Does a Healthy Diet a Day Keep the Economic Burden Away?

A Multi-Pathway Modelling Assessment of the Economic Benefits of Healthy Diets

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Contents

Tables

No table of figures entries found.

Figures

No table of figures entries found.

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Abstract

Diet-related health burdens entail major adverse economic implications, notably through hidden losses in labour productivity. Advances in nutrition science and epidemiology have strengthened evidence on the multiple pathways through which diets affect health over the life course. These pathways extend beyond diet-related diseases to include micronutrient deficiencies and immune impairment, mental-health deterioration, metabolic dysfunction and noncommunicable disease risk, as well as developmental and intergenerational effects. Yet most economic assessments capture only a subset of these pathways or their dynamic interactions, despite the fact that some generate long-lasting human capital losses while others primarily affect individuals during periods of unhealthy dietary intake.

To address this gap, this paper provides an integrated assessment of the potential economic benefits of healthy diets. The analysis combines a recursive-dynamic global computable general equilibrium (CGE) model (MIRAGRODEP) with a household microsimulation module (POVANA) and health pathway models grounded in nutrition and epidemiology. By differentiating labour by sex and birth cohort, the framework captures key diet-related health pathways and their life-course effects. These pathways influence economic outcomes through short- and long-term labour productivity, labour-force participation, health expenditures, and human capital formation. This integrated approach enables the estimation of direct and indirect economic gains from dietary improvements, including higher labour productivity and labour supply alongside lower health-related costs. Beyond aggregate GDP effects, the analysis examines public-sector implications such as reduced healthcare spending, lower opportunity costs of public expenditure, and moderated public-debt trajectories, as well as distributional impacts across income groups and sex.

Keywords: unhealthy diets; economic benefits; diet–health pathways; computable general equilibrium; labour productivity; distributional impacts

1 Introduction

A growing economic literature has estimated the costs associated with diet-related health burdens (Bertram et al. 2018; Bommer et al. 2017; Candari et al. 2017; Custodio et al. 2025; Galasso et Wagstaff, s. d.; Susan Horton et al. 2024; Kuiper et al. 2025; Nugent et al. 2020a; Okunogbe et al. 2022; Shekar et al. 2024; Shekar et Popkin 2020; Siekmans et al. 2024; Springmann et al. 2021; Victora et al. 2008; World Bank et FAO 2025), with particular attention to the hidden costs arising from reduced labour productivity (Brooks et Diaz-Bonilla 2025; FAO 2023, 2024; S. Lord 2024; Steven Lord 2024; S. Lord 2023). In parallel, advances in nutrition science and epidemiology have strengthened the evidence on the multiple pathways through which diets influence health outcomes over the life course (Khaliq et al. 2022; Shekar et Popkin 2020; Victora et al. 2008). These pathways extend beyond diet-related diseases and include micronutrient deficiencies and immune impairment, mental-health deterioration, metabolic dysfunction and noncommunicable disease risk, and developmental and intergenerational effects (Monteiro et al. 2025; Swinburn et al. 2019; Wells et al. 2020; Jan et al. 2018; Georgiadis et Penny 2017; Arlinghaus et al. 2018). However, most existing economic assessments capture only a subset of these pathways or only part of their dynamic and interacting effects. These dynamic dimensions are essential: some pathways generate long-lasting consequences for human capital, while others primarily affect individuals during periods of unhealthy dietary intake.

This paper provides an integrated and comprehensive assessment of the potential economic benefits associated with the consumption of healthy diets. The analysis combines the global computable general equilibrium (CGE) model MIRAGRODEP (Bouët et al. 2022), a detailed household module (POVANA) (Laborde et al. 2021), and pathway-specific modelling approaches grounded in the nutrition and epidemiology literature. The modeling uses the recursive-dynamic version of MIRAGRODEP and explicitly differentiates labour endowments by sex and birth cohort, allowing diet-related health effects to evolve over time and across generations. Through this structure, the four pathways influence economic outcomes via distinct channels, including temporary and permanent labour productivity losses, changes in labour-force participation, increased health expenditures, and long-term effects on skilled human capital formation. This integrated approach enables the estimation of both direct and indirect economic gains, including changes in labour productivity, labour supply, and health-related expenditures.

The study also examines a "no-action" dynamic baseline, calibrated on existing demographic, epidemiological, and income trends and reflecting the projected evolution of the economic costs associated with unhealthy diets in low and middle-income countries, in line with recent patterns observed in upper-middle and high-income countries. This business-as-usual trajectory represents an augmented baseline relative to the standard CGE practice — driven by population growth and productivity — and therefore provides a robust counterfactual against which the potential economic gains from healthier diets can be assessed.

Beyond aggregate GDP effects, the analysis explores public-sector implications, including reductions in healthcare spending, lower opportunity costs of public expenditure, and potential effects on public-debt trajectories. It also examines distributional impacts, recognising that diet-related health burdens and the associated economic gains from healthier diets vary across income groups and sex, generating differentiated health and economic outcomes.

This paper therefore provides a structured and integrated assessment of the economic benefits of healthy diets, offering a comprehensive analytical basis for understanding how dietary improvements may influence economic outcomes across multiple pathways and population groups. It also advances the modelling of labour-force dynamics within the CGE framework, allowing diet-related health effects to be reflected more accurately in economic projections.

2 Literature review

Unhealthy diets have become a leading cause of disease worldwide, with profound economic implications. A large medical and nutritional literature has established that poor diets contribute substantially to morbidity and mortality from non-communicable diseases (NCDs) such as cardiovascular disease, diabetes, and certain cancers, while undernutrition persists in many low- and middle-income countries (Afshin et al. 2019; Black et al. 2013a). Translating these health impacts into economic terms reveals that societies incur substantial hidden costs arising from inefficiencies in agrifood systems - costs that are not reflected in market transactions but ultimately constrain economic growth.

Recent assessments by the Food and Agriculture Organization of the United Nations (FAO) estimate that the global hidden costs of agrifood systems - including environmental, social, and health-related costs - amount to approximately USD 10 trillion annually (FAO 2023, 2024; Steven Lord 2024; S. Lord 2023). Poor dietary patterns are estimated to account for the majority of these hidden costs, particularly through productivity losses associated with diet-related morbidity and premature mortality (S. Lord 2023).

This review examines the hidden economic costs of unhealthy diets and the potential economy-wide gains from healthier dietary patterns, with a focus on non-market impacts typically omitted or only partially captured in macroeconomic frameworks. Four pathways are considered, reflecting both the strength of the evidence and the scale of the associated economic stakes: (1) short-term micronutrient deficiencies and immune impairment; (2) diet-related mental health deterioration; (3) chronic diet-related disease; and (4) developmental and intergenerational impacts of early-life undernutrition. For each pathway, the analysis distinguishes between uncertainty in attributing diet-related health effects and uncertainty in translating those effects into economic outcomes relevant for economy-wide and CGE modelling.

2.1 Analytical Framework

Estimating the hidden economic costs of unhealthy diets requires linking epidemiological evidence with economic valuation and modelling.

First, the epidemiological foundation quantifies diet-related health impacts. Global comparative risk assessments (e.g. the Global Burden of Disease studies) estimate how dietary factors contribute to mortality and disability, providing a standardized measure of the health burden of unhealthy diets (Afshin et al. 2019; IHME 2026). Such data, alongside cohort studies and clinical trials, form the foundation by identifying how many cases of illness or premature deaths are diet-attributable. While the evidence here is generally strong for many diet–disease links (particularly for metabolic NCDs and micronutrient deficiencies), there remain uncertainties in attribution (Mathers 2020) – e.g., precisely isolating diet's effect from other lifestyle factors - or gauging newer suspected links such as diet's role in mental illness (Lassale et al. 2019).

Second, economists use cost accounting and valuation approaches to translate those health outcomes into monetary terms.

A common approach is the cost-of-illness (COI) analysis, which tallies the total economic burden of diet-related diseases on society (Bommer et al. 2017; Okunogbe et al. 2022). COI includes direct costs (medical expenditures on treatment of diet-induced conditions) and indirect costs (the value of lost productivity, income, and human capital from morbidity and early mortality). COI studies consistently find these indirect costs to be a dominant component of diet-related economic losses (Shekar et Popkin 2020; Okunogbe et al. 2022)). To quantify such losses, COI analyses draw on diverse data sources: labor-force surveys and firm data to measure absenteeism and productivity deficits; longitudinal studies linking early-life nutrition to adult earnings (Hoddinott et al. 2013); and demographic statistics to value working years lost to diet-related deaths. There is some valuation uncertainty in this step – for instance, whether to monetize a lost life year based on foregone earnings (the human capital approach) or using a higher willingness-to-pay metric for mortality risk (the value of a statistical life). Different valuation methods can yield different dollar estimates. Moreover, care must be taken to avoid double counting when aggregating costs of multiple diet-related diseases that overlap (Nugent et al. 2020a); advanced studies use statistical attribution or limit valuations to the net combined burden of all diet-related conditions.

A variant of this valuation approach is the "true cost" accounting frameworks designed to capture the full societal cost of food systems, of which diet-related health losses are a major part. The FAO's State of Food and Agriculture 2023 report uses this approach to estimate hidden health costs by valuing all diet-attributable healthy life years lost in monetary terms (FAO 2023, 2024; Steven Lord 2024; S. Lord 2023). all diet-attributable healthy life years lost are monetized by multiplying the total DALYs due to unhealthy diets by a shadow price (approximated by GDP per capita in purchasing-power terms), to express the aggregate welfare loss as a share of economic output.

A complementary approach is economy-wide modelling, which embeds diet-health effects into multisector or macroeconomic models (often Computable General Equilibrium models) to simulate broader impacts (OECD 2019; Springmann et al. 2016; Galasso et Wagstaff 2019). Rather than summing costs, these models integrate diet–health effects into a multi-sector economic framework. They typically use the same epidemiological inputs (e.g. population risk exposure, disease prevalence) and similar economic parameters (e.g. percent reduction in labor productivity per case of illness) as the above valuations, but apply them as shocks to a dynamic economic model. For example, a CGE model can simulate a scenario where a certain fraction of the workforce is removed (or has reduced productivity) due to diet-related NCDs. The model then endogenously calculates impacts on GDP, consumption, investment, and even trade, while maintaining internal consistency (resources reallocate across sectors and time). The strength of this approach is capturing feedback effects and interdependencies that a static COI cannot – for instance, how lower labor supply might raise wages. These models, however, require many assumptions (on behavioral responses, fiscal adjustments, etc.), so parameter uncertainty is a concern here as well. Importantly, CGE models provide insight into timing (short- vs long-run effects), distribution (impacts on different sectors or social groups), and fiscal dynamics. They also help avoid double counting by ensuring each effect (labor, capital, etc.) is modeled once in a unified framework, rather than independently summing costs.

In the following sections, we review each of the four health–economy pathways in turn, discussing (a) the health impacts of diets (including strength of evidence and measurement challenges), (b) the economic costs of those health impacts and how they are calculated, and (c) the extent to which each pathway is represented in current macroeconomic models and policy analyses. **Error! Reference source not found.** provides a summary of the pathways, their health effects, associated economic impact mechanisms, and key uncertainties.

Table 1 - Major diet-health-economy pathways: health outcomes, economic impact channels, and key uncertainties in evidence and modelling

Pathway (diet-related health impact)	Key health outcomes (evidence base & gaps)	Economic impact mechanisms (integration in models & uncertainties)
1. Short-term micronutrient deficiencies & immune impairment	• **Outcomes**: Anemia; weakened immune function; higher infection risk; impaired physical work capacity; maternal and child mortality from severe deficiencies • **Evidence**: Well-established causal links between specific micronutrient deficiencies and health outcomes (Black et al. 2013b). Global prevalence remains high (over 2 billion people with one or more micronutrient deficits). • **Evidence Gap**: Hard to isolate the productivity loss from each nutrient deficiency versus general undernutrition; co-occurring infections and caloric/protein shortage make measurement difficult. Risk of double counting.	• **Impact on labour**: Micronutrient deficiencies reduce worker productivity and effective labour time. For example, iron-deficient adults suffer fatigue and can have 5–17% lower output in manual labor, while immune impairment from poor nutrition leads to frequent illnesses (malaria, diarrhoea, etc.) that increase absenteeism. (S. Horton et Ross 2003). • **Integration in models**: Partially captured in some models via reduced labour efficiency (anemia proxy) or using cohort models. Many CGE models still omit immunity effects • **Challenges**: Quantifying mild vs. severe deficiency impacts on cognitive development and work capacity; risk of overlapping with Pathway 4 (childhood undernutrition) effects if not carefully separated.
2. Diet-related mental health and cognition deterioration	• **Outcomes**: Depression and anxiety disorders; possibly faster cognitive decline and higher dementia risk linked to poor diet quality • **Evidence**: Emerging but mixed. Observational studies find healthier diets are associated with lower depression incidence (Lassale et al. 2019) and slower cognitive decline (Lourida et al. 2013). However, causality is not confirmed – mental illness can also lead to poor diet. RCT eivndence is limited. • **Evidence Gap**: Unclear biological mechanisms (chronic inflammation and altered gut microbiota from unhealthy diets are proposed links to depression, and vascular effects of diet might influence dementia risk); effect sizes vary and diet's contribution to disorders like dementia is still being studied.	• **Impact on labour**: Impaired mental health can substantially reduce productivity and labour participation. Mental disorders (all causes) are a major source of lost productivity globally (~$1 trillion annual productivity lost)(Chisholm et al. 2016). Serious mental illness and cognitive decline can also reduce labour force participation (disability, early retirement). • **Integration in models**: Largely absent. • **Challenges**: Very high – both in the diet–mental health link and in how to quantify the economic effects. Risk of double counting: e.g., some productivity loss attributed to obesity (Pathway 3) might actually be due to comorbid depression, or vice versa.

Pathway (diet-related health impact)	Key health outcomes (evidence base & gaps)	Economic impact mechanisms (integration in models & uncertainties)
3. Chronic diet-related diseases	• **Outcomes**: Overweight and obesity; diet-related non-communicable diseases (NCDs) including type 2 diabetes, coronary heart disease, stroke, hypertension, and certain cancers. Also musculoskeletal issues due to obesity. • **Evidence**: Very strong for many links. Unhealthy diet is a leading global risk factor for NCD morbidity and mortality (Afshin et al. 2019). Excess body weight is clearly associated with higher mortality and morbidity (Okunogbe et al. 2022). Causality is well-established for most relationships, though precise contribution of diet vs. physical inactivity and genetics in obesity can vary • **Evidence Gap**: Measuring the combined impact of multiple coexisting conditions without double-counting is challenging. GBD methods partition these, but uncertainty remains in attributing portions of multi-causal diseases to diet alone. Current evidence is stronger for mortality than for morbidity and productivity impacts.	• **Impact on labour**: Diet-related NCDs in the working-age population reduce on-the-job productivity (fatigue, complications) and increase sick leave; they also cause disability and premature death, shrinking the labour force. Global studies indicate these conditions currently decrease total labour output by several percent (Nugent et al. 2020b; Okunogbe et al. 2022). • **Integration in models**: Commonly included in macro models via reduced labour productivity or workforce size (OECD 2019). • **Challenges**: Key uncertainties in modeling are how to value presenteeism (working while sick) and capturing behavioral feedbacks (a healthier population might save/invest differently). Potential overlap with other pathways (e.g. some NCD costs stem from childhood undernutrition or from mental health comorbidities). Models often assume uniform productivity loss per sick worker, but in reality impacts vary by job type and disease severity. Morbidity (non-fatal illness) is harder to measure and value than mortality – e.g., how to value "presenteeism" (working while ill) is debated.
4. Developmental & intergenerational impacts	• **Outcomes**: Fetal growth restriction (low birthweight); childhood stunting (chronic undernutrition) and wasting (acute starvation); impaired cognitive and physical development; elevated child mortality; and intergenerational effects (malnourished mothers more likely to have undernourished, low-birthweight babies, perpetuating the cycle). • **Evidence**: Very strong. Decades of studies show early-life undernutrition causes lasting deficits in terms of irreversible height and IQ losses, and increase in mortality (Victora et al. 2008; Hoddinott et al. 2013). Stunted children complete fewer years of schooling and earn less as adults (Galasso et Wagstaff 2019). Intergenerational links are also documented: malnourished mothers more often give birth to malnourished infants, perpetuating poverty. • **Evidence Gap**: Hard to quantify milder forms of undernutrition (e.g. moderate stunting or sporadic hunger) on adult outcomes – most data are from severe cases. Long-term follow-ups are limited to specific cohorts, so generalizing across diverse settings involves uncertainty. Also, potential partial catch-up growth or cognitive recovery is not fully understood. Some pathways (such as impaired immune development leading to more adult illness, or epigenetic changes raising NCD risk) are not fully quantified.	• **Impact on Labour**: Early undernutrition reduces the quality of the future workforce, leading to less skilled labor and lower productivity decades later (Galasso et Wagstaff 2019), associated to lower income (Hoddinott et al. 2013). These losses persist across generations until the cycle is broken. Severe malnutrition lead to higher child mortality – reducing the size of future labor workforce. • **Integration in models**: Increasingly included in long-run economic models by embedding nutrition into human capital formation through "cohort quality" adjustments (Galasso et Wagstaff 2019). Many standard growth models, however, still assume labor productivity gains come only from education or technology, missing the nutrition dimension. • **Challenges**: Key uncertainties involve the extent of recovery possible (if nutrition improves at later ages) and ensuring no double-counting between this pathway and Pathway 1 (since micronutrient deficiency is a component of undernutrition's effect). Additionally, estimating intergenerational effects (e.g. how maternal stunting affects grandchildren's outcomes) is complex and rarely done, potentially underestimating true societal benefits of breaking cycles of malnutrition.

2.2 Pathway 1: Short-term micronutrient Deficiencies and Immune Impairment

Micronutrient deficiencies (e.g. iron, vitamin A, iodine, zinc) remain pervasive in many populations and impose an immediate economic burden by curtailing work capacity. Recent global estimates indicate that about 50% of preschool children and 67% of women of reproductive age have at least one key micronutrient deficiency (Stevens et al. 2022). Iron-deficiency anemia alone affects roughly one-quarter of the world's population (Balarajan et al. 2011). These deficiencies lead to well-documented health impairments: anemia causes fatigue and reduced muscular and cognitive performance, while insufficient vitamin A, zinc, or other micronutrients compromise immune function and elevate infection risks (Black et al. 2013b).

Empirical evidence shows that micronutrient deficits notably diminish labor productivity. Even moderate anemia reduces adult productivity by about 5–15% for physical work, and cross-country studies estimate iron-deficiency anemia costs economies ~0.6% of GDP on average (up to 2–4% in high-prevalence countries) (S. Horton et Ross 2003). Workers with nutrient deficiencies have lower effective labor output (e.g. anemia reduces strength and endurance, impairing agricultural and manual labor productivity) and higher morbidity (leading to excess sick leave and reduced work time). In sectors requiring physical labor, the output penalty is especially pronounced. For instance, anemia prevalence rates above 30% – common in South Asia and sub-Saharan Africa – can translate into a sizable fraction of the agricultural labor force operating below peak capacity, hindering overall economic performance (Horton & Ross, 2003; World Bank, 2012). Further, severe anemia can increase maternal and neonatal mortality, affecting future labor supply.

Most standard economic models do not fully capture the productivity cost of micronutrient deficiencies. Partial approaches exist – for example, embedding an "anemia coefficient" to down-scale labor productivity in proportion to anemia rates – but comprehensive integration is rare. Immune impairment from deficiencies is typically omitted, meaning the impact of recurrent infections on labor supply is overlooked. Furthermore, many models rely on older parameter estimates from a few case studies (e.g. 1990s–2000s data) and may not reflect current epidemiology or workforce structures. There is also an analytic overlap with other pathways: micronutrient deficiencies in early childhood contribute to stunting and lower adult human capital.

2.3 Pathway 2: Diet-related mental health and cognition deterioration

A growing body of research examines diet quality as a potential factor in mental disorders, though evidence is still emerging. Large observational studies have found that people with healthier diets tend to show a lower risk of depression. For example, individuals who closely follow a Mediterranean-style diet have about a 30% lower risk of developing depressive symptoms compared to those with very unhealthy diets(Lassale et al. 2019). Mental health disorders have complex, multifactorial causes – including genetics, stress, and trauma – so isolating diet's role is challenging. The association is likely bi-directional as well: depression can lead to poor appetite or unhealthy eating, complicating causal inference. Thus, while diet is a plausible risk factor for mental health via biological pathways (inflammation, oxidative stress, gut–brain axis), its precise impact on mood disorders remains uncertain and difficult to quantify.

Mental ill-health imposes substantial economic costs through lost productivity and reduced labor participation. Depression and anxiety together account for roughly $1 trillion in global output lost annually due to absenteeism, presenteeism, and work disability[1] (Chisholm et al. 2016). In the workplace, employees

[1]This figure reflects the overall burden of these conditions, not the fraction caused by diet.

with depression have higher rates of sick leave and lower on-the-job productivity. Untreated depression is associated with lower earnings and employment; for example, an OECD analysis found that depressed individuals have significantly lower labor force participation and, if employed, earn 10–30% less than comparable workers without depression (OECD 2021). Beyond immediate productivity effects, early-onset mental health issues can disrupt education and skill formation, potentially reducing the human capital of the future labor force (e.g. adolescents with poor diets and concomitant mental health or attention difficulties may attain less schooling). Although such long-term effects are hard to quantify, they suggest an additional channel by which unhealthy diets might indirectly impair economic prospects via mental wellbeing.

Diet–mental health pathways are virtually absent from current economic models. Existing cost estimates of poor diets focus on physical diseases; none allocates a portion of mental disorder costs to nutrition, given the high uncertainty. Incorporating this channel is challenging due to the scarcity of data available. Moreover, attempting to integrate this pathway raises double-counting risks: some productivity losses attributed to obesity or cardiovascular disease (Pathway 3) may in fact result from co-occurring depression, and vice versa.

2.4 Pathway 3: Chronic diet-related diseases

Unhealthy diets are a primary upstream driver of obesity and diet-related non-communicable diseases (NCDs), including type 2 diabetes, cardiovascular diseases (CVDs), several cancers, and musculoskeletal disorders. The epidemiological evidence linking unhealthy diet to NCDs is extensive and robust. Using comparable risk-assessment methods across 195 countries, the Global Burden of Disease (GBD) study identified dietary risks as the leading contributor to premature adult mortality, associated with approximately 11 million deaths globally in 2017 (Afshin et al. 2019). In parallel, excess body weight has become widespread: roughly 2 billion adults are overweight or obese, and high body-mass index was associated with an estimated 4–5 million deaths globally in 2019, mainly via CVD and diabetes (Okunogbe et al. 2022)

A key analytic challenge is that diet-related NCDs frequently co-occur within individuals (e.g. obesity with hypertension and diabetes). The GBD framework addresses this by applying population-attributable fractions and assigning each death or disability-adjusted life year to a single underlying cause, thereby avoiding double counting at the health-outcome stage (Afshin et al. 2019). Economic analyses that aggregate disease-specific costs without such adjustment risk overstating total burdens. More recent WHO and OECD models therefore estimate the joint impact of multiple NCDs, explicitly accounting for overlap (Bertram et al. 2018; OECD 2019).

The economic consequences of diet-related NCDs arise primarily through reduced labour productivity and lower labour-force participation. Chronic NCDs increase absenteeism and presenteeism, raise the likelihood of early retirement and disability, and shorten working lives through premature mortality. Because these conditions often emerge in mid-life, productivity losses accumulate over many years. Focusing specifically on obesity and overweight, recent global modelling suggests an economic impact equivalent to about 2.2% of global GDP in 2019, projected to rise to around 3.3% of global GDP by 2060 if current trends persist (Okunogbe et al. 2022). These losses are increasingly concentrated in low- and middle-income countries undergoing rapid dietary transition, where NCD prevalence is rising among working-age populations. Beyond labour markets, widespread chronic illness can indirectly dampen growth by reducing household savings, diverting public resources, and constraining fiscal space (Suhrcke et Urban 2010).

Compared with other diet–health pathways, the NCD channel is relatively well represented in economic models, though often in simplified form. Many macroeconomic and CGE studies incorporate NCDs as reductions in effective labour supply or productivity. Nevertheless, important gaps remain. Models frequently apply uniform productivity penalties across workers, overlooking heterogeneity by age, skill, and sector, and long-term dynamic feedbacks (e.g. effects on savings or human capital accumulation) are often omitted. Ensuring consistency with epidemiological attribution is also critical to avoid double counting across diseases and pathways (Nugent et al. 2020b)

2.5 Pathway 4: Developmental & intergenerational impacts

Inadequate nutrition during critical periods – from pregnancy through early childhood – can cause stunting (chronic growth faltering), wasting (acute severe thinness) and micronutrient deficiencies, with irreversible impacts on physical growth, cognitive development and survival. In 2020, an estimated 149 million children (22% of under-5) were stunted worldwide, with the vast majority living in low- and lower-middle-income countries. Maternal and child undernutrition is a leading contributor to child mortality, implicated in roughly 45% of all under-5 deaths (about 3 million fatalities annually). Surviving malnourished children face long-term deficits: undernourishment in the first 1,000 days impairs immune function, irreversibly harms brain development, and raises the risk of chronic diseases later in life (Victora et al. 2008). Stunted children grow up with lower cognitive abilities, attain less schooling, and ultimately earn less in adulthood. Undernutrition can also perpetuate an intergenerational cycle of poverty: malnourished girls often become stunted, anemic mothers who are more likely to have low-birthweight, undernourished infants, locking communities into lower productivity over multiple generations (Black et al. 2013b; Victora et al. 2008).

The economic cost of early-life undernutrition manifests over decades in the form of foregone human capital and reduced labour productivity. Stunting in childhood leads to a less educated and less productive workforce years later. Cross-country estimates in a development accounting framework indicate that, on average, per capita incomes in developing countries would be about 7% higher today if adult workers had not suffered stunting in childhood. In regions with the highest prevalence – notably South Asia and sub-Saharan Africa – historical childhood stunting is estimated to have depressed current GDP by 9–10%. These losses occur because undernourished children grow into adults with diminished productivity and earnings. One study found that each additional centimeter of adult height (a proxy for better childhood nutrition) is associated with a 4% increase in wage rates, especially in manual labor (Sue Horton et Steckel 2013). Likewise, each additional year of early-childhood stunting is associated with fewer years of schooling and lower cognitive skills, thereby reducing the future share of skilled labor and innovation in an economy (Galasso et Wagstaff 2019). Galasso and Wagstaff (2019) estimate that for the developing world as a whole, childhood stunting lowers aggregate income by the equivalent of about 7% of GDP.

Fully capturing developmental and intergenerational impacts in economic models is challenging due to the lagged and cumulative impacts. Recent efforts address this by using cohort-based models and overlapping-generations frameworks. Such approaches show modest short-term effects but increasingly large gains over time as healthier cohorts replace previously undernourished ones. For example, one global simulation suggests eliminating child stunting would yield relatively small income gains by 2030 but very large gains by 2050. Methodological challenges also include parameterizing the relationship between early nutrition and adult productivity for diverse contexts and accounting for partially reversible damage. There are finally interactions to navigate: early undernutrition can lead to higher adult NCD burdens (linking to Pathway 3), but care must be taken to avoid double-counting such costs across pathways.

2.6 Cross-Cutting Issues: Uncertainty and Double-Counting

A consistent theme across the four pathways is the interdependence of diet-health effects and the difficulty of isolating their economic costs. In reality, multiple forms of malnutrition and diet-related disease often co-occur in the same populations – even within the same households or individuals (Nugent et al. 2020b). For instance, a stunted child (Pathway 4) is likely to also suffer from micronutrient deficiencies (Pathway 1); an obese adult with diabetes (Pathway 3) may also experience depression (Pathway 2). These overlapping etiologies mean that simply summing cost estimates from each pathway would double-count shared losses.

Sophisticated analyses therefore use integrated approaches – such as comparative risk assessment in epidemiology and true cost accounting in economics – to attribute proportions of outcomes to primary diet-related causes. For example, the GBD study assigns each diet-attributable death or disability-adjusted life-year to a single dietary risk (Afshin et al. 2019), and the FAO's hidden costs methodology models all diet-related health outcomes jointly to ensure that co-morbid disease burdens are not counted twice (S. Lord 2023).

Diet-related costs fall disproportionately on low-income countries and vulnerable groups (S. Lord 2023) – for instance, undernutrition remains concentrated among the poor, while obesity and related NCDs are rising fastest in many lower-middle-income populations (Nugent et al. 2020b). Standard macro models, focused on aggregate GDP, may overlook these inequalities, failing to differentiate impacts by gender or socioeconomic status.

To accurately capture diet-related costs, analyses increasingly use system-wide, life-course models. These include microsimulations and dynamic CGE models that track individuals or cohorts over time, linking early-life nutrition to adult outcomes and simulating how reductions in current malnutrition yield future economic benefits (Galasso et Wagstaff 2019). Such models allow simultaneous representation of multiple pathways and their interactions – for example, showing how reducing child stunting today lowers both direct human capital losses (Pathway 4) and future NCD prevalence (Pathway 3). They also facilitate scenario analysis of "double-duty actions" that address under- and overnutrition together (Nugent et al. 2020b).

In summary, accurately assessing the economic cost of unhealthy diets demands careful handling of overlapping effects and uncertainties. The four pathways operate jointly, not in isolation. A comprehensive accounting requires methods to apportion costs to primary dietary causes and to capture the interactions between undernutrition and NCDs (the "double burden") without double-counting (Nugent et al., 2020). It also calls for attention to distributional dimensions – which subpopulations bear the brunt of hidden costs, and who stands to gain most from interventions. Ongoing efforts like true cost accounting and cohort-based macro models are steps toward a more holistic quantification.

3 Analytical modelling framework

3.1 Introduction

The MIRAGRODEP model is a multi-region, multisector computable general equilibrium model (CGE model). It builds on MIRAGE, a multisector CGE model of the global economy (Decreux et Valin 2007) and is specifically designed for the analysis of agricultural sectors, food systems, and distributional impacts. MIRAGRODEP can be linked to complementary modules to assess impacts on poverty, food security, nutrition, land use, and greenhouse gas emissions, making it particularly suitable for the assessment of agrifood system reforms.

The MIRAGRODEP model is based on an input–output framework and its theoretical structure is derived from optimizing behavior of economic agents, particularly households and firms. The model assumes perfect competition in all sectors, enabling detailed geographic and sector decompositions. A detailed and complete documentation of the model is provided in (Bouët et al. 2022)

The primary data source for MIRAGRODEP is the GTAP12 database (fully documented in (Aguiar et al. 2025), a database comprising world macroeconomic accounts and trade flows for the reference year 2023). Specifically, it describes the values of production, intermediate and final consumption of commodities and services for 145 countries or regions and 65 sectors. The database also provides information on global bilateral trade patterns, international transport margins, and protection matrices allowing for an accurate representation of linkages between individual countries and regions.

Regarding the supply side, the production function is modeled for each sector as a Leontief function of intermediate inputs and value-added. The intermediate input bundle is represented by a constant elasticity of substitution (CES) function across commodities, allowing for substitution among intermediate inputs in response to relative price changes. The level of substitutability is assumed to be constant for any pair of intermediate goods. Similarly, value added is a CES function of unskilled labor, land, natural resources, and a bundle of skilled labor and capital. This nesting structure enables the model to capture the lower substitutability between capital and skilled labor compared to other factors.

In each region the demand side is modeled with a representative agent who is assumed to own all factors of production, have a constant propensity to save and seek to maximize utility by purchasing goods. The agent’s utility function is a linear expenditure system-constant elasticity of substitution (LES-CES) function to capture his preferences between goods and determine the allocation of expenditure across those goods. The LES-CES function allows for different income elasticities of demand across goods. It can therefore capture changes in consumption patterns as income levels evolve across regions. This is particularly relevant for modeling agricultural and non-agriculture sectors since income elasticities for food tend to be lower than those for manufactured goods and services. Additionally, the elasticity of substitution among sectoral consumptions is constant only above a minimum level, which can vary by region, such as developing versus developed countries.

The model captures international economic linkages through international trade in goods and services, and capital flows, thereby providing a comprehensive and consistent representation of economic and trade relations between countries. MIRAGRODEP is a bilateral trade model that is consistent with the Armington assumption, which posits that commodities are heterogeneous based on their origin and thus, imperfect substitutes for each other. The model uses nested CES functions to reflect preferences for varieties originating from different countries. This approach allows for countries to import and export the same product simultaneously, reflecting consumers' preferences for specific varieties.

MIRAGRODEP also explicitly models the government as a separate agent from private households and firms. The government's income comprises taxes on production, factors of production, exports, imports, consumption, and households’ income. The model assumes that the government aims to maximize a Cobb-Douglas utility function, where its expenditure on each commodity is a fixed share, in value, of the total public expenditure on goods and services.

The recursive dynamic version of the model was used for this analysis. The model incorporates a capital accumulation framework, where the capital for year t+1 is based on the capital of year t, increased by the previous year's investment and adjusted for depreciation. The model is then solved sequentially, moving the equilibrium from one year to another.

3.2 Four main assumptions

The model rests on four major assumptions: the factor market closure, the private account closure, the external account closure and the government account closure.

With respect to the factor market closure, total employment is assumed to remain constant as a proportion of the active population, which is defined as individuals aged between 15 and 60 years old. Labor markets are differentiated by gender, accounting for an imperfect substitution between male and female labor within each skill category. Skilled labor is perfectly mobile, while unskilled labor has imperfect mobility between agricultural and non-agricultural sectors, as determined by a constant elasticity of transformation (CET) function. Natural resources are the only factor with a fixed supply over time. Land supply, on the contrary, is endogenous and depends on the real remuneration of land. Additionally, it is assumed that land has imperfect mobility between agricultural sectors. Finally, installed capital and natural resources are modeled as sector specific.

Regarding the private account closure, the model makes the neo-classical assumption that the marginal propensity to save remains constant. Consequently, variations in income result in variations in savings, which, in turn, bring variations in investment. Investments are therefore savings-driven.

For the external account closure, it is assumed that the current account balance remains stable as a proportion of global GDP. Hence, the real exchange rate adjusts through different changes in domestic prices across various regions, referred to as competitive disinflation.

Finally, with respect to the public account closure, it is assumed that the government budget remains a fixed share of the national GDP, and that the real public expenses per capita are held constant to ensure a constant provision of public goods. There are therefore no changes in the size of the public sector and no crowding-out effect on private investment. To keep the government budget and the real public expenses per capita at a constant level, a consumption tax is adjusted. The magnitude of this tax thus measures the cost of maintaining a steady supply of public goods.

3.3 Poverty aspects

The poverty analyses were carried out using the POVANA household model, as documented in (Laborde et al. 2021). The POVANA model is based on the POVANA dataset which provides the full income distribution for approximately 300,000 households. The model captures the endogenous evolution of households' real income in response to changes in multiple factors, such as employment, prices of goods and services, wages, and productivity. Additionally, the comprehensive information on household consumption patterns within the POVANA dataset enables the estimation of the effects of economic shocks on household costs, including the costs of food.

4 Data

Forthcoming.

5 Scenario

Forthcoming.

6 Results

Forthcoming.

7 Conclusion

Forthcoming.

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