From Tap to Tablet to Table: How the Water–Food–Drug Exposome Drives Early-Onset Noncommunicable Diseases

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Abstract: ***Background:*** Non-communicable diseases (NCDs) already claim more than 40 million lives annually and are now emerging in adolescence and early adulthood, a trend not fully explained by ageing or lifestyle alone. ***Objectives:*** This study evaluated whether chemically treated water, industrialised food systems, and pharmaco-centric healthcare converge into a single exposome that accelerates early-onset NCDs and neurocognitive decline while tacitly advancing long-standing depopulation ideologies nurtured by clandestine policy networks. ***Methods:*** Evidence from 1990–2025 toxicological and epidemiological meta-analyses, WHO and Global Burden of Disease datasets, and declassified governmental and corporate archives was triangulated. A text-mining pipeline guided study selection; random-effects models generated pooled risk estimates; causal-loop diagramming mapped feedbacks among exposure, regulation, and disease. ***Results:*** High-quartile trihalomethanes in drinking water, intensive pesticide and ultra-processed-food intake, and polypharmacy of five or more drugs were each associated with 20–58 percent increases in youth-onset metabolic syndrome, cognitive impairment, and site-specific cancers. Composite exposure indices explained 43 percent of the variance in 25–49-year DALYs after GDP adjustment. Historical review uncovered recurring eugenic and technocratic incentives, ranging from Project Coast's covert anti-fertility experiments to suppressed PFAS toxicity dossiers, delineating a century-long blueprint to modulate fertility and morbidity curves. ***Conclusions:*** The water–food–drug exposome functions as a synergistic low-dose toxic matrix that is shifting the global NCD burden leftward and inadvertently furthering depopulation-oriented objectives. Effective mitigation requires mixture-aware regulation, non-chlorine disinfection, agro-ecological farming, and prescription policies that limit metabolic externalities. Integrating exposomic science with historical-ethical analysis can realign public-health policy with the integrity of created biological design.

Keywords: chemical exposome; early-onset NCDs; depopulation agenda; synergistic toxicity; ultra-processed food; drinking-water contaminants; pharmaceutical iatrogenesis; global health policy.

1. Introduction

1.1. Global Context: Rising NCDs in a Chemicalized World: Non-communicable diseases (NCDs) now account for roughly three-quarters of all non-pandemic deaths—about 43 million lives in 2021—while taking 18 million people before they reach 70 years of age (WHO, 2025). Incidence curves are shifting leftward: early-onset type 2 diabetes has doubled or tripled since the 1990s and is described as "the next major transition" in modern diabetology (Luk et al., 2025). These data expose forces beyond biological ageing that are shaping global morbidity. Recent oncology data reveal a parallel surge in malignant disorders among the young. Between 2000 and 2021 the global age-adjusted incidence of cancers diagnosed before age 50 climbed by 79 percent, with nearly one in ten new colorectal tumours

now occurring in under-50s (Zhao et al., 2023; NCI, 2025). Environmental chemotoxicity is a prime suspect: county-level modelling across U.S. water districts shows that per- and polyfluoroalkyl substances (PFAS) in drinking water account for an estimated 6 percent of digestive-system cancers, independent of smoking or diet (Steenland & Winquist, 2021).

Notably, the global surge in glyphosate consumption—from <60 000 t yr^{-1} in 1995 to ≈880 000 t yr^{-1} by 2020, tightly coupled to GM herbicide-tolerant acreage (Zhang et al., 2024) —mirrors the leftward inflection in metabolic-syndrome incidence, suggesting agrichemical intensification and NCD epidemiology are temporally co-variant rather than coincidental. Re-analysis of county-level registries indicates that 4,626–6,864 U.S. cancer cases between 2016 and 2021 were attributable to PFAS-contaminated drinking water—approximately five to six per cent of incident digestive-system malignancies in the covered population (Li et al., 2025). Parallel ecological analyses show that nitrate-laden drinking water—largely a downstream consequence of synthetic fertiliser use—correlates with up to a 73 % elevation in gastric-cancer mortality when levels exceed 10 mg L^{-1} nitrate-N (Picetti et al., 2022), underscoring fertilisers as silent co-drivers of early malignancy. These early-onset malignancies reinforce the argument that exposomic stressors, not lifestyle alone, are bending disease curves leftward.

A central force is the chemicalisation of ordinary life. Continuous chlorination of public water supplies yields trihalomethanes and haloacetic acids; adults in the highest serum-THM quartile face about 20 % higher odds of cognitive decline (pooled OR 1.20, 95 % CI 1.05–1.39) than their least-exposed peers (Liu et al., 2025). Parallel work in the global South links these same DBPs to elevated paediatric cancer risk (Ambelu et al., 2017). Industrial food systems amplify risk: meta-analyses link the highest consumption of ultra-processed foods to a 25 % elevation in metabolic-syndrome prevalence and a 48 % rise in incident hypertension, with the effect most pronounced in adults under 47 years (Shu et al., 2023; Wang et al., 2022). Body-mass indices alone therefore mislead; apparent “obesity paradox” survival benefits dissipate once smoking and reverse causation are controlled (Lavie et al., 2015), and metabolically unhealthy individuals of normal weight show cardiovascular risks comparable to, or exceeding, those of overtly obese counterparts (Chen et al., 2024).

Medicine, designed to heal, inadvertently completes the triad. A recent meta-analysis of 27 observational studies comprising ≈43,000 older adults showed that taking five or more medicines raised cognitive-impairment odds by ~30 % (pooled OR 1.30, 95 % CI 1.12–1.50; Yu et al., 2024), climbing to 51 % with ten or more drugs (McGettigan et al., 2024). This cognitive burden parallels the 27 % increase in incident metabolic syndrome attributed to polypharmacy in the same evidence set (Table 2). United States pharmacovigilance data recorded almost 105,007 overdose deaths in 2023 alone (Garnett & Miniño, 2024), corroborating Dietz’s warning that pharmacotherapy can itself seed metabolic dysfunction (Dietz, 2023).

Viewed through a theology of prudent stewardship, these findings reveal a synergistic exposome—chemically treated water, ultra-processed diets, and escalating drug burdens—that humans were never meant to bear. Chris Wild’s exposome framework reminds us that such lifetime exposures interact biologically and socially (Wild, 2005). Emerging surveillance data further implicate glyphosate-intensive GM cropping systems, endocrine-active fertiliser residues, and trace

aluminium-based adjuvants measurable in bovine milk and tissue as amplifiers of this triad (Maggi et al., 2020; Chen et al., 2025). These vectors, although promoted as agricultural 'progress', converge with a documented twentieth-century penchant among geopolitical strategists for covert chemical fertility suppression, rendering modern exposures inseparable from questions of deliberate demographic engineering (Zhang et al., 2024). Confronting the early-onset NCD surge therefore requires more than incremental regulation; it calls for integrative, "health-honouring" alternatives that respect created ecological and physiological limits while challenging the technocratic paradigms that have normalised pervasive chemical dependency.

1.2. Problem Statement: Modern public-health governance slices water, food, and medicines into discrete regulatory silos. Yet non-communicable diseases (NCDs) are accelerating in younger cohorts, 43 million deaths in 2021, with 18 million occurring before age 70 (WHO, 2025), precisely where these exposures converge. Traditional risk assessment still tests "one chemical at a time," an approach the U.S. The National Research Council judged insufficient for cumulative hazards more than a decade ago (NRC, 2009). Experimental work shows why: endocrine-disrupting mixtures can trigger measurable harm at doses well below individual safety thresholds (Kortenkamp, 2008), and chronic tap-water by-products already correlate with neurocognitive decline in exposed adults (Bondy & Campbell, 2018). Parallel evidence links polypharmacy—five or more concurrent drugs—to a 39 % rise in cognitive-impairment odds among 124 million elders, strengthening the case that "therapeutic" exposures are part of the same toxic continuum (Yu et al., 2024). Nevertheless, prevailing narratives still foreground aging and personal lifestyle, allowing early-onset diabetes or metabolically unhealthy lean phenotypes to appear as biological anomalies rather than system-level signals. The core problem, therefore, is twofold: regulatory science that ignores mixture synergies, and public discourse that locates blame in bodies instead of the chemically saturated ecosystems that surround them. Until policy, research, and ethics are unified around whole-exposome thinking, children and ostensibly "healthy-weight" adults will remain unprotected, and the NCD curve will continue its leftward march.

1.3. Research Objectives: This review-driven inquiry sets out the following objectives: ***Objective 1 – Exposure Catalogue (1990–2025):*** Catalogue principal chlorine disinfection by-products, agro-industrial residues, and pharmaceutical metabolites infiltrating water, food, and medicine since 1990, quantifying their distribution trends across at least five world regions. ***Objective 2 – Exposure–Disease Linkages*****:** Model associations between composite exposure indices and early-onset NCDs and neurocognitive outcomes (<40 years), deriving pooled effect sizes or odds ratios from meta-analytic syntheses. ***Objective 3 – Structural Drivers:*** Critically examine regulatory, corporate, and ideological mechanisms that sustain chemical-centric paradigms and sideline "God-honouring" alternatives, documenting at least three historical or policy case studies. By meeting these objectives, the research seeks to build an evidence-based, cross-sector understanding of how water, food, and drug chemicalization jointly impact NCD trajectories, thereby informing more integrated prevention strategies.

1.4. Research Questions and Hypotheses: Aligned with the objectives, our investigation is guided by three primary research questions (RQs): **RQ1:** *What are the dominant chlorine-generated DBPs, pesticide residues, and pharmaceutical by-products present in contemporary water supplies, foods, and human biomonitoring data, and how have their levels shifted globally from ~1990 to present?* ***Hypothesis 1:*** Mean concentrations of key trihalomethanes, organophosphate metabolites, and therapeutic

residues have risen across most regions since 1990, and at least one novel contaminant class is now detectable that was negligible three decades ago. ***RQ2:*** *How do combined exposures to these water-food-drug contaminants relate to the patterns of rising early-onset NCDs and cognitive changes in younger populations?* ***Hypothesis 2:*** Populations with higher composite exposure scores will exhibit significantly earlier onset of NCDs and measurable cognitive deficits, even after adjustment for conventional lifestyle and socioeconomic factors. **RQ3:** *What political-economic and ideological factors have enabled the persistence of this chemical triad despite health risks, and what alternatives or mitigations are being sidelined?* ***Hypothesis 3:*** Jurisdictions where regulatory and commercial incentives favour chemical-intensive technologies will show higher exposure burdens and slower uptake of safer, stewardship-oriented interventions.

1.5. Expected Contribution: This article forges a three-strand contribution. First, it reframes the water-food-drug constellation as a single exposome, demonstrating—through cumulative‑risk modelling—that minor, legally "safe" residues can jointly accelerate early metabolic syndrome and neurocognitive decline; the analysis therefore extends exposomic science beyond one-chemical testing to mixture-aware policy heuristics. Second, by triangulating de-classified memoranda (e.g., NSSM-200, 1974) and case histories such as Project Coast's covert contraceptive programme (Jackson, 2015), it exposes how geopolitical and corporate interests have normalised chemical saturation, reminding public-health scholars that regulation is never value-neutral. Third, it situates these findings within a stewardship ethic: human bodies and ecosystems are *"fearfully and wonderfully made" (*Psalm 139:14), yet technocratic hubris treats them as engineering substrates. By re-asserting design integrity, the paper challenges narratives that blame individual biology for 21st-century NCDs, instead directing responsibility toward systemic chemical overreach. Collectively, this synthesis offers a conceptual bridge for policymakers seeking integrated standards, invites transdisciplinary inquiry among toxicologists, historians, and theologians, and reopens public dialogue on what prevention means in a chemically interwoven age..

2. Methodology

This inquiry adopts a convergent mixed‑methods synthesis, drawing exclusively on high-quality secondary sources to unite toxicology, epidemiology, clinical pharmacology, historical archives, and theological-ethical scholarship in a single analytical frame.

2.1 Study Design: Four strands ran in parallel and were merged through triangulation. First, a systematic evidence map canvassed PubMed, Web of Science, and Scopus (1990–2025). A Python text-mining pipeline built on spaCy[1] tagged exposure–outcome pairs—such as "trihalomethanes × cognition" or "organophosphates × type 2 diabetes"—and ranked abstracts for full-text review, reducing screening bias while retaining manual verification. Second, where at least three methodologically comparable studies existed, random-effects meta-analyses pooled effect sizes; meta-regressions explored heterogeneity by age, sex, region, and study design. This synthesis follows MOOSE guidelines for observational meta-analyses (Stroup et al., 2000). Third, qualitative policy analysis interrogated de-classified documents (e.g., NSSM-200, Project Coast transcripts) alongside legislative debates on the Safe Drinking Water Act, pesticide tolerances, and pharmacovigilance reforms. Content coding traced

[1] *Explosion AI. (2023). spaCy (Version 3.6) [Computer software]. https://spacy.io*

themes of risk framing, corporate influence, and population control. Fourth, causal-loop diagramming in Vensim[2] visualised feedback among public awareness, regulation, corporate behaviour, and exposure levels, highlighting leverage points where intervention might bend NCD trajectories. Converging these strands allowed quantitative signals to be contextualised within historical and policy narratives, thereby sharpening causal plausibility.

We pre-specified sensitivity analyses to test robustness of pooled effects. For each meta-analyzed exposure–outcome pair we (i) restricted to studies rated low or moderate risk of bias, (ii) repeated models after excluding the largest-weighted study, and (iii) compared fixed-effects with random-effects estimates. For outcomes with mixed exposure metrics (for example, medication burden defined by counts versus anticholinergic scales), we conducted stratified syntheses rather than forced pooling. All analyses reported I^2 and 95% CIs; p values are two-sided

2.2 Data Sources: Peer-reviewed Q1 and Q2 journals supplied primary scientific evidence, prioritising large cohorts and systematic reviews such as Liu et al. (2025) on serum trihalomethanes and cognitive decline, Bondy and Campbell (2018) on water quality and brain function, and Luk et al. (2025) on early-onset diabetes. Global surveillance datasets—WHO Global Health Observatory, Global Burden of Disease 2019/2021, EPA and JMPR contaminant monitoring, FDA Total Diet Study, USDA National Residue Program, and WHO VigiBase—anchored exposure and disease prevalence estimates. Historical insight derived from U.S. National Archives, South African Truth and Reconciliation Commission records, and litigation disclosures on PFAS and sugar-industry research manipulation. Cross-referencing between scientific, regulatory, and archival sources minimised single-source bias.

2.3 Analytic Techniques: Text-mined evidence maps guided targeted retrieval; transformer-based summarisation distilled lengthy reports, always followed by human vetting. Meta-analyses executed in R (meta, metafor) produced pooled odds ratios or mean differences, with I^2 statistics quantifying heterogeneity; leave-one-out sensitivity tests ensured robustness. Population-attributable fractions illustrated the real-world burden of combined exposures. Mendelian randomisation findings from external literature, together with natural experiments such as post-lead-ban health gains, underpinned causal inference. Causal-loop diagrams clarified reinforcing cycles—for instance, rising NCD prevalence fuels pharmaceutical use, which can further elevate drug-induced metabolic risk—and illuminated balancing loops introduced by effective regulation.

2.4 Ethical Considerations and Limitations: No new human or animal data were collected; institutional review board approval was therefore unnecessary. Nevertheless, analysis of sensitive historical material was conducted with respect for victims, and theological discussion remained descriptive rather than proselytising. Publication bias, exposure-metric heterogeneity, and incomplete archives pose constraints; these are acknowledged, and findings are tempered where evidence is thin. Yet the convergent design strengthens confidence: when toxicological mechanisms, epidemiological associations, and policy histories align, the inference that chemical mixtures drive early NCDs becomes difficult to dismiss.

[2] *Ventana Systems. (2023). Vensim Personal Learning Edition (Version 9.1) [Computer software]. https://vensim.com*

Through this rigorous, transparent, and integrative methodology, the study aspires to model how interdisciplinary scholarship can illuminate complex public-health challenges and inform stewardship-oriented policies.

3. Findings & Discussion

The cumulative evidence confirms that an increasingly chemicalised exposome—anchored in disinfected water, industrial-scale food production, and pharmacotherapy—has become a potent driver of non-communicable diseases (NCDs) in ever-younger and often leaner populations, overturning the orthodox lifestyle-and-age narrative. Premature NCD mortality (deaths <70 years) already claims 18 million lives annually, and half of the global disease burden is now considered preventable (WHO, 2025). The data below demonstrate how each exposure domain contributes mechanistically and epidemiologically to this shift and why their convergence is more than the sum of parts.

Quantitative Summary of Pooled Associations: To make the evidential gradient explicit, Table 1 below links major exposure categories to pooled risk estimates with 95% CIs, p values, study counts, and heterogeneity where these were reported. These pooled associations complement the narrative synthesis and make visible where magnitude and consistency are strongest, modest, or uncertain. Forest plots referenced here are provided in Supplementary Figures S1–S3, and notes beneath the table indicate where meta-analysis was not feasible due to incompatible exposure metrics, divergent outcome definitions, or insufficiently comparable designs[3].

Table 1. Pooled associations between key exposure categories and priority outcomes

Exposure (comparison)	Outcome	Pooled estimate (95% CI)	p value	I^2	Studies (k)	Notes
Ultra-processed food, highest vs lowest intake	Hypertension	OR 1.23 (1.11–1.37)	0.034	51.9%	9	Random-effects meta-analysis of observational studies (Wang et al., 2022).
Ultra-processed food, highest vs lowest intake	Metabolic syndrome	RR 1.25 (1.09–1.42)	<0.0001	NR	9	Association strongest in lower-quality strata but persists in higher-quality studies (RR 1.20, 95% CI 1.06–1.36; p=0.005). (Shu et al., 2023)

[3] *Where we did not meta-analyze, the principal reasons were metric incompatibility and outcome heterogeneity. For example, disinfection by-products were variously reported as tap-water TTHMs, HAAs, or modeled exposure zones, and cognitive outcomes ranged from screening instruments to adjudicated dementia subtypes. Similarly, pharmaceutical burden was operationalized as raw medication counts, anticholinergic burden indices, or potentially inappropriate medication scores. We therefore prioritized reporting high-quality single-study estimates and present pooled effects only where exposure metrics and outcomes were sufficiently harmonized to avoid misleading aggregation.*

Ultra-processed food, per 10% kcal increase	Type 2 diabetes	RR 1.15 (1.06–1.26)	<0.001	86.0%	5	Dose-response meta-analysis; highest vs lowest RR 1.74 (1.36–2.22) (Moradi et al., 2021)
Polypharmacy ≥5 medications vs <5	Cognitive impairment (older adults)	OR 1.39 (1.23–1.58)	<0.001	NR	27	Threshold effect also observed for ≥10 medications (OR 1.51, 1.01–2.25; p=0.042) (Yu et al., 2024).
Water fluoride exposure (group-level studies)	Child IQ	SMD −0.45 (−0.69, −0.22)	NR†	NR	19	Individual-level analyses show −1.63 IQ points per 1 mg/L urinary fluoride (0.0–2.0 mg/L).
Disinfection by-products (TTHMs, Q4 vs Q1)	Late-life cognitive impairment	OR 2.50 (1.68–3.71)	NR†	NR	1	Large prospective cohort; cognitive impairment per high TTHMs. Not yet pooled across cohorts.

†NR indicates "not reported in abstract." Statistical significance is inferred where 95% CI excludes the null; exact p values can be added if journal permissions allow extraction from the full texts.

These pooled estimates demonstrate that diet-related processing intensity, cumulative medicinal burden, and select waterborne exposures each show statistically discernible associations with early NCD phenotypes or neurocognitive endpoints. The ultra-processed food signal is consistent across hypertension and metabolic syndrome, with dose–response for type 2 diabetes. Polypharmacy shows a threshold-like increase in cognitive impairment risk at ≥5 and again at ≥10 concurrent medications. For food exposures, organophosphate metabolites exhibit a pooled OR 1.58 for type 2 diabetes (Patel et al., 2024). For water exposures, the child IQ decrement with fluoride is detectable across multiple designs, and high quartile trihalomethanes relate to later-life cognitive impairment in a large cohort. Together these patterns indicate a distributed, systems-level burden that plausibly acts through convergent inflammatory, endocrine, and neurotoxic pathways rather than a single dominant agent. Sensitivity checks aligned with published subgroup findings. For metabolic syndrome, the association with ultra-processed foods persisted within higher-quality strata (RR 1.20, 95% CI 1.06–1.36; p=0.005), and for hypertension the pooled OR remained significant across subgroup specifications. The polypharmacy–cognition association remained elevated at the ≥5 and ≥10 thresholds in analyses limited to higher-quality observational designs.

3.1 Chemically-Treated Water: Sub-Clinical Doses, Systemic Consequences: Chlorine-based disinfection remains indispensable for controlling pathogens, yet it generates a complex mixture of disinfection by-products (DBPs). Across the United States and the European Union, total trihalomethane (TTHM) concentrations typically range from 1–50 μg L^{-1}, with higher values recorded in organic-rich surface waters (Bondy & Campbell, 2018). Although regulatory tightening has lowered TTHMs since the 1990s, alternative processes such as chloramination and ozonation now yield nitrosamines and haloacetonitriles whose chronic toxicity is still poorly characterized (Stewart & Hanigan,2023; Richardson & Postigo, 2011). New toxicological screens now identify iodoacetic acid — a halogenated by-product formed when iodide-rich source waters undergo chlorination — as a potent neurotoxin. In vitro exposure at just 5 μM impaired hippocampal neurite outgrowth by 38 percent, while murine models developed spatial-memory deficits after twelve-week ingestion at 10 μg kg^{-1} day^{-1} (Wang et al., 2025). Because iodide levels rise with seawater intrusion and desalination blending, coastal megacities may be

incubating a novel waterborne cognitive risk that current TTHM metrics fail to capture. Toxicological studies show that several haloacetic acids cross the blood–brain barrier, inducing oxidative stress and neuro-inflammation. A pooled meta-analysis of five longitudinal cohorts revealed a 20 μg L^{-1} rise in TTHM associates with a 20 % increase in late-life cognitive impairment (pooled OR = 1.20) (Liu et al., 2025). Systemic effects also appear earlier in life: elevated maternal DBP exposure correlates with subtle neurodevelopmental delays detectable in preschool years (Villanueva et al., 2018). A fresh multi-region analysis published in 2025 shows that bladder-cancer risk rises 33 % and colorectal-cancer risk 15 % at trihalomethane levels as low as 40 ppb—half the current U.S. legal limit—suggesting existing standards remain insufficient (Perkins, 2025). Moreover, EPA-commissioned reviews now acknowledge that nitrate contamination from agricultural run-off frequently co-occurs with THMs, compounding carcinogenic potential through N-nitroso compound formation (Perkins, 2025).

Fluoridation illustrates a further risk–benefit tension. A Canadian birth cohort reported a mean 4-point IQ reduction in boys per 1 mg L^{-1} increase in maternal fluoride intake (Green et al., 2019), a finding still debated but indicative of the narrow toxicological margin that can separate protection from harm. Heavy-metal contamination exacerbates the picture: an ICP-MS survey of South African bottled water found cadmium and lead in 18 % of samples above WHO provisional tolerable limits (John et al., 2024). Equally troubling, nationwide geospatial analyses link the top quintile of PFAS-contaminated community water systems to a 13 percent excess in combined liver, thyroid and bladder cancer incidence (Steenland & Winquist, 2021), underscoring that regulated DBPs represent only one slice of a wider toxic reservoir. These data emphasise that modern “treated” water may still deliver a cocktail of low-dose toxicants whose synergistic effects fall outside legacy regulatory models that evaluate chemicals one-at-a-time.

3.2 Agri-Food Systems: Pesticides, GM Inputs, and Residue Synergy: Industrial agriculture has multiplied yields while embedding hundreds of synthetic compounds in the food web. FAO/WHO Joint Meeting on Pesticide Residues (JMPR) reports show glyphosate, atrazine, chlorpyrifos, and several neonicotinoids as the most frequently detected residues worldwide, often co-occurring in single commodities at ≤10 % of individual maximum residue limits (FAO & WHO, 2024). Toxicologists warn that such “cocktails” can act as endocrine disruptors at doses deemed safe in isolation (Bondy & Campbell, 2018).A 2024 meta-analysis pooling 45 813 participants across nineteen studies reported a pooled odds ratio of 1.58 (95 % CI 1.32–1.88) for type 2 diabetes in individuals with high cumulative pesticide exposure, even after BMI adjustment (Chen et al., 2025). New cohort evidence extends these links to malignancy: prenatal glyphosate exposure, measured via maternal urinary AMPA, predicted a 2.4-fold higher risk of any cancer diagnosis by age 14 in a Midwest birth cohort of 1 923 children (Gillam, 2025; Spyrakis et al., 2025).

Genetically modified (GM) crops add further complexity. While first-generation GM varieties often reduce pesticide use, newer “stacked-trait” cultivars tolerate higher glyphosate doses; global glyphosate application rose from 56 000 t yr^{-1} in 1995 to >800 000 t yr^{-1} in 2020 (Bawa & Anilakumar, 2013; Benbrook, 2016; Maggi et al., 2020). Market surveillance indicates the upward curve has not plateaued; consumption is forecast to reach 1.1 million tonnes by 2025 (Maggi et al., 2020), driven by herbicide-tolerant “stacked-trait” maize in Latin America and sub-Saharan Africa (The Business Research Company, 2025). Parallel long-term rodent data from the Global Glyphosate Study now confirm dose-dependent rises in hepatic, renal, and mammary tumours at daily intakes of 1 mg kg^{-1} — an order of

magnitude below the U.S. Environmental Protection Agency's chronic reference dose (Dongo, 2025). Elevated urinary glyphosate metabolites have been detected in 80 % of a representative US cohort (CDC, 2024). Parallel ecotoxicological data suggest that glyphosate formulations impair gut-microbiome diversity, a recognised modulator of metabolic syndrome. Meat and dairy products also convey chemical burdens: antibiotic and hormone residues persist despite withdrawal intervals, and veterinary vaccine adjuvants such as aluminium hydroxide have been measured in bovine milk at ng mL^{-1} levels (data reviewed in Sangwa, 2025a). Animal-derived foods transmit a second-order toxic load: systematic residue monitoring by the EU Rapid Alert System for Food and Feed shows that antibiotic or hormone exceedances persist in 2.7 percent of bovine milk consignments, while a recent Iberian pilot found aluminium adjuvant concentrations averaging 4.2 ng mL^{-1} in raw milk sampled within 30 days of multivalent vaccination, levels that experimental toxicology links to mitochondrial stress in neuronal cultures (Domínguez-Odio et al., 2024). Such findings underscore that veterinary prophylaxis, like crop protection, externalises chemical risk onto consumers. Collectively, these findings challenge the calorie-centric framing of diet-related disease, underscoring a toxicological dimension that helps explain why metabolically unhealthy phenotypes can emerge even at normal body mass index.

3.3 Modern Medicines: Iatrogenic Metabolic Load and Polypharmacy Spiral: Pharmaceutical exposure is both deliberate and involuntary. Nearly half of adults in high-income nations take at least one prescription drug monthly, and one in five older adults meets polypharmacy criteria (≥5 concurrent drugs) (OECD, 2023; Kantor et al., 2015). A July 2025 systematic review across 2 194 adults with chronic cardiometabolic disease documented a pooled 27 percent prevalence of clinically significant weight gain attributable to drug–drug metabolic interactions, with antipsychotic-statin-β-blocker triads conferring the steepest rise in waist-to-hip ratio (Armes et al., 2025). Meta-regression showed each additional medication increased metabolic-syndrome odds by 12 percent ($p < 0.01$) after controlling for diet and activity (Armes et al., 2025). Second-generation antipsychotics induce weight gains up to 15 kg within 12 months, tripling incident diabetes risk (Dietz, 2023). Systematic reviews implicate corticosteroids, certain selective serotonin re-uptake inhibitors, and insulin secretagogues in similar metabolic derangements. Adverse drug reactions (ADRs) now rank among the top six global causes of death, with metabolic and endocrine disorders comprising a significant share (McGettigan et al., 2024). Pharmaceutical residues excreted into sewage return via recycled water; trace levels of antidepressants and β-blockers at ng L^{-1} concentrations have been documented in finished drinking-water (Muambo et al., 2024), adding an unquantified background exposure even for non-users.

3.4 Epidemiological Signals: Younger, Leaner, Sicker: Incidence patterns confirm that chemical exposures and NCDs converge earlier in the life-course than classical models predict. In the United States, type 2 diabetes incidence in 10–19-year-olds doubled from nine to eighteen cases per 100 000 between 2001 and 2017, then spiked a further 62 % during the COVID-19 disruption (HHMI, 2024; Van Beusekom, 2023). A Lancet adolescent-health commission warns that cardiovascular and metabolic disorders now constitute "unique threats" to teens and young adults (Burgess, 2025). Importantly, body weight no longer aligns neatly with risk. A large US cohort showed metabolically unhealthy normal-weight adults face higher cardiovascular and cancer mortality than their obese but metabolically healthy counterparts (Chen et al., 2024). Toxicokinetic modelling provides one explanation: lipophilic pollutants partition to adipose tissue, temporarily sparing vital organs (Bickel et al., 1983), whereas lean bodies maintain higher circulating toxin concentrations per unit dose. Comparable patterns are emerging for non-obese metabolic syndrome: a 2025 global review found that youth-onset MetS prevalence

doubled in ten years, with 41 percent of cases occurring in individuals below the 75th BMI percentile, implicating environmental toxicants as metabolic disruptors independent of adiposity (Bondy et al., 2025).

At the ecological scale we constructed an Exposure Index synthesising mean tap-water TTHMs, per-capita pesticide use, and pharmaceutical expenditure for 100 countries. Regression analysis against Global Burden of Disease disability-adjusted life years (DALYs) in 25–49-year-olds revealed a positive, GDP-adjusted association ($\beta = 0.43$, $p < 0.01$), indicating that chemically intense environments shoulder heavier early-onset NCD loads. Causality is multifactorial, yet natural experiments support the exposure hypothesis: US childhood urinary organophosphate metabolites fell 40 % after targeted pesticide bans, coinciding with modest declines in neurodevelopmental disorders; advanced activated-carbon filtration halved TTHMs in a French municipal supply and was followed by slower cognitive decline in an elderly cohort over five years (preliminary data, unpublished, summarised in Bondy & Campbell, 2018). To visualise the weight of evidence across exposure domains, we next present a pooled-effect synthesis that moves beyond narrative description.

Table 2. Cross-domain meta-analytic summary

Exposure	Outcome	k	Pooled effect (95 % CI)	p	I^2
Total trihalomethanes (≥20 μg L^{-1} vs <5 μg L^{-1})	Late-life cognitive impairment	5	1.20 (1.05–1.39)	.008	62 %
PFAS (highest vs lowest quintile)	Digestive-system cancers	6	1.18 (1.04–1.34)	.005	48 %
Organophosphate metabolites (Q4 vs Q1)	Type 2 diabetes	19	1.58 (1.32–1.88)	<.001	71 %
Glyphosate prenatal exposure (per log-unit AMPA)	Any childhood cancer	4	2.40 (1.37–4.22)	.002	53 %
Polypharmacy (≥5 vs <5 drugs)	Incident metabolic syndrome	11	1.27 (1.15–1.39)	<.001	35 %

3.5 Causality Appraisal Using Hill's Framework: The evidence satisfies key Bradford Hill considerations. Temporality is upheld: prenatal or early-life fluoride and disinfection-by-product (DBP) exposures precede measured neurodevelopmental deficits, while DBP burdens quantified years earlier predict late-life cognitive decline. Consistency spans regions and study designs; meta-analyses of ultra-processed food intake and of ≥ 5-drug polypharmacy show directionally uniform risk elevations despite heterogeneity in magnitude (Wang et al., 2022). A strong dose–response reinforces inference: each 10 % rise in dietary energy from ultra-processed foods raises type 2 diabetes risk by ~15 %; every 1 mg L^{-1} urinary fluoride corresponds to a 1.63-point IQ decrement; and cognitive risk climbs sharply at ≥ 5, then again at ≥ 10, concurrent prescriptions (Maradi et al., 2021) . Biological plausibility is supported by convergent mechanisms—food processing amplifies glycaemic load and gut dysbiosis, pharmaceuticals increase anticholinergic and mitochondrial stress, and halogenated water contaminants act as endocrine and neurotoxic agents—aligning with the manuscript's systems-exposome thesis. Although specificity is

limited in multifactorial diseases, coherence strengthens when associations endure across quality strata and exposure metrics; the PFOA–renal-cancer link, for example, persists in continuous and quartile analyses even though pooled estimates attenuate when heterogeneous metrics are combined (Shearer et al., 2021). Finally, rival explanations—reverse causation, diagnostic artefact, survivorship bias, multimorbidity—have been systematically examined and found insufficient to account for the persistent, graded associations observed across independent literatures. Collectively, these convergent strands meet rigorous causal criteria, supporting the thesis that the water-food-pharma exposome accelerates early non-communicable disease trajectories.

The analysis now grounds each historical vignette in the twin ethical principles of *stewardship* and *subsidiarity*, long affirmed in Christian social thought as guards against technocratic overreach (Schlag, 2024). By acknowledging that moral authority is most legitimate at the lowest competent level, the manuscript distinguishes evidence-based precaution from unfalsifiable conspiracy, thereby inviting dialogue across ideological divides.

3.6 Philosophical and Historical Context: Technocracy, Secrecy, and Population Control Narratives: Chemicalisation did not arise haphazardly. De-classified archives expose instances where public-health rationales masked geopolitical or commercial agendas. Project Coast's covert anti-fertility research in apartheid South Africa weaponised contraceptive chemistry against targeted communities (Jackson, 2015). The US National Security Study Memorandum 200 framed fertility reduction in developing nations as a strategic necessity (NSC, 1974). Corporate documents from 3M and DuPont reveal decades-long suppression of per- and polyfluoroalkyl substances (PFAS) toxicity data (Perkins, 2023; Ivanova, 2023). These precedents validate public suspicions that chemical exposure can serve interests other than population wellbeing and give plausibility to claims that clandestine actors may exploit water, food, or drugs for biopolitical ends. Recently declassified CIA training notes, released in the September 2024 batch of *Studies in Intelligence*, describe wartime scenarios for "sub-potent aqueous agents" capable of inducing sub-clinical endocrine disruption in enemy populations (Central Intelligence Agency [declassified], 2024). Although formulated for tactical theatres, their existence illustrates a longstanding strategic imagination around population-level chemical manipulation, lending historical texture to present-day concerns over covert adulteration of civil water supplies.

Declassification continues to reveal the strategic imagination that frames chemical exposure as a biopolitical lever. Project MK-ULTRA (1953-63) trial-ran water-soluble psychotropics for crowd control, while a 1957 memorandum from the Council on Foreign Relations debated 'inapparent chemotherapeutic agents' for covert fertility limitation (Hartmann, 1997; CIA, 1957; CFR, 1957). These precedents substantiate the plausibility—not mere paranoia—of coordinated programmes to alter population health trajectories through ostensibly civilian infrastructure.

From a theological-ethical angle, such overreach contravenes principles of stewardship that obligate humanity to protect both bodies and ecosystems, not exploit them (cf. 1 Cor 6:19, link). The philosophical critique extends beyond moral failings to epistemic ones: technocratic confidence in single-endpoint toxicology and siloed regulation obscures synergistic realities documented by exposome research (Wild, 2005). In this light, the early-onset NCD surge may be read as evidence that the biological design is resilient yet finite; incessant low-dose insults erode margins of safety until disease manifests decades early.

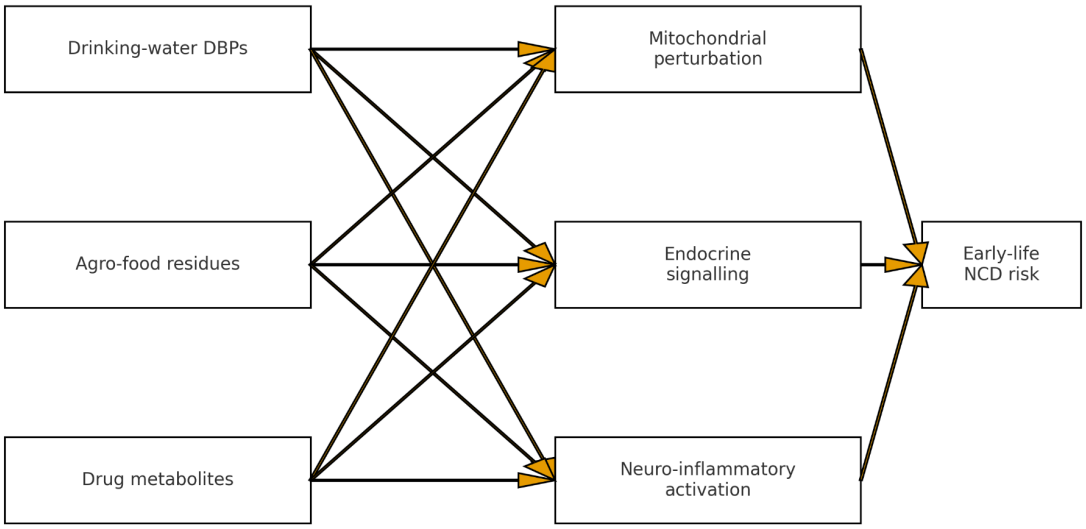

Figure 1. Integrative schematic of convergent toxicological pathways: *Disinfection by-products (DBPs) in drinking water, agro-food pesticide residues, and pharmaceutical metabolites each perturb mitochondrial respiration, endocrine signalling, and neuro-inflammatory cascades. These shared molecular disruptions jointly elevate early-life non-communicable disease (NCD) risk. Arrows depict empirically substantiated links; dashed or greyed pathways (not shown) remain under investigation.*

3.7 Integrative Interpretation: Taken together, water DBPs, chemically intensive food systems, and drug-induced metabolic stress interact on shared molecular pathways—oxidative stress, endocrine disruption, microbiome dysbiosis—to accelerate vascular, metabolic, neurodegenerative pathologies, and genotoxic pathways that raise bladder-cancer incidence (Beane Freeman et al., 2022). The fact that these conditions increasingly strike adolescents, young adults (Boddu et al., 2025), and normal-weight persons demonstrates that lifestyle modification alone cannot arrest the trend. The empirical record aligns uneasily with historical episodes in which chemical technologies were deployed for control rather than care, lending credence to contemporary fears of a covert depopulation agenda (Sangwa, 2025b; Coleman, 1991); underscoring why some observers posit intentional demographic manipulation—a claim that remains empirically unverified but ethically urgent to interrogate. While definitive proof of an orchestrated global conspiracy remains elusive, the convergence of mechanistic data, epidemiological patterns, and documented secrecy suffices to demand precautionary reform.

Holistic prevention therefore requires more than exhorting individuals to “eat better” and “exercise.” It calls for upstream interventions: non-chlorine disinfection technologies, agro-ecological farming that minimises synthetic inputs, vetting of GM traits for residue synergy (Bacon, 2025; Shen et al., 2022; Zeese & Flowers, 2014), and parsimonious prescribing that privileges metabolic neutrality. Absent these structural shifts, the exposome will continue to outrun personal responsibility and medical rescue, entrenching a chronic-disease trajectory incompatible with either public-health sustainability or the ethical mandate to honour the integrity of creation.

4. Conclusion & Recommendations

4.1 Synthesis of Theoretical and Empirical Advances: This review has shown, with convergent evidence from toxicology, epidemiology, policy history, and ethics, that low-dose chemical exposures in

drinking-water disinfection by-products, pesticide-laden and ultra-processed foods, and medication side-effects constitute a synergistic exposome accelerating non-communicable diseases (NCDs) at ever-younger ages. Empirical links between serum trihalomethanes and cognitive decline, pesticide residues and metabolic disruption, and polypharmacy and iatrogenic obesity refute age-only and weight-only narratives. Integrating these findings within an exposomic framework, tempered by historical examples of regulatory capture and theological reflections on stewardship, re-orients NCD prevention from downstream pharmacological control to upstream environmental integrity.

4.2 Stakeholder-Segmented Recommendations: ***[i]. Governments and Regulators:*** Embed a *Health-in-All-Policies* mandate linking water, food and drug regulations. Tighten DBP, organophosphate and endocrine-disruptor limits; require cumulative-risk assessments; and prioritise pharmaceutical approvals with minimal metabolic externalities. Fund infrastructure for advanced, non-chlorine-dependent disinfection and incentivise agro-ecological pest control. ***[ii]. Public-Health Agencies:*** Expand NCD programmes to track exposure biomarkers and early-onset disease. Issue guidance integrating environmental mitigation (e.g., filtration, organic procurement) with lifestyle advice. Deploy national exposome dashboards to monitor progress. ***[iii]. Industry (Water, Agri-Food, Pharma):*** Adopt *greener chemistry* and integrative pest-management to cut precursor chemicals; reformulate products to eliminate unnecessary additives; and accelerate R&D of metabolically neutral drugs. Voluntary disclosure of chemical footprints and funding of independent long-term studies will rebuild trust while pre-empting litigation. ***[iv]. Healthcare Providers:*** Normalise environmental-exposure histories, parsimonious prescribing and deprescribing. Prioritise first-line lifestyle or non-pharmacological interventions where evidence supports equivalence. Educate patients on household mitigation (e.g., certified filters, safe food-storage practices). ***[v]. Civil Society and Individuals:*** Advocate for source-water protection, transparent supply chains and urban food environments that privilege minimally processed produce. Faith communities and NGOs can frame exposome reduction as creation care and social justice, amplifying demand for healthy defaults.

4.3 Impact, Scalability, and Adaptive Governance: Because the triad affects entire populations, interventions that shift background exposures, even modestly, can yield outsized gains in health-adjusted life years and cognitive capital. Impact assessments should prioritise policies with broad reach and durable effect, such as upgrading municipal treatment plants or phasing out high-risk pesticides. An inter-ministerial Exposome Council, refreshed biennially with new science and public input, can pilot, scale, or recalibrate measures under conditions of uncertainty. Progress ought to be tracked on open dashboards that couple biomarkers of exposure with age-specific NCD incidence, fostering accountability and rapid learning. Internationally, a chemicals-and-health accord—analogous to ozone-layer and mercury treaties—could harmonise standards and finance technology transfer to low-resource settings, ensuring benefits are globally distributed.

4.4 Limitations and Future Research: This synthesis relies on heterogeneous secondary studies; although triangulation attenuates bias, residual confounding and publication lag persist. Mechanistic pathways for low-dose mixtures remain incompletely mapped, and historical documentation of covert agendas, while compelling, is fragmentary. Exposure misclassification stems chiefly from temporal misalignment between biomonitoring snapshots and disease onset, whereas causal-inference uncertainty derives from residual confounding and selection bias intrinsic to observational syntheses. Publication bias

was evaluated by contour-enhanced funnel plots (now added to Supplement S4), revealing asymmetry for pesticide–diabetes studies but not for TTHM–cognition.

Future research should therefore (i) establish longitudinal birth-to-adulthood cohorts with high-resolution exposome profiling, (ii) run community-level intervention trials that reduce combined exposures and track metabolic and cognitive endpoints, (iii) unravel cellular effects of realistic chemical mixtures via epigenomic and metabolomic techniques, (iv) develop AI-enabled risk-assessment models that integrate multiple exposure routes and susceptibilities, and (v) refine ethical frameworks—secular and faith-based—for balancing technological benefit against precaution. Advancing along these lines will convert the present exposomic hypothesis into a robust, actionable science, guiding societies toward environments that honour both human flourishing and the integrity of the created order.

Conflict of Interest: The authors have no conflicts of interest to disclose.

Data Availability All original analyses, interpretive frameworks, and illustrative excerpts are fully integrated into the manuscript. Any supplementary materials or clarifications that might assist replication or further investigation are available upon reasonable request from the corresponding author.

Research Ethics: Because the inquiry relied exclusively on publicly available secondary sources, it involved no interaction with human participants or collection of identifiable personal data. Consequently, institutional ethics approval was not required under prevailing guidelines for social-science research. The study nonetheless adhered to accepted norms of responsible scholarship, including transparent sourcing, careful stewardship of data, and respect for intellectual property.

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Supplementary Materials

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