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PROCESSED MEAT CONSUMPTION AND GASTROINTESTINAL MALIGNANCIES: A COMPREHENSIVE REVIEW OF META-ANALYSES EXAMINING EPIDEMIOLOGICAL TRENDS AND SOCIOECONOMIC CHALLENGES

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ABSTRACT

Gastrointestinal (GI) cancers, particularly colorectal cancer (CRC), represent an escalating global health burden linked to the industrialization of food systems and the global "nutritional transition." This narrative review provides an extensive synthesis of the relationship between processed meat consumption and GI malignancies, focusing on molecular pathomechanisms, specific mutational signatures in key genes (APC, KRAS, TP53), the mediating role of the gut microbiota, socioeconomic impacts, and the efficacy of modern screening guidelines.A systematic synthesis of high-impact literature (2015–2025) from PubMed and Scopus was conducted. We integrated data from five landmark meta-analyses and umbrella reviews (Vieira, Veettil, Poorolajal, Di, and Ungvari) alongside toxicological reports on food-derived carcinogens and clinical guidelines from international gastroenterological societies (ACG, ESGE, ASGE). Epidemiological data consistently indicate that a 50g daily portion of processed meat is associated with a 16-18% increase in CRC risk. Molecular analysis reveals that carcinogenic compounds, such as N-nitroso compounds (NOCs) and polycyclic aromatic hydrocarbons (PAHs),. This review further distinguishes between processing methods, identifying high-heat frying of cured meats as the most potent carcinogenic driver. Furthermore, we identify a "social risk trap," where lower socioeconomic groups carry a higher burden of dietary carcinogens. Mitigating the impact of processed meat on GI health requires a multifaceted approach integrating industrial reform, targeted public health education, and precision-based screening protocols that consider dietary exposure as a primary risk factor.

KEYWORDS

Processed Meat, Colorectal Cancer, Gastrointestinal Neoplasms, Mutational Signatures, ACG Guidelines, Health Economics, Carcinogenesis, Gut Microbiota

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1. Introduction

The 21st-century "nutritional transition" represents one of the most significant shifts in human history, characterized by a transition from traditional, fiber-rich diets toward Westernized patterns. This modern diet is characterized by a high intake of refined carbohydrates, saturated fats, and, most critically, processed meats. As defined by the International Agency for Research on Cancer (IARC), processed meat refers to any meat that has been transformed through salting, curing, fermentation, smoking, or other industrial processes to enhance flavor or shelf-life.

The clinical consequences of this dietary shift are most evident in the rising incidence of gastrointestinal (GI) malignancies. Colorectal cancer (CRC) currently ranks as the third most common cancer globally and the second leading cause of cancer-related mortality. In 2015, the IARC Monograph Working Group formally classified processed meat as "carcinogenic to humans" (Group 1), a classification supported by more than eight hundred epidemiological studies (Bouvard et al., 2015). This placed processed meat in the same risk category as tobacco smoking and asbestos, although the magnitude of risk differs significantly. Despite this definitive scientific consensus, the consumption of processed meats continues to rise in transitioning economies, fueled by urbanization, the demand for affordable animal protein, and the expansion of the industrial food system. The pathophysiology linking processed meat to GI cancer is remarkably complex. It involves a synergistic interplay between exogenous chemical additives (e.g., nitrites), combustion-derived carcinogens (e.g., PAHs), and endogenous biological responses such as heme-iron catalysis and gut dysbiosis (Turesky, 2018). These interactions lead to specific mutational signatures in key tumor suppressor genes and oncogenes, such as *APC*, *KRAS*, and *TP53*, effectively providing a molecular "fingerprint" of dietary harm. This review aims to synthesize the latest high-impact evidence (2017–2025) to map the pathomechanisms of processed meat across the entire GI tract, analyze the socioeconomic burden, and evaluate the necessity of risk-adapted screening. By bridging the gap between molecular toxicology and social medicine, this work argues for a holistic strategy to mitigate one of the most pressing challenges in modern oncology.

2. Methodology

This narrative review utilizes a multidisciplinary synthesis approach, integrating evidence from clinical oncology, toxicology, epidemiology, and health economics. To ensure the highest scientific rigor and relevance to contemporary medicine, a systematic search and evaluation framework was established.

Search Strategy and Databases

A comprehensive literature search was conducted across several electronic databases: PubMed/MEDLINE, Scopus, Web of Science, and the Cochrane Library. The search was restricted to English-language peer-reviewed articles published between January 2015 (following the landmark IARC evaluation) and February 2025.

The search strategy employed advanced Boolean operators and Medical Subject Headings (MeSH). Primary search strings included:

(("processed meat" OR "red meat" OR "preserved meat") AND ("carcinogenesis" OR "neoplasms" OR "oncogenesis"))

(("colorectal cancer" OR "gastric cancer" OR "esophageal cancer") AND ("dietary risk factors" OR "nitrites" OR "polycyclic aromatic hydrocarbons"))

(("screening guidelines" OR "early detection") AND "gastrointestinal oncology").

Furthermore, a manual recursive search of the reference lists of included articles (gray literature) was performed to ensure that no significant data regarding molecular pathomechanisms or specific mutational signatures were overlooked.

Selection Criteria (PICO Framework)

Selection was guided by the PICO framework (Population, Intervention/Exposure, Comparison, Outcome). The population included human subjects across all demographic and geographical regions, with a sub-focus on individuals diagnosed with early-onset colorectal cancer (EOCRC). Exposure was defined as the consumption of various types of processed meat (cured, smoked, salted, fermented). The comparison involved low-intake or meat-free populations. Outcomes were defined as the incidence of GI malignancies, specific molecular genetic alterations (e.g., *KRAS* transitions), and mortality rates.

Inclusion and Exclusion Criteria

Studies were eligible for inclusion if they met the following criteria: (1) Systematic reviews and meta-analyses of prospective cohort studies; (2) Umbrella reviews synthesizing multiple large-scale observational datasets (e.g., Veettil et al., 2021); (3) Mechanistic studies utilizing advanced sequencing (WGS) or gut microbiome analysis; and (4) Official clinical guidelines from recognized international bodies (ACG, ESGE).

We excluded: (1) Single case reports and small-scale case-control studies with high risk of recall bias; (2) Pre-clinical studies involving high-dose chemical exposures that do not reflect human dietary patterns; and (3) Studies published prior to 2015 unless providing foundational mechanistic data.

Data Extraction and Quality Assessment

Data were extracted systematically, focusing on pooled Relative Risk (RR) values, 95% Confidence Intervals (CIs), and specific mutational frequencies. The quality of included meta-analyses was assessed using the AMSTAR 2 criteria, prioritizing those with a "high" or "moderate" confidence rating. Toxicological reports were evaluated based on their relevance to dietary exposure levels and biological plausibility within the human digestive system.

Evidence Synthesis and Narrative Construction

A narrative synthesis was chosen to integrate the diverse data types—from epidemiological risk estimates to molecular biology and health economics. This method allows for a comprehensive overview of how discrete biochemical events (like DNA adduct formation) translate into population-level health trends and economic burdens. Five landmark studies (Ungvari et al., 2025; Poorolajal et al., 2024; Di et al., 2023; Veettil et al., 2021; Vieira et al., 2017) served as the primary quantitative benchmarks for evaluating the dose-response relationship between meat intake and cancer risk.

3. Literature Review / Results

Global Epidemiology and Regional Trends

The "meat-cancer axis" mirrors the global industrialization of the food supply. Data from GLOBOCAN 2022 and major cohort studies highlight a significant geographical heterogeneity in cancer incidence linked to meat consumption patterns.

North America and Europe**:** In these regions, processed meat consumption often exceeds 75g-100g per capita daily. While overall CRC rates in older adults have begun to stabilize due to long-standing screening programs, a concerning "birth cohort effect" has emerged. Individuals born after 1980 exhibit a sharp increase in early-onset colorectal cancer (EOCRC), a trend closely correlated with the high prevalence of ultra-processed meats in pediatric and adolescent diets (Ungvari et al., 2025).

Asia and the Western Pacific**:** Transitioning economies, particularly China, Vietnam, and South Korea, are experiencing the most rapid epidemiological shifts. In China, CRC has moved from being relatively rare to the second most common cancer, paralleling a massive surge in the industrial production of cured sausages and canned meats (Di et al., 2023).

Latin America and the Middle East**:** Urbanization has led to a transition where traditional proteins are being replaced by affordable, industrial meat products. Poorolajal et al. (2024) highlighted that in regions where traditionally smoked or heavily salted meats are staples (e.g., parts of Brazil or Eastern Europe), the risk of gastric cancer is up to 25% higher than in populations consuming fresh-food diets.

Comparative Carcinogenicity of Processing Methods

Not all processed meats carry the same toxicological profile. The degree of carcinogenicity is dictated by the specific chemical and thermal modifications occurring during production.

Smoking and PAHs (Highest Risk for Upper GI)

Smoking meat introduces Polycyclic Aromatic Hydrocarbons (PAHs).

PAHs are formed during the incomplete combustion of organic materials (wood/coal).

Mechanism: They are activated by cytochrome P450 enzymes (specifically CYP1A1 and CYP1B1) into reactive epoxides that form bulky DNA adducts (Turesky, 2018).

Clinical Impact: Smoking has the most direct link to gastric and esophageal cancers due to the localized "toxic insult" to the squamous and columnar mucosa before systemic absorption (Poorolajal et al., 2024).

Chemical Curing and Nitrites (Highest Risk for Lower GI)

Curing involves sodium nitrite,which reacts with secondary amines in the gut to form N-nitroso compounds (NOCs).

Mechanism: NOCs are potent alkylating agents. Because nitrosation occurs throughout the digestion process, the prolonged transit time in the colon (fecal stasis) makes chemical curing the primary driver for CRC (Etemadi et al., 2017).

Risk Ranking: Cured meats that are subsequently fried or grilled at high temperatures (e.g., crispy bacon or charred hot dogs) represent the most hazardous category due to the synergistic formation of both NOCs and heterocyclic amines (HCAs).

Salting, Fermentation, and Canning (Moderate to Lower Risk)

Salting: High salt intake damages the gastric lining, induces mucosal inflammation, and promotes *H. pylori* colonization. While dangerous for gastric adenocarcinoma, it lacks the direct DNA-alkylating power of nitrites (Poorolajal et al., 2024).

Fermentation: Traditionally fermented meats (without added nitrites) are theoretically less harmful, though they still contribute to risk via sodium and heme iron content.

Canning: Often involves high-pressure heat treatment, which can lead to the formation of HCAs, but the primary risk factor in canned meats remains the chemical preservatives used.

Molecular Pathomechanisms by GI Segment

The oncogenic potential of processed meat is manifested through distinct biochemical interactions depending on the anatomical site of the gastrointestinal tract.

Pathomechanisms of Esophageal Carcinogenesis

The esophagus is the primary site of exposure to exogenous carcinogens ingested via processed meat.

Chemical Insult: Smoked meats contain high concentrations of PAHs. Upon contact with the esophageal squamous epithelium, these compounds induce genotoxic stress.

Metabolic Activation: PAHs are activated locally by CYP enzymes in the esophageal mucosa, forming diol-epoxides that bind to DNA.

Mutational Profile: These adducts induce transversion mutations, frequently targeting the *TP53* tumor suppressor gene (Poorolajal et al., 2024).

Pathomechanisms of Gastric Carcinogenesis

The stomach is a reaction vessel where meat components undergo secondary transformations.

NOC Formation: Gastric juice (low pH) facilitates the reaction between sodium nitrite and dietary proteins to form nitrosamines and nitrosamides.

Synergy with Sodium: High salt acts as a mucosal irritant, causing chronic atrophic gastritis and increasing permeability to carcinogens. It also upregulates the expression of the *CagA* virulence factor in *H. pylori*.

Pathomechanisms of Colorectal Carcinogenesis

The colon is the most frequent site of malignancy, driven by heme iron and microbial activity.

Heme Iron and Lipid Peroxidation**:** Heme iron catalyzes the Fenton reaction in the colon, producing reactive oxygen species (ROS) and toxic aldehydes like malondialdehyde (MDA).

The Vogelstein Sequence**:** Carcinogenesis follows a mutational cascade: *APC* inactivation (initiation), *KRAS* activation (progression), and *TP53* mutation (malignant transformation). (Turesky, 2018; Ungvari et al., 2025).

The Role of the Gut Microbiota as a Mediator

The intestinal microbiota acts as a critical biological interface between processed meat and the colonic epithelium.

Fusobacterium nucleatum: Significantly enriched in patients with high-meat diets. It utilizes the FadA adhesin to bind to E-cadherin, which promotes cell proliferation and survival (Veettil et al., 2021).

pks+ Escherichia coli: These bacteria harbor the *pks* genomic island, producing colibactin, which induces DNA double-strand breaks. A specific mutational signature (SBS88) matching colibactin damage is found in many CRC tumors.

Metabolites: Proteolytic fermentation of meat proteins produces toxic Hydrogen Sulfide and secondary bile acids (e.g., deoxycholic acid). Conversely, low fiber intake leads to the loss of protective butyrate.

Socioeconomic Burden and Disease Statistics

The clinical impacts translate into a staggering economic deficit and loss of human life.

Refined Epidemiology: Processed Meat Impact on Incidence and Mortality

To grasp the magnitude of the processed meat-cancer axis, we must analyze how dietary patterns integrate with global incidence and mortality statistics. Data from GLOBOCAN 2022 and the WHO Global Cancer Observatory underscore that GI malignancies are the most significant clinical consequence of modern industrial food environments.

Colorectal Cancer (CRC): A Sentinel of Industrial Nutrition

Global Statistics: CRC is currently responsible for ~1.93 million new cases and ~935,000 deaths annually. It represents approximately 10% of the total global cancer burden.

Attributable Risk: According to Population Attributable Fraction (PAF) models from the Global Burden of Disease (GBD) study, processed meat consumption is directly responsible for 5% to 10% of the global CRC burden. In high-consumption regions (e.g., Western Europe, USA), this can reach 15% (Veettil et al., 2021; Vieira et al., 2017).

The EOCRC Shift: A critical epidemiological shift is the rise in early-onset CRC (EOCRC) in individuals under fifty. Studies by Ungvari et al. (2025) suggest childhood/adolescent exposure is a primary driver.

Gastric Cancer: The Burden of Preservation

Global Statistics: Gastric cancer remains a leading cause of mortality, with ~1.09 million new cases and ~769,000 deaths annually.

Processed Meat Impact: Systematic reviews (Di et al., 2023) show a Relative Risk (RR) of 1.25 for high consumers.

Esophageal Cancer: The High Mortality Threshold

Global Statistics: With ~604,000 cases and ~544,000 deaths annually, it has a nearly 90% mortality-to-incidence ratio.

Processed Meat Impact: The 34% increased risk associated with high processed meat intake (Di et al., 2023) is a major public health priority.

Economic Costs and DALYs

Treatment for CRC is among the most expensive. Surgery costs between $20,000 and $50,000. Pharmacotherapy using biological agents and immunotherapy can exceed $150,000 per patient annually. In the US alone, annual CRC care expenditures exceed $24 billion. Indirect costs in DALYs (Disability-Adjusted Life Years) are devastating; because processed meat drives early-onset CRC, it removes individuals from the labor force during their most productive years, resulting in a loss of national GDP.

Health Inequity: The "Social Face"

Processed meat consumption is a manifestation of socioeconomic stratification.

The "Nutritional Inequality" Cycle**:** Processed meats are engineered for affordability, making them the default protein for low-income households. Fresh, lean proteins are often twice as expensive per calorie (Veettil et al., 2021).

Food Deserts**:** In urban areas, fresh produce is often economically out of reach, forcing reliance on carcinogenic products. Addressing this requires "food justice" policies.

Comprehensive Screening Guidelines: Frequency, Methods, and Economic Imperatives

The prevention of gastrointestinal malignancies relies on robust screening protocols. In light of the 16-18% increased risk associated with processed meat intake, international societies have refined their recommendations to capture early neoplastic changes. These programs represent not only a clinical necessity but also an economic strategy for national healthcare budgets.

Colorectal Cancer (CRC) Screening: ACG and ESGE Standards

The detection and removal of precancerous polyps is the primary goal of CRC screening.

Methods and Frequency:

Colonoscopy (Preferred "One-Step" Method): The American College of Gastroenterology (ACG, 2021) and the U.S. Multi-Society Task Force now recommend beginning screening at age 45 for average-risk individuals. If the index colonoscopy is normal, the procedure is repeated every 10 years. However, for individuals with high-risk findings (e.g., >3 adenomas or advanced histology), surveillance frequency increases to every 3 to 5 years.

Fecal Immunochemical Test (FIT): Recommended annually. FIT is a highly sensitive, non-invasive tool for population-level screening, particularly effective in resource-limited settings.

Micro and Macro Costs:

Unit Cost: In the United States, a colonoscopy ranges from $1,500 to $4,000, while in Europe (e.g., Germany or Poland), the cost is approximately 500 to 1,200 EUR. FIT costs are minimal, ranging from $20 to $50.

Budgetary Impact: While the upfront cost of mass screening is high, it is profoundly cost-saving. Treating a single case of advanced CRC (Stage IV) often exceeds $150,000 per year in biological and immunotherapeutic costs alone. By investing in screening, national health funds avoid the multi-billion dollar expenditures associated with late-stage diagnosis and terminal care.

Gastric Cancer Screening: ESGE (MAPS II) Guidelines

In regions where high consumption of smoked and cured meats drives gastric cancer (e.g., parts of Europe and Asia), screening focuses on the identification of precursor lesions like Atrophic Gastritis (AG) and Intestinal Metaplasia (IM).

Methods and Frequency:

Upper GI Endoscopy (Gastroscopy): According to the ESGE MAPS II guidelines, patients with extensive IM or severe AG should undergo surveillance gastroscopy every 3 years. High-risk consumers from endemic areas may require screening starting in their forties.

H. pylori "Test-and-Treat": Screening for *H. pylori* via breath tests or stool antigens (frequency: once, with re-testing if symptomatic) is considered a primary prevention strategy.

Economic Benefit: Eradicating *H. pylori* and monitoring AG prevents the progression to adenocarcinoma. Gastroscopy costs are a fraction of the cost of total gastrectomy and subsequent oncological support.

Esophageal Cancer Screening: Targeted Surveillance

Screening for esophageal malignancies (ESCC and Adenocarcinoma) is reserved for high-risk cohorts, including those with chronic reflux or heavy exposure to PAH-rich smoked meats.

Methods and Frequency:

Endoscopy with Biopsy: For patients with Barrett's esophagus, the ASGE and ACG recommend surveillance every 3 to 5 years depending on the presence and degree of dysplasia.

Cytosponge (Emerging Non-Invasive Tool): A novel screening tool being evaluated for 5-year intervals in high-risk meat consumers.

Budgetary Perspective: Early detection allows for endoscopic mucosal resection (EMR), which avoids the high morbidity and cost (often >$100,000) of esophagectomy and multi-modal chemo-radiotherapy.

Summary of Precision Screening Imperatives

Gastroenterological societies increasingly recognize that "one-size-fits-all" age-based screening may miss individuals with high dietary risk. We propose that a lifetime history of high processed meat consumption (>100g daily for 10+ years) should trigger screening 5 to 10 years earlier than standard guidelines, effectively treating high dietary intake as a risk factor equivalent to a minor family history.

Macro-Economic Impact of National Screening Programs

The financial burden of gastrointestinal malignancies on national budgets is one of the most significant challenges for modern healthcare systems. However, a comprehensive analysis reveals a stark contrast between the "price of prevention" and the "cost of neglect."

Return on Investment (ROI) of Mass CRC Screening

While the implementation of a national screening program—such as Poland's *Program Badań Przesiewowych* or the UK's bowel cancer screening—requires substantial initial investment, it represents a high-return economic strategy.

National Budget Translation: A shift from reactive treatment to proactive screening drastically reduces long-term oncology expenditures. Treating a Stage IV colorectal cancer patient can cost a national health fund over $150,000 annually in biological agents and palliative care. In contrast, the systemic cost of identifying and removing a precancerous adenoma via colonoscopy (even at a macro-scale of thousands of procedures) is a fraction of that expenditure.

Cost-Efficiency of Fit Programs: For emerging economies, the adoption of annual FIT (Fecal Immunochemical Test) programs offers a highly cost-efficient budget model. By spending approximately $20–$50 per citizen, states can avoid the astronomical surgical and chemotherapy costs associated with late-stage diagnosis.

The Macro-Economic Benefit of Early Detection in Gastric and Esophageal Cancer

In regions where high consumption of smoked and cured meats drives upper GI malignancies, targeted surveillance (e.g., every 3 years for high-risk cohorts) protects the national human capital.

Preventing Productivity Loss: Gastric and esophageal cancers have high mortality rates. By utilizing Gastroscopy and Cytosponge technologies at a national level, governments prevent the permanent removal of citizens from the workforce. The "Social Return on Investment" is measured in the continued contribution of these individuals to the national GDP and the avoidance of disability pension payments.

Budgetary Comparison: Prevention vs. Treatment

A rigorous economic analysis demonstrates that mass screening programs are budget-neutral or budget-positive over a 10-year horizon.

Stage I Diagnosis (via Screening): 5-year survival >90%; treatment cost is low (one-time surgery).

Stage IV Diagnosis (via Symptomatic Detection): 5-year survival <15%; treatment cost is astronomical (chronic biological therapy, repeated hospitalizations). By allocating funds to modalities, national health departments effectively "buy" years of productive life for the population while protecting the fiscal integrity of the healthcare system.

Precision Budgeting for High-Meat-Consumption Regions

Governments in regions with high industrial meat consumption (e.g., Central Europe, US Midwest) must recognize that their oncology budgets are being drained by modifiable dietary risks. We advocate for a "Prevention Tax" model, where revenues from ultra-processed food taxes are directly reinvested into the national screening infrastructure, ensuring that the cost of screening is covered by the industry that contributes to the cancer burden.

4. Discussion

The "NutriRECS" Controversy and Evidence Certainty

The evidence linking processed meat to GI cancers is classified as "convincing" according to the WCRF and IARC. However, the 2019 NutriRECS publication sparked debate by suggesting individual risk reduction was minimal (Han et al., 2019). This review contends that such an "individual-centric" view is flawed. Extrapolated to a global population, even a small risk reduction prevents millions of cases and saves billions in healthcare spending.

The Early-Onset CRC (EOCRC) Crisis and the Birth Cohort Effect

EOCRC is the most pressing challenge in modern oncology. Individuals born after 1980 have been exposed to industrial nitrites throughout their developmental period, accelerating the mutation rate in the colonic epithelium (Ungvari et al., 2025). This requires a reconsideration of age-based screening models in favor of exposure-based triggers.

Global Dietary Recommendations and Substitution Strategies

The synthesis of molecular and epidemiological evidence has led to a major alignment in international dietary guidelines. The World Cancer Research Fund (WCRF) and the American Institute for Cancer Research (AICR) provide the most authoritative benchmarks, recommending that individuals should eat little, if any, processed meat. For red meat (beef, pork, lamb), the recommendation is limited to no more than three portions per week, equivalent to approximately 350–500g of cooked weight. This threshold is based on evidence that risk increases significantly above these levels due to the cumulative load of heme iron and metabolic byproducts (Vieira et al., 2017).

Furthermore, public health strategies are shifting toward promoting "nutritional substitution." Meta-analyses of prospective cohort studies indicate that replacing a single daily serving (50g) of processed meat with legumes, nuts, whole grains, or moderate amounts of poultry and fish is associated with a 10-15% reduction in CRC risk. These findings suggest that GI cancer prevention is not merely about exclusion but about a fundamental restructuring of the protein matrix in the human diet to favor anti-inflammatory and fiber-rich substrates.

Industrial Reform and Policy Regulation

The formation of carcinogenic compounds is a result of industrial processing. The burden of prevention should not rest solely on the consumer. We advocate for:

Industry Reform: Reducing nitrite levels and adopting smoke-free flavoring.

Transparent Labeling: Clear labels identifying processed meat as a Group 1 carcinogen (Bouvard et al., 2015).

Subsidization: Making plant-based proteins more economically competitive.

Future Directions: From Microbiomics to Liquid Biopsies

The future of GI oncology lies in the integration of dietary data with advanced diagnostic tools. Emerging research into the gut microbiota (e.g., detection of *pks+ E. coli*) could provide a "biological screening" tool. Liquid biopsies (ctDNA) may eventually allow for non-invasive monitoring of individuals with high dietary risk profiles.

Limitations of the Review

This narrative review is limited by the observational nature of dietary studies, susceptible to recall bias. However, the identification of specific mutational signatures provides the necessary biological "proof of concept" to support epidemiological associations.

Conclusions

The synthesis of molecular and epidemiological data established over the last decade provides substantiated evidence of a direct association between the consumption of processed meat and the increased risk of gastrointestinal malignancies. The classification of these products by the IARC as Group 1 carcinogens is underpinned by mechanistic findings, including the identification of specific mutational signatures in the *APC*, *KRAS*, and *TP53* genes, primarily induced by N-nitroso compounds and polycyclic aromatic hydrocarbons. These biological markers act as a modifiable environmental footprint, reflecting the impact of industrial food processing on the human genome. Beyond individual biological risk, the socioeconomic analysis indicates that the management of gastrointestinal neoplasms represents a significant and escalating demand on national healthcare budgets. The high direct costs associated with late-stage oncological care—including advanced surgical resections and high-cost systemic therapies—stand in stark contrast to the fiscal efficiency of early detection and primary prevention. Macroeconomic data suggest that the rising incidence of

early-onset colorectal cancer results in substantial human capital depreciation, as individuals are removed from the labor force during their most productive years, thereby impacting national GDP. Consequently, transitioning from a reactive treatment model to a proactive, precision-based screening infrastructure is a necessary strategy for ensuring the long-term sustainability of public health systems. Mitigating the burden of diet-induced malignancies requires a systemic approach that integrates industrial regulation, transparent public labeling, and risk-adapted screening protocols based on dietary exposure history. By prioritizing preventative intervention over symptomatic care, global health authorities can significantly reduce the incidence of preventable gastrointestinal cancers while securing the economic stability of future healthcare delivery.

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