend on the rules governing what foods are available, especially the ultra-processed, how they are priced and promoted, and whether healthier options are made more accessible than competing products.

Thus, particularly in Global South settings undergoing rapid nutrition and food-system transitions, the dietary implications of school food modalities are shaped by the interaction between formal meal provision and the wider commercial food environment. Overall, the evidence on canteens and vendor-based provision is suggestive: it rests on cross-sectional and pre-post designs and, in one case, a feasibility protocol.

5.3.2 Behavioural outcomes

This domain was represented in 78 of the 203 outcome assessments, spanning approximately 67% of the 116 unique studies, and was concentrated in HICs (61 assessments). Overall, the evidence, graded as suggestive because it relies mainly on observational and qualitative studies, points to associations between school meal programmes and dietary patterns, adherence to school meals, and food-purchasing behaviours. However, these findings capture distinct dimensions of dietary behaviour and do not establish the mechanisms through which school meals may influence food preferences, acceptance, or eating habits. In Japan, studies of centralised catering models describe programmes that combine meal provision with structured food education, illustrating how institutional and educational arrangements may support the development of dietary norms, although their specific effects are not directly established by the evidence reviewed. In Brazil, Froelich et al. [7] found that both satisfactory and unsatisfactory adherence to school meals were positively associated with a “healthy” dietary pattern, suggesting that dietary patterns cannot be explained by meal adherence alone. In Ghana, Fernandes et al. [21] found that receiving free school meals was associated with a lower likelihood of leaving campus to purchase food, indicating that meal provision may influence students’ food-purchasing behaviour and their exposure to food environments beyond the school.

Taken together, these studies suggest that school meal programmes can be associated with dietary behaviours through potentially different pathways, including the relationship between meal participation and overall dietary patterns, and the availability of alternatives outside school. They do not, however, directly test whether repeated exposure, social eating, or programme design drives these associations. This distinction is important: while the organisation of meal provision and its integration with educational activities may help shape the school food environment, the available evidence does not establish which programme features are most effective in influencing dietary norms. Evidence from LICs remains more limited and focuses more on attendance than on dietary behaviour itself [6], further constraining conclusions about the consistency of these associations across contexts.

Despite these limitations, this evidence remains relevant to understanding how school feeding modalities may contribute to NCD prevention. Dietary patterns, adherence to school meals, and exposure to food environments outside school are important intermediate dimensions through which programme modalities may be associated with longer-term health outcomes. Different arrangements for food procurement, preparation, service, and educational engagement may create different opportunities to

improve dietary quality and support healthier eating practices, although their specific effects require further investigation. Examining these dimensions alongside the characteristics of each modality helps identify plausible pathways linking programme implementation to NCD prevention without assuming that behavioural changes or health benefits have been demonstrated. This perspective is particularly important because the potential of school meals to support healthier diets depends not only on the provision of meals, but also on the foods served, the wider school food environment, and the social and material conditions shaping students' food-related behaviours. The evidence therefore provides a basis for identifying promising programme features and research priorities, while underscoring the need for stronger longitudinal and intervention studies to establish whether, and under what conditions, different modalities contribute to improved dietary and cardiometabolic outcomes.

5.3.3 Health and NCD outcomes

Present in 34 of the 203 outcome assessments (about 29% of the 116 unique studies), with the greatest heterogeneity. In LICs, evidence concerns child growth, stunting reduction, and micronutrient status: Beressa et al. [8] documented 26.6% stunting and 28.9% thinness prevalence among Ethiopian schoolchildren (baseline context rather than an effect of any modality), highlighting that undernutrition, itself a pathway to later NCD risk, remains the dominant nutritional concern in this setting. Sebba et al. [9] found in Tanzania that school meal participation was associated with a 45% higher likelihood of achieving minimum dietary diversity, a dietary rather than a health outcome. School meals in Tanzania are delivered through a mix of government coordination and international organisations. While implementation is decentralised and coverage uneven, the programme is guided by national policies such as the 2020 School Feeding and Nutrition Guidelines, which connect school meals to broader health and nutrition objectives. This description of the programme does not show that its procurement or delivery arrangements explain the association.

The only phased cluster-RCT examining HGSF in a LIC context, the WFP evaluation of the Home-Grown School Meals Programme in The Gambia, found a 22% rise in dietary diversity, a 12% improvement in food security, a 13% reduction in self-reported depression, and a 20% reduction in medium-to-high stress, with the largest effects observed among girls [22]. Dietary diversity and food security are diet-related rather than strictly health outcomes; they are reported here because the study assessed them alongside health outcomes. International actors play an important role alongside the government in delivering this programme. The study is among the first to link HGSF in a LIC to mental health outcomes, a domain with no prior experimental LIC evidence. As it evaluated the HGSF programme as a package, it provides evidence on that package rather than on procurement modality as such. As a single cluster-RCT, it is graded suggestive.

In UMICs, Andrade et al. [10] found that participation in Brazil's HGSF was positively associated with height-for-age z-score improvement in socioeconomically vulnerable

adolescents, while body mass index (BMI) results in Brazil have been mixed [23]. In HICs, post-2010 evidence from the United States is the most direct: Taber et al. [11] found that states with stricter nutrition standards had a 12.3 percentage-point smaller obesity prevalence disparity between free/reduced-price lunch recipients and other students. Furthermore, Wang et al. [24] modelled that new nutritional standards could prevent up to 10,600 adult deaths per year from cardiovascular disease, diabetes, and cancer, saving 355,000 disability-adjusted life years (DALYs) and USD19.3 billion annually — the most explicit estimate of the school-meals-to-NCD pathway in the literature reviewed, although it is model-based and therefore suggestive.

Cohort studies from LMICs and UMICs — including Brazil, Guatemala, India, the Philippines, and South Africa — document a dual challenge in contexts where chronic undernutrition persists among vulnerable populations while overweight and NCD prevalence among school-age children is rising [25,26]. The cohort studies do not describe the school meal modalities operating in these countries, so modality-specific inferences cannot be drawn from them. In these settings, stunting and micronutrient deficiency in childhood predispose towards NCD-relevant metabolic risk markers in adulthood. In such contexts, school meal programmes could in principle address both nutritional deficits and emerging obesogenic pressures, although the studies reviewed do not show that particular modalities do so; the NCD framing is therefore context-dependent rather than universally applicable. These findings point to the interaction between programme design quality, food environment, and the governance capacity to sustain standards over time, which is in turn shaped by the social conditions children bring to school. Across these settings, the health gains were greater where programme design combined local procurement with nutritional standards and attenuated where implementation quality was inconsistent [17,41]. This pattern is suggestive at most, as it rests on few studies that did not compare designs directly.

More evidence from the Global South suggests that the relationship between school meal modalities and health and NCD-related outcomes is heterogeneous, extending beyond HGSF and canteens and vendors to school milk programmes and mixed food environments. Wijesinha-Bettoni et al. [17], examining 18 middle-income countries, documented substantial variation in nutritional standards, fruit and vegetable provision, school gardens, “tuck shops” and external vendors, with cost, storage and procurement constraints limiting access to healthier foods.

Importantly, some configurations may also reinforce dietary risk factors for NCDs, particularly where commercially supplied foods and beverages are readily available. The findings below concern dietary risk factors rather than health outcomes; they are reported here because, in the absence of direct health outcomes, dietary exposure is the NCD-relevant pathway most often documented for these configurations.

From an NCD prevention perspective, evidence from the Eastern Mediterranean Region (EMR) highlights the importance of examining how school meal modalities interact with wider school nutrition policies and food environments. Evidence from the Eastern

Mediterranean Region (EMR) illustrates the diversity of government-led school nutrition programmes and the limited evidence available on their impacts. A systematic review by Al-Jawaldeh et al. documented government-led school nutrition programmes across all 22 countries of the WHO Eastern Mediterranean Region (EMR): Afghanistan, Bahrain, Djibouti, Egypt, Iran, Iraq, Jordan, Kuwait, Lebanon, Libya, Morocco, Oman, Pakistan, Palestine, Qatar, Saudi Arabia, Somalia, Sudan, Syria, Tunisia, the United Arab Emirates, and Yemen [54]. The review identified 298 interventions, including school meal and feeding programmes in all countries, fruit and vegetable schemes in 12 of 22 countries, milk schemes in nine, and take-home rations in Egypt, Iraq, and Yemen. These food provision approaches were complemented by interventions targeting nutrition education, school canteen standards, food availability, and food safety.

However, few programmes had undergone impact assessment, limiting conclusions about their effectiveness in improving dietary or health outcomes. The review also documented a double burden of malnutrition across the region, with undernutrition coexisting with increasing overweight, obesity, and diet-related chronic disorders. This context underscores the importance of examining not only whether meals are provided, but also how their composition and the surrounding food environment may support healthier dietary patterns.

In parallel, evidence from China illustrates one pathway through which the organisation of the school food environment may influence dietary risk factors. Liang et al. [54] found that digital nudges in school canteens increased consumption of fruits, coarse grains, seafood, and soy products while reducing consumption of sweets, sugary drinks, and fast food. Although this intervention does not necessarily represent a distinct school meal modality, it demonstrates how modifications to the food environment in which meals are selected and consumed can complement meal provision. Taken together, these studies point to two complementary dimensions for understanding the potential contribution of school meals to NCD prevention: the modality through which meals are procured, prepared, and provided, and the wider food environment and accompanying interventions that shape what children eat.

Different modalities may create different opportunities to influence food quality, menu composition, and the integration of nutrition education, but the available evidence does not establish which configurations produce the greatest health benefits. Neither the regional review nor the Chinese study demonstrates reductions in NCD incidence. Rather, they highlight the need to assess how specific modality characteristics, in combination with food environment measures, influence dietary quality and whether these changes translate into sustained improvements in cardiometabolic health.

For the health section of this chapter, it is important to note that the epidemiological context in which school meals operate is not differentiated only by national income level; it can also vary substantially within countries, particularly in the Global South. Metropolitan and rapidly urbanising populations may experience a nutrition transition

in which undernutrition and micronutrient deficiencies coexist with increasing overweight, obesity and other NCD risk factors, while rural and geographically isolated populations may continue to experience endemic hunger and acute food insecurity, which can intensify during economic, climatic or other crises.

The health and dietary concern is shared across settings and original studies, but the nutritional problems to which school meals respond, and consequently the appropriate programme priorities, can differ markedly between and within countries. This does not imply fundamentally different health objectives, but rather different combinations of objectives and programme responses according to the epidemiological and food-system context.

Within the evidence reviewed here, HGSF programmes in the Global South and some catering-based school meal programme with home-grown components in the Global North are particularly relevant because they bring agriculture and health agendas into the school food systems, while placing school meals within a governance arrangement in which government has an important role in coordinating or regulating provision and the education sector provides the institutional setting for delivery. These configurations illustrate how school meals can function not only as nutrition interventions, but also as instruments through which agricultural, health, social protection and education policies intersect.

The enabling environment is therefore critical: the capacity of school meals to respond to different nutritional challenges depends on the policy and legal framework, institutional coordination, financing, procurement and food-system infrastructure, as well as the governance capacity to maintain standards and regulate the wider food environment over time.

Therefore, particularly in Global South settings undergoing rapid nutrition and food-system transitions, the health effects of school food modalities depend on how formal meal provision interacts with the wider food environment and on the governance of what foods are available, affordable, promoted and consumed.

5.3.4 Economic and systems outcomes

In LICs and LMICs, HGSF models account for most of the available evidence, which is largely observational and suggestive: evidence from Ghana illustrates the potential nutritional benefits of school feeding, while highlighting the importance of distinguishing overall programme effects from subgroup findings. In a cluster-randomised trial, Gelli et al. [14] found no overall effect of the national school meals programme on height-for-age or BMI-for-age among children aged 5–15 years. However, subgroup analyses identified improvements in height-for-age among children aged 5–8 years, girls, and children from households living below the poverty line, suggesting that nutritional benefits may vary across population groups. Silva et al. [15] reported that the majority of Brazilian municipalities evaluated met the national HGSF's 30% family-farming procurement target then in force (since raised to 45%), indicating that the target

is operationally achievable at scale, although meeting it shows compliance rather than downstream food-system or health effects. In HICs, centralised catering systems demonstrate cost-efficiency advantages but face criticism for supply-chain industrialisation and portion misalignment: Balzaretti et al. [16] found substantial discrepancies across Italian regions, with healthy options (fish, vegetables) consistently undersized relative to reference values, and cluster analysis linking these patterns to regional obesity rate variation. Bandoni et al. [27] synthesised evidence from Brazil and India to show that both Brazil's HGSF and India's PM POSHAN (formerly the Mid-Day Meal Scheme) are associated with improved nutritional status, better academic outcomes, and support for local agriculture — suggesting the dual role of school meals as a health and food systems intervention.

Multi-sectoral benefit-cost analyses across 14 low- and middle-income countries estimate that each US dollar invested in school feeding generates between USD 7 and USD 35 in combined benefits across health, education, social protection and local agricultural sectors [55]. In Brazil, the School Feeding Observatory estimates that each monetary unit of demand generated through HGSF produces GDP multipliers of 1.52 in family farming and 1.66 in family livestock production [29]. These economic effects are relevant to NCD/CMD prevention indirectly, through the conditions that shape household food access and dietary choices.

Increased income among smallholder households may improve purchasing power and access to diverse foods, but this pathway is not automatic: its potential nutritional effect depends on what foods are available and affordable, how additional income is allocated, and whether other determinants of diet and health are addressed. Brazil's institutional framework illustrates how these pathways can be intentionally connected. HGSF is embedded within broader food and nutrition policies, including requirements for purchases from family farming and restrictions on ultra-processed foods in public schools, linking agricultural production, school food provision, nutrition and social protection within a common policy framework [15,27]. The relevance of HGSF to NCD/CMD prevention therefore lies not in the economic multiplier itself, but in how programme design and policy can connect agricultural development with healthier food availability and consumption.

WFP-supported programmes extend this evidence in two directions. In Jordan, women formally employed within the National School Feeding Programme were five times more likely to be employed and reported household income increases of one-third, with gains in savings and non-food expenditures — linking the programme to the social determinants of health in surrounding communities [30]. In Burundi, decentralised commodity voucher procurement reduced per-child cost (USD 40.61 vs. USD 46.85) but at a cost to dietary diversity, underscoring that economic efficiency and nutritional adequacy must be optimised jointly [20]. Taken together, these systems-level effects, on agricultural incomes, women's employment and community purchasing power, may link

institutional design choices to the upstream conditions that shape children's health, although none of these studies measured children's diets or health as a result.

The economic pathways documented here bear on the NCD/CMD prevention argument only indirectly, through household income and food access, and do not by themselves establish a health benefit. In Brazil, where the nutrition transition has already taken place, procurement mandates may raise smallholder incomes and expand household access to diverse foods, potentially counteracting food pressures that in-school provision alone cannot address, particularly in settings where local food systems maintain availability of minimally processed foods [14,15,29]; in Jordan, women's formal employment within the programme improved household material conditions through which children's health is produced [30]. The economic effects may be more modality-dependent than the health ones, but this is a hypothesis: the multipliers reported for HGSF and hybrid models come from separate studies rather than direct comparisons across modalities, so it is not established that these models generate the largest economic effects.

Taken together, the studies reviewed suggest that the effects of SMPs on economic and wider food-system outcomes are shaped by an enabling environment in which biomedical, social, governance and food-supply determinants interact. Biomedical and nutritional conditions shape the needs that SMPs are expected to address, while social determinants—including household income, food insecurity, socioeconomic position and access to food—condition both programme participation and the marginal contribution of school meals to children's diets and nutritional status [8,27]. At the governance level, the evidence shows that procurement rules, nutritional standards, institutional capacity and the regulation of canteens, vendors and marketing determine how programme resources translate into food access and dietary outcomes [17,47,52].

These arrangements also shape food-supply effects: local procurement and SMPs programmes can create demand for domestic agricultural production, but storage constraints, supply-chain complexity, fiscal capacity and seasonality can limit these linkages [17,38]. Conversely, decentralised procurement can improve meal availability while producing trade-offs in dietary diversity, as illustrated by the Burundi comparison of commodity vouchers and centralised in-kind provision. At the same time, school meals can generate broader system effects by shifting demand towards healthier foods and local producers, while reforms to school food environments may alter participation and the economic viability of meal provision [48,51]. Thus, SMPs do not generate economic or food-system outcomes through procurement alone; their effects depend on the interaction between the nutritional and biomedical needs of populations, social and economic conditions, governance capacity, and the reliability and structure of food supply chains.

5.3.5 Methodological and conceptual analyses

This section does not report programme outcomes. Its aim is to characterise how the design and governance literature is framed and where it limits the interpretation of Sections 5.3.1 to 5.3.4. It is the smallest cluster (13 of 203 outcome assessments; about 11% of the 116 unique studies), but analytically significant for interpreting the rest of the evidence base. This literature identifies the same limitation of the evidence base: heavy concentration in epidemiology and quantitative health and nutrition disciplines, with limited engagement from the social sciences, public policy, and implementation research. Boehm et al. [31] and Klotz-Silva et al. [32] both note that studies frequently examine isolated programme components without situating them within the broader architecture of programme design or the governance structures that shape implementation.

Wijesinha-Bettoni et al. [17] found that while 12 of 18 middle-income countries had nutrient-based standards, only seven recommended daily vegetables and five recommended daily fruit targets — revealing a gap between policy framework and nutritional ambition that cannot be detected from outcome studies alone. This fragmentation constrains evidence-to-policy translation, motivating the design-centred analytical framework developed in Section 5.4. What the methodological literature ultimately reveals is that the gap between policy intent and measurable outcomes is not a data problem alone: it is an analytical problem, produced by the failure to situate programme components within governance architectures and food environments. The framework in Section 5.4 addresses this gap directly, making the interaction between modality, model, component, and food environment the unit of analysis rather than the residual.

A further methodological limitation is the limited use of theory-driven and context-sensitive approaches to understand how SMPs can achieve their expected effects differently across settings. Much of the evidence evaluates individual interventions or outcomes without explicitly theorising how programme design interacts with local enabling environments, including social determinants, governance arrangements, food-supply systems and institutional capacity. This can obscure how similar programme components operate across settings. A methodological approach requires localised, design-centred analysis, in which modality, programme components and implementation features are examined in relation to the social, economic, political and food-system conditions in which SMPs are embedded. Such an approach would move beyond asking whether a programme works towards understanding how, for whom, and under what enabling conditions particular designs generate nutritional, health, economic and food-system effects. The analysis in this chapter was not structured around a formal implementation-science framework: the design-centred framework in Section 5.4 was derived inductively from the evidence, and applying implementation-science approaches is identified as a research priority (Section 5.5).

5.4 A proposed framework to foster enabling environments and improve programme design

The evidence reviewed in Section 5.3 suggests that outcomes depend less on programme modality than on the models, components and implementation arrangements through which a modality is operationalised, although few studies compared these directly. In HGSE, local procurement does not by itself guarantee consistent nutritional or food-system outcomes: procurement rules, supplier arrangements, technical assistance, institutional capacity and the stability of local supply chains shape what schools are able to provide [15,20]. Evidence from Brazil, Ghana and some European countries similarly shows that the effects of formal school meals are conditioned by nutritional standards and the menus based on them, procurement requirements and regulation of competitive foods [17,18,33–35]. Modality is therefore not a sufficient explanation of programme effects; effects emerge from the interaction between modality, programme design and the enabling environment.

Figure 2 is an illustrative analytical device, not a prescriptive toolkit or a universal causal model. It is a conceptual typology of how programmes can be organised, not a summary of demonstrated effects: only the links between meal composition, the regulation of competing foods and dietary outcomes are supported by consistent evidence (Section 5.5), and the advantages attributed to particular modalities and models are hypotheses. It was constructed inductively from the scoping review and grey literature to organise the factors that appear to shape how modalities operate and the outcomes they may generate. It separates enabling conditions from programme design, two domains that interact and are not ranked in order of importance. The enabling conditions comprise four environments that recur across the evidence:

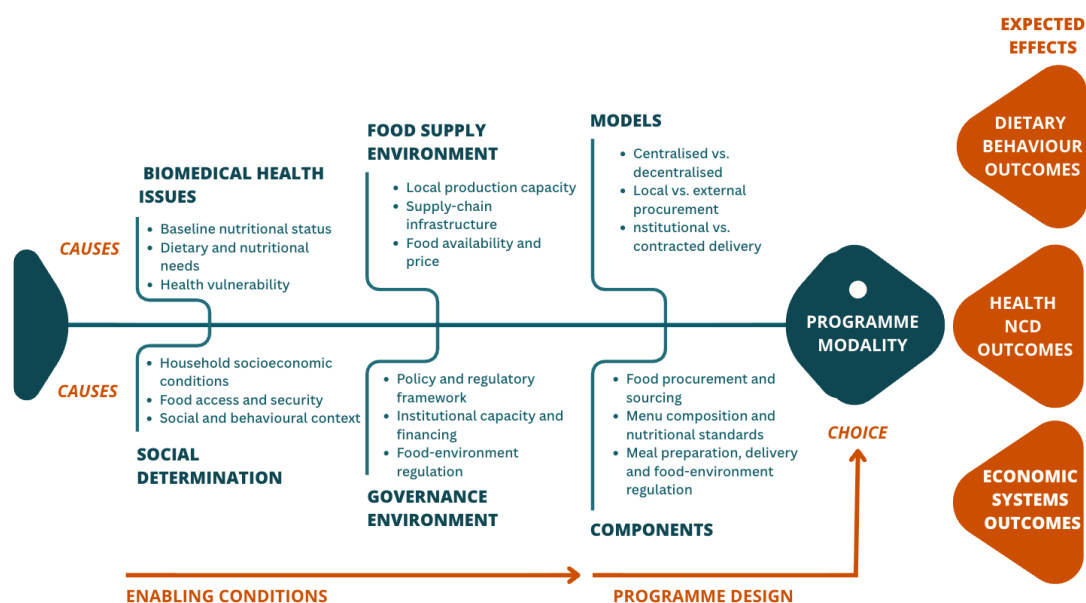


Figure 2. Illustrative synthesis of enabling environments, programme design features and outcome pathways identified in the evidence. Source: developed inductively from the scoping review (2026) and associated grey literature (Section 5.3).

- **Biomedical health issues:** children's baseline nutritional status, dietary needs and health vulnerabilities, which define what SMPs are expected to address.
- **Social determinants:** household socioeconomic position, food access and security, and the social and behavioural contexts that shape how much school meals contribute to overall intake.
- **Food-supply environment:** local production capacity, supply-chain infrastructure, and food availability and prices. Price volatility is a particular risk where budgets are fixed in advance, because a rise in food costs can reduce the consistency and nutritional adequacy of meals.
- **Governance environment:** the policy and regulatory framework, institutional capacity, financing and regulation of the wider school food environment, which determine how procurement is organised, which standards apply and how competing food sources are regulated. Political economy cuts across all four environments, including the influence of commercial actors, the presence of policy champions and civil society, and the capacity to implement, monitor and enforce standards after they are adopted.

On the design side, models describe how a modality is organised (centralised or decentralised, local or external procurement, institutional or contracted delivery), and components are the features that operationalise them: procurement and sourcing, menu composition and nutritional standards, preparation and delivery, and regulation of the surrounding food environment. The diagram places “choice” between enabling conditions, design and modality because configurations are not made in a vacuum; they emerge from the institutional, social, biomedical and food-system conditions in which programmes are implemented. They may influence dietary and behavioural, health and NCD, and economic and food-system outcomes, but not through linear or guaranteed pathways. Local procurement may strengthen links with agriculture where production and institutional capacity suffice, yet meet supply constraints where they do not; centralised catering may facilitate standardisation and scale while weakening links with local producers; and better meal composition may be attenuated where less healthy foods remain available through canteens and vendors. The same component can therefore operate differently across settings.

The diagram does not rank its factors, and context-dependence of this kind is not unique to school meals. What the evidence adds is a view of how the three pathways introduced in Section 5.1 are conditioned by design. Through the meal itself, what is served and how the wider school food environment is regulated shape dietary intake, nutritional status and dietary risk markers during school age: the evidence is consistent for meal composition and regulation of competing foods, and suggestive for the size of any effect on health outcomes. Through the food supply chain and food environment, procurement source and supply chain shape the stability and diversity of the food basket, local demand and household income: the evidence is suggestive, and local sourcing does not by itself determine nutritional quality, since a locally procured food of low nutritional value remains of low nutritional value. Through dietary patterns established at school and their persistence into adulthood, the wider food environment and access to nutritious foods may matter, but the evidence does not follow children beyond school age in relation to

programme design, and this remains a critical gap. Governance, standards and financing cut across all three by determining the coherence of design choices over time. Section 5.5 returns to which conditions appear to matter most.

Table 14 grounds the framework in empirical evidence. It brings together country examples from the scoping review and grey literature to show how modalities and models have been configured in practice, the enabling conditions they depend on, their principal design features, and the potential effects and limitations documented. The figure illustrates the relationships identified across the literature; the table shows how they have manifested in different country and programme contexts.

Table 14. Modalities, models, and their potential implications, constraints, and key limitations for the school food environment

PROGRAMME MODALITY	ENABLING CONDITIONS		DESIGN		EXPECTED EFFECTS	
	Food supply	Governance features	Meal preparation & delivery (EXAMPLE OF COMPONENT)	Food procurement (EXAMPLE OF MODEL)	Implications for the school food environment	Key limitations and risks
Home-Grown School Feeding (HGSF) — decentralised model	Supply is tied directly to local agricultural cycles and smallholder output; requires minimal formal warehousing.	Local autonomy; direct links with family farming. Food availability and diversity fluctuate with harvest seasons, local production capacity, policies in place and proximity to markets.	Prepared at school or a nearby kitchen with locally procured goods.	School-level procurement from local smallholder farmers within a defined radius (e.g. Malawi, Brazil).	Greater availability of fresh foods; strongest links between schools and local food systems; supports dietary diversity [13,14].	Exposed to seasonality and supply shocks; consistency of the food basket depends on complementary procurement and market-access mechanisms (Section 5.4).
Home-Grown School Feeding (HGSF) — centralised model	Supply is tied directly to local agricultural cycles and smallholder output; requires minimal formal warehousing.	Central coordination with domestic sourcing mandate. Food availability and diversity fluctuate with harvest seasons, local production capacity, policies in place and proximity to markets.	Prepared at school from centrally distributed local produce.	National-level procurement from domestic smallholders, aggregated and distributed to schools (e.g. Madagascar, Senegal).	Wider menu range and easier quality standardisation than the decentralised model.	Greater logistics and pipeline risk; local food-system linkage is weaker than the decentralised model.
Centralised School Meals programmes	Staple or non-perishable items typically move through	Central coordination with local	School-level preparation guided by	Combination of centralised procurement and local	Balance between standardisation and local	Can inherit the weaknesses of both HGSF and traditional

(no or little homegrown component)	centralised logistics, while fresh/perishable items are sourced locally; overall reliability depends on how well the two supply streams are coordinated and sequenced.	implementation.	national menu guidelines.	sourcing (e.g. The Gambia, São Tomé e Príncipe).	flexibility; moderate dietary diversity potential [17].	models if coordination between central and local actors is poor.
Centralised catering	Relies on large-scale, often contracted supply chains (wholesalers, food-service distributors) with standardised specifications; high consistency and shelf stability, but sourcing is generally decoupled from local production and seasonality.	Strong national oversight and nutrition standards possible.	Meals prepared in central kitchens or by contracted catering companies.	Large-scale centralised procurement (common in Europe, the US, parts of Eastern Asia).	High scalability; consistent standards possible.	Risk of supply-chain industrialisation and portion misalignment [16]; weak link to local food systems.
Decentralised catering	Local caterers or community providers source independently from nearby markets or small suppliers; supply reliability rests on each provider's individual purchasing relationships rather than a centrally managed system, so it can vary considerably between sites.	Central coordination with local delivery, often with explicit employment goals.	Prepared at school or local kitchen level by locally employed staff.	Meals prepared by local small businesses or community groups (e.g. Jordan).	Generates local income opportunities ; can combine catering with social-protection objectives [19,30].	Standardisation and quality-control challenges; outcomes depend on training and oversight of local providers.
Food transfer / take-home rations	Supply chain resembles conventional food-aid or social-protection logistics — centrally procured, often fortified or long-shelf-life commodities distributed in bulk; not tied to local food production or	Often used in humanitarian or social protection contexts.	Distributed to households rather than consumed at school.	Centrally procured food baskets or fortified products.	Improves household food security; limited control over the in-school food environment.	Weak influence on in-school dietary practices; not designed to shape the school food environment directly.

	the school food environment at all.					
Non-institutionalised / competitive foods (canteens, vendors, retail)	No programme-level supply chain exists; whatever is available depends entirely on private vendors' or retailers' own sourcing decisions, which sit outside any programme control.	Often weakly regulated.	Foods purchased independently by students.	Private vendors, school canteens, retail outlets, food brought from home.	Dominates near-school environments with energy-dense, ultra-processed foods; risk amplified with adolescent autonomy [18,34].	Sits outside formal programme structures, so is not directly addressable through programme design alone; requires food-environment regulation.

Source: Based on scoping review (2026). Limitations column developed for this White Paper from the narrative synthesis in Sections 5.3 and 5.4.

Table 14 sets seven modalities and models against the four design dimensions introduced in Section 5.1. On who procures food and from where, the rows run from school-level purchase from smallholders (decentralised HGSP) and national aggregation of domestic produce (centralised HGSP) to mixed central and local streams, large contracted supply chains (centralised catering), local caterers sourcing independently (decentralised catering) and centrally procured baskets (take-home rations). Competitive foods have no programme supply chain at all – as they are not considered as programmes in this paper. On preparation and service, meals are cooked at school or in a nearby kitchen, in central kitchens or by contractors, or at home.

On the food environment, the rows with locally sourced or standards-based provision are associated with fresher foods, more diversity and easier standardisation. Non-institutionalised and competitive foods dominate the near-school environment with energy-dense, ultra-processed options. On policy and legal structures, governance ranges from local autonomy to strong national oversight, and competitive foods are weakly regulated, so they cannot be addressed through programme design alone.

Read against the pathways in Section 5.1, the table supports the first two. Through the meal itself, composition, standards and regulation of competing foods are the design conditions most consistently linked to dietary quality. Through the food supply chain, procurement source shapes links to local food systems, but the evidence is suggestive, and local sourcing does not by itself determine nutritional quality. The third pathway, persistence of school dietary patterns into adulthood, is not addressed by any row, and the evidence does not allow it to be assessed here.

The table does not estimate effects on NCD/CMD outcomes. It maps which design and governance conditions may enable or constrain the pathways, and the gaps are set out in Section 5.5.

5.5 Summary

This chapter asked under what programme design, governance and food environment conditions those preventive benefits are most likely to be realised. Its answer is that the benefits depend on design: the same modality produces different dietary and health effects depending on what is served, how food is procured and prepared, how the surrounding food environment is regulated and what governance sustains it. Design is therefore a lever for NCD/CMD prevention, not a detail of delivery. The scoping review (116 unique studies, 203 outcome assessments) identifies which conditions warrant priority. It does not estimate the effect of any design on NCD/CMD incidence, and it should be read as identifying supporting conditions, not as proving prevention. Figure 2 and Table 14 organise these conditions as an illustrative synthesis, not a prescriptive model.

What the evidence supports. Read against the three pathways introduced in Section 5.1, the evidence is uneven.

- The meal itself. This is the best-supported pathway. The evidence is consistent that the composition of the meal actually served and the regulation of competing foods within and around schools shape dietary outcomes, and that an unregulated school food environment can offset the gains of a well-designed meal [4,11,18,19,20,46–49]. It is suggestive that these dietary effects extend to health outcomes during school age, and evidence linking them to NCD/CMD incidence is limited.
- The food supply chain and food environment. The evidence is suggestive that local procurement, including HGSF, strengthens local food systems and smallholder income and, where menus are designed to include them, the availability of fresh foods [13–15,20,29]. Procurement source does not by itself determine nutritional quality: a locally procured food of low nutritional value remains of low nutritional value. Price volatility is a further risk where budgets are fixed in advance.
- Dietary patterns established at school and their persistence. The evidence reviewed does not follow children beyond school age in relation to programme design, so this pathway cannot be assessed and remains a critical gap.

Across the four design dimensions, the strongest signals concern what is served and the regulation of the surrounding food environment (consistent). Who procures food and from where, how meals are prepared and served, and the policy and legal structures that support design carry suggestive evidence. Centralised, decentralised and hybrid arrangements trade off cost, meal frequency, standardisation and dietary diversity, and governance capacity, through standards, financing and monitoring, conditions all of these effects. The evidence does not allow the relative weight of modality, sourcing, governance and food environment to be estimated.

Gaps. Four gaps limit these conclusions.

1. The composition, cost and standards of the meals studied are rarely reported in enough detail to compare programmes, so effects cannot be attributed to design choices rather than to what was served.
2. Modality is seldom stated explicitly and often had to be inferred from programme descriptions or legal frameworks, and no formal assessment of risk of bias was possible.
3. Evidence from LICs and LMICs, where many implementing agencies operate, is scarce (4 of 203 outcome assessments come from LICs and 40 from UMICs and LMICs combined, against 141 from HICs), and most of the experimental evidence comes from externally supported programmes that are not representative of national programmes.
4. Dietary patterns are not followed from school age into adulthood in relation to programme design, and there is little guidance on how programmes withstand food-price shocks.

Research priorities. The gaps point to five priorities, each tied to a pathway or dimension above:

1. Direct comparisons of design components, for example ration composition versus procurement source, with and without regulation of competing foods, in LICs and LMICs.
2. Routine reporting of meal composition, cost and the standards applied in evaluations.
3. Explicit classification of modality, model and component in evaluations, using the vocabulary of Section 5.1.1.
4. Implementation-science studies, including on how programmes buffer food-price shocks and sustain standards over time.
5. Longitudinal studies linking design features to dietary patterns and cardiometabolic risk into adulthood.

Until these gaps are addressed, Chapter 5 gives the NCD/CMD discussion a practical reading of Chapters 2 to 4: school meals are most likely to contribute to healthier dietary patterns (the intermediate pathway to NCD/CMD risk) where enforceable standards define what is served, competing foods are regulated and governance sustains both. Local procurement and other modality choices can strengthen this contribution, but their effect on NCD/CMD risk depends on the conditions around them. Design is therefore part of how school meals contribute to prevention, and it is where policy attention and future research are best directed.

6. Conclusions and recommendations

This White Paper examined the role of school meals in NCD prevention across four chapters: the immediate impacts of school meal programmes on nutrition, education, agriculture, and social protection (Chapter 2); the behavioural pathways through which school meal exposure shapes children's eating beyond the school gate (Chapter 3); the biological markers and longer-term health consequences of school meal exposure across the life course (Chapter 4); and the programme design, governance, and food environment conditions under which these benefits are most likely to be realised (Chapter 5).

This concluding chapter synthesises the combined evidence from 705 data sources in three sections, moving from what the evidence shows to where the author team takes a position. The first section sets out conclusions that can be drawn from the evidence at varying levels of confidence, alongside the gaps that constrain what the evidence can yet support (Figure 3). The second translates the evidence to implications for policy, research, and funders. The third makes explicit where the White Paper takes a position that goes beyond what the evidence directly demonstrates.

Two features of the evidence base qualify all of the conclusions below. First, the composition of the meals studied, including whether they met a defined standard, is rarely reported, so the role of meal quality can often be inferred but not measured. Second, the evidence is likely to over-represent programmes that were evaluated because an effect was expected, and it comes from a limited number of countries, so it may not represent school meals as they are typically delivered.

6.1 Conclusions based on the evidence

Where the evidence is most consistent: supported by many studies, including experimental and quasi-experimental designs, across multiple contexts.

The evidence covered in this White Paper suggests that school meals can improve children's immediate dietary intake, diet diversity, diet quality and micronutrient status. This is the largest body of evidence in the White Paper, with 248 studies on nutrition outcomes in Chapter 2, 151 of them on dietary intake. Gains are largest in LICs and LMICs, where baseline nutritional deficits are greatest. In LICs, the few available studies consistently show that school meals increase energy and protein intake, and in LICs and LMICs, meals with higher micronutrient content, for example through fortification, improve haemoglobin and reduce anaemia; evidence for school feeding without such enhancement is less clear. In HICs, school meals improve what children eat during school hours, and participation is associated with better diet quality, such as higher Healthy Eating Index scores in Canada and lower sodium and ultra-processed food intake than

packed lunches in the UK. In LMICs, however, intake often still falls short of recommended levels, and in HICs, children's total daily intake often stays the same, possibly because they eat less at other meals. Improvements in dietary diversity and micronutrient status are also often confined to school days and may fade during holidays, and infection can reduce the benefit for micronutrient status.

School meals also seem to act as a direct and reliable incentive for school participation, particularly in food-insecure regions with low participation at baseline. In LICs, studies repeatedly report improvements in attendance and enrolment, in some cases specifically for girls. In LMICs the effects are positive but less consistent, and stronger for attendance than for enrolment. In HICs they are smaller: in the United States, for example, universal free breakfast increased breakfast participation by 20–25% but produced only modest gains in attendance. Evidence on cognitive performance and academic attainment points in the same direction, with the largest effects in LICs and LMICs, but it is less consistent. These outcomes also depend on household circumstances, teaching quality and other concurrent interventions, so they cannot be attributed to school meals alone.

Where the evidence is suggestive but more limited: supported by fewer studies, narrower geographical bases, or by designs that limit causal inference.

School meal exposure may influence children's eating behaviour beyond the school meal itself. In a Danish trial, high-quality school meals providing about half of daily intake improved children's overall weekly diet, although behaviour outside school was not measured separately. Observational and qualitative studies suggest positive effects on what children request at home and on how families organise food provisioning. This evidence is limited in number, design and geographical scope.

The evidence on growth and anthropometric outcomes in middle-income settings is positive and reasonably consistent, particularly for stunting and weight-for-age, though much of it is observational. The few experimental and quasi-experimental studies of metabolic markers, mainly from Denmark, Thailand and Brazil, point to beneficial effects on blood pressure, cholesterol and insulin resistance when meals are of high nutritional quality. Evidence on mental wellbeing, inflammation and the gut microbiota is sparser still and too inconsistent to draw firm conclusions. However, school meals appear to reduce household food insecurity in HICs, which suggests an indirect pathway to mental wellbeing that is rarely studied directly within the school meals literature.

In middle-income settings, home-grown school feeding programmes embedded in strong governance frameworks are associated with dietary diversity gains and with food-system benefits such as demand for produce from family farms, although evidence comparing them with other modalities is limited. Evidence from Brazil and Jordan suggests that school meal programmes with local procurement or local employment, when consistently financed and delivered, can generate income for smallholder farmers

and locally employed school cooks; whether this contributes to NCD prevention through household economic conditions has not been examined.

Direct evidence on NCD outcomes in adulthood is scarce (see the outcome gap below), so the long-term question rests mainly on modelling. Wang et al. (2023) estimate that aligning US school meal standards with the 2020–2025 Dietary Guidelines for Americans could prevent approximately 10,600 NCD deaths annually, and Springmann et al. (2025) estimate that providing every child of school age with a healthy meal by 2030 could reduce undernourishment in LMICs by 24% and avert 0.8–1.2 million deaths annually. The scenario analysis commissioned for this White Paper (Box 4.3) suggests that the benefit would be modest and would emerge only after several decades, depending on how far improvements in children’s diets persist into adulthood and on the nutritional quality of the meals. All three rest on assumptions about meal composition, participation and adherence, the same parameters the design and methodological gap identifies as poorly documented. They are therefore best read as estimates of what well-specified programmes could achieve, rather than as forecasts for programmes as currently delivered. This does not weaken their value as a basis for action, but it does place baseline dietary quality assessment at the convergence of both research agendas: better documentation of what is served would strengthen the modelling evidence as much as the empirical evidence.

A cross-cutting finding: meal quality

One finding cuts across all four evidence chapters: meal quality matters for the size and direction of effects, not only whether meals are provided. In LIC and LMIC contexts, a small number of trials found that enhanced school meals (fortified, more nutrient-rich, or supplemented with animal-source foods) had greater effects on dietary intake, participation and cognitive outcomes than the standard meal or snack provided by the same programme. In HIC contexts, binding nutritional standards (nutritional requirements that schools are legally required to follow) are associated with measurable improvements in diet quality and reductions in BMI among overweight children, while food provision without nutritional standards shows null or mixed effects on overweight and obesity. This evidence comes mainly from a single policy, the US Healthy, Hunger-Free Kids Act, and from England, and may not generalise to systems with long-standing universal provision and standards. What matters therefore appears to be both what is in the meal and likely also how consistently children receive it over time (although this was not studied in this report). The independent contribution of meal quality cannot, however, be conclusively established, because the composition of the meals studied is rarely reported in sufficient detail.

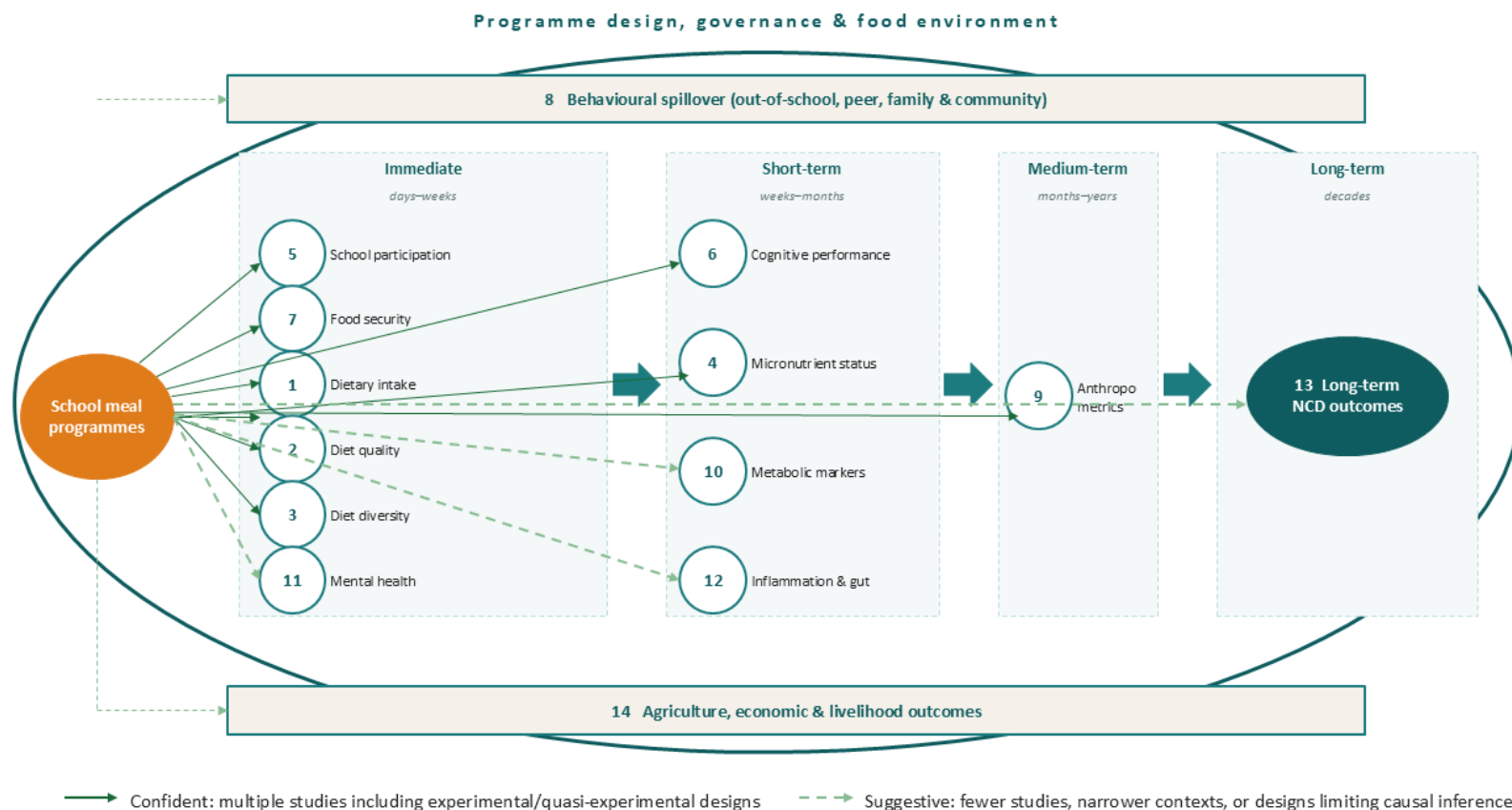


Figure 3. Causal pathway from school meals to long-term NCD prevention with links to the evidence found in this review.

Thin arrows from school meal programmes show direct associations examined in this White Paper, coloured by strength of evidence. Large arrows between time-period quadrants represent the biological pathway through which earlier outcomes mediate later ones (e.g. dietary intake → micronutrient status → anthropometric → NCD; food security → mental health → NCD; cognitive performance → educational attainment → NCD). These mediating relationships are established in other literature rather than examined directly here. The numbers of the outcomes are in the order that they appear in the White Paper's evidence chapters.

Where there are critical gaps – three distinct types of gaps that constrain what the synthesis can support.

Geographical gap. Low-income countries are systematically underrepresented and entirely absent from the evidence base for metabolic markers, inflammation, gut microbiota, social protection, and medium- and long-term outcomes, and represented by very few studies for mental wellbeing and behavioural pathways. Where LIC anthropometric evidence exists, it is largely observational and concentrated in a small number of countries. Countries within the same income group also differ widely, for example Denmark and the USA in their school meal systems, so findings should not be generalised across an income group. Evidence from middle-income countries is documented through a small number of contexts. Research on large programmes such as India's PM POSHAN, which serves about 120 million children, is barely visible in the global databases searched; it is unclear whether this reflects an absence of research or research published in national journals and databases. The evidence is therefore densest in high-income countries and sparsest in low-income countries, where undernutrition is most common and health systems are least equipped to manage a rising NCD burden.

A further observation follows from this geography. The programme configurations the evidence identifies as most promising — home-grown school feeding embedded in binding legal and governance frameworks — already operate at national scale in a number of middle-income countries, financed domestically and institutionalised in law, in several cases for decades; Latin America offers the clearest concentration of such cases. That these programmes generate comparatively little internationally indexed evidence may reflect the distribution of research infrastructure, funding and publication practice, including language of publication, rather than an absence of programme experience. This makes the middle-income gap qualitatively different from the low-income one: the exposure has already occurred, the cohorts exist, and participation records are held by the governments that finance the programmes. Where that is the case, linking those records to health and educational outcomes may be among the faster available routes to strengthening the long-term evidence base.

Design and methodological gap. Across short-term outcome categories, cross-sectional and observational study designs predominate, limiting causal inference. A further cross-cutting limitation is that the nutritional composition of school meals is rarely reported in sufficient detail across studies to allow meaningful assessment of what children actually consumed. This constrains the extent to which outcome differences can be attributed to meal quality specifically, and limits comparability across studies and contexts. Many programmes also lack a defined standard for meal composition in the first place, so the gap lies in programmes as well as in publications. The behavioural evidence base is also notably thin — most studies measure dietary intake or food selection rather than behavioural change as such, and few are designed to isolate the specifically behavioural contribution of school meal exposure. Mental wellbeing, inflammation and gut

microbiota have very few experimental studies. Behavioural literature relies heavily on self-reported measures, including intended food choices, which can correspond poorly with actual food behaviour. The evidence is also likely to be affected by publication and selection bias: programmes were often evaluated because they were designed to achieve a particular effect, so the studies may not represent school meals as typically delivered.

Outcome gap. Long-term NCD endpoints are scarcely empirically documented. Of the medium- and long-term studies identified, only one (Lundborg et al. 2022) examines NCD mortality or morbidity empirically. No longitudinal study tracks dietary behaviour patterns from school age into adulthood as a function of school meal participation. Evidence that dietary patterns and risk factors track from childhood into adulthood (Chapter 1) does not by itself show that changing school meals will change adult NCD outcomes, so the full chain from school meals to adult NCD outcomes cannot yet be demonstrated directly. Social protection, agriculture, behavioural, mental wellbeing, inflammation, and gut microbiota are also poorly studied outcome categories overall.

Headline conclusions from the evidence

- The evidence is the most consistent when it comes to the role of school meals in improving immediate dietary intake, diversity, quality, and micronutrient status, and in improving school participation, particularly in food-insecure regions.
- The evidence also suggests that meal quality, and not provision alone, is a key driver of school meals' contribution to nutritional and educational outcomes, in LIC and LMIC contexts (where quality includes fortification and the addition of nutrient-dense foods such as animal-source foods) and in HIC contexts (where it shows up as binding nutritional standards); the metabolic evidence points the same way but is limited.
- The evidence is suggestive that school meal exposure influences children's eating beyond the school meal, or at least is not offset by compensation outside school; that improvements in household food security may provide an indirect pathway to mental wellbeing; and that, under assumptions about meal quality, compliance and the persistence of healthier eating into adulthood, school meals could contribute to long-term NCD prevention at scale.
- The evidence base contains structural gaps: geographical (LICs and many MICs severely underrepresented), design and methodological (cross-sectional study designs dominance, incomplete reporting of meal nutritional composition, likely publication bias, scarcity of behavioural research, reliance on self-report), and outcome (long-term NCD outcomes, mental wellbeing, inflammation, behavioural pathways).

6.2 Implications across the evidence

Implications for policy

Where the evidence is most consistent or suggestive, several directions appear well-supported.

The evidence is consistent with school meal programmes delivering reliable short-term benefits across multiple domains, provided that meals meet nutritional standards and are delivered consistently, particularly in low- and middle-income contexts where the potential to benefit appears greatest. The evidence also suggests that these benefits depend on prioritising meal quality, not just provision. Healthy diet principles are universal, but what meal quality requires depends on how far current diets are from them, so it differs by context. In LIC and LMIC contexts where undernutrition is the primary concern, it points to fortification, nutrient-richness, and the inclusion of animal source foods or other nutrient-dense components. In HIC contexts where overnutrition dominates, it points to limiting added sugars, sodium, and ultra-processed foods. Many MIC contexts face both challenges simultaneously and require attention to both undernutrition and overnutrition. Across all settings, the evidence supports the case for binding nutritional standards aligned with dietary guidelines as a means of ensuring that essential nutrients are present where they are lacking and limiting harmful dietary components where these dominate. Investing in coverage without investing in quality is unlikely to deliver the NCD-prevention potential the modelling evidence projects, and poor-quality meals could add to NCD risk.

The evidence suggests that no single modality is universally optimal and that programme design is likely to benefit from being context-dependent. In HICs, binding nutritional standards and regulation of competitive food environments are associated with improvements in diet quality and, in some studies, BMI, which are risk factors for NCDs. In MICs, particularly UMICs, home-grown school feeding embedded in strong governance frameworks seems to offer integrated nutritional, agricultural, and economic benefits. In LICs and LMIC, where undernutrition remains a primary concern, programmes likely benefit from design that addresses baseline nutritional deficits while building governance capacity, which requires financial and institutional resources. Governance and institutional architecture appear integral to programme design rather than background conditions; programme effects on diet quality and NCD-relevant outcomes are observed in conjunction with the legal, financial, and governance frameworks that surround them.

The behavioural evidence further suggests that programme acceptability and the social eating context appear to be more consequential for behavioural outcomes than is typically reflected in programme design. The qualitative dimensions of school meal delivery, such as taste, cultural fit, peer norms and relief of family burden, may influence whether the dietary benefits documented at the point of consumption translate into behavioural change in households and communities,

alongside household resources and the wider food environment, although this rests mainly on observational and qualitative evidence.

Realising these benefits also depends on commitment and resources. Programmes need explicit nutrition objectives with measurable targets, such as compositional targets for the meals served, and the budget, institutional capacity and financing to deliver meals of that quality consistently. Funders of programmes, not only of research, have a role in making nutritional quality a condition of investment.

Meal quality alone is unlikely to be enough where children can readily buy or bring less healthy foods. Policies are likely to be most effective when binding standards cover not only the meals served but also competitive foods, vending, canteens, vendors and marketing within and around schools, and when they are backed by procurement specifications, monitoring and enforcement (Chapters 3 and 5).

The evidence also has implications for targeting. Effects on growth and other outcomes are often largest among children who were most disadvantaged at baseline, such as those from the poorest households or with the poorest nutritional status (Chapters 2 and 4). Where universal provision is not yet feasible, as in many LICs, prioritising the areas and groups with the greatest need is likely to yield the largest gains. Evidence from universal and near-universal programmes suggests that benefits also concentrate among the most disadvantaged children when provision is universal, without the stigma and administrative costs of means-testing, so the choice between targeted and universal provision depends on context and resources.

These implications are likely not of equal urgency, and few governments are likely to be able to act on all of them at once. The evidence profile suggests an order of operations. The first step is diagnostic and comparatively inexpensive: documenting what is currently served, as planned and as delivered. Without this baseline no subsequent change can be attributed or evaluated, which is why the research implications set out below identify it as foundational — and it is a step that requires no legislative change and can begin immediately. The second is normative: establishing binding nutritional standards aligned with national dietary guidelines, which the evidence associates with measurable improvement in the settings where they have been introduced. The third is operational: building the monitoring capacity, kitchen infrastructure, workforce and supplier relationships without which standards remain aspirational, and which the implementation evidence identifies as the point at which programmes most often fail to convert intention into delivery. The fourth extends beyond the plate to the food environment within and around the school, where the evidence indicates that the gains from high-quality meals can be substantially offset. Governments are not choosing among these steps but ordering them, and the order matters: each step makes the next both cheaper to execute and possible to verify.

Implications for research

The three gap types each generate distinct research priorities.

The cross-cutting finding that meal quality matters for health outcomes has a direct implication for the research agenda: understanding what is in the meals being delivered is foundational to any further analysis. Yet the studies reviewed in this White Paper are concentrated in a small number of country contexts, meaning baseline dietary quality data — both as planned and as delivered — remain sparse for the majority of school meal programmes globally, and meal composition is rarely reported in the studies themselves. Without this foundation, attribution, comparison, and evaluation across programme contexts remain difficult. A theory of change set out at the start of an evaluation, linking what is served to the outcomes expected, would also make clear which outcomes a given menu can plausibly affect. The three gap types identified in Section 6.1 each generate distinct research priorities, all of which are made more tractable by a stronger foundation of baseline dietary quality assessment in under-evidenced settings.

To address the geographical gap, research investment is likely to deliver the most value in low-income countries and the under-documented middle-income contexts. Strengthening the LIC evidence base appears to require mapping of current programmes and their components, baseline and longitudinal studies, implementation research, and policy analysis grounded in social science as well as epidemiology — research designed for the institutional, infrastructural, and governance realities of low-income contexts, rather than an extension of HIC research designs into new geographies.

To address the design and methodological gap, designs capable of supporting causal inference are likely to be of particular value across the outcome categories where the field currently relies on cross-sectional evidence. RCTs and quasi-experimental designs appear especially needed for metabolic markers, mental wellbeing, inflammation, gut microbiota outcomes, and behavioural pathways. Observational and behavioural measurement methods that move beyond self-report — including biomarkers, plate waste, administrative records, and direct observation — appear especially needed in behavioural research. A common but adaptable evaluation protocol, with a minimum core set of outcomes that can be measured in all income settings and extended with biomarker and long-term modules where resources allow, would improve comparability and move evaluation beyond BMI. Evaluations of long-standing universal systems with binding standards, for example in Central and Eastern Europe, Japan and Finland, and of how far the effects of standards depend on enforcement rather than adoption, would show whether findings from the USA generalise.

To address the outcome gap, longer follow-up periods and a focus on NCD endpoints are likely to be needed to address what the field cannot yet evidence empirically. The mismatch between current empirical follow-up (typically under 20 years) and the time horizons over which NCD risk

develops can plausibly be partly addressed through prospective/retrospective cohort designs and stronger linkage of school meal exposure to long-term health and administrative records. The role of household food security and economic conditions, and the behavioural pathways through shared mealtimes, household food practices and children's eating at home, appear to deserve greater attention. Such studies face long latency periods, confounding by socioeconomic and lifestyle factors, and the difficulty of attributing long-term outcomes to one dietary intervention that provides only part of children's daily intake; reporting the share of daily energy and nutrients provided by school meals would help interpret their effects. Comparative evidence on how school meals perform against, and alongside, other interventions to change dietary practices across the life course is also needed.

Implications for funders of research and programmes

Funders are well-positioned to address asymmetries that individual researchers and institutions cannot easily correct. Five priorities follow from the evidence profile. First, investment in documenting the composition of school meals, as planned and as delivered, using established metrics such as food-group diversity or the Global Diet Quality Score for meals, appears to be a foundational priority for the field. The cross-cutting finding that meal quality matters for nutritional outcomes makes baseline dietary quality assessment a logical foundation for the entire research agenda. Without it, attribution, comparison, and evaluation across programme contexts remain difficult. Second, funding is likely to add the most value where it rebalances the geographical asymmetry of the evidence base, because the potential benefits of school meals for nutrition and later NCD risk appear greatest where evidence is scarcest; closing the LIC evidence gap is likely to require sustained investment in research capacity, infrastructure, and partnerships in under-evidenced contexts. Third, broadening the evidence base on long-term NCD outcomes requires cohort studies, implementation research, and linkage with health-record systems—designs that take years to deliver results but are essential to building the empirical base, including on whether and under what conditions school meal programmes lead to sustained dietary change. In the meantime, modelling evidence already provides a defensible basis for investment, and waiting for empirical confirmation is likely to be costly given the burden of disease projected by the models. Funding both modelling and longitudinal designs in parallel, rather than treating them as alternatives, appears to be the most pragmatic path forward. Fourth, the under-evidenced outcome categories — mental wellbeing, inflammation, gut microbiota, behavioural pathways, and indirect pathways through food security and household economic conditions — are disproportionately underfunded relative to anthropometric and educational outcomes and thus warrant attention. Fifth, funders of programmes can make nutritional quality, consistent delivery and the budgets these require a condition of their investment.

Implications for implementers

The White Paper findings suggest that nutritional quality (and likely also implementation fidelity, meaning whether meals are delivered as planned on the days planned) are key to NCD-relevant outcomes, with direct implications for those responsible for delivering programmes on the ground. Ensuring that what is planned is what is delivered, and that what is delivered meets nutritional standards, requires adequate and sustained financing, monitoring systems, supplier relationships and kitchen capacity that extend well beyond food procurement alone. Where these systems are weak, the gap between intended and actual meal quality is likely to be consequential for the health outcomes programmes can achieve.

The behavioural evidence further suggests that meal acceptability—shaped by taste, cultural resonance, presentation, and the social context of eating—plays an important role in determining whether children consume what is offered and whether dietary benefits extend beyond the school gate. Investing in the qualitative dimensions of meal delivery, including involving children and families in menu design and fostering positive shared mealtime environments, appears to matter more for behavioural outcomes than is typically reflected in programme design.

The evidence on competitive foods and informal vendors also suggests that the benefits of high-quality school meals can be undermined by the wider food environment within and around school premises. Where programme design can account for what children are exposed to beyond the plate—including vendor access and what children eat before and after school—this appears worth attending to. This is an implementation and regulatory task as well as a design one, requiring coordination with school leadership, local authorities and vendors, and mechanisms to monitor and enforce restrictions on the sale and marketing of less healthy foods.

Furthermore, the agriculture evidence suggests that home-grown school feeding can generate food system and livelihood benefits, but that these do not emerge automatically from local sourcing mandates. Realising these benefits appears to require supply chain infrastructure, reliable payment systems, and institutional coordination that implementers and their partners need to actively build and maintain.

Finally, a dimension of quality that this White Paper identifies but that programme monitoring rarely captures is continuity of exposure. Section 6.1 concludes that the active ingredient is continuity as well as composition, yet the number of days on which meals are actually served — as distinct from the number planned or budgeted — is seldom recorded in either programme monitoring or the research literature. The distinction is consequential: a programme whose menu meets national standards but which serves meals on two-thirds of the school year delivers two-thirds of the exposure those standards were designed to produce, and no assessment of meal composition will detect the shortfall. Interruptions arise from ordinary operational causes — delayed disbursement, procurement failures, breakdowns in the relationship between schools and

local suppliers — and are therefore tractable wherever they are measured. Recording days served alongside meal composition would strengthen programme management and the evidence base together, and would express the cost of quality in the terms in which programmes are actually budgeted.

Headline implications

- **For policy:** the evidence suggests that meal quality, not provision alone, is a key driver of nutritional and educational outcomes, with more limited evidence for metabolic outcomes; investment in coverage without parallel investment in quality is unlikely to realise the NCD-prevention potential projected in the modelling evidence.
- **For policy:** programme design is likely to benefit from being context-dependent (nutritional standards and food environment regulation in HICs; home-grown school feeding embedded in strong governance in MICs; design addressing baseline deficits in LICs), with governance, financing and institutional architecture as integral components.
- **For policy:** acceptability and social eating context appear to be more consequential for behavioural outcomes than typically reflected in programme design. Structured food and nutrition education linked to the meal, as in Japan, may strengthen these effects.
- **For policy:** programmes need explicit nutrition objectives with measurable targets and the budget to deliver them, binding standards that extend to the whole school food environment, and, where universal provision is not yet feasible, prioritisation of the children and areas with the greatest need.
- **For research:** baseline dietary quality assessment is foundational to interpreting outcomes; the geographical, design and methodological, and outcome gaps each generate distinct priorities, with particular value in LIC research, causal designs, behavioural measurement beyond self-report, and long-term NCD endpoints, as well as multi-domain designs that assess dietary, health, behavioural, economic and food-system outcomes in the same programme, and stronger research capacity and equitable partnerships in LIC and LMIC contexts.
- **For funders:** baseline dietary quality research, rebalancing the geographical asymmetry, supporting modelling and longitudinal designs in parallel, and investing in under-evidenced outcomes are the highest-leverage priorities; for funders of programmes, making nutritional quality and consistent delivery a condition of investment.
- **For implementers:** adequate financing, monitoring of implementation fidelity (including the days on which meals are actually served), kitchen capacity and supplier relationships

are likely necessary conditions for translating nutritional standards into practice; meal acceptability and the social eating context deserve greater attention in programme design; and the benefits of home-grown school feeding require active investment in supply chain infrastructure and institutional coordination to be realised.

6.3 Closing positional statements

The conclusions set out in sections 6.1 and 6.2 are grounded in the evidence reviewed across the four substantive chapters of this White Paper. This section makes explicit where the White Paper takes a position that goes beyond what the evidence directly demonstrates — reflecting the collective judgement of the author team rather than conclusions that can be drawn from the synthesised literature alone.

School meals should be treated as a health intervention, not only an education one. The evidence reviewed in this White Paper spans nutrition, behaviour, metabolic health, agriculture, and social protection—outcomes that fall as much within the mandate of health, social protection, and food systems as of education systems. Yet in most countries, school meal programmes are designed, funded, and governed primarily by Ministries of Education, with limited systematic involvement from Ministries of Health, Agriculture, and Social protection. We take the position that this governance gap is consequential: the NCD-prevention potential documented in this White Paper is unlikely to be fully realised without greater multi-sector engagement in programme design, nutritional standard-setting, and monitoring. Other food system actors, including producers, processors and retailers, also shape what reaches schools, and their role needs to be governed in line with programme objectives. Greater multi-Ministry involvement in school feeding is not merely desirable but structurally necessary if school meals are to function as the beneficial intervention the evidence suggests they can be across diverse outcomes.

Meal quality is a non-negotiable component of NCD-prevention potential. The cross-cutting finding that meal quality, not provision alone, matters for NCD-relevant outcomes has a policy implication that goes beyond what individual studies demonstrate: programmes that deliver meals without adequate attention to nutritional quality are unlikely to contribute meaningfully to NCD prevention, regardless of their scale or coverage. We take the position that coverage targets without accompanying quality standards are insufficient, and that funders and governments should treat nutritional quality as a condition of investment rather than an optional enhancement. Quality in this sense includes cultural acceptability, which determines whether meals are eaten, and the environmental sustainability of what is procured, alongside nutritional adequacy.

Schools should be healthy food environments as a whole. We take the position that the aim should be a school food environment in which healthy foods are available and less healthy foods, including ultra-processed foods and sweetened beverages, are not routinely sold, promoted or made readily available through competitive channels. Nutritional standards achieve this only if

they are implemented, monitored and enforced, and they need to be defended after adoption, since they can be relaxed under commercial pressure or because meals are poorly accepted. Transparent, evidence-informed standard-setting with safeguards against conflicts of interest is therefore part of programme governance.

School meals are an equity intervention as much as a nutrition one. The structural conditions that drive NCD risk—poverty, food insecurity, constrained food environments—are conditions to which school meals can contribute, alongside other interventions. We take the position that school meals represent one of the few preventive strategies capable of reaching children who face the highest NCD risk precisely because of their structural circumstances, and that this equity dimension should be central to how programmes are designed, evaluated, and funded, not treated as a secondary benefit. Where universal provision is not yet feasible, prioritising the children and areas with the greatest need is consistent with this position (Section 6.2).

Uncertainty about long-term effects should not be read as evidence of no effect. Direct evidence on whether school meals prevent adult NCDs is scarce and mixed, and long-term effects remain uncertain because the available studies are too few and too short to determine them. This largely reflects the time horizons over which NCD risk develops and the rarity of cohorts followed for the decades required. We take the position that this uncertainty should be weighed alongside the stronger evidence on dietary and nutritional outcomes, the more limited but directionally consistent evidence on metabolic markers, and modelling studies that project population-level benefits under stated assumptions. The pathway in Figure 3 combines evidence synthesised in this White Paper with mediating relationships established in other literature; it is a proposed causal pathway, not one this review demonstrates. Taken together, these provide a principled basis for investing in high-quality school meals in advance of direct empirical confirmation, while long-term evaluations are built. Waiting for that confirmation before acting is likely to be costly relative to the burden of disease the modelling projects.

The window for prevention closes fastest where populations are ageing fastest. Countries in accelerated demographic transition face a compressed interval between the childhood in which dietary risk is established and the later life in which its costs are realised — an interval that unfolded over several generations in most high-income countries. We take the position that in these settings the case for investing now in the nutritional quality of school meals rests not only on the biological argument for early intervention but on its timing: the cohort now in school will reach the ages of highest NCD incidence while their health systems are still building the capacity to manage chronic disease at scale. The countries where school meal programmes are most consolidated, most domestically financed and most legally embedded are, in a number of cases, also the countries with the least time — and those programmes are the instrument most immediately available to them.

Prevention over treatment deserves greater investment. The current trajectory of NCD management is increasingly oriented toward pharmacological intervention — most visibly in the growing use of weight-loss injections as a response to obesity and metabolic disease. While such treatments represent important advances, they address consequences rather than causes, and their cost and accessibility mean they are unlikely to reach the majority of people carrying the greatest NCD burden globally. This White Paper did not compare prevention and treatment directly; this position rests on the judgement of the author team. We take the position that structural, upstream prevention, of which school meals are one important example, deserves greater and more sustained investment alongside treatment-focused approaches. Prevention starts before school age, in the first 1,000 days from conception, and school meals can extend it through childhood and adolescence. A child whose dietary patterns are shaped early, in an environment that supports healthy eating, is less likely to require intensive intervention later.

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Introduction

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Chapter 2

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Chapter 3

Primary studies cited as evidence in Sections 3.3.1–3.3.3: references 1–7, 9–17, 19–25, 27–30, 32, 40–42 and 45. Reviews and guidelines cited for context in Sections 3.1 and 3.4 only: references 18, 26, 33, 38, 39, 43 and 44. Grey literature: references 8, 31 and 34–37 (programme reports, evaluations and institutional surveys). References 12, 13, 16 and 24 were replaced during revision with verified sources identified outside the scoping searches; renumber the list in order of first citation before submission.

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Chapter 4

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Chapter 5

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Annex 1: Methodological approach

The White Paper draws on a series of scoping reviews undertaken by four research teams, each examining one component of the causal pathway through which school meal programmes may contribute to non-communicable disease (NCD) prevention. To support transparency and consistency across chapters, the reviews followed a common methodological approach informed by established frameworks for scoping reviews and reported using a PRISMA flow diagram. This annex describes the shared approach; chapter-specific details on outcome definitions, included databases, and time periods are provided within each chapter or its sub-annex where relevant. The four reviews were conducted by separate teams, so databases, search strategies and time windows differed between chapters; Chapter 5, for example, added two databases based in the Global South.

Review approach

A scoping review approach was adopted across all four reviews. Scoping reviews are appropriate where the aim is to map the breadth and nature of the available evidence on a topic — including the types of studies conducted, the populations and settings examined, the outcomes measured, and the gaps that remain — rather than to derive a pooled quantitative estimate of effect. Given the heterogeneity of school meal interventions, study designs, and outcome measures across the literature, this approach allowed the reviews to characterise the existing evidence base in a way that meta-analysis would not have permitted. A systematic review with formal risk-of-bias and certainty assessment was not feasible across the breadth of questions covered; the limitations this implies are set out in Chapter 1.

Eligibility criteria (PICO)

Population (P) - The target population for all reviews was school-aged children (5–19 years). Studies focused exclusively on preschool-age children or participants over 19 years were excluded. Where outcomes pertained to children's nutrition, education, behaviour, or health but were reported through other respondents — for example, parents, caregivers, teachers, school food authorities, or community members — these studies were included provided the primary outcome concerned the target population or the household and community linked to it. Agriculture and food-system outcomes, such as farmer income or procurement, were included where they arose from a school meal programme serving this population.

Intervention (I) - The intervention of interest was participation in, or exposure to, a school meal programme of any modality — including in-school provision (breakfast, lunch or snacks) and take-home rations, whatever the procurement and preparation arrangement (for example home-grown school feeding, centralised catering, decentralised on-site preparation or hybrid models). Studies of interventions that incorporated nutritional standards, food fortification, behaviour change

components, or related programme design features as part of a school meal programme were also included. Where a programme included several components, effects are attributed to the programme as delivered rather than to the meal alone (see the definition of school meal exposure in Chapter 1). Chapters 3 and 5 also draw on some studies of school food interventions outside school meal programmes to illustrate pathways, and identify them as such.

Comparator (C) - Comparators varied by study design and outcome. They included children not participating in a school meal programme, children participating in a less intensive or differently designed programme, pre-intervention or pre-policy periods within longitudinal or natural experimental designs, and modelled counterfactuals in projection studies.

Outcomes (O) - The outcomes of interest varied by chapter and reflected the distinct stage of the causal pathway under examination. They covered, across chapters, immediate impacts on nutrition, education, agriculture, and social protection; behavioural impacts on children's eating outside school, peer dynamics, and family and community food behaviours; biological markers including anthropometric measures, metabolic markers, mental wellbeing, and inflammation and gut microbiota; and medium- and long-term NCD-relevant outcomes. To avoid duplication across chapters, each review focused on the outcomes specifically within its scope, with chapter introductions making cross-references where outcomes are addressed elsewhere in the White Paper.

Types of studies included

Across all reviews, the following study types were eligible for inclusion:

- Experimental studies (randomised controlled trials and quasi-experimental designs)
- Observational studies (cohort, case-control, and cross-sectional studies)
- Modelling studies where these projected the health, economic, or population-level impact of school meal programmes
- Some chapters also included qualitative research, published systematic and scoping reviews, which are identified as reviews where they are cited.

Non-peer-reviewed academic outputs, such as commentaries, opinion pieces, study protocols, and unpublished theses, were excluded, except where used as supplementary grey literature as described below.

Grey literature

Each review included grey literature from recognised international organisations operating at scale in school feeding — including the World Food Programme (WFP), the Global Child Nutrition Foundation (GCNF), the World Bank, UNICEF, the International Labour Organization (ILO), the

World Health Organization (WHO), the OECD, and national-level institutional sources. Grey literature was included as a supplementary evidence stream to capture programme-level insights and context, particularly from low- and middle-income settings where peer-reviewed evidence is sparse. Grey literature sources were explicitly distinguished from peer-reviewed evidence throughout the synthesis. No formal assessment of methodological quality or risk of bias was carried out for either grey literature or peer-reviewed studies; study design was used as the main indicator of the strength of evidence (see the evidence grades described in Chapter 1).

Search strategy

A structured search was conducted across at least three databases per review, with chapter teams selecting databases appropriate to the disciplinary scope of their outcomes. Databases used across the four reviews included PubMed (MEDLINE), Scopus, Google Scholar, ERIC, PsycINFO, CAB Abstracts, EconLit, AgEcon, and Web of Science. Each review developed a search strategy tailored to the included databases, combining terms for school meal provision with terms specific to the outcomes under examination. Boolean operators and Medical Subject Headings (MeSH) were used where applicable.

Grey literature was identified through targeted hand-searches of institutional repositories, supplemented by reviews of the websites and publications of the international organisations listed above. Country-specific supplementary searches were undertaken where required to capture evidence on programmes that are systematically under-indexed in global databases — for example, an India-targeted supplementary search was conducted in Chapter 3 to identify evidence on PM POSHAN.

Eligibility window and language

Search windows began between 2000 and 2011 depending on the chapter, and extended to February 2026 for all chapters. Start dates were selected to capture approximately two decades of policy-relevant evidence on school meal programmes and child nutrition outcomes; in chapters where the literature base was particularly large, a later start date was applied to ensure the volume of results was manageable within available screening capacity. Evidence predating this period was considered less relevant to contemporary programme design, nutritional contexts, and policy frameworks. English-language publications were prioritised across all reviews; some chapters additionally included French- or other language publications where translation capacity was available within the team.

Screening and data extraction

Title, abstract, and full-text screening were conducted using systematic review software or comparable platforms, against each review's inclusion and exclusion criteria. Records were screened individually with uncertainties resolved by inquiry from other team members. In Chapter

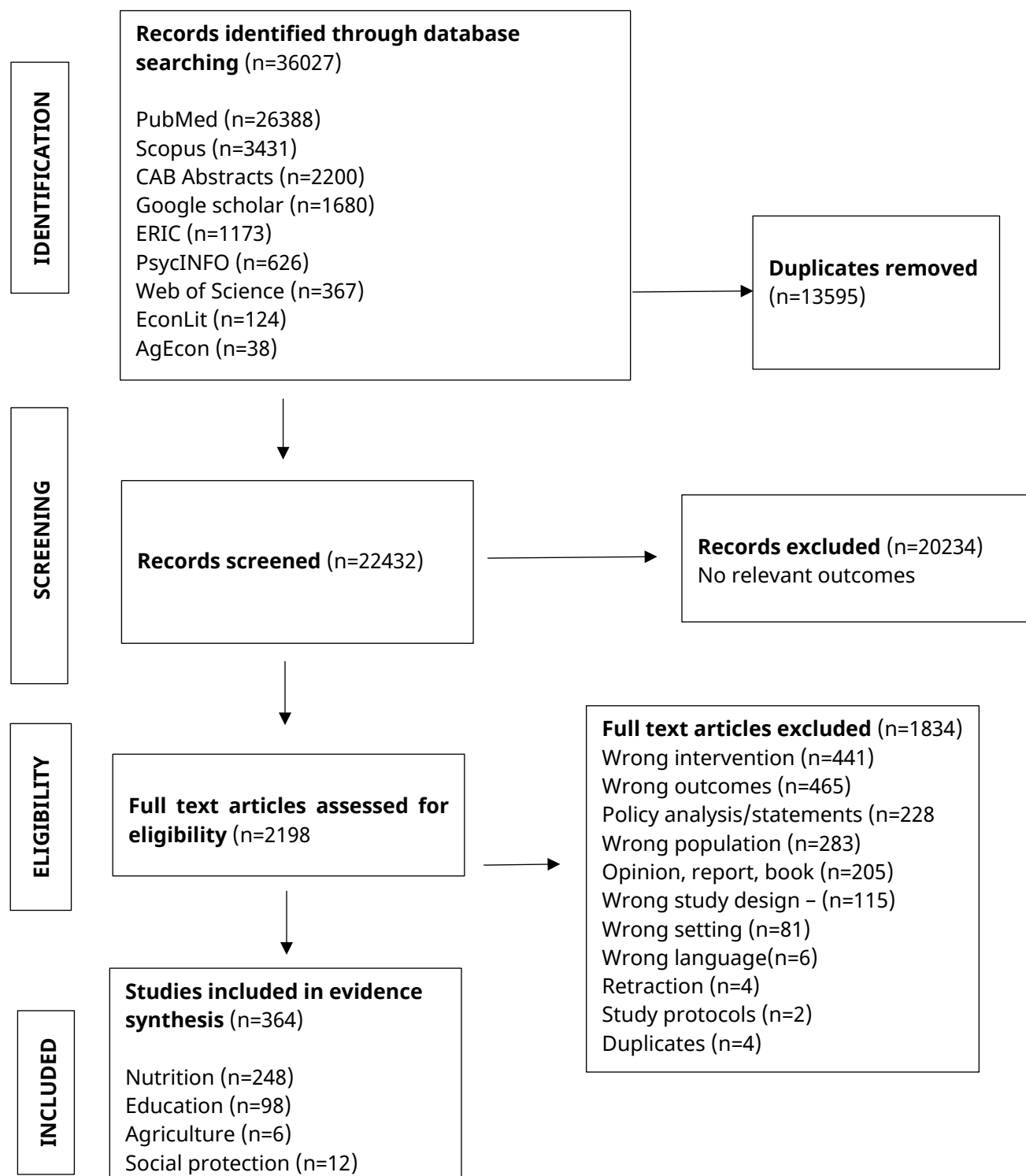
5, two reviewers screened records independently, with a third resolving disagreements. A standardised Excel-based data extraction template was developed for each review, capturing study characteristics (author, year, country, design, sample size), intervention characteristics, outcome definitions and measurement approaches, and key findings. PRISMA flow diagrams were prepared for each review and are provided in chapter-specific annexes.

Synthesis approach

Due to the heterogeneity of study designs and outcome measures across the included studies, quantitative pooling and meta-analysis were not possible in any of the four reviews. A qualitative narrative synthesis was therefore used to summarise findings within each chapter, organised by outcome domain and stratified by World Bank country income setting (LIC, LMIC, UMIC, HIC) where appropriate. This approach is well-suited to characterising the existence, direction, and nature of effects across diverse contexts but is less precise than statistical pooling in quantifying effect magnitudes. Findings were then graded across chapters as consistent, suggestive or limited according to the number of studies, their designs (in particular whether experimental or quasi-experimental studies were available) and the range of contexts (Chapter 1). These grades reflect the judgement of the author team rather than a formal certainty assessment.

Annex 2: Prisma flow chart of included studies for each evidence chapter

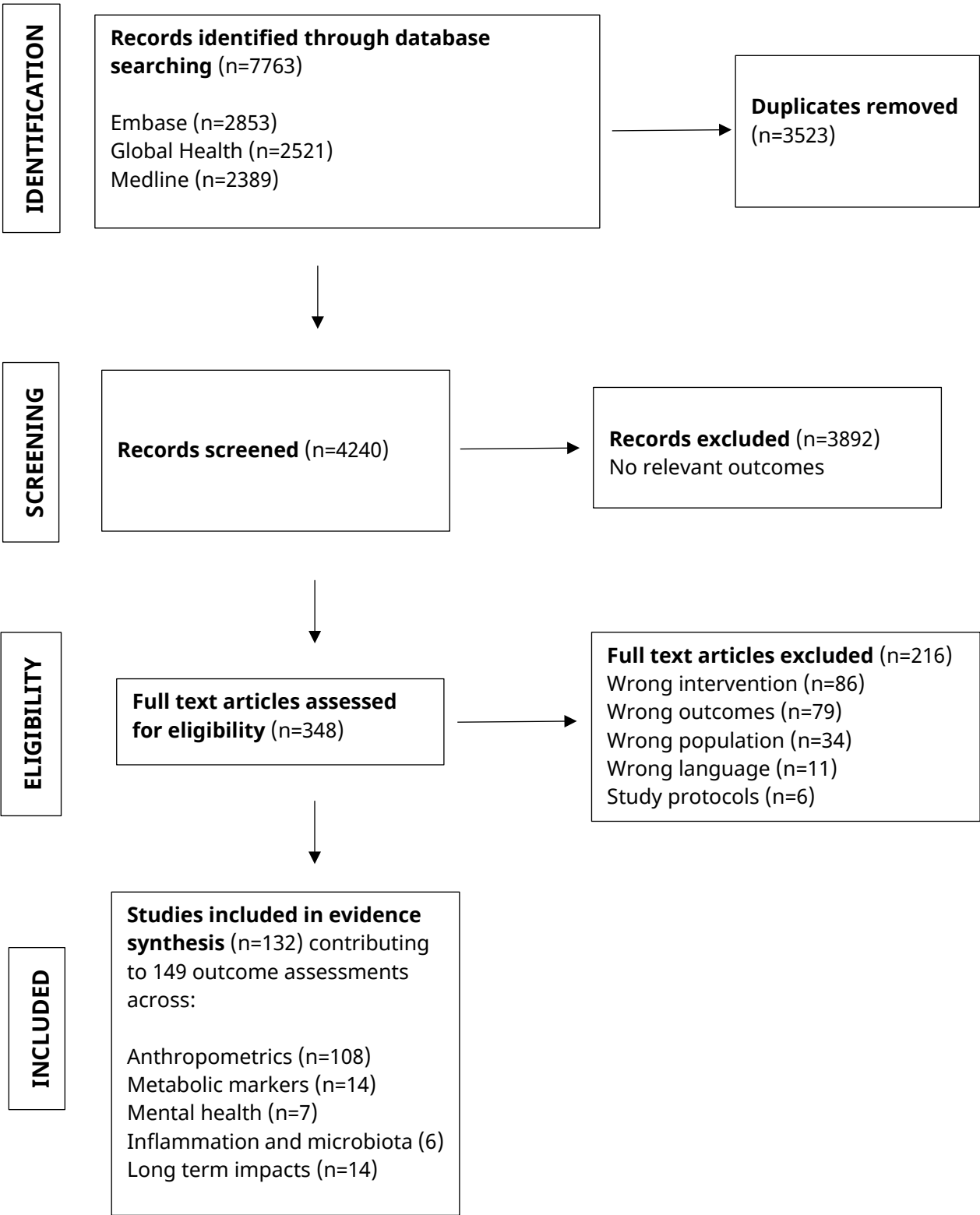
Chapter 2



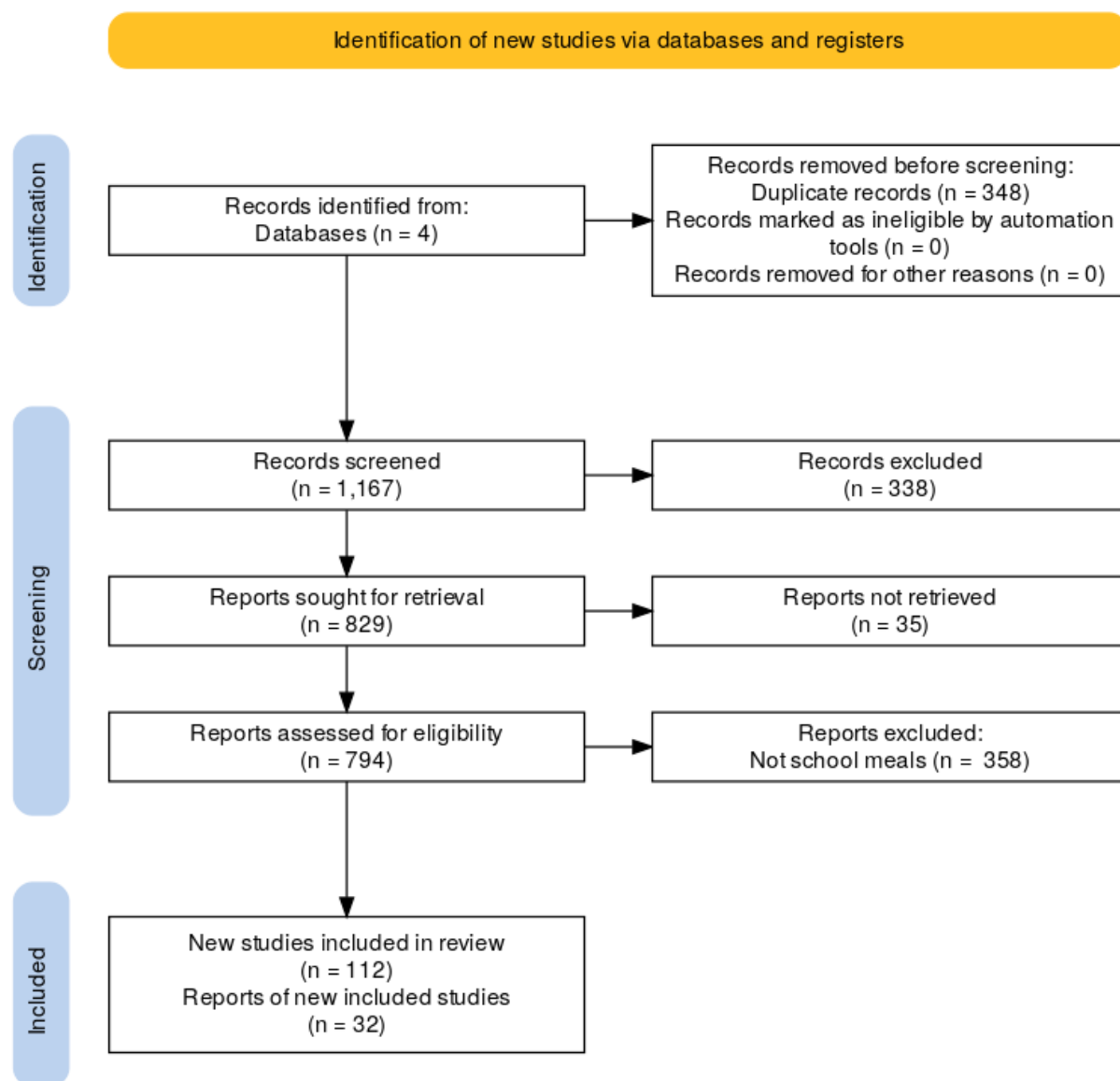
Chapter 3

Stage	n	Notes
Records identified (4 database searches)	~2,200	PubMed, SCOPUS, and supplementary databases
After deduplication	~1,500	Estimate across four contributing teams
Title/abstract screening excluded	~1,300	No out-of-school behavioural outcome; wrong population; wrong exposure; protocol/letter
Full-text assessed for eligibility	~200	Full-text reviewed across all teams
Full-text excluded	~126	Wrong intervention; wrong outcomes; no primary data; biomarker/weight only
Primary peer-reviewed studies included across 3 behavioural domains	41	Spillover 19, Peer/Social 12, Family/Community 10 (study-domain entries; some studies appear in multiple domains)
Grey literature sources included	24	WFP, World Bank, UNICEF, GCNF, Nielsen, ILO, OECD, CDC, EEF, AU, WHO, EC, IDB

Chapter 4



Chapter 5



Annex 3: Extraction tables for included studies

The extraction tables for Chapters 2-5 can be found [via this link](#).

Annex 4: Technical details for the comparative risk scenario analysis

School Feeding and Long-Run Diet-Linked NCD Mortality: Model Details, Findings and Limitations

William Msemburi & David Watkins, 12 August 2026

1. Overview of analytic approach

The analysis asked how improvements in school meals could translate into adult diet-linked mortality benefits from noncommunicable diseases (NCDs) after exposed children would age into adulthood. It proceeded in two parts. Part 1 established the timing and sensitivity of the pathway using illustrative dietary shifts applied across ten countries. Part 2 replaced the illustrative shift, for risks that could be mapped, with contributions derived from observed, food-composition-table (FCT)-matched school menus in five countries and compared those contributions against benchmarks of moving adult diets partway or all the way to a theoretical minimum-risk exposure level (TMREL).

Guiding questions

- How large and how delayed was the mortality effect? If school meals improved child diets during 2025–2050, and a specified share of the gain persisted, how much lower would diet-linked mortality be as these children aged beyond 30?
- What did observed menus contribute? For the five Part 2 countries, which menu components could be mapped to Global Burden of Disease (GBD) Study dietary risks, and how large were their annualised contributions relative to the adult baseline diet?
- How much headroom remained? How did mapped menu contributions compare with moving each sex- and age-specific adult exposure mean 50% or 100% of the way to that risk's TMREL, and did either construction reach a level that was meaningful in terms of population health (which we defined as a 20% reduction in mortality from diet-related NCDs)?

Interpretive frame

The estimate throughout was a conditional scenario effect, not a causal estimate of an existing programme and not a forecast. The primary endpoint, diet-linked 40q30, the probability of dying between ages 30 and 70 from nine diet-linked causes, was a single-decrement life-table probability built from nine diet-linked cause hazards. The range of values presented in the analyses can be interpreted as upper and lower bounds, not as statistical uncertainty intervals.

2. Methods and input data

2.1 Data sources and terms

Three data sources fed the model:

- United Nations World Population Prospects (WPP), 2024 revision: population, fertility, migration and all-cause mortality.
- GBD 2023: cause-specific mortality and adult dietary exposure distributions used directly in the projection. Published population-attributable fractions (PAFs) from GBD were used only for an internal concordance diagnostic, not to parameterise the model. GBD study estimates of risk-cause relative risks and TMREs were also used to estimate impact and to specify the upper bound on impact, respectively.
- Dietary survey data: For Part 2, ingredient-level school menus from a handful of countries were matched to food-composition values and classified using GDQS food groups.

Of 15 GBD dietary risks originally prepared, the relative-risk table and potential-impact-fraction (PIF) calculations used 11; four risks — low nuts and seeds, low omega-6 polyunsaturated fatty acids, low seafood omega-3 fatty acids, and high trans fatty acids — were prepared upstream but not included in the impact model.

Key terms used consistently through the analysis:

- TMREL: as per the GBD Study terminology, this is the theoretical minimum-risk exposure level for a dietary risk.
- PIF: this is the signed proportional change in mean relative risk after shifting an exposure distribution. Positive values reduce the cause hazard; negative values increase it.
- Persistence (p): a term specific to this analysis that indicates the fraction of the childhood dietary change that this analysis assumes is retained in adult diet.
- Programmatic benchmark: this refers to the fact that a shift was applied only to exposed school cohorts (2025-2030), with cohort dose and lag applied.

Table 1 lists the countries included in each part of the analysis, and Table 2 sets out the relative-risk structure used throughout — direction of effect, TMREL and RR unit, coefficient of variation, and the causes each risk affected.

2.2 Model logic common to both parts

Programme exposure. Part 1 placeholder deltas were already annualised per-person changes in the risk's exposure unit and entered the PIF calculation directly. Part 2 contributions began as per-meal amounts and were annualised using school days, programme coverage, uptake and net addition (see section 2.4).

Cohort dose. For each calendar-year and age cell, the model counted years spent at ages 5–15 during 2025–2050 and divided by the maximum 11 school-age years. Boundary cohorts therefore received fractional weights, and ages below the selected effect-onset age received zero.

Potential impact fraction. For each dietary risk and cause, $PIF = [E\{RR(X_0)\} - E\{RR(X_1)\}] / E\{RR(X_0)\}$. Expectations were evaluated over a lognormal exposure distribution using 200 points between its 0.1st and 99.9th percentiles. Part 1 and the observed-menu scenario retained the baseline absolute standard deviation when shifting the mean.

GBD exposure selection. The most recent available exposure year was selected separately for every sex-age-risk stratum, drawn from an extract covering adults aged 25 and older. Five-year age-group values and their PIFs were assigned to single years of age by nearest age-group midpoint, producing a step function rather than smooth interpolation.

Cause-fraction projection. For each country, sex, age and modelled cause, GBD cause-specific mortality was divided by GBD all-cause mortality at six anchor years (2000, 2005, 2010, 2015, 2019 and 2023). The secular slope was the mean of consecutive interval slopes in logit space after a Smithson–Verkuilen boundary correction ($c = 1/12$); absolute slopes were symmetrically winsorised at the country-specific 99th percentile. The 2023 fraction was advanced two years to 2025, then a parabolic taper through 2100 produced half the cumulative displacement of a continued linear trend, and the fraction was frozen thereafter.

Cause rates and joint risks. Projected cause fractions were multiplied by WPP-based all-cause mortality to obtain cause rates; if the nine fractions summed above 0.99 in a given year, age and sex, they were scaled down proportionally. Risk-specific effects for one cause, sex and age were combined as $1 - \prod(1 - \text{delta_PAF})$, an independence construction.

Causal lag and adjusted mortality. The joint impact fraction was multiplied by a logistic age-lag (steepness 0.30; cause-class midpoints of 10 years for cardiovascular disease, 15 years for type 2 diabetes, 25 years for cancer, 12 years for chronic kidney disease) and by the cohort-dose weight. Adjusted cause rates equalled baseline cause rates multiplied by one minus this final fraction; adjusted all-cause mortality was baseline all-cause mortality minus the summed cause-rate reductions, floored at 10^{-10} .

Population projection. The demographic model used WPP 2024 population, mortality, fertility and sex-ratio inputs through 2100, back-solved migration as a cohort-component residual, and

extended arrays to 2155 by holding 2100 fertility, sex-ratio and migration schedules and extrapolating mortality using the final annual log change. Every executed run, however, called a projection function with a calibration setting under which age-specific fertility, sex ratio at birth and net migration were reset after the first step and held at their 2026 schedules for the remaining horizon, so the prepared post-2100 holds for those three inputs did not in fact govern the executed runs. Baseline and adjusted scenarios shared fertility and migration; only mortality differed between them.

Primary endpoint. Diet-linked $40q30 = 100 \times [1 - I[70]/I[30]]$ was calculated from life tables built from the sum of the nine baseline or adjusted diet-linked cause-rate arrays. It was a single-decrement probability, not a conventional competing-risk probability and not the official SDG 3.4 indicator. Figures and endpoint tables used the simple arithmetic mean of the male and female probabilities (not population-weighted); death counts were summed over both sexes. This analysis did not adjust for quality of life or report health-adjusted life expectancy or healthy life-years.

2.3 Part 1: ten countries, illustrative dietary shifts

Part 1 applied the same illustrative dietary-shift scenarios to ten countries. The 18-run grid combined three delta levels, three persistence values ($p = 0.25, 0.50, 1.00$) and two effect-onset ages (25 and 35). The reference scenario used moderate deltas, full persistence and onset at age 25. These numerical levels were scenario inputs, not estimates of an actual school-meal effect.

The placeholder dietary deltas used in Part 1, annualised per-person shifts in each risk's own exposure unit, are given in Table 3.

2.4 Part 2: FCT-matched menus and distance to TMREL

Part 2 used India, Nepal, Cambodia, Guatemala and Lesotho, since these 5 counties had sufficiently complete data on school menus and their components to model changes in the above dietary risks. For risks represented in the menu data, the Part 1 placeholder shift was replaced with the annualised contribution of the food actually served.

Ingredient rows were grouped by day, menu number, age and meal type, then averaged across all observed meal records; a mapped food group absent from a meal contributed zero. The per-meal contribution was annualised using the model's meal-delta parameters: 180 school days, 80% programme coverage, 90% uptake and 75% net addition. Part 1 placeholder deltas did not undergo this step because they were already annualised.

Fruits, vegetables, legumes, whole grains and milk were mapped from GDQS food-group weights; fibre, calcium and sodium were mapped from nutrient columns. A net-of-mapped-harms sensitivity also included red meat where present. Processed meat had no GDQS equivalent, and sweets and juice were not treated as GBD sugar-sweetened beverages.

GBD adult baseline exposure for the eleven modelled risks, by country, is given in Table 4, and the corresponding scale check, comparing the annualised menu-derived delta against the GBD adult mean exposure for the same risk, is given in Table 5.

Three comparable programmatic scenarios were compared on the same basis. All three applied the exposed-cohort dose and causal lag, and Part 2 fixed persistence at $p = 1.00$:

- Mapped protective menu components: observed served-food contributions for protective foods and beneficial nutrients that could be mapped, annualised under the model's implementation assumptions. This was the headline menu scenario, and was partial by construction.
- 50% of mean gap to TMREL: for every sex-age-risk stratum whose adult mean lay on the risk-bearing side of TMREL, the mean moved halfway to the TMREL boundary; strata already on the low-risk side were unchanged. This was specified as an intermediate benchmark against which to compare the mapped menu data.
- 100% of mean gap to TMREL: the adult mean reached the TMREL boundary for every risk-bearing stratum, with the impact fraction capped at the model-implied baseline attributable fraction. This was an upper benchmark against which the mapped menu data were compared.

A separate whole-adult-population scenario removed the exposed-cohort weight and causal lag (retaining only the effect-onset age gate) and was used purely as context.

3. Findings from Part 1

Timing of the modelled benefit

The finite intervention window produced a delayed rise and subsequent decline in the modelled benefit, as exposed cohorts entered and then left the 30–70 risk window. The first materially visible divergence from baseline occurred decades after programme start, because adult disease onset, cause-specific biological lags, and cohort ageing were all applied in sequence before an effect appeared. Figure 1 illustrates this pattern for Kenya under the 18-scenario Part 1 grid.

Country variation and its drivers

Figure 2 shows country variation in the modelled benefit, and Figure 3 shows the assumptions that drove that variation.

Across the ten countries, the reference scenario spanned 0.28–1.24 percentage points off the mortality endpoint at 2095; the full scenario envelope (across all 18 runs) spanned 0.04–1.67 points. The same assumed dietary shifts were applied in every country, so differences in country position reflected baseline cause burden, adult diet gaps and demography. Across the grid, moving persistence from 0.25 to 1.00 increased the mean effect roughly 3.2-fold, compared with about 2.5-

fold from conservative to ambitious assumed deltas. This confirmed that persistence, rather than the size of the assumed dietary shift, was the single most influential parameter in the analysis. The effect also declined once cohorts exposed during 2025–2050 aged beyond 70, producing the finite, hump-shaped pattern seen in Figure 1.

Burden scale under the reference scenario

Figure 4 shows the burden scale (deaths) under the Part 1 reference scenario; counts remained conditional on illustrative dietary shifts and long-horizon demographic projection. Full Part 1 endpoint values at selected reporting years, by country, are given in Table 6.

4. Findings from Part 2

What the observed menus contained

Figure 5 shows the mapped protective-food content of the observed school menus, on a mean grams-per-meal-day basis, by country; this reported served-food content and did not represent consumption or displacement of foods eaten elsewhere.

Timing under Part 2

Figure 6 shows that the timing of the modelled Part 2 benefit remained delayed in the same way as Part 1; the main new information was the size of the gap between the mapped menu contribution and the TMREL benchmarks, discussed below.

Mapped menu contribution against TMREL benchmarks

Figure 7 compares the mapped menu contribution against the 50%- and 100%-to-TMREL benchmarks, by country, at 2095, and Figure 8 shows the same comparison across the full projection horizon.

At 2095, the mapped protective-menu scenario spanned 0.020–0.037 percentage points off the mortality endpoint across the five countries, equivalent to only 1.3%–7.5% of the 100% TMREL benchmark. Mapped menu components sat well below the TMREL benchmarks in every country studied.

Contribution against each risk's own distance to TMREL

Figure 9 shows the mapped menu contribution against each risk's own distance to TMREL; percentages made risks measured in different units comparable. Two country-risk combinations were omitted from the chart because the baseline already sat at or very near TMREL, which made a percentage of the remaining gap unstable; absolute gram-per-day baselines, gaps and menu contributions for all country-risk combinations, including those two, are given in Table 7.

Target-seeking analysis: how far was 20%?

Figure 10 shows the target-seeking analysis: relative reduction in the diet-linked mortality endpoint at 2095 as a common gap-to-TMREL fraction was swept from 0% to 100% in ten-point steps, by country. Figure 11 shows the required gap-to-TMREL fraction to reach the 20% relative reduction that was considered meaningful from a population health impact standpoint.

No focus country reached a 20% relative reduction in the diet-linked mortality endpoint, even when every modelled risk's sex- and age-specific mean was moved fully to TMREL on the risk-bearing side. The dose-response sweep applied one common gap fraction to all modelled risks simultaneously; it did not optimise different shifts by risk, so it should be read as a standardised diagnostic rather than an achievable policy path.

Net effect once mapped harms were included

Figure 12 compares the headline protective-component result against the net-of-mapped-harms sensitivity, by country, at 2095; the sensitivity added menu-contributed sodium and any mapped red meat, and the gap between the paired bars was the endpoint cost of the harms a menu carried.

When the sensitivity also included mapped sodium and any mapped red meat, the projected benefit fell in all five countries, and was lowest in Cambodia at -0.017 percentage points at 2095, against +0.022 points on the protective-component headline. In Cambodia the net effect turned negative; i.e., the mapped harms outweighed the mapped protective components on this endpoint. This sensitivity still did not represent a complete net programme effect, because consumption, substitution, energy balance and unmapped dietary harms remained outside the analysis.

Cumulative deaths averted

Figure 13 shows cumulative diet-linked deaths averted, ages 30–69, across completed annual intervals 2025–2149; India dominated the absolute scale because of population size. Figure 14 shows the same comparison with India removed, rescaling the axis so the remaining four countries were visible. Full Part 2 benchmark values at all reporting years, by country and scenario, are given in Table 8.

5. Limitations and caveats

The model was most informative about the structure, timing, and comparative scale of a direct diet-to-mortality pathway. The limitations below defined the claims that could be carried forward and the issues that this analysis did not address.

Menu data and country selection

Part 2 covered five analyst-designated countries. A reproducible adequacy threshold and country-level inclusion audit were not encoded in the supplied materials. The complete menu workbook and food-composition source notes were not part of this review.

Served food versus realised intake

The menus described food served. Coverage, uptake and net-addition scalars attenuated the served-food contribution, but the model did not directly observe or separately identify consumption, plate waste, attendance, sharing, or displacement of home food, snacks or vendor purchases.

Persistence and causal timing

Persistence, effect-onset age and cause-specific lag midpoints were assumptions. Part 2 used full persistence, which was a high benchmark rather than an empirically established central estimate.

Comparative-risk structure

Adult GBD exposure distributions were used for childhood programme scenarios; risks were combined as independent; means and marginal standard deviations could not represent the joint distribution of diet.

Long-horizon projection

Cause fractions and mortality were extended far beyond observed data. In the executed calibration, age-specific fertility, sex ratio at birth and net migration were frozen at their 2026 schedules after the first projection step. Population and exposed-cohort sizes still evolved through births, survival, migration and ageing, but under those fixed schedules rather than the time-varying WPP trajectories. No probabilistic uncertainty was propagated anywhere in the model.

Endpoint and omitted pathways

The endpoint covered nine diet-linked causes and mortality only. BMI-mediated effects, early-life programming, ultra-processed foods, refined grains, micronutrients beyond calcium, and nonfatal outcomes were outside the estimate.

What the results supported, and what remained out of scope

Results supported:

- A delayed cohort pattern when childhood dietary gains persist.
- Country heterogeneity under a common set of assumptions.
- Persistence of dietary patterns into adulthood as a major structural driver of the modelled effect (persistence itself was an assumption, not an estimate).

- Mapped protective menu contributions are small relative to the all-risk TMREL benchmark.
- Additionally, in no cases did the school meals achieve a 20% reduction in diet-linked mortality risk, so their impact on later-life NCD profile would be modest at best.

Claims that remained outside scope:

- A measured causal impact of current school-meal programmes.
- Programme-quality rankings between countries.
- A complete net effect of each menu.
- Impacts on nonfatal outcomes.
- The economic value of these health improvements to society.
- Statistical confidence intervals or posterior uncertainty.

6. Tables and Figures

Tables

Table 1. Countries included in each part of the analysis. The Part 2 script designated India, Nepal, Cambodia, Guatemala and Lesotho as having adequate FCT-menu coverage; the supplied materials did not encode a quantitative country-adequacy threshold.

Country	Part 1 scenario model	Part 2 FCT-menu analysis
Kenya	Included	Not included in this pass
Nigeria	Included	Not included in this pass
Bangladesh	Included	Not included in this pass
India	Included	Included
Nepal	Included	Included
Lesotho	Included	Included
Honduras	Included	Not included in this pass
Guatemala	Included	Included
Cambodia	Included	Included
Uzbekistan	Included	Not included in this pass

Table 2. Relative-risk structure, read directly from the model script: direction of effect, TMREL and RR unit (in the risk's own exposure unit), coefficient of variation, and the causes each risk affected. A blank cause meant no relative risk was defined for that risk-cause pair, so the pathway carried no effect in the model.

Dietary risk	Direction	TMREL	RR unit	Coefficient of variation	Causes affected
Diet low in fruits	Increase intake	250.00	100.00	0.50	Ischaemic heart disease; Ischaemic stroke; Oesophageal cancer
Diet low in vegetables	Increase intake	360.00	100.00	0.50	Ischaemic heart disease; Ischaemic stroke; Oesophageal cancer
Diet low in legumes	Increase intake	100.00	50.00	0.60	Ischaemic heart disease
Diet low in whole grains	Increase intake	125.00	50.00	0.55	Ischaemic heart disease; Ischaemic stroke; Type 2 diabetes; Colorectal cancer
Diet low in milk	Increase intake	435.00	200.00	0.55	Colorectal cancer
Diet low in calcium	Increase intake	1.20	0.30	0.55	Colorectal cancer
Diet low in fiber	Increase intake	25.00	5.00	0.60	Colorectal cancer
Diet high in sodium	Reduce intake	3.00	1.00	0.30	Ischaemic heart disease; Ischaemic stroke; Haemorrhagic stroke; Subarachnoid haemorrhage; Chronic kidney disease
Diet high in red meat	Reduce intake	0.00	100.00	0.70	Type 2 diabetes; Colorectal cancer; Stomach cancer; Chronic kidney disease
Diet high in processed meat	Reduce intake	0.00	50.00	0.70	Type 2 diabetes; Colorectal cancer; Stomach cancer
Diet high in sugar-sweetened beverages	Reduce intake	0.00	250.00	0.65	Type 2 diabetes

Table 3. Part 1 placeholder dietary deltas, read directly from the model script. Values were annualised per-person shifts in the risk's own exposure unit; note that calcium and sodium rows were on the same gram-per-day scale as the exposure data.

Dietary risk	Direction	TMREL	Conservative	Moderate	Ambitious
Diet low in fruits	Increase intake	250.00	40.00	80.00	120.00
Diet low in vegetables	Increase intake	360.00	50.00	100.00	150.00
Diet low in legumes	Increase intake	100.00	20.00	40.00	60.00
Diet low in whole grains	Increase intake	125.00	30.00	60.00	90.00
Diet low in milk	Increase intake	435.00	80.00	160.00	240.00
Diet low in calcium	Increase intake	1.20	0.06	0.12	0.18
Diet low in fiber	Increase intake	25.00	5.00	10.00	15.00
Diet high in sodium	Reduce intake	3.00	-0.10	-0.20	-0.30
Diet high in red meat	Reduce intake	0.00	0.00	0.00	0.00
Diet high in processed meat	Reduce intake	0.00	-10.00	-20.00	-30.00
Diet high in sugar-sweetened beverages	Reduce intake	0.00	-60.00	-120.00	-180.00

Table 4. GBD adult baseline exposure for the eleven modelled risks, by country. Values were simple, unweighted means across male and female five-year exposure strata with age midpoints 32–67 years, using the most recent available exposure year in each stratum. Units were the risk's own model unit; calcium and sodium were grams per day.

Dietary risk	Bangladesh	Cambodia	Guatemala	Honduras	India	Kenya	Lesotho	Nepal	Nigeria	Uzbekistan
Diet low in fruits	46.07	32.86	102.52	89.54	19.35	79.84	21.70	92.88	87.23	174.63
Diet low in vegetables	46.46	33.22	86.28	48.91	120.74	81.34	31.80	99.13	91.75	416.59
Diet low in legumes	13.07	12.86	25.28	19.20	36.05	30.74	6.98	21.36	19.70	1.53
Diet low in whole grains	4.53	1.91	120.34	103.14	44.18	104.05	163.39	62.49	87.61	39.21
Diet low in milk	47.14	6.38	37.20	62.54	33.03	133.70	188.48	95.97	4.46	277.36

Dietary risk	Bangladesh	Cambodia	Guatemala	Honduras	India	Kenya	Lesotho	Nepal	Nigeria	Uzbekistan
Diet low in calcium	0.23	0.27	0.35	0.35	0.53	0.49	0.36	0.33	0.30	0.90
Diet low in fiber	8.33	6.14	25.86	20.65	18.03	25.04	25.99	20.77	31.51	23.34
Diet high in sodium	3.49	4.55	4.06	4.06	3.96	2.59	2.91	3.49	2.65	3.53
Diet high in red meat	4.35	13.49	17.93	13.08	3.05	15.78	10.99	18.12	9.19	44.52
Diet high in processed meat	2.97	1.00	2.60	1.81	1.24	2.52	1.26	1.89	5.66	9.26
Diet high in sugar-sweetened beverages	4.91	18.69	52.43	56.53	25.42	20.93	47.41	8.75	17.94	36.78

Table 5. Scale check for the five Part 2 countries: the annualised menu-derived delta against the GBD adult mean exposure for the same risk, in the same unit. This check existed specifically to catch unit mismatches; for example, a delta that was an implausible share of the adult mean, the signature of a milligram-against-gram error that this project had previously encountered.

Country	Dietary risk	Per-meal shift (risk unit)	Annualised delta (per day)	GBD adult mean exposure	Delta as % of GBD mean
India	Sodium	0.06	0.02	3.59	0.40
India	Calcium	0.09	0.02	0.46	5.20
India	Fibre	5.80	1.54	17.51	8.80
India	Fruits	5.50	1.46	26.98	5.40
India	Legumes	10.50	2.80	35.10	8.00
India	Milk	5.00	1.33	40.33	3.30
India	Vegetables	38.63	10.29	117.76	8.70
Nepal	Sodium	0.79	0.21	3.48	6.00
Nepal	Calcium	0.04	0.01	0.31	3.80
Nepal	Fibre	2.71	0.72	20.17	3.60

Country	Dietary risk	Per-meal shift (risk unit)	Annualised delta (per day)	GBD adult mean exposure	Delta as % of GBD mean
Nepal	Whole grains	33.33	8.88	63.42	14.00
Nepal	Legumes	20.00	5.33	20.07	26.50
Nepal	Vegetables	33.33	8.88	93.01	9.50
Cambodia	Red meat	6.67	1.78	12.00	14.80
Cambodia	Sodium	0.49	0.13	4.33	3.00
Cambodia	Calcium	0.08	0.02	0.25	8.50
Cambodia	Fibre	1.13	0.30	5.97	5.00
Cambodia	Fruits	0.33	0.09	33.41	0.30
Cambodia	Vegetables	65.03	17.32	31.20	55.50
Guatemala	Sodium	0.10	0.03	3.94	0.70
Guatemala	Calcium	0.17	0.04	0.33	13.60
Guatemala	Fibre	4.71	1.25	25.13	5.00
Guatemala	Whole grains	33.21	8.84	122.13	7.20
Guatemala	Legumes	23.72	6.32	23.76	26.60
Guatemala	Milk	12.96	3.45	38.00	9.10
Guatemala	Vegetables	14.18	3.77	80.88	4.70
Lesotho	Sodium	0.27	0.07	2.71	2.70
Lesotho	Calcium	0.36	0.10	0.34	28.90
Lesotho	Fibre	9.09	2.42	25.24	9.60
Lesotho	Legumes	27.00	7.19	6.58	109.30
Lesotho	Vegetables	6.00	1.60	29.87	5.30

Table 6. Full Part 1 endpoint values at selected reporting years [2025, 2050, 2070, 2095, 2100], reference scenario, sex-averaged, by country. Values are given for the diet-linked 40q30 endpoint and, for context, the corresponding all-cause 70q0.

Country	Metric	Year	Baseline (%)	Adjusted (%)	Absolute reduction (pp)	Relative reduction (%)
Bangladesh	All-cause 70q0	2025	27.03	27.03	0.00	0.00
Bangladesh	All-cause 70q0	2050	16.74	16.73	0.00	0.02
Bangladesh	All-cause 70q0	2070	11.26	11.09	0.17	1.48
Bangladesh	All-cause 70q0	2095	7.12	6.65	0.47	6.47
Bangladesh	All-cause 70q0	2100	6.52	6.17	0.35	5.18
Bangladesh	Diet-linked 40q30, ages 30 to 70	2025	12.05	12.05	0.00	0.00
Bangladesh	Diet-linked 40q30, ages 30 to 70	2050	9.37	9.37	0.00	0.05
Bangladesh	Diet-linked 40q30, ages 30 to 70	2070	6.96	6.78	0.18	2.45
Bangladesh	Diet-linked 40q30, ages 30 to 70	2095	4.62	4.14	0.48	10.28
Bangladesh	Diet-linked 40q30, ages 30 to 70	2100	4.24	3.88	0.35	8.25
Cambodia	All-cause 70q0	2025	34.13	34.13	0.00	0.00
Cambodia	All-cause 70q0	2050	27.32	27.31	0.01	0.03
Cambodia	All-cause 70q0	2070	22.19	21.94	0.25	1.08
Cambodia	All-cause 70q0	2095	16.50	15.78	0.72	4.30
Cambodia	All-cause 70q0	2100	15.49	14.95	0.54	3.48
Cambodia	Diet-linked 40q30, ages 30 to 70	2025	13.61	13.61	0.00	0.00
Cambodia	Diet-linked 40q30, ages 30 to 70	2050	13.20	13.19	0.01	0.08
Cambodia	Diet-linked 40q30, ages 30 to 70	2070	11.62	11.33	0.29	2.36

Country	Metric	Year	Baseline (%)	Adjusted (%)	Absolute reduction (pp)	Relative reduction (%)
Cambodia	Diet-linked 40q30, ages 30 to 70	2095	8.95	8.17	0.78	8.71
Cambodia	Diet-linked 40q30, ages 30 to 70	2100	8.39	7.80	0.59	7.01
Guatemala	All-cause 70q0	2025	31.02	31.02	0.00	0.00
Guatemala	All-cause 70q0	2050	24.03	24.02	0.00	0.01
Guatemala	All-cause 70q0	2070	18.72	18.60	0.11	0.60
Guatemala	All-cause 70q0	2095	13.17	12.85	0.31	2.38
Guatemala	All-cause 70q0	2100	12.23	11.99	0.24	1.94
Guatemala	Diet-linked 40q30, ages 30 to 70	2025	13.42	13.42	0.00	0.00
Guatemala	Diet-linked 40q30, ages 30 to 70	2050	13.27	13.27	0.00	0.03
Guatemala	Diet-linked 40q30, ages 30 to 70	2070	11.75	11.62	0.12	1.03
Guatemala	Diet-linked 40q30, ages 30 to 70	2095	8.88	8.55	0.33	3.68
Guatemala	Diet-linked 40q30, ages 30 to 70	2100	8.27	8.02	0.25	2.98
Honduras	All-cause 70q0	2025	31.46	31.46	0.00	0.00
Honduras	All-cause 70q0	2050	22.82	22.82	0.00	0.01
Honduras	All-cause 70q0	2070	16.70	16.62	0.08	0.45
Honduras	All-cause 70q0	2095	11.03	10.77	0.26	2.25
Honduras	All-cause 70q0	2100	10.17	9.97	0.20	1.88
Honduras	Diet-linked 40q30, ages 30 to 70	2025	11.96	11.96	0.00	0.00
Honduras	Diet-linked 40q30, ages 30 to 70	2050	8.61	8.60	0.00	0.03
Honduras	Diet-linked 40q30, ages 30 to 70	2070	6.22	6.13	0.09	1.44
Honduras	Diet-linked 40q30, ages 30 to 70	2095	4.03	3.75	0.28	6.97

Country	Metric	Year	Baseline (%)	Adjusted (%)	Absolute reduction (pp)	Relative reduction (%)
Honduras	Diet-linked 40q30, ages 30 to 70	2100	3.70	3.49	0.21	5.81
India	All-cause 70q0	2025	33.14	33.14	0.00	0.00
India	All-cause 70q0	2050	23.03	23.03	0.00	0.02
India	All-cause 70q0	2070	16.69	16.53	0.15	0.89
India	All-cause 70q0	2095	10.95	10.45	0.49	4.39
India	All-cause 70q0	2100	10.06	9.70	0.37	3.58
India	Diet-linked 40q30, ages 30 to 70	2025	14.33	14.33	0.00	0.00
India	Diet-linked 40q30, ages 30 to 70	2050	12.17	12.17	0.00	0.03
India	Diet-linked 40q30, ages 30 to 70	2070	9.67	9.50	0.17	1.68
India	Diet-linked 40q30, ages 30 to 70	2095	6.62	6.11	0.52	7.67
India	Diet-linked 40q30, ages 30 to 70	2100	6.09	5.70	0.39	6.23
Kenya	All-cause 70q0	2025	52.57	52.57	0.00	0.00
Kenya	All-cause 70q0	2050	44.69	44.69	0.00	0.01
Kenya	All-cause 70q0	2070	38.97	38.84	0.13	0.33
Kenya	All-cause 70q0	2095	31.45	30.96	0.48	1.54
Kenya	All-cause 70q0	2100	29.89	29.51	0.38	1.25
Kenya	Diet-linked 40q30, ages 30 to 70	2025	16.84	16.84	0.00	0.00
Kenya	Diet-linked 40q30, ages 30 to 70	2050	17.63	17.63	0.00	0.02
Kenya	Diet-linked 40q30, ages 30 to 70	2070	16.76	16.59	0.17	1.04
Kenya	Diet-linked 40q30, ages 30 to 70	2095	13.86	13.25	0.62	4.42
Kenya	Diet-linked 40q30, ages 30 to 70	2100	13.09	12.62	0.47	3.57

Country	Metric	Year	Baseline (%)	Adjusted (%)	Absolute reduction (pp)	Relative reduction (%)
Lesotho	All-cause 70q0	2025	63.54	63.54	0.00	0.00
Lesotho	All-cause 70q0	2050	49.38	49.38	0.00	0.00
Lesotho	All-cause 70q0	2070	41.04	40.98	0.06	0.15
Lesotho	All-cause 70q0	2095	31.54	31.23	0.31	0.99
Lesotho	All-cause 70q0	2100	29.71	29.46	0.25	0.84
Lesotho	Diet-linked 40q30, ages 30 to 70	2025	16.11	16.11	0.00	0.00
Lesotho	Diet-linked 40q30, ages 30 to 70	2050	14.59	14.59	0.00	0.01
Lesotho	Diet-linked 40q30, ages 30 to 70	2070	13.07	12.98	0.09	0.68
Lesotho	Diet-linked 40q30, ages 30 to 70	2095	10.27	9.84	0.42	4.01
Lesotho	Diet-linked 40q30, ages 30 to 70	2100	9.62	9.29	0.33	3.36
Nepal	All-cause 70q0	2025	35.37	35.37	0.00	0.00
Nepal	All-cause 70q0	2050	24.77	24.76	0.00	0.02
Nepal	All-cause 70q0	2070	18.06	17.94	0.12	0.64
Nepal	All-cause 70q0	2095	11.86	11.52	0.34	2.75
Nepal	All-cause 70q0	2100	10.89	10.64	0.25	2.22
Nepal	Diet-linked 40q30, ages 30 to 70	2025	13.61	13.61	0.00	0.00
Nepal	Diet-linked 40q30, ages 30 to 70	2050	10.76	10.76	0.00	0.04
Nepal	Diet-linked 40q30, ages 30 to 70	2070	8.26	8.12	0.14	1.58
Nepal	Diet-linked 40q30, ages 30 to 70	2095	5.49	5.12	0.37	6.43
Nepal	Diet-linked 40q30, ages 30 to 70	2100	5.02	4.75	0.27	5.17
Nigeria	All-cause 70q0	2025	60.72	60.72	0.00	0.00

Country	Metric	Year	Baseline (%)	Adjusted (%)	Absolute reduction (pp)	Relative reduction (%)
Nigeria	All-cause 70q0	2050	55.01	55.00	0.00	0.01
Nigeria	All-cause 70q0	2070	50.50	50.33	0.18	0.35
Nigeria	All-cause 70q0	2095	45.31	44.49	0.83	1.80
Nigeria	All-cause 70q0	2100	44.35	43.67	0.68	1.52
Nigeria	Diet-linked 40q30, ages 30 to 70	2025	18.31	18.31	0.00	0.00
Nigeria	Diet-linked 40q30, ages 30 to 70	2050	19.61	19.60	0.01	0.04
Nigeria	Diet-linked 40q30, ages 30 to 70	2070	19.50	19.22	0.29	1.47
Nigeria	Diet-linked 40q30, ages 30 to 70	2095	18.34	17.10	1.24	6.72
Nigeria	Diet-linked 40q30, ages 30 to 70	2100	18.00	16.99	1.01	5.56
Uzbekistan	All-cause 70q0	2025	33.46	33.46	0.00	0.00
Uzbekistan	All-cause 70q0	2050	25.93	25.92	0.01	0.02
Uzbekistan	All-cause 70q0	2070	19.97	19.76	0.21	1.01
Uzbekistan	All-cause 70q0	2095	13.68	12.99	0.69	4.86
Uzbekistan	All-cause 70q0	2100	12.61	12.09	0.52	3.98
Uzbekistan	Diet-linked 40q30, ages 30 to 70	2025	20.70	20.70	0.00	0.00
Uzbekistan	Diet-linked 40q30, ages 30 to 70	2050	16.03	16.02	0.01	0.04
Uzbekistan	Diet-linked 40q30, ages 30 to 70	2070	12.38	12.15	0.23	1.83
Uzbekistan	Diet-linked 40q30, ages 30 to 70	2095	8.46	7.73	0.73	8.49
Uzbekistan	Diet-linked 40q30, ages 30 to 70	2100	7.78	7.23	0.55	6.94

Table 7. Baseline mean adult exposure (ages 30–70), the shift required to reach TMREL, half of that shift, and the observed annualised menu contribution, by country and risk, all in the risk's own exposure unit.

Country	Dietary risk	Risk direction	TMREL	Baseline mean exposure, ages 30-70	Shift required to reach TMREL	Half of the shift to TMREL	Observed annualised delta	Observed as % of TMREL gap
India	Fruits	Protective	250.00	19.35	230.65	115.33	1.46	0.60
India	Vegetables	Protective	360.00	120.74	239.26	119.63	10.29	4.30
India	Legumes	Protective	100.00	36.05	63.95	31.97	2.80	4.40
India	Whole grains	Protective	125.00	44.18	80.82	40.41	0.00	0.00
India	Milk	Protective	435.00	33.03	401.97	200.99	1.33	0.30
India	Calcium	Protective	1.20	0.53	0.67	0.33	0.02	3.60
India	Fibre	Protective	25.00	18.03	6.97	3.48	1.54	22.20
India	Sodium	Harmful	3.00	3.96	-0.96	-0.48	0.02	-1.60
India	Red meat	Harmful	0.00	3.05	-3.05	-1.53	0.00	0.00
India	Processed meat	Harmful	0.00	1.24	-1.24	-0.62	0.00	0.00
India	Sugar-sweetened beverages	Harmful	0.00	25.42	-25.42	-12.71	0.00	0.00
Nepal	Fruits	Protective	250.00	92.88	157.12	78.56	0.00	0.00
Nepal	Vegetables	Protective	360.00	99.13	260.87	130.44	8.88	3.40
Nepal	Legumes	Protective	100.00	21.36	78.64	39.32	5.33	6.80
Nepal	Whole grains	Protective	125.00	62.49	62.51	31.26	8.88	14.20
Nepal	Milk	Protective	435.00	95.97	339.03	169.52	0.00	0.00
Nepal	Calcium	Protective	1.20	0.33	0.87	0.44	0.01	1.40
Nepal	Fibre	Protective	25.00	20.77	4.23	2.11	0.72	17.10

Country	Dietary risk	Risk direction	TMREL	Baseline mean exposure, ages 30-70	Shift required to reach TMREL	Half of the shift to TMREL	Observed annualised delta	Observed as % of TMREL gap
Nepal	Sodium	Harmful	3.00	3.49	-0.49	-0.24	0.21	-43.00
Nepal	Red meat	Harmful	0.00	18.12	-18.12	-9.06	0.00	0.00
Nepal	Processed meat	Harmful	0.00	1.89	-1.89	-0.95	0.00	0.00
Nepal	Sugar-sweetened beverages	Harmful	0.00	8.75	-8.75	-4.38	0.00	0.00
Cambodia	Fruits	Protective	250.00	32.86	217.14	108.57	0.09	0.00
Cambodia	Vegetables	Protective	360.00	33.22	326.78	163.39	17.32	5.30
Cambodia	Legumes	Protective	100.00	12.86	87.14	43.57	0.00	0.00
Cambodia	Whole grains	Protective	125.00	1.91	123.09	61.55	0.00	0.00
Cambodia	Milk	Protective	435.00	6.38	428.62	214.31	0.00	0.00
Cambodia	Calcium	Protective	1.20	0.27	0.93	0.47	0.02	2.30
Cambodia	Fibre	Protective	25.00	6.14	18.86	9.43	0.30	1.60
Cambodia	Sodium	Harmful	3.00	4.55	-1.55	-0.77	0.13	-8.40
Cambodia	Red meat	Harmful	0.00	13.49	-13.49	-6.74	1.78	-13.20
Cambodia	Processed meat	Harmful	0.00	1.00	-1.00	-0.50	0.00	0.00
Cambodia	Sugar-sweetened beverages	Harmful	0.00	18.69	-18.69	-9.35	0.00	0.00
Guatemala	Fruits	Protective	250.00	102.52	147.48	73.74	0.00	0.00
Guatemala	Vegetables	Protective	360.00	86.28	273.72	136.86	3.77	1.40
Guatemala	Legumes	Protective	100.00	25.28	74.72	37.36	6.32	8.50
Guatemala	Whole grains	Protective	125.00	120.34	7.28	3.64	8.84	121.50
Guatemala	Milk	Protective	435.00	37.20	397.80	198.90	3.45	0.90

Country	Dietary risk	Risk direction	TMREL	Baseline mean exposure, ages 30-70	Shift required to reach TMREL	Half of the shift to TMREL	Observed annualised delta	Observed as % of TMREL gap
Guatemala	Calcium	Protective	1.20	0.35	0.85	0.43	0.04	5.30
Guatemala	Fibre	Protective	25.00	25.86	0.55	0.27	1.25	229.60
Guatemala	Sodium	Harmful	3.00	4.06	-1.06	-0.53	0.03	-2.60
Guatemala	Red meat	Harmful	0.00	17.93	-17.93	-8.96	0.00	0.00
Guatemala	Processed meat	Harmful	0.00	2.60	-2.60	-1.30	0.00	0.00
Guatemala	Sugar-sweetened beverages	Harmful	0.00	52.43	-52.43	-26.21	0.00	0.00
Lesotho	Fruits	Protective	250.00	21.70	228.30	114.15	0.00	0.00
Lesotho	Vegetables	Protective	360.00	31.80	328.20	164.10	1.60	0.50
Lesotho	Legumes	Protective	100.00	6.98	93.02	46.51	7.19	7.70
Lesotho	Whole grains	Protective	125.00	163.39	0.00	0.00	0.00	—
Lesotho	Milk	Protective	435.00	188.48	246.52	123.26	0.00	0.00
Lesotho	Calcium	Protective	1.20	0.36	0.84	0.42	0.10	11.50
Lesotho	Fibre	Protective	25.00	25.99	0.46	0.23	2.42	526.00
Lesotho	Sodium	Harmful	3.00	2.91	-0.10	-0.05	0.07	-72.20
Lesotho	Red meat	Harmful	0.00	10.99	-10.99	-5.50	0.00	0.00
Lesotho	Processed meat	Harmful	0.00	1.26	-1.26	-0.63	0.00	0.00
Lesotho	Sugar-sweetened beverages	Harmful	0.00	47.41	-47.41	-23.71	0.00	0.00

Table 8. Full Part 2 benchmark values at all reporting years, by country and scenario (mapped protective menu; 50% and 100% of mean gap to TMREL).

Country	Scenario	Year	Baseline (%)	Adjusted (%)	Absolute reduction (pp)	Relative reduction (%)
India	50% of mean gap to TMREL	2050	12.17	12.17	0.00	0.04
India	50% of mean gap to TMREL	2070	9.67	9.50	0.17	1.70
India	50% of mean gap to TMREL	2095	6.62	6.14	0.49	7.17
India	50% of mean gap to TMREL	2100	6.09	5.73	0.36	5.78
India	100% of mean gap to TMREL	2050	12.17	12.17	0.01	0.06
India	100% of mean gap to TMREL	2070	9.67	9.39	0.27	2.71
India	100% of mean gap to TMREL	2095	6.62	5.85	0.78	11.42
India	100% of mean gap to TMREL	2100	6.09	5.51	0.57	9.20
India	100% of mean gap to TMREL, whole adult population	2050	12.17	10.37	1.80	14.46
India	100% of mean gap to TMREL, whole adult population	2070	9.67	8.25	1.42	14.30
India	100% of mean gap to TMREL, whole adult population	2095	6.62	5.65	0.97	14.32
India	100% of mean gap to TMREL, whole adult population	2100	6.09	5.19	0.90	14.35
Nepal	50% of mean gap to TMREL	2050	10.76	10.76	0.00	0.04
Nepal	50% of mean gap to TMREL	2070	8.26	8.14	0.12	1.36
Nepal	50% of mean gap to TMREL	2095	5.49	5.19	0.30	5.24
Nepal	50% of mean gap to TMREL	2100	5.02	4.80	0.22	4.21
Nepal	100% of mean gap to TMREL	2050	10.76	10.76	0.01	0.07
Nepal	100% of mean gap to TMREL	2070	8.26	8.06	0.19	2.19
Nepal	100% of mean gap to TMREL	2095	5.49	5.00	0.49	8.49
Nepal	100% of mean gap to TMREL	2100	5.02	4.66	0.36	6.82
Nepal	100% of mean gap to TMREL, whole adult population	2050	10.76	9.52	1.24	11.15

Country	Scenario	Year	Baseline (%)	Adjusted (%)	Absolute reduction (pp)	Relative reduction (%)
Nepal	100% of mean gap to TMREL, whole adult population	2070	8.26	7.30	0.95	11.03
Nepal	100% of mean gap to TMREL, whole adult population	2095	5.49	4.85	0.64	11.04
Nepal	100% of mean gap to TMREL, whole adult population	2100	5.02	4.44	0.58	11.06
Cambodia	50% of mean gap to TMREL	2050	13.20	13.19	0.01	0.10
Cambodia	50% of mean gap to TMREL	2070	11.62	11.23	0.39	3.17
Cambodia	50% of mean gap to TMREL	2095	8.95	7.86	1.09	12.00
Cambodia	50% of mean gap to TMREL	2100	8.39	7.57	0.83	9.70
Cambodia	100% of mean gap to TMREL	2050	13.20	13.18	0.02	0.16
Cambodia	100% of mean gap to TMREL	2070	11.62	11.01	0.61	4.93
Cambodia	100% of mean gap to TMREL	2095	8.95	7.26	1.70	18.58
Cambodia	100% of mean gap to TMREL	2100	8.39	7.11	1.28	15.00
Cambodia	100% of mean gap to TMREL, whole adult population	2050	13.20	10.10	3.10	22.89
Cambodia	100% of mean gap to TMREL, whole adult population	2070	11.62	8.78	2.84	23.78
Cambodia	100% of mean gap to TMREL, whole adult population	2095	8.95	6.70	2.25	24.42
Cambodia	100% of mean gap to TMREL, whole adult population	2100	8.39	6.28	2.11	24.49
Guatemala	50% of mean gap to TMREL	2050	13.27	13.27	0.00	0.03
Guatemala	50% of mean gap to TMREL	2070	11.75	11.63	0.12	0.97
Guatemala	50% of mean gap to TMREL	2095	8.88	8.57	0.32	3.44
Guatemala	50% of mean gap to TMREL	2100	8.27	8.03	0.24	2.80
Guatemala	100% of mean gap to TMREL	2050	13.27	13.26	0.01	0.05
Guatemala	100% of mean gap to TMREL	2070	11.75	11.55	0.20	1.66

Country	Scenario	Year	Baseline (%)	Adjusted (%)	Absolute reduction (pp)	Relative reduction (%)
Guatemala	100% of mean gap to TMREL	2095	8.88	8.35	0.54	5.85
Guatemala	100% of mean gap to TMREL	2100	8.27	7.86	0.41	4.76
Guatemala	100% of mean gap to TMREL, whole adult population	2050	13.27	12.20	1.07	7.85
Guatemala	100% of mean gap to TMREL, whole adult population	2070	11.75	10.82	0.93	7.65
Guatemala	100% of mean gap to TMREL, whole adult population	2095	8.88	8.18	0.70	7.63
Guatemala	100% of mean gap to TMREL, whole adult population	2100	8.27	7.62	0.65	7.65
Lesotho	50% of mean gap to TMREL	2050	14.59	14.59	0.00	0.01
Lesotho	50% of mean gap to TMREL	2070	13.07	12.98	0.10	0.72
Lesotho	50% of mean gap to TMREL	2095	10.27	9.81	0.45	4.27
Lesotho	50% of mean gap to TMREL	2100	9.62	9.26	0.36	3.59
Lesotho	100% of mean gap to TMREL	2050	14.59	14.59	0.00	0.02
Lesotho	100% of mean gap to TMREL	2070	13.07	12.92	0.15	1.16
Lesotho	100% of mean gap to TMREL	2095	10.27	9.54	0.73	6.83
Lesotho	100% of mean gap to TMREL	2100	9.62	9.05	0.57	5.73
Lesotho	100% of mean gap to TMREL, whole adult population	2050	14.59	13.42	1.18	7.94
Lesotho	100% of mean gap to TMREL, whole adult population	2070	13.07	12.01	1.07	7.97
Lesotho	100% of mean gap to TMREL, whole adult population	2095	10.27	9.41	0.85	8.06
Lesotho	100% of mean gap to TMREL, whole adult population	2100	9.62	8.82	0.80	8.09

Figures

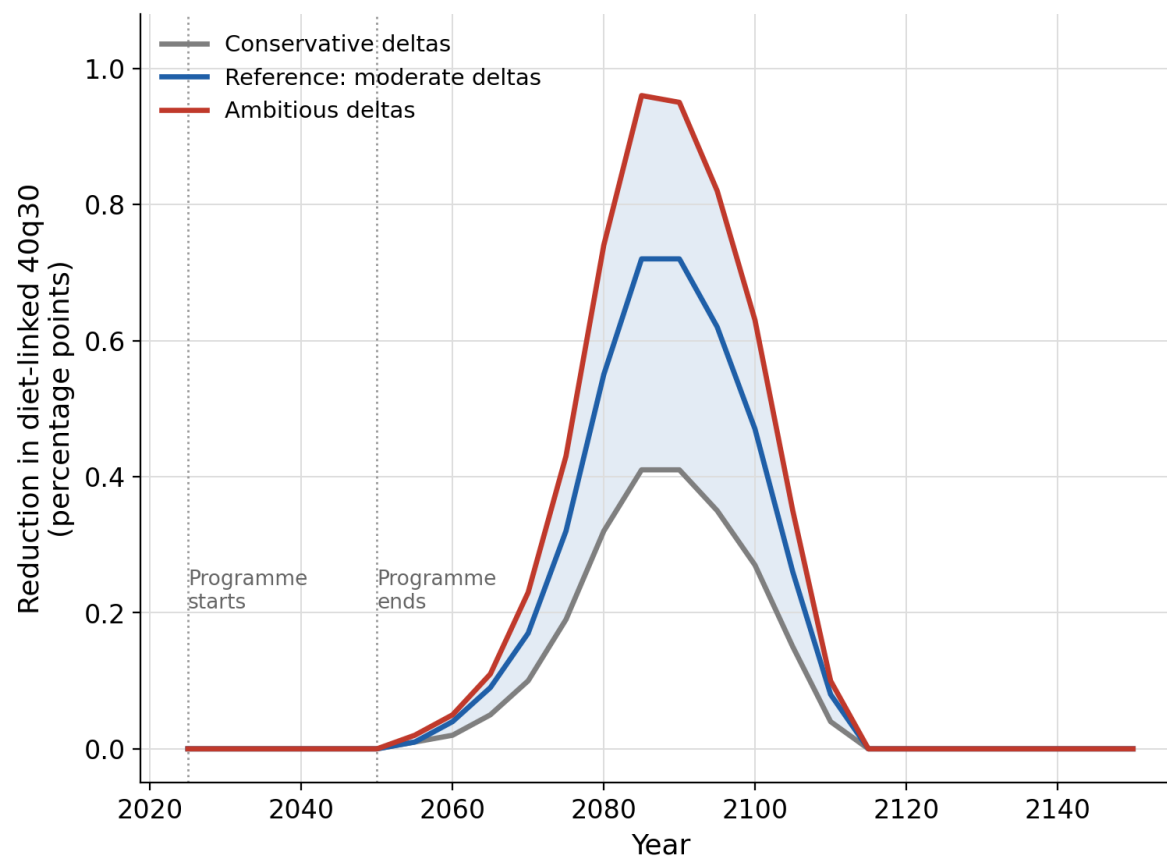


Figure 1. Timing of the modelled benefit, illustrated for Kenya under the Part 1 grid. Lines show the conservative, reference (moderate) and ambitious dietary-delta scenarios at full persistence; shading spans the conservative-to-ambitious range.

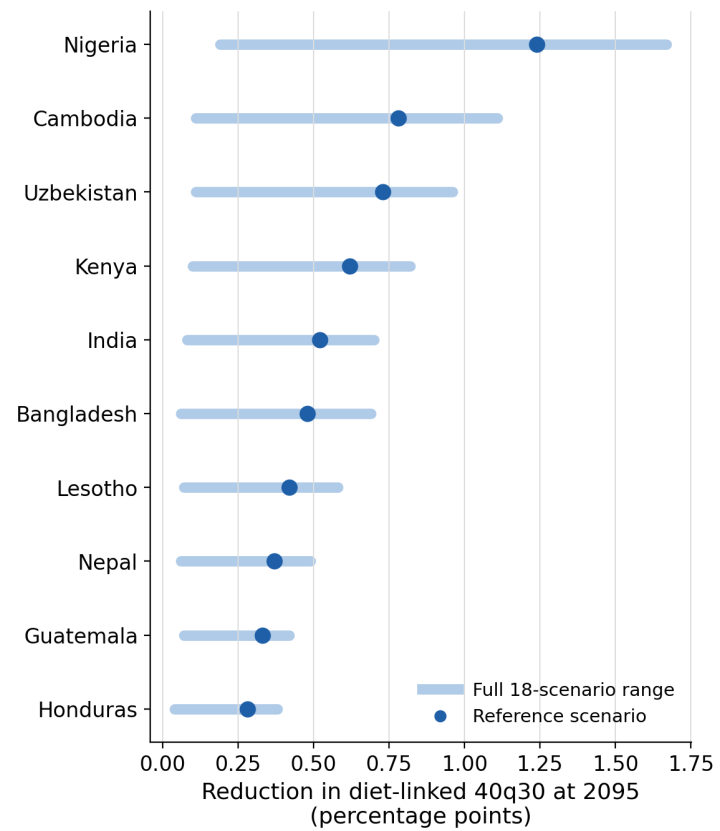


Figure 2. Country variation in the modelled Part 1 benefit in 2095. Dots mark the reference scenario; bars span the full 18-scenario range.

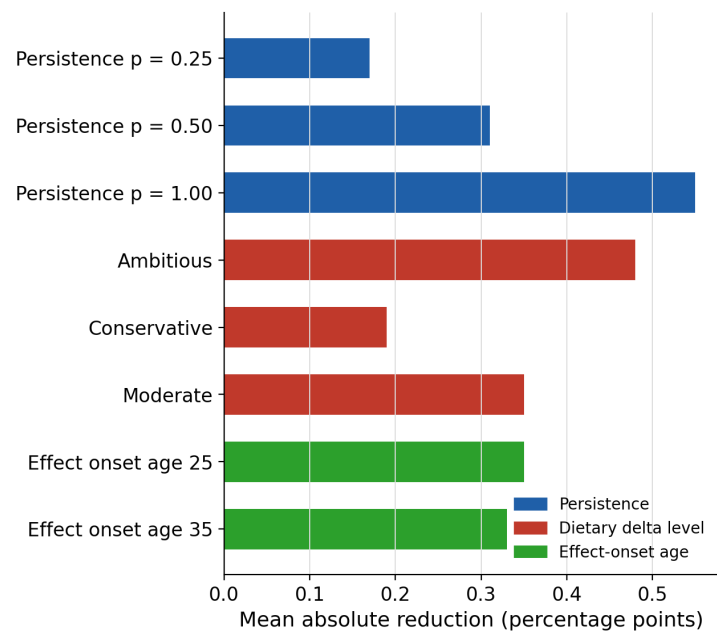


Figure 3. The assumptions that drove cross-country variation: mean absolute reduction by persistence level, dietary delta level and effect-onset age, averaged across the 18-scenario grid.

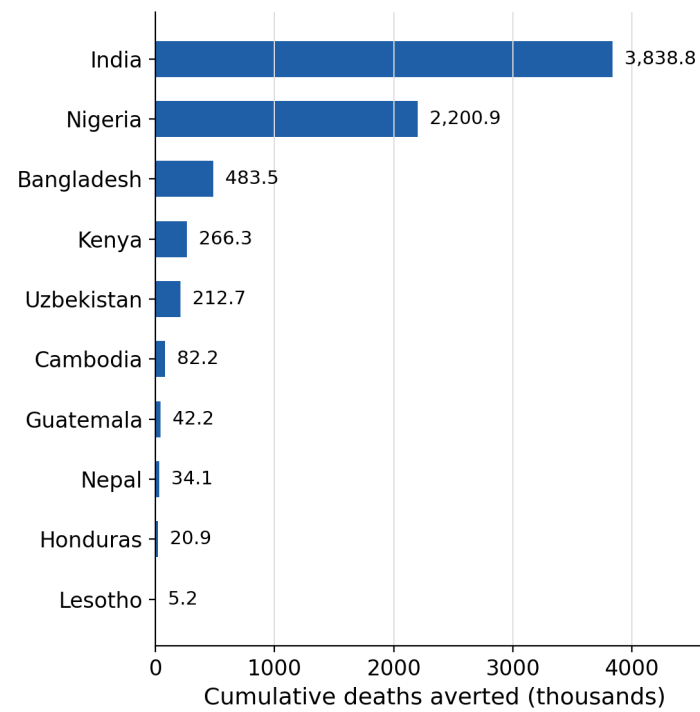


Figure 4. Burden scale (deaths) under the Part 1 reference scenario.

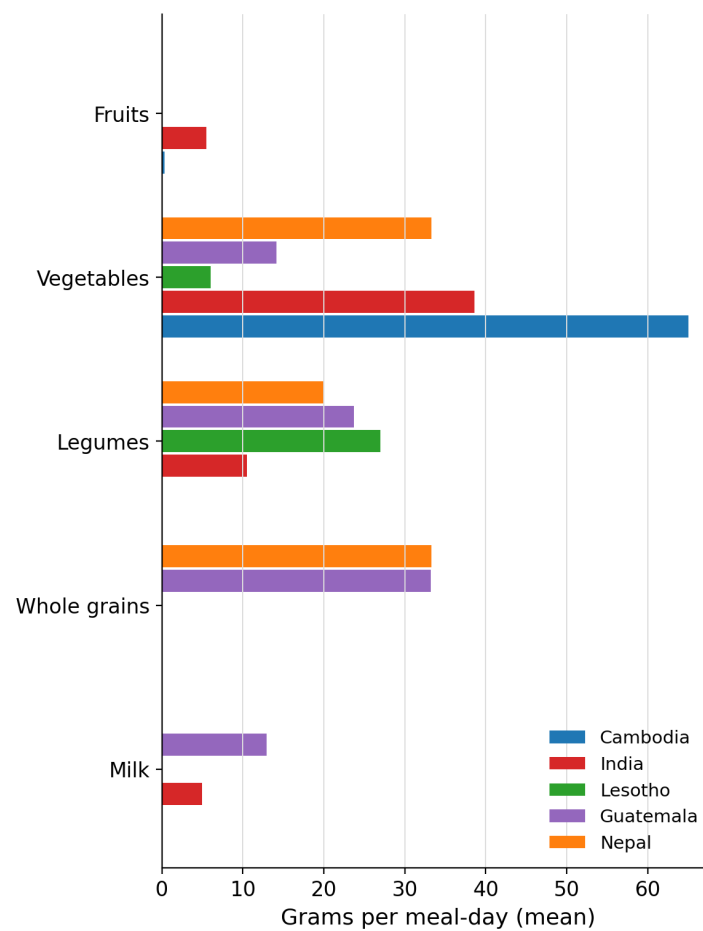


Figure 5. Mapped protective-food content of the observed school menus, on a mean grams-per-meal-day basis, by country.

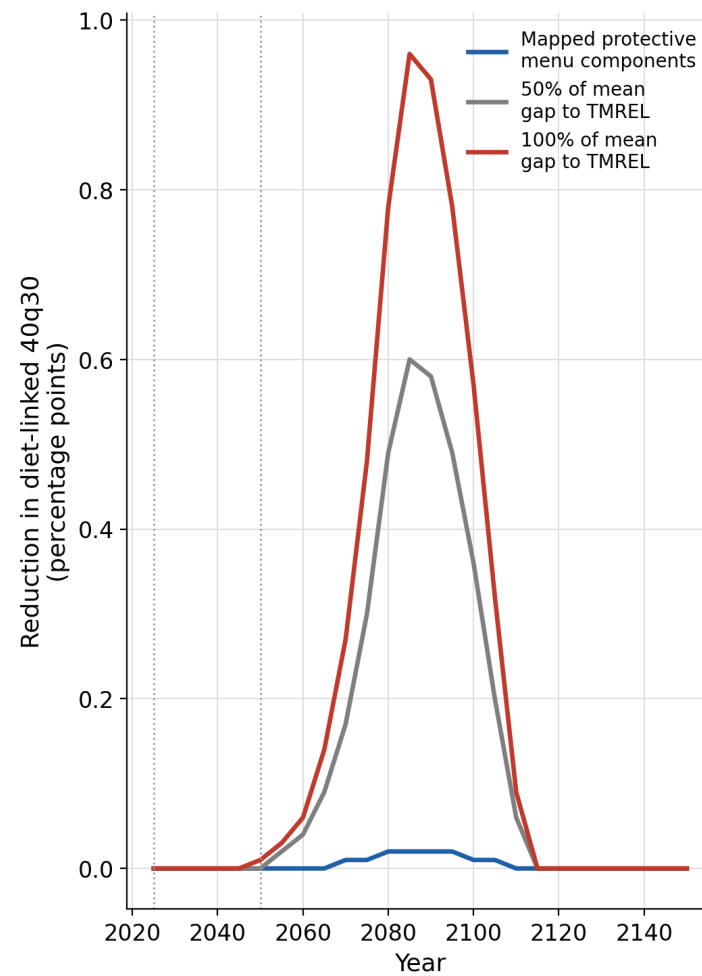


Figure 6. Timing of the modelled Part 2 benefit for India, which remained delayed in the same way as Part 1; the main new information was the size of the gap between the mapped menu contribution and the TMREL benchmarks.

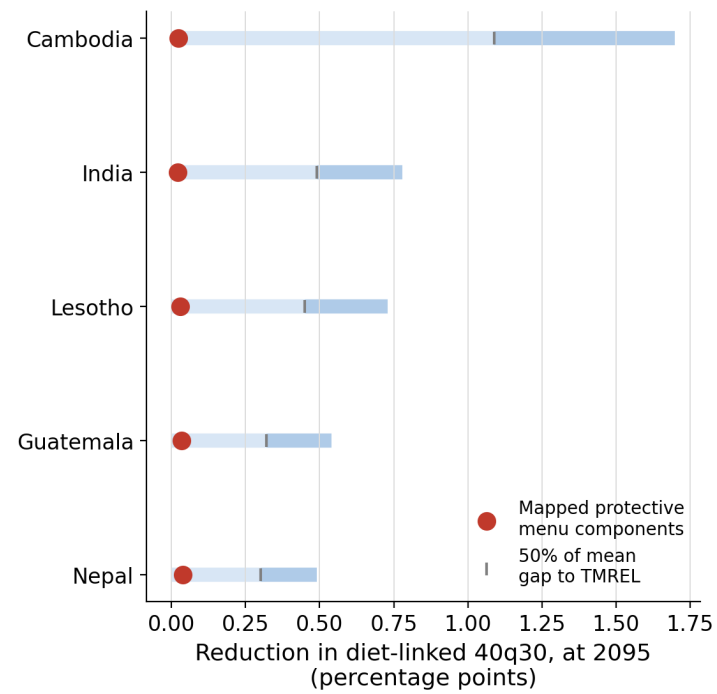


Figure 7. Mapped menu contribution against the 50%- and 100%-to-TMREL benchmarks, by country, in 2095.

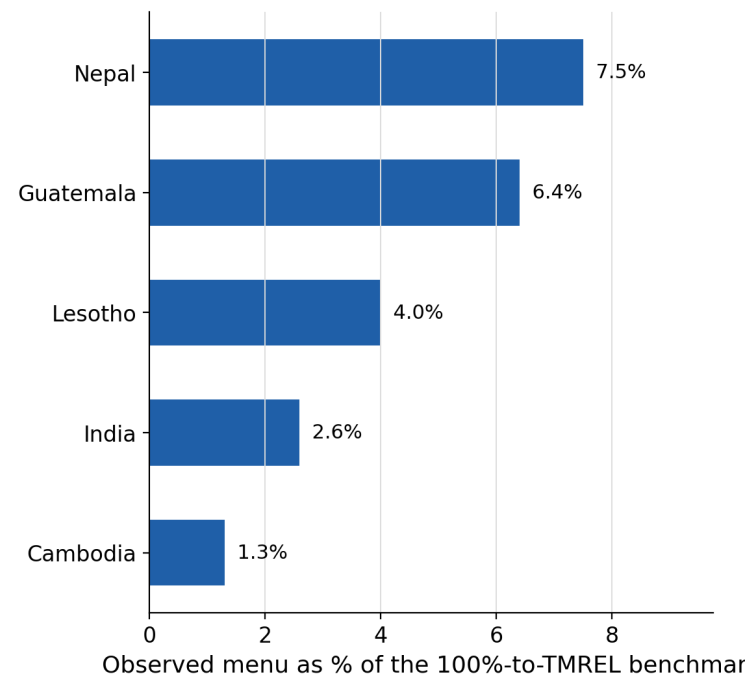


Figure 8. The same comparison viewed as the observed menu's share of the 100%-to-TMREL benchmark, across the full projection horizon.

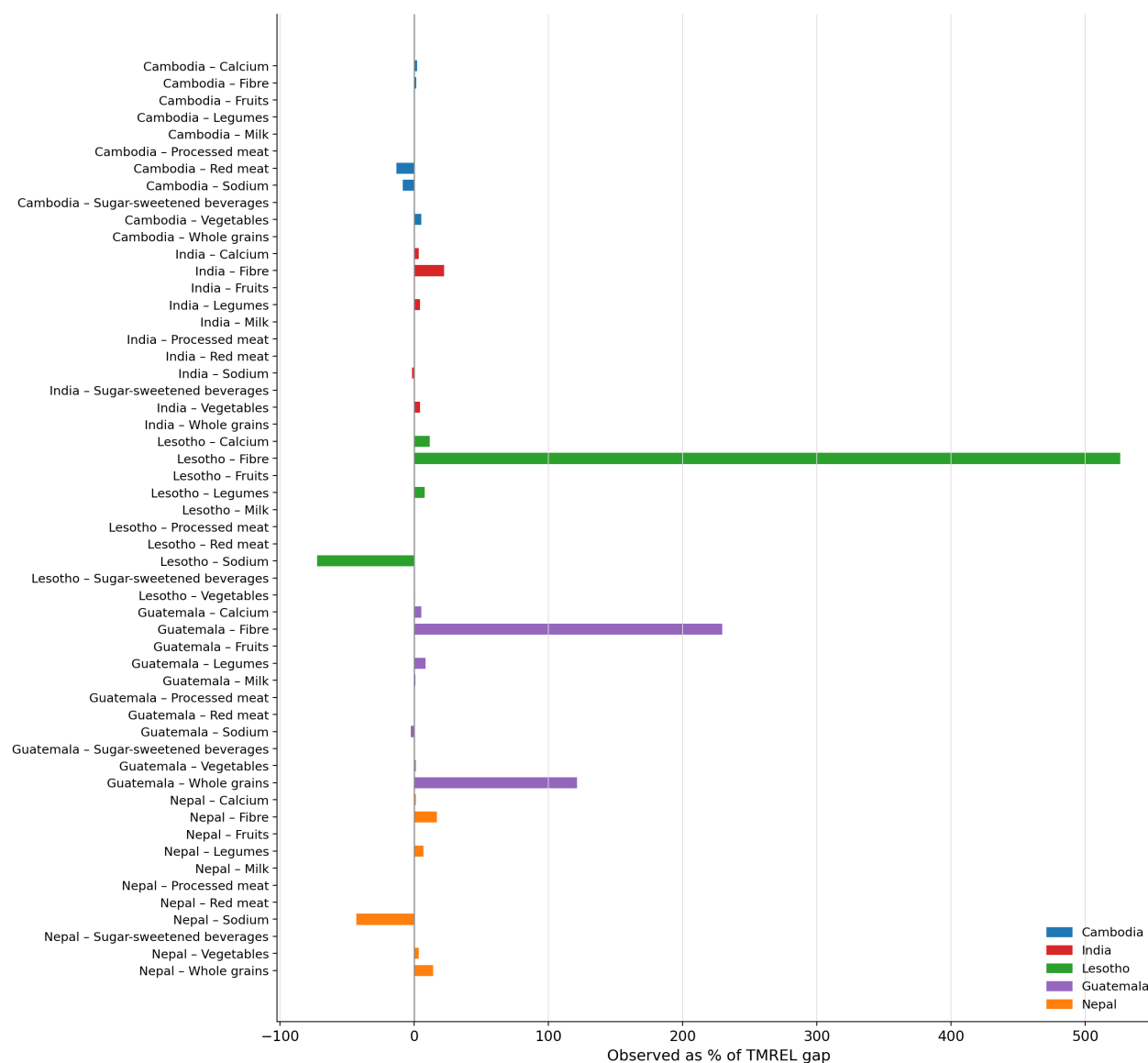


Figure 9. Mapped menu contribution against each risk's own distance to TMREL, by country and dietary risk. Absolute gram-per-day baselines, gaps and menu contributions are given in Table 7.

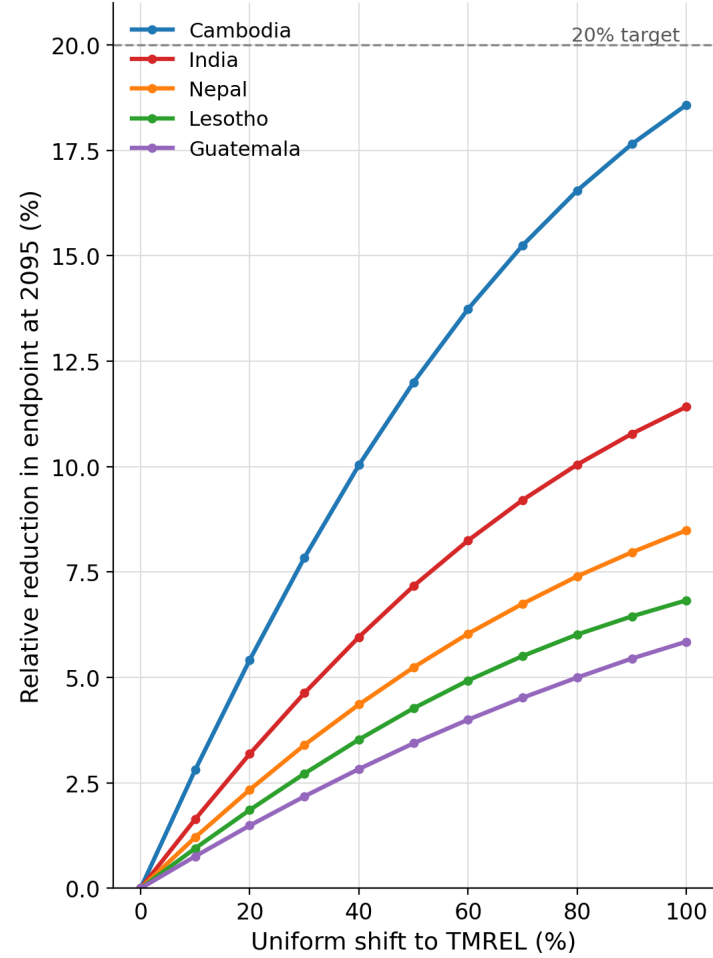


Figure 10. Target-seeking analysis: relative reduction in the diet-linked mortality endpoint in 2095 as a common gap-to-TMREL fraction.

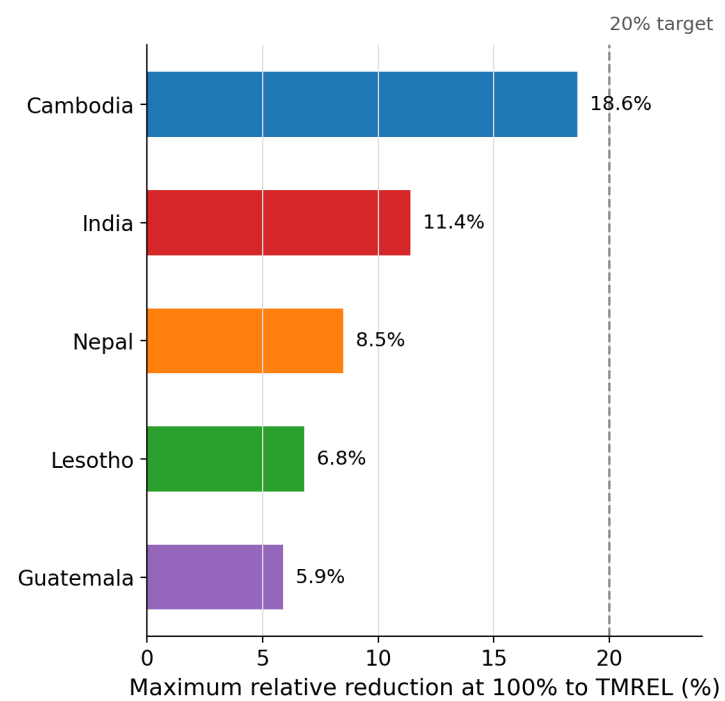


Figure 11. How far short of the 20% relative-reduction target each country remained, even at 100% of the mean gap to TMREL.

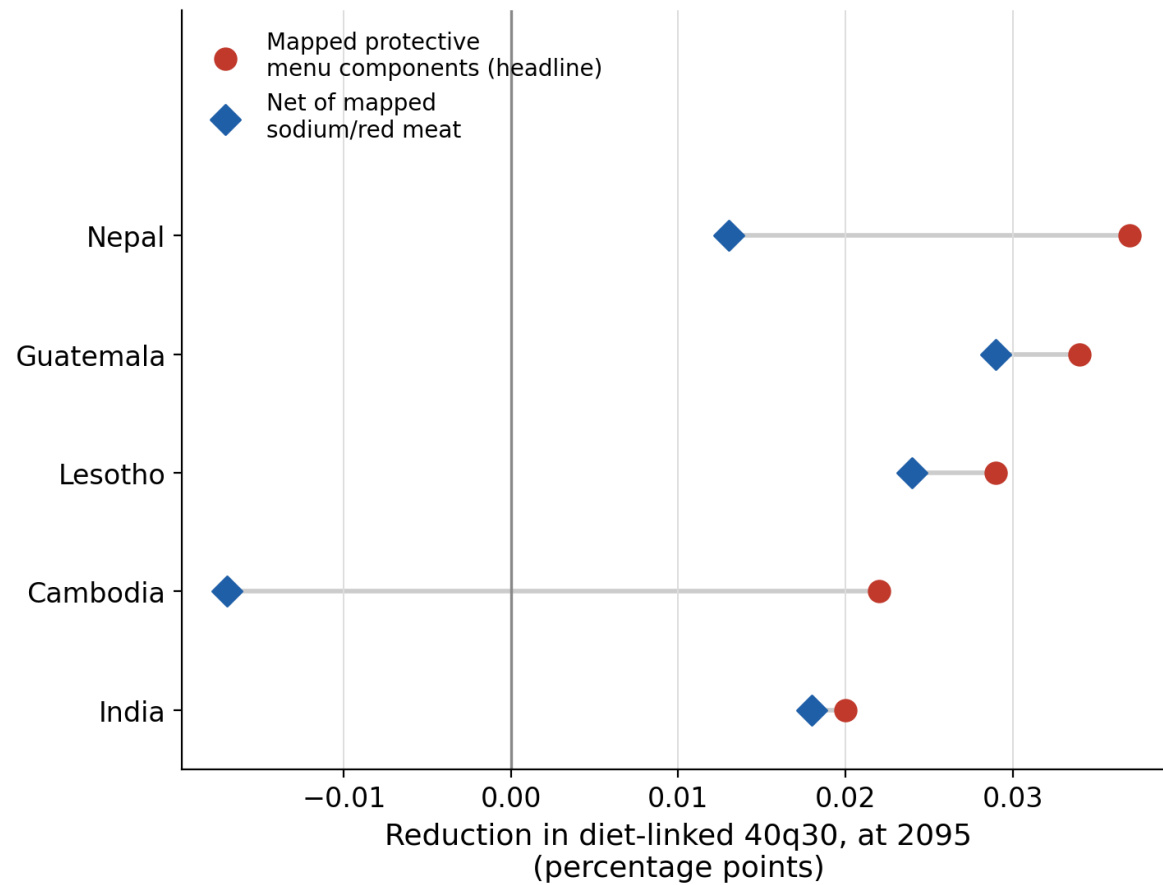


Figure 12. Headline protective-component result against the net-of-mapped-harms sensitivity, by country, in 2095. The sensitivity added menu-contributed sodium and any mapped red meat; Cambodia's net effect turned negative.

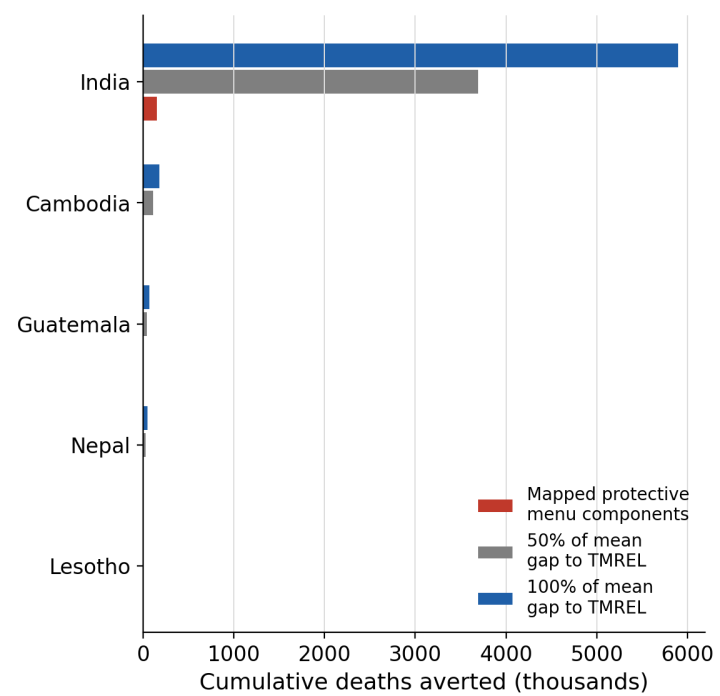


Figure 13. Cumulative diet-linked deaths averted, ages 30–69, across completed annual intervals 2025–2149, by scenario.

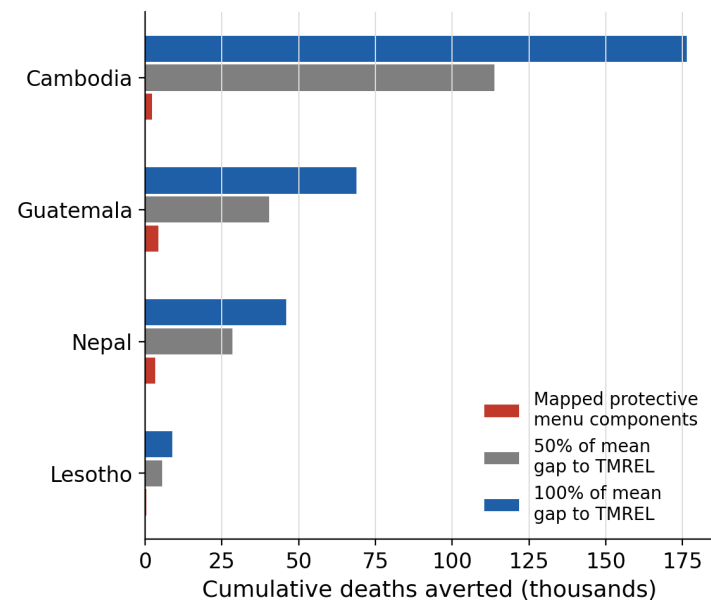


Figure 14. The same comparison with India removed, rescaling the axis so the remaining four countries were visible.

7. References

7.1 Model inputs and methods

Institute for Health Metrics and Evaluation. Global Burden of Disease Study 2023. Seattle, WA. Cause-specific mortality and adult dietary exposure distributions were projection inputs; published dietary attributable fractions were retained for an internal concordance diagnostic.

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FACT-matched school-menu data prepared for this project by A. Kante. Publication note: insert the complete food-composition-table source, matching protocol and data-preparation citation before external publication.s

7.2 School-meal and modelling literature

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