



HEALTH IMPACTS OF THE 4F NEXUS (FUEL, FOOD, FERTILISERS AND FINANCE) IN THE GLOBAL SOUTH, WITH EMPHASIS ON CANCER RISK: A NARRATIVE REVIEW

Community Medicine

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ABSTRACT

Background: Energy-market volatility, geopolitical conflict and climate extremes transmit through fertiliser and food markets and public finances, a convergence of pressures described by policymakers as the "4F" (fuel, food, fertiliser, finance) crisis. The health consequences for low- and middle-income countries (LMICs) have been examined mostly for nutrition; cancer-relevant pathways have not been synthesised. **Objective:** To synthesise evidence on the pathways linking the 4F nexus to health in the Global South, distinguishing pathways with direct epidemiological support from those that are biologically plausible but unquantified. **Methods:** Non-systematic narrative review of International Agency for Research on Cancer (IARC) Monographs, United Nations reports and key epidemiological studies selected by the authors for relevance, size and recency. Strength of evidence for each pathway was graded qualitatively. **Findings:** The best-quantified pathway is food inflation: in 1.27 million children from 44 LMICs, a 5% rise in real food prices was associated with a 9% higher risk of wasting and a 14% higher risk of severe wasting. Household air pollution from solid fuels is an established lung carcinogen (coal: IARC Group 1; biomass: Group 2A); a prospective cohort of 323,794 Chinese never-smokers reported a 4% higher lung-cancer mortality per 5 years of exposure. Ingested nitrate and nitrite under conditions of endogenous nitrosation are classified Group 2A; human evidence links drinking-water nitrate mainly to colorectal cancer and, less consistently, to thyroid and kidney cancer. The evidence that 4F shocks will increase cancer incidence is indirect: it rests on extrapolating exposure–response relationships and has not been tested in studies of shock exposure and cancer outcomes. **Conclusions:** The 4F nexus affects health through acute nutritional pathways with strong evidence and through carcinogenic exposures with moderate evidence but long latency. Priorities include nutrition-sensitive social protection, protection of clean-cooking transitions and cancer-screening capacity during fiscal stress, and nitrate-aware fertiliser management.

KEYWORDS

Food Security; Fertilisers; Household Air Pollution; Nitrates; Neoplasms; Low- And Middle-income Countries; Child Nutrition

INTRODUCTION

Around 645 million people (7.8% of the global population) faced hunger in 2025, and the 2026 State of Food Security and Nutrition report projects that higher energy and fertiliser prices could leave 510–520 million people hungry in 2030, compared with 503 million in a pre-conflict scenario.¹ Fluctuations in energy prices, armed conflict and climate extremes now repeatedly pass through fertiliser markets into food prices, household energy choices and government budgets. In September 2026, India's External Affairs Minister described a "4F crisis of fuel, food, fertiliser and finance" facing the Global South.² Most analyses of the 4F nexus address macroeconomic and food-security outcomes; its health consequences, and especially its cancer-relevant consequences, have received little systematic attention.

Cancer is a long-latency outcome, so a causal link from a price shock to cancer incidence cannot be shown directly in the short term. The question this review addresses is therefore narrower and testable: through which pathways does the 4F nexus alter exposure to established or suspected carcinogens, dietary protection against cancer and access to cancer care, and how strong is the evidence for each pathway? Shock propagation through the global food production network has been modelled and shown to depend on the connectivity of trading partners and their import dependence.³ This review synthesises the available evidence on these pathways, with particular attention to carcinogenic hazards classified by IARC.

Methods

This is a non-systematic narrative review of the pathways linking fuel, food, fertiliser and finance shocks to health outcomes, with emphasis on cancer risk, in the Global South. Evidence was selected by the authors from IARC Monograph evaluations, United Nations agency reports (FAO, WHO), and key epidemiological studies (prospective cohorts, randomised trials, systematic reviews and meta-analyses), prioritising the largest and most recent studies relevant to LMIC settings. Searches were not conducted in a protocolised, reproducible way, and the review is not intended to be exhaustive. No meta-analysis was performed.

Strength of evidence for each pathway was graded qualitatively as strong, moderate or limited according to study design, consistency of findings, and whether the evidence addresses the 4F context directly or is extrapolated from other settings (Table 1). IARC Monograph evaluations were used as the primary source for carcinogen hazard classification. The review is reported in accordance with the SANRA (Scale for the Assessment of Narrative Review Articles) recommendations.

Conceptual framework

The principal transmission chain is: energy or fuel price shock → higher fertiliser production and logistics costs (natural gas is the main feedstock and energy source for nitrogen fertiliser synthesis⁴) → reduced fertiliser application or higher farm-gate costs → lower yields and higher food prices → constrained household diets and incomes → undernutrition and excess morbidity.

Three parallel channels are relevant to cancer:

- 1. Fuel → household energy substitution:** affordability of clean cooking fuel falls, and households revert to solid fuels, increasing household air pollution.
- 2. Finance → fiscal compression:** subsidies, social protection and health budgets contract, and out-of-pocket costs rise, delaying screening, diagnosis and treatment.
- 3. Fertiliser use → water quality:** high application rates, rather than shortages, drive nitrate leaching into groundwater. Shocks may change application patterns in either direction.

Food-price pathway Acute and chronic undernutrition

Food inflation is the best-documented health channel. In a study linking 130 Demographic and Health Surveys in 44 LMICs (2000–2021) with monthly national food-price data, a 5% increase in the real food price over the preceding three months was associated with a 9% higher risk of wasting and a 14% higher risk of severe wasting among 1.27 million children aged 0–59 months.⁵ Effects were larger among boys, rural children and children from asset-poor, landless households (26% higher wasting risk for a 5% price rise).⁵ Food inflation during pregnancy and the first year of life was also associated with a higher risk of stunting at 2–5 years.⁵ These are observational associations from repeated cross-sectional survey data and should be interpreted as such.

Undernutrition accounts for a large share of child mortality in LMICs, and early-life growth faltering has lasting consequences for cognitive development, adult health and economic productivity.^{6,7} Dietary diversity declines as prices rise, as households protect energy intake by reducing animal-source foods, fruits and vegetables, producing micronutrient deficiencies.⁵

Diet quality and cancer

A lower intake of fruit, vegetables and fibre-rich foods is consistent with the evidence that dietary fibre and whole grains reduce colorectal cancer risk, and the World Cancer Research Fund concluded that wholegrains, fibre-containing foods, and non-starchy vegetables and fruits protect against several cancers.^{8,9} Whether price-driven dietary deterioration in LMICs translates into measurable cancer incidence has not been studied, and the link should be regarded as plausible but unquantified.

Double burden

Where price shocks shift diets toward cheap, energy-dense, ultra-processed foods, overweight and metabolic disease may follow the earlier undernutrition; excess body fatness is an established cause of several cancers.¹⁰ This remains a hypothesis for the 4F context and requires cohort evidence from affected populations.

Fuel and household-energy pathway

WHO estimated that household air pollution from polluting fuels and technologies was responsible for about 3.2 million deaths in 2020, including more than 237,000 deaths in children under five.¹¹ Women and young children bear a disproportionate burden because of time spent near cooking fires.

Carcinogen classification

The IARC Monographs classify indoor emissions from household coal combustion as carcinogenic to humans (Group 1; sufficient evidence for lung cancer) and emissions from household biomass combustion, mainly wood, as probably carcinogenic (Group 2A).^{12,13}

Epidemiological evidence

A meta-analysis of case–control studies reported pooled odds ratios for lung cancer of 1.82 (95% CI 1.60–2.06) for coal use and 1.50 (95% CI 1.17–1.94) for biomass use,¹⁴ while a later review focused on biomass found a smaller pooled estimate (OR 1.17, 95% CI 1.01–1.37), with substantial heterogeneity and potential confounding.¹⁵ Evidence for coal derives substantially from Xuanwei, China, where very high lung-cancer mortality was observed among users of smoky coal.¹⁶

The China Kadoorie Biobank (CKB) cohort provides prospective evidence: among 323,794 never-smokers (89% women) followed for a

median of 10.2 years, there were 979 lung-cancer deaths, and each 5-year increase in cumulative exposure was associated with a 4% higher risk of lung-cancer death (HR 1.04, 95% CI 1.01–1.06).¹⁷ The highest risk was seen at 40–50 years of exposure (HR 1.53, 95% CI 1.13–2.07), whereas the HR for more than 50 years was lower (1.27, 95% CI 0.93–1.73), which the authors attributed to survival or healthy-volunteer bias, and "ever versus never" exposure was not statistically significant (HR 1.15, 95% CI 0.91–1.45).¹⁷ The outcome was mortality rather than incidence, and exposure was self-reported solid-fuel history rather than measured. In the same cohort, solid-fuel use was also associated with cardiovascular and all-cause mortality and with respiratory disease.^{18,19}

Links to the 4F shock

The inference that energy-price shocks reverse clean-fuel adoption and thereby increase lung-cancer risk is plausible but is an extrapolation. In the CKB cohort, a statistically significant excess lung-cancer mortality emerged only after about 30 or more years of cumulative exposure,¹⁷ so a short-term reversion would change cumulative exposure only modestly, whereas sustained energy poverty would matter. A multicountry randomised trial of liquefied petroleum gas (LPG) stoves (Guatemala, India, Peru and Rwanda), in which uptake was nearly complete, did not show a significant improvement in birth weight (adjusted mean difference 19.6 g, 95% CI –10.1 to 49.2).²⁰ Clean-fuel adoption therefore cannot be assumed to benefit every outcome, and outcome-specific evidence is needed.

Other indirect effects include disrupted vaccine cold chains, reduced facility functionality, and higher transport costs for patients seeking cancer care. These are plausible but lack direct quantification. Generalisability beyond Chinese settings with high smoky-coal exposure requires caution, as fuel types, ventilation and smoking patterns differ across LMICs.

Fertiliser and water-nitrate pathway

Direction of effect

Fertiliser shortages and price spikes primarily harm health through reduced yields and higher food prices. Their relationship to nitrate contamination is more complex than a simple secondary pathway: nitrate leaching is driven by high or inefficient application, so a price shock that curtails use would be expected to reduce nitrate loading in the short term, while subsidised or policy-driven over-application (as in many intensive systems) sustains it. The nitrate pathway is therefore a feature of the fertiliser system in general, and its relationship to 4F shocks is conditional on how application patterns respond.

Carcinogen classification and human evidence

IARC classified ingested nitrate and nitrite under conditions that result in endogenous nitrosation as probably carcinogenic to humans (Group 2A).²¹ The classification rests largely on animal and mechanistic evidence; human evidence was limited or inadequate for individual cancer sites.²¹ Subsequent reviews found the most consistent human associations of drinking-water nitrate for colorectal cancer, with weaker and less consistent evidence for thyroid and kidney cancer, and inconsistent findings for other sites such as bladder and stomach.²² A Danish nationwide cohort reported increased colorectal-cancer risk at nitrate concentrations below the current drinking-water standard of 50 mg/L.²³ A modelling study estimated that nitrate in US public water supplies could account for about 12,500 cancer cases annually; this is a US-specific, model-based estimate and should not be extrapolated to the Global South without local exposure data.²⁴ Vitamin C and other inhibitors of nitrosation modify risk, which is relevant where diets deteriorate.^{21,22}

Finance pathway and amplifying mechanisms

Currency depreciation, tighter external financing and rising debt-service costs constrain subsidies, food reserves, cash transfers and health budgets, while households face higher real prices and greater out-of-pocket health costs. Cancer care already imposes a heavy financial burden in LMICs: a systematic review and meta-analysis reported a pooled prevalence of catastrophic health expenditure among cancer patients of 47% in middle- and high-income countries and 74.4% in low-income countries.²⁵ It is therefore plausible that fiscal and household financial stress would delay screening, diagnosis and treatment, but, to our knowledge, studies directly linking macroeconomic shocks to cancer stage at diagnosis or treatment abandonment are lacking, and this pathway is graded as limited.

Women and girls often absorb shocks through reduced food intake and increased unpaid work, with consequences for maternal and child nutrition.^{5,7}

Summary of evidence

Table 1. Strength of evidence by pathway

Pathway	Exposure → outcome	Key evidence	Strength	Main caveat
Food price → child wasting/stunting	5% real price rise → 9% (wasting), 14% (severe wasting)	1.27 million children, 44 LMICs ⁵	Strong (observational, large, consistent)	Repeated cross-sectional design
Food price → diet quality → cancer	Reduced fibre, fruit, vegetables → colorectal and other cancers	WCRF, meta-analysis ^{8,9}	Moderate for diet-cancer; limited for shock-to-cancer	No shock-exposure cancer studies
Fuel → solid-fuel use → lung cancer	Coal Group 1; biomass Group 2A; 4% per 5 years in never-smokers	IARC ^{12,13} ; CKB cohort ¹⁷ ; meta-analyses ^{14,15}	Strong (coal), moderate (biomass)	Mortality outcome; long latency; extrapolation to shocks; limited generalisability beyond high-exposure Chinese settings
Fertiliser → water nitrate → cancer	Group 2A; colorectal, possibly thyroid and kidney	IARC ²¹ ; review and cohort ^{22,23}	Moderate (colorectal), limited (other sites)	Human evidence limited; exposure context-specific; direction of effect under shocks is conditional
Finance → delayed cancer care	Out-of-pocket costs and fiscal compression → later diagnosis	Financial toxicity of cancer care in LMICs ²⁵ ; no direct shock studies	Limited	Evidence on stage migration and treatment abandonment during shocks is lacking

Vulnerable populations

Children under two years, pregnant and lactating women, asset-poor and landless households, and populations in import-dependent or conflict-affected regions face the greatest risks.⁵ Early-life nutritional deficits have lasting consequences for growth, cognition and adult health.⁷ Women face combined exposure to household air pollution and reduced dietary quality. Urban poor households face rapid price transmission, whereas rural households experience both price and production shocks together with higher solid-fuel and nitrate exposure. The suggestion that early-life undernutrition raises later cancer susceptibility should be stated as a hypothesis, because adiposity and attained adult height, rather than stunting itself, are the better-established anthropometric correlates of cancer risk.^{10,26}

DISCUSSION

The evidence synthesised here shows that the 4F nexus exerts its clearest and most immediate effects on child nutritional status through food-price inflation, with large, consistent associations demonstrated across dozens of LMICs.⁵ In contrast, the carcinogenic pathways are better characterised as established hazards whose activation by 4F shocks remains inferential. Household solid-fuel combustion is an IARC Group 1 (coal) or Group 2A (biomass) carcinogen with

prospective cohort support for lung-cancer mortality,^{12–17} yet the step from a temporary energy-price shock to a meaningful increase in cumulative lifetime exposure, and therefore to excess lung-cancer incidence, has not been demonstrated. Similarly, nitrate under conditions favouring endogenous nitrosation is a Group 2A carcinogen with moderate human evidence for colorectal cancer,^{21–23} but fertiliser price spikes may reduce rather than increase application rates in the short term, so the net effect on population nitrate exposure is context-dependent.

These distinctions matter for policy, and five implications follow. First, nutrition-sensitive social protection that scales automatically with food-price shocks (targeted cash or food transfers, school feeding, micronutrient supplementation) is justified by strong observational evidence and is likely to protect the first 1,000 days.^{5,7} Second, clean-cooking support addresses a well-established lung carcinogen, but its evidence for specific outcomes such as birth weight is mixed,²⁰ so programmes should be evaluated against measured exposure and health outcomes. Third, nitrate-aware fertiliser management (efficiency-focused support, soil testing) and groundwater monitoring are warranted where application rates are already high, independent of short-term price shocks.^{21,22} Fourth, market monitoring, strategic reserves and regional trade arrangements can dampen price volatility and its propagation through food networks.³ Fifth, preservation of cancer-screening and treatment capacity during fiscal stress is logical, given the existing financial burden of cancer care in LMICs,²⁵ but currently rests on limited direct evidence.

Research gaps

First, no study has directly examined cancer incidence or stage at diagnosis in relation to exposure to fuel, fertiliser or food-price shocks; quasi-experimental and cohort designs are needed. Second, India-specific and other LMIC data should be added on clean-fuel programme uptake and sustained use, groundwater nitrate in high-input agricultural districts, and cancer-screening continuity. Third, exposure measurement for household air pollution is largely self-reported, and measured exposure-response data in LMIC settings would strengthen causal inference. Fourth, the contribution of dietary change during shocks to cancer risk requires prospective cohorts with dietary assessment. Fifth, the finance pathway requires direct evidence on catastrophic health expenditure, stage migration and treatment abandonment during 4F-type shocks.

Limitations

This is a non-systematic narrative review: study selection was not protocolised or reproducible and may be subject to selection bias, and no formal quality appraisal was performed. Much of the carcinogen evidence derives from China or high-income settings and may not transfer to other LMICs with different fuel types, ventilation, smoking patterns or agricultural intensity. Exposure assessment for household air pollution is largely self-reported, and the leading cohort evidence concerns mortality rather than incidence. The linking of acute shocks to long-latency outcomes is inferential, and even well-designed cohort studies of current shocks will need years of follow-up before incidence effects can be observed. The finance pathway lacks 4F-specific studies. Effect sizes are from observational studies and are subject to confounding and measurement error.

CONCLUSIONS

The 4F nexus affects health through a strongly evidenced nutritional pathway and through carcinogenic pathways whose underlying hazards are well established but whose link to 4F shocks is inferential. Even modest food-price rises are associated with clinically meaningful increases in child wasting. Household solid-fuel use is an established lung carcinogen with prospective cohort support, and nitrate in drinking water is a probable carcinogen with moderate human evidence for colorectal cancer. Integrated responses should combine upstream resilience (energy, fertiliser and trade) with downstream buffers (nutrition-sensitive transfers, clean energy, water-quality protection and uninterrupted cancer care). Studies that directly link shock exposure to cancer outcomes are the key research priority.

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