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Synergistic Effects of NO_2, SO_2, and Particulate Matter on Plant Physiology and Human Respiratory Health

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Abstract: Nitrogen dioxide (NO_2), sulfur dioxide (SO_2), and particulate matter (PM) are co-emitted from vehicles, industrial facilities, and fossil fuel combustion. Urban and industrial populations are exposed to these pollutants simultaneously, yet most air quality standards treat them in isolation. This review synthesizes evidence from plant biology, atmospheric chemistry, epidemiology, and toxicology to show that their combined exposure produces synergistic effects, meaning interaction coefficients exceeding 1.0, in both plant and human systems. In plants, pollutant mixtures amplify photosynthetic inhibition beyond 60% (versus 15 to 50% for individual pollutants), overwhelm superoxide dismutase and catalase defenses, impair stomatal regulation, and reduce crop yields by 20 to 40% in heavily polluted regions. In humans, concurrent NO_2, SO_2, and PM exposure synergistically activates NF-κB and MAPK inflammatory cascades, reduces FEV_1 beyond additive predictions, and exacerbates asthma and chronic obstructive pulmonary disease (COPD). Molecular mechanisms converge on oxidative stress, epithelial barrier dysfunction, and epigenetic reprogramming. Current single-pollutant regulatory frameworks underestimate these mixture risks. Integrated emission controls and multi-pollutant standards are needed to adequately protect public and ecosystem health.

1. Introduction

Air pollution kills approximately 7 million people annually (WHO, 2021), with NO_2, SO_2, and PM among the primary culprits. These pollutants share emission sources, including vehicular exhaust, coal-fired power plants, and industrial smelters, ensuring that real-world exposures are invariably mixed rather than single-compound (Lelieveld *et al.*, 2015). Motor vehicles alone contribute 50 to 60% of urban NO_X emissions, while coal combustion accounts for 50 to 70% of anthropogenic SO_2 in industrialized regions (Lu *et al.*, 2010; Carslaw and Rhys-Tyler, 2013). Secondary PM, formed through atmospheric reactions among NO_X, SO_2, ammonia, and volatile organic compounds, contributes 40 to 70% of fine PM in urban air (Seinfeld and Pandis, 2016).

A bibliometric analysis of 972 Scopus-indexed documents (2015 to 2024) shows annual publication output growing from about 20 in 2015 to a peak of approximately 211 in 2022, with contributions from 69 countries. China, India,, and the United States lead output, followed by the South Korea, and Iran (Dilanjani *et al.*, 2025; Kachbou *et al.*, 2025; Hammouti *et al.*, 2025; Nandiyanto *et al.*, 2026). China accounts for about 450 articles, followed by India (143 articles), an emerging contributor, reflecting growing concern over industrialization-driven air quality

deterioration, though its research output remains secondary to China and Western nations. This finding can be presented via VOSviewer using the 972 articles collected from Scopus (Aichouch *et al.*, 2025; Salghi *et al.*, 2025; Lakhlifi *et al.*, 2025). Countries are visualized by colored circles called nodes, and the size indicates the number of articles. The largest blue node represents China as the top-published country, and India (green node) is in the second position, and the US is shown by a blue node. The collaboration is visualized by the lines interconnecting nodes.

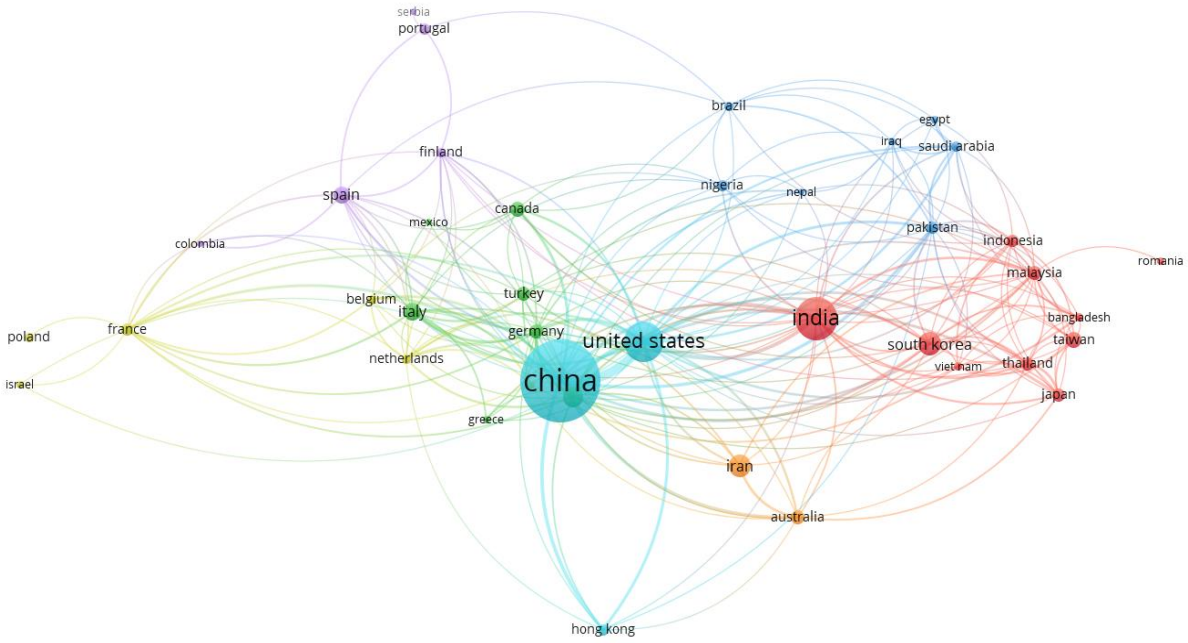

Figure 1: VOSviewer network visualization (air pollution, NO_2, SO_2, and PM) from 2015 to 2024

Synergism occurs when the combined effect of pollutants exceeds the sum of their individual effects. Growing evidence shows that interactions among NO_2, SO_2, and PM produce outcomes that simple additive risk models underestimate (Liu *et al.*, 2021). Plants act as both sentinels and victims of this pollution, exhibiting measurable physiological dysfunction at field-relevant concentrations (Agathokleous *et al.*, 2020). Vulnerable human populations, including children, the elderly, and individuals with pre-existing respiratory disease, face compounded risks from these mixtures (Kampa and Castanas, 2008; Saini *et al.*, 2019).

This review examines the mechanistic basis of synergistic pollutant interactions in plant and human systems, quantifies dose-response relationships, and evaluates the implications for environmental management and public health policy. Sections 2 and 3 address atmospheric chemistry and plant physiology. Sections 4 through 6 cover human respiratory health and molecular mechanisms. Sections 7 through 10 discuss modifying factors, ecosystem consequences, regulatory challenges, and research priorities.

2. Sources and Atmospheric Chemistry of NO_2, SO_2, and PM

2.1 Emission Sources

NO_2 arises from high-temperature combustion where atmospheric nitrogen reacts with oxygen. Its atmospheric lifetime ranges from hours to a few days, depending on oxidant availability and meteorology. SO_2 emissions have declined substantially in developed nations following regulatory interventions and fuel switching, but remain significant wherever coal combustion is predominant. Power plants, metal smelters, and petroleum refineries are the primary SO_2 point sources (Lu *et al.*,

2010). PM is classified by aerodynamic diameter: PM_{10} (particles ≤10 μm) and $PM_{2.5}$ (particles ≤2.5 μm). Primary PM is emitted directly from combustion, construction, and natural sources. Secondary PM forms through atmospheric chemical reactions and accounts for 40 to 70% of total fine PM in urban environments (Heal *et al.*, 2012).

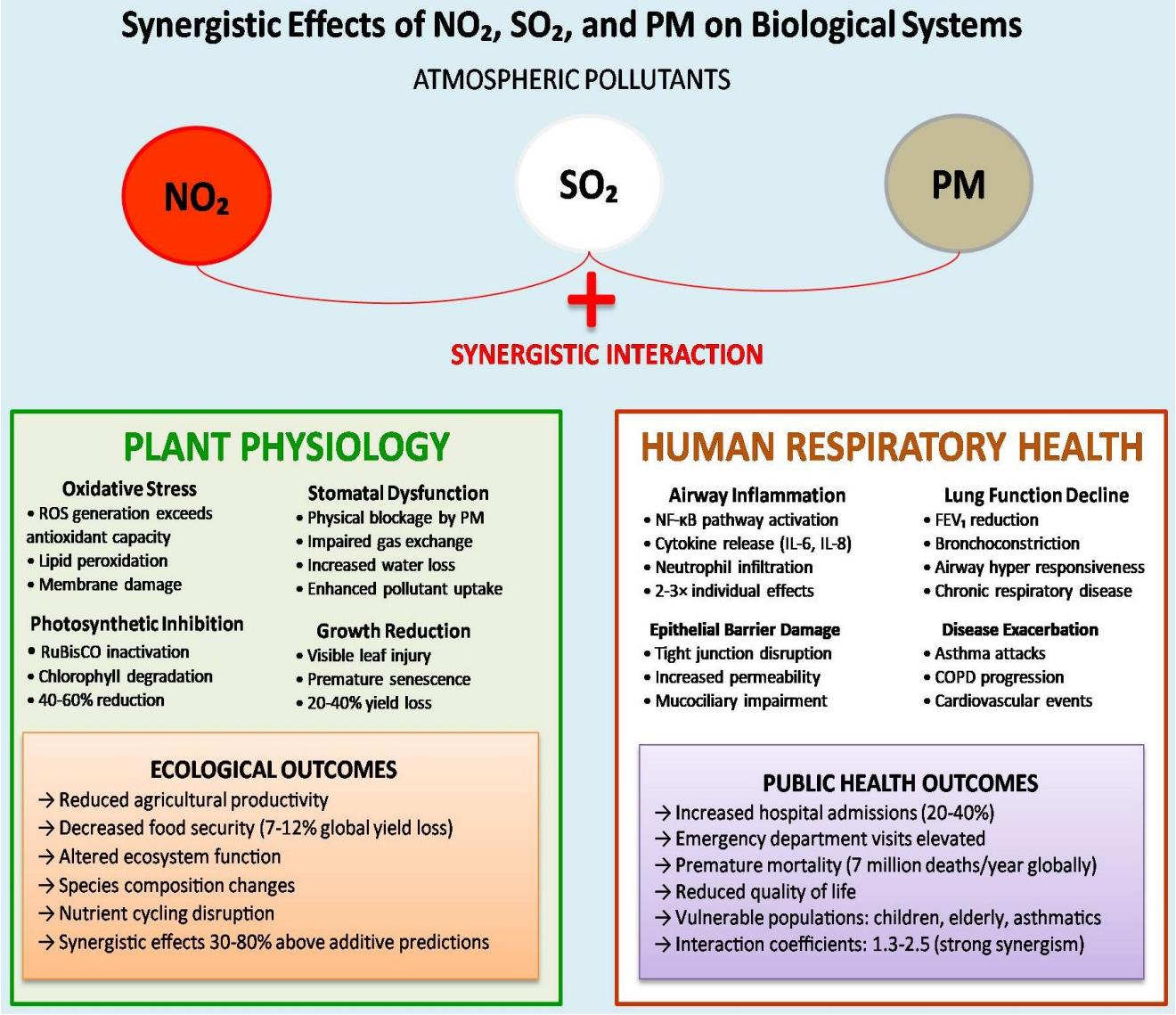

Figure 2: Synergistic effects of NO_2, SO_2, and PM on both plant physiology and human respiratory health

2.2 Atmospheric Interactions and Secondary Pollutant Formation

The atmospheric chemistry of these three pollutants is tightly coupled. NO_2 drives tropospheric ozone formation and contributes to nitrate aerosol production. SO_2 oxidation generates sulfate aerosols, a significant fraction of fine PM. Heterogeneous reactions on particle surfaces catalyze NO_2 and SO_2 transformations that would proceed slowly in the gas phase (Abbatt *et al.*, 2012). PM can also absorb and transport gaseous pollutants, delivering them more efficiently to biological surfaces. The hygroscopic growth of sulfate and nitrate aerosols modifies particle size distribution, altering respiratory deposition patterns in humans and foliar deposition in plants. These interdependencies mean that reducing one pollutant often shifts the formation and toxicity of others, underscoring the need for integrated emission strategies.

3. Effects on Plant Physiology

3.1 Individual Pollutant Effects on Plants

NO_2 enters leaf tissue through stomata, dissolving in apoplastic fluid to form nitrite and nitrate. At low concentrations it can serve as a nitrogen source, but at elevated levels it generates reactive nitrogen species (RNS) and causes leaf chlorosis, necrosis, and premature senescence. SO_2 similarly enters via stomata, forming sulfite and bisulfite ions that inhibit photosynthetic enzymes, alter membrane permeability, and reduce chlorophyll content. Sensitive species exhibit visible injury at 0.1 to 0.3 ppm SO_2 during extended exposure. PM reduces light transmittance, physically obstructs stomata, and delivers toxic metals and organic compounds to leaf surfaces. Fine particles penetrating sub-stomatal cavities cause direct cellular damage (Grantz *et al.*, 2003; Saini *et al.*, 2021).

3.2 Synergistic Effects on Photosynthesis

Combined NO_2 and SO_2 exposure produces photosynthetic inhibition greater than additive predictions, with interaction coefficients exceeding 1.3 in wheat fumigation experiments (Rai, 2016). Both pollutants inactivate ribulose-1,5-bisphosphate carboxylase/oxygenase (RuBisCO), attacking different functional sites simultaneously. They also generate distinct reactive oxygen species (ROS) that act together to damage photosystem II reaction centres and thylakoid membranes. Photosynthetically active radiation (PAR) reaching leaves declines further as PM accumulates on surfaces, forcing stomata to open to maintain CO_2 uptake. This paradoxically increases gaseous pollutant uptake, amplifying their toxic effects. Acidic sulfate- and nitrate-bearing particles additionally damage leaf cuticles, increasing pollutant penetration and water loss (Nanos and Ilias, 2007). The net result is photosynthetic inhibition exceeding 60% under combined exposure, compared to 15 to 50% for individual pollutants.

3.3 Oxidative Stress and Antioxidant Response

Each pollutant triggers ROS production through distinct pathways, but their combined load overwhelms enzymatic (SOD, CAT, APX, GR) and non-enzymatic (ascorbic acid, glutathione, tocopherols) antioxidant defenses. Pea plants exposed to combined NO_2 and SO_2 showed initial spikes in SOD and CAT activity followed by precipitous decline as cellular damage progressed; malondialdehyde content, a marker of lipid peroxidation, increased synergistically (Pandey *et al.*, 1992). This transition from adaptive defense to irreversible injury represents a key threshold in plant toxicology. Antioxidant depletion under mixed-pollutant conditions occurs more rapidly than under single-pollutant stress, reducing the window during which reversible protective responses can operate.

3.4 Stomatal Function and Water Relations

NO_2 and SO_2 can induce either stomatal closure or opening depending on concentration and exposure duration. Chronic exposure impairs stomatal responsiveness, leading to unregulated water loss and increased pollutant uptake (Robinson *et al.*, 1998). Physical blockage by PM prevents stomatal closure, compromising both water-use efficiency and pollutant exclusion (Farmer, 1993). Chemical interactions between particles and leaf surfaces can also disrupt abscisic acid signaling, the primary regulator of stomatal aperture. Plants exposed to combined gaseous pollutants and PM deposition show greater transpirational water loss and reduced water-use efficiency than plants under individual stressors, exacerbating drought stress and reducing tolerance to other environmental challenges (Honour *et al.*, 2009).

3.5 Growth, Yield, and Reproduction

Chronic exposure to pollutant mixtures reduces leaf area, stem elongation, and root development, with biomass declines of 15 to 40% depending on species and exposure intensity. Yield losses are documented for wheat, rice, maize, and legumes (Emberson *et al.*, 2018). Timing is critical: exposure during flowering and grain filling causes disproportionate yield losses compared to vegetative-stage exposures. Ground-level ozone and associated pollutants already account for 7 to 12% annual wheat yield losses and 6 to 16% for soybeans globally (Avnery *et al.*, 2011); synergistic NO_2, SO_2, and PM effects may push these losses to 15 to 25% in heavily polluted regions.

Reproductive impairment adds another layer of risk. NO_2 and SO_2 reduce pollen germination rates by 30 to 50% (Speranza *et al.*, 2004). PM physically obstructs stigmatic surfaces, disrupts pollinator attraction by altering floral chemistry, and compromises seed quality through oxidative damage. These reproductive bottlenecks reduce recruitment in sensitive plant populations, with long-term implications for ecosystem structure and genetic diversity.

4. Effects on Human Respiratory Health

4.1 Individual Pollutant Effects on the Respiratory System

Each pollutant targets different anatomical sites in the respiratory tract. NO_2 has relatively low water solubility, allowing deep lung penetration. It generates nitrogen-centered radicals and peroxynitrite at the respiratory epithelium, triggering lipid peroxidation and neutrophilic inflammation (Ghio *et al.*, 2015). SO_2, being more water-soluble, is predominantly absorbed in the upper airway, where it stimulates sensory nerves and reflex bronchoconstriction through cholinergic pathways.

Table 1: Major physiological effects of NO_2, SO_2, and PM on plants

Pollutant	Primary entry route	Cellular effects	Physiological impacts	Visible symptoms
NO_2 (nitrogen dioxide)	Stomatal uptake; dissolves in apoplastic fluid	Nitrite/nitrate accumulation; RNS generation; enzyme inhibition	15-40% photosynthesis decline; impaired N metabolism; stomatal dysfunction	Leaf chlorosis; interveinal necrosis; premature senescence
SO_2 (sulfur dioxide)	Stomatal uptake; forms sulfite/bisulfite	Sulfite accumulation; ROS production; membrane disruption; chlorophyll degradation	20-50% photosynthetic rate reduction; enzyme inactivation	Bleaching; marginal necrosis; growth suppression
PM (particulate matter)	Surface deposition; potential stomatal penetration	Light reduction; physical blockage; toxic metal release; cuticle damage	10-30% reduced light interception; impaired gas exchange; increased water loss	Dust accumulation; discoloration; accelerated aging
Combined (synergistic)	Multiple pathways; enhanced penetration	Amplified ROS/RNS; antioxidant depletion; metabolic collapse	>60% photosynthesis inhibition; disrupted C/N balance; yield losses 20-40%	Early abscission; extensive necrosis; stunted growth

Synergistic interaction coefficients typically >1.2. Percentages represent ranges from controlled studies at moderate to high concentrations; actual effects vary by species, exposure duration, and environmental conditions. Adapted from Sharma *et al.*, 2012; Rai, 2016; Agathokleous *et al.*, 2020.

Sulfite formed from SO_2 dissolution can trigger mast cell degranulation in sensitized individuals, releasing histamine and pro-inflammatory mediators (Linn *et al.*, 1994). PM effects depend on particle size: PM_{10} deposits in upper airways, while $PM_{2.5}$ and ultrafine particles (<0.1 μm) reach the

alveolar region. Particles carry adsorbed polycyclic aromatic hydrocarbons, heavy metals (lead, cadmium, arsenic), and biological components that amplify toxicity. Ultrafine particles may translocate across the alveolar-capillary barrier into systemic circulation, reaching the heart, brain, and liver (Pope III and Dockery, 2006; Saini and Kumar, 2022).

4.2 Synergistic Effects on Respiratory Inflammation

A time-series study in China found that combined NO_2, SO_2, and PM_{10} exposure raised hospital admissions for respiratory disease beyond additive predictions, with the strongest synergy during high-pollution episodes and in elderly patients (Meng *et al.*, 2013). Mechanistically, gaseous pollutants enhance particle toxicity by generating more reactive particle surfaces, while increasing epithelial permeability to facilitate deeper particle penetration (Kelly and Fussell, 2012). At the cellular level, mixtures simultaneously activate NF-κB, MAPK, and NLRP3 inflammasome pathways. Transcriptomic studies show that combined NO_2 and $PM_{2.5}$ exposure upregulates IL-6, IL-8, and TNF-α far beyond levels seen with individual pollutants, while more profoundly suppressing antioxidant defense and DNA repair genes (Li *et al.*, 2018).

4.3 Lung Function Impairment

Spirometric data from Chinese adults show that combined NO_2 and $PM_{2.5}$ exposure produces greater FEV_1 decrements than additive models predict, with the strongest interactions in smokers and those with pre-existing respiratory disease (Guo *et al.*, 2018). Children are particularly vulnerable: their developing airways, higher ventilation rates per body mass, and greater outdoor activity time increase pollutant doses. Longitudinal studies confirm that chronic childhood exposure to elevated NO_2, SO_2, and PM reduces lung function growth trajectories and increases asthma incidence, with effects persisting into adulthood (Schultz *et al.*, 2016).

4.4 Exacerbation of Asthma and COPD

Asthma affects over 300 million people worldwide. Gaseous pollutants increase airway reactivity to allergens by enhancing allergen penetration, amplifying inflammatory responses, and disrupting mucociliary clearance (D'Amato *et al.*, 2016). When combined with PM, which delivers oxidative stress and pro-inflammatory stimuli directly to the airways, the inflammatory response is substantially amplified. Analysis of over 10,000 asthma emergency visits showed significant synergistic effects between SO_2 and PM on visit rates, strongest in warm seasons (Dales *et al.*, 2000). In COPD patients, the pro-inflammatory effects of NO_2, SO_2, and PM synergize with underlying chronic airway inflammation, accelerating lung function decline. Hospitalization and mortality rates for COPD are strongly associated with ambient pollutant levels, especially when respiratory infections also peak in winter (Zemp *et al.*, 1999).

4.5 Cardiovascular and Systemic Effects

Pollutant-induced pulmonary inflammation elevates circulating C-reactive protein, interleukin-6, and fibrinogen, promoting endothelial dysfunction, atherosclerosis, and thrombosis. Ultrafine particles that access the bloodstream through alveolar translocation can directly damage cardiovascular tissues (Rückerl *et al.*, 2011). A comprehensive multi-city Chinese study found that combined NO_2, SO_2, and $PM_{2.5}$ produced positive interaction terms for cardiovascular mortality, particularly for ischemic heart disease and stroke (Chen *et al.*, 2020). These findings extend the health burden of pollutant mixtures well beyond the respiratory tract.

Table 2: Respiratory health effects of NO_2, SO_2, and PM in human populations

Pollutant	Respiratory deposition	Acute health effects	Chronic health effects	Vulnerable populations
NO_2	Deep lung penetration (50-80% alveolar)	Airway inflammation; increased infection susceptibility; bronchial hyperresponsiveness	Reduced lung function growth in children; increased asthma incidence; COPD exacerbation	Children, asthmatics, elderly, outdoor workers
SO_2	Upper airway deposition (70-90% nasal/pharyngeal)	Bronchoconstriction; mucus hypersecretion; chest tightness	Chronic bronchitis; accelerated lung function decline	Asthmatics; pre-existing respiratory disease; exercising individuals
$PM_{2.5}$	Alveolar deposition (70-90%)	Inflammatory responses; oxidative stress; acute symptom exacerbation	Lung cancer; cardiovascular disease; premature mortality; reduced life expectancy	Cardiovascular patients; children; pregnant women; elderly
Combined (synergistic)	Enhanced penetration; increased epithelial permeability	Amplified inflammation (2-3× individual effects); emergency visits 20-40% above additive	Accelerated disease progression; greater lung function decline; interaction coefficients 1.3-2.1	All vulnerable groups; cumulative lifetime exposure compounds risk

Interaction coefficients >1.0 indicate synergistic effects. Adapted from Meng *et al.*, 2013; Brook *et al.*, 2010; D'Amato *et al.*, 2016.

5. Molecular and Cellular Mechanisms of Synergistic Effects

5.1 Oxidative Stress as a Unifying Mechanism

Oxidative stress links pollutant exposure across plant and mammalian systems. NO_2 generates nitrogen-centered radicals and peroxynitrite; SO_2 metabolites produce superoxide and hydrogen peroxide; PM-associated transition metals (iron, copper) catalyze hydroxyl radical formation via Fenton and Haber-Weiss reactions (Nel *et al.*, 2006). Organic compounds on particle surfaces, including quinones and polycyclic aromatic hydrocarbons, undergo redox cycling that depletes cellular reducing equivalents while generating additional ROS (Li *et al.*, 2008). Combined, these mechanisms create a multi-pronged oxidative assault that depletes glutathione, vitamins C and E, and antioxidant enzyme activity. Unchecked ROS then damage lipids, proteins, and nucleic acids, generating reactive aldehydes that propagate injury and impair cellular signaling.

5.2 Inflammatory Signaling Pathways

NF-κB is a master regulator of inflammatory gene expression activated by both gaseous pollutants and PM through oxidative stress-dependent mechanisms (Baulig *et al.*, 2003). Pollutant mixtures converge multiple upstream signals on NF-κB, producing greater and more sustained activation than individual exposures. MAPK pathways (ERK, JNK, p38) are similarly synergistically activated, amplifying downstream inflammatory gene expression (Rodríguez-Cotto *et al.*, 2013). The NLRP3 inflammasome, which activates caspase-1 to mature IL-1β and IL-18, is potentiated when gaseous pollutant-enhanced oxidative stress combines with PM-driven lysosomal disruption and potassium efflux. The result is amplified cytokine production that extends well beyond the sum of individual exposures (Kayalar *et al.*, 2024).

5.3 Epithelial Barrier Dysfunction

Tight junction proteins (occludin, claudins, ZO-1) maintain the respiratory epithelial barrier. Both gaseous pollutants and PM reduce their expression and disrupt their distribution, increasing

paracellular permeability and allowing greater allergen, pathogen, and pollutant penetration into subepithelial tissues (Xiao *et al.*, 2011). Ciliary beat frequency decreases following NO_2 and SO_2 exposure, and PM physically obstructs ciliary function. Mucin glycoprotein structure is altered, with increased sulfation and reduced water content impairing mucociliary transport (Antunes and Cohen, 2007). Combined exposure synergistically worsens these deficits, creating conditions that favor particle accumulation and bacterial colonization, contributing to recurrent respiratory infections in chronically exposed individuals.

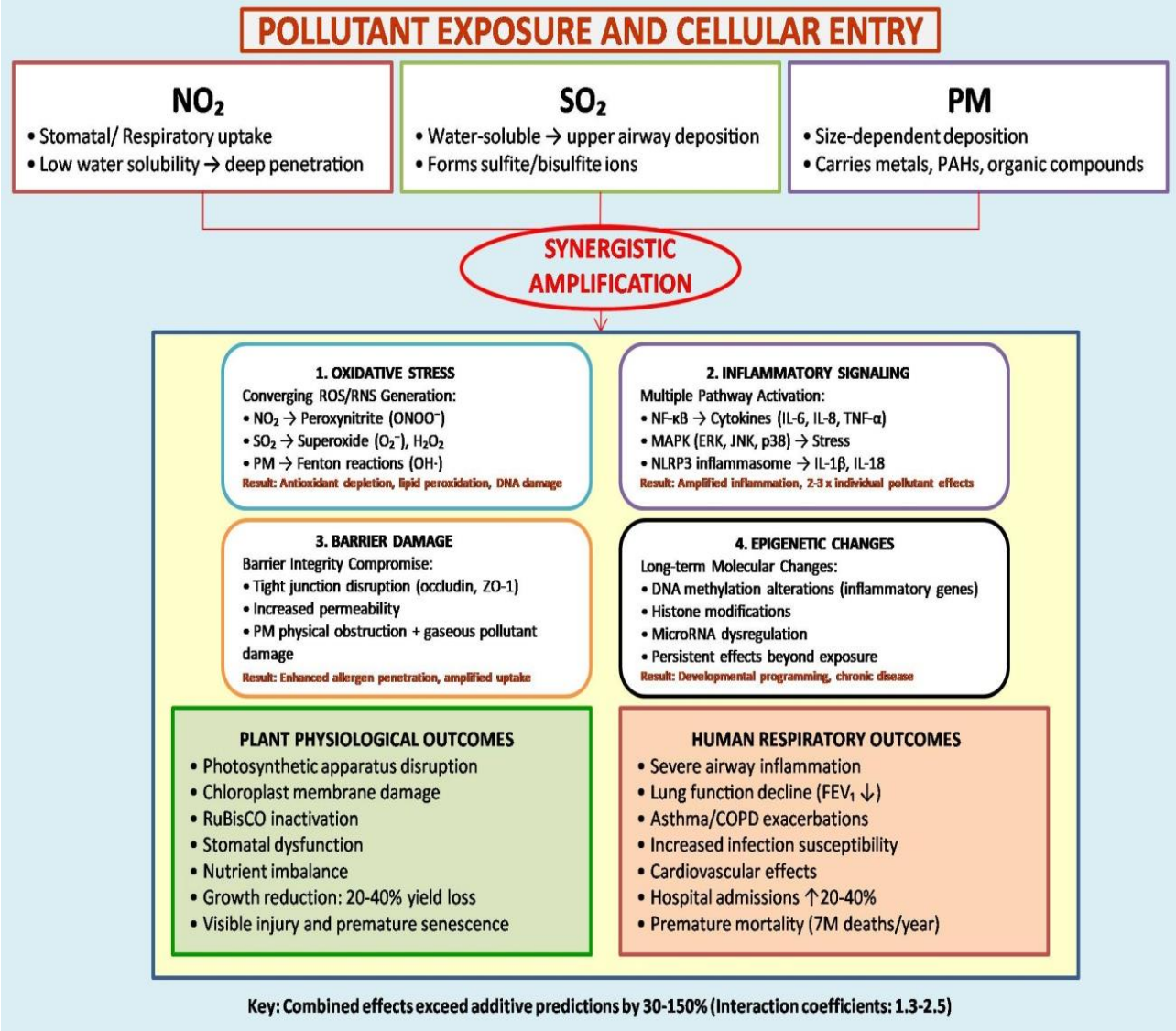

Figure 3: Molecular mechanisms of synergistic pollutant effects

5.4 Genetic and Epigenetic Modifications

DNA damage from oxidative stress, peroxynitrite, sulfite metabolites, and PM-associated polycyclic aromatic hydrocarbon adducts can overwhelm base excision, nucleotide excision, and double-strand break repair pathways. Persistent lesions in tumor suppressors (p53, BRCA1) or oncogenes may drive carcinogenesis, with pollutant mixtures creating synergistic genomic instability when repair capacity is concurrently compromised (DeMarini, 2004). Epigenetic changes, including altered DNA methylation at IL-6, TNF-α, NRF2, and SOD2 loci, can persist after exposure ends and may propagate intergenerationally (Baccarelli and Bollati, 2009). Mouse models show that maternal air

pollution exposure alters offspring DNA methylation patterns, predisposing subsequent generations to asthma and metabolic disorders. MicroRNAs miR-21, miR-146a, and miR-155 are consistently upregulated by pollutant exposure, sustaining inflammatory responses, while let-7 family members that suppress oncogenes are downregulated (Fossati *et al.*, 2014). These coordinated epigenetic and genetic changes represent mechanisms through which short-term pollutant mixture exposure can produce long-lasting pathological consequences.

6. Dose-Response Relationships and Threshold Effects

6.1 Concentration Ranges and Exposure Scenarios

Urban areas in developing nations frequently record NO_2 at 40 to 100 µg/m^3, SO_2 at 20 to 150 µg/m^3, and $PM_{2.5}$ at 50 to 200 µg/m^3, all substantially exceeding WHO guidelines (annual means: NO_2 <40 µg/m^3, SO_2 <40 µg/m^3, $PM_{2.5}$ <10 µg/m^3) (WHO, 2021). **Table 3** shows how synergistic interaction coefficients intensify nonlinearly with increasing pollutant concentrations. Background concentrations produce minimal interaction, whereas severe pollution episodes generate coefficients of 1.5 to 2.5 or higher. This nonlinearity underscores the disproportionate health and ecological risk posed by episodic pollution peaks. Even at moderate urban concentrations, epidemiological evidence detects health effects, challenging the concept of safe thresholds. Each 10 µg/m^3 increase in $PM_{2.5}$ is associated with approximately 6 to 7% increases in cardiopulmonary mortality with no evidence of a threshold (Pope III *et al.*, 2002).

Table 3: Dose-response relationships for pollutant mixtures across exposure scenarios

Exposure scenario	Pollutant concentrations	Plant effects	Human health effects	Synergistic interaction
Background/clean air	NO_2 <10, SO_2 <5, $PM_{2.5}$ <5 µg/m^3	No observable injury; normal physiology	No increased symptoms; normal lung function	Minimal to none; below detection threshold
Moderate urban	NO_2 40-60, SO_2 20-40, $PM_{2.5}$ 25-50 µg/m^3	Subclinical stress; 5-15% photosynthesis reduction; visible injury in sensitive species	Mild irritation; 2-3% FEV_1 decline; increased medication use in asthmatics	Weak to moderate; coefficients 1.1-1.3; effects 10-30% above additive
High urban/industrial	NO_2 80-120, SO_2 50-100, $PM_{2.5}$ 75-150 µg/m^3	20-40% photosynthetic inhibition; visible foliar injury; 15-25% growth reduction	ED visits up 15-25%; significant FEV_1 decline; increased symptom burden	Moderate to strong; coefficients 1.3-1.8; effects 30-80% above additive
Severe pollution episodes	NO_2 >150, SO_2 >150, $PM_{2.5}$ >200 µg/m^3	>50% photosynthesis loss; acute injury; potential mortality in sensitive species	Hospital admissions up 40-100%; excess mortality; COPD/asthma decompensation	Strong; coefficients 1.5-2.5+; nonlinear responses; effects >100% above additive

Interaction coefficients = (observed combined effect)/(sum of individual effects). Concentrations are 24-hour averages. Effects vary by species/individual susceptibility, exposure duration, and co-pollutants. Adapted from Pope III *et al.*, 2002; Meng *et al.*, 2013; Agathokleous *et al.*, 2020; WHO, 2021.

6.2 Temporal Patterns and Cumulative Effects

Acute, high-concentration episodes trigger different biological responses than chronic moderate exposures, yet both demonstrate synergistic effects. Mortality displacement studies show that many acute pollution victims were already compromised by chronic exposure, with episodes triggering final decompensation (Zanobetti *et al.*, 2000). This represents a form of temporal synergism where chronic exposure creates vulnerability that acute peaks exploit. For plants, brief intense exposures cause acute visible injury, while chronic exposures progressively degrade photosynthetic capacity. Exposure during sensitive developmental windows, such as seedling establishment or flowering, creates disproportionate fitness impacts.

6.3 Nonlinear Responses and Tipping Points

At very low concentrations, compensatory mechanisms can maintain homeostasis. Low NO_2 concentrations may even stimulate plant growth by serving as a nitrogen source, while mild oxidative stress can upregulate protective antioxidant defenses. These hormetic effects disappear as concentrations rise (Agathokleous, 2018). Beyond critical thresholds, systems transition abruptly from compensation to decompensation. Plants maintaining photosynthesis under single-pollutant stress may collapse when a second pollutant is added, exceeding adaptive capacity. Similarly, individuals with subclinical respiratory impairment may rapidly shift from asymptomatic status to requiring emergency care during multi-pollutant episodes.

7. Modifying Factors and Vulnerable Populations

7.1 Environmental Modifiers

Temperature accelerates photochemical pollutant formation and secondary aerosol production, enhancing synergistic interactions (Jacob and Winner, 2009). High humidity promotes SO_2 conversion to sulfuric acid and NO_2 to nitric acid, increasing acidity-related toxicity, while also altering particle hygroscopic growth and respiratory deposition patterns. Solar radiation drives photochemical ozone formation from NO_X and generates secondary organic aerosols, adding to the pollutant mixture experienced biologically. These environmental modifiers vary seasonally and geographically, contributing to temporal and spatial patterns in synergistic health and ecological effects.

7.2 Species and Individual Susceptibility

Among plants, agricultural crops are generally more sensitive to mixed-pollutant stress than wild species, having been selected for yield rather than stress tolerance (Mills *et al.*, 2011). Legumes show greater sensitivity to NO_2 and SO_2 than cereals. In humans, children face elevated risk due to developing airways, higher minute ventilation per body mass, and greater outdoor exposure. Elderly individuals have reduced physiological reserve, making them vulnerable to acute decompensation. Pre-existing respiratory conditions (asthma, COPD, pulmonary fibrosis) and cardiovascular disease further amplify risk by providing a baseline of chronic inflammation and functional impairment that pollutant mixtures can rapidly worsen (Brook *et al.*, 2010; Schultz *et al.*, 2016).

7.3 Genetic Susceptibility

Polymorphisms in antioxidant enzyme genes, including glutathione S-transferases and superoxide dismutase, modify individual capacity to neutralize pollutant-induced oxidative stress (Li *et al.*, 2013). Variants in cytokine genes (TNF-α, IL-6, IL-1β) influence inflammatory response magnitude. Phase I and II metabolic enzyme variants (cytochrome P450s, conjugating enzymes) affect the

formation and elimination of toxic pollutant metabolites (Gilliland *et al.*, 2004). These gene-environment interactions explain why individuals with equivalent exposures can experience substantially different health outcomes, and they underscore the importance of genetic susceptibility data in risk assessment for pollutant mixtures.

7.4 Socioeconomic and Behavioral Factors

Lower-income communities frequently experience higher pollutant concentrations due to proximity to traffic corridors, industrial facilities, and ports, compounding socioeconomic health inequalities (Hajat *et al*., 2015; Rana and Saini, 2025b, 2026). Residential segregation and weak environmental enforcement further concentrate NO_2, SO_2, and PM exposure in marginalized neighborhoods, while limited healthcare access worsens health outcomes. Outdoor workers, including agricultural laborers and traffic police, face elevated doses during physical activity, when breathing rates increase. Smoking creates chronic airway inflammation that synergizes with ambient pollutants. Poor nutrition compromises antioxidant defenses, reducing capacity to neutralize ROS. These behavioral and socioeconomic factors create cumulative vulnerability that amplifies the health impact of pollutant mixtures beyond what pollutant concentrations alone would predict, particularly among populations with the least political influence over environmental decision-making (Rana and Saini, 2025a).

8. Implications for Ecosystem Function and Food Security

8.1 Agricultural Productivity and Food Quality

Global wheat yield losses from ground-level ozone and associated pollutants are estimated at 7 to 12% annually; when synergistic NO_2, SO_2, and PM effects are included, total losses may reach 15 to 25% in heavily polluted regions (Avnery *et al.*, 2011; Van Dingenen *et al.*, 2009). Beyond yield, air pollution degrades nutritional quality, reducing protein, vitamin, and micronutrient content in grains and vegetables. For horticultural crops, PM deposition reduces marketability through visible surface contamination. Fine particles embedded in trichomes or stomata during early development can become incorporated into expanding leaf tissue, posing potential food safety concerns regarding toxic metals and organic contaminants (Broberg *et al.*, 2017). These combined quantity and quality impacts threaten food security in developing nations already facing rapid population growth and limited resources for adaptation.

8.2 Natural Ecosystem Impacts

Forest decline in Europe, North America, and Asia has been partially attributed to air pollution, though distinguishing pollution effects from climate change and pests is challenging (Bytnerowicz *et al.*, 2007). Differential sensitivity across plant functional types can reshape community composition, with cascading effects on herbivores, pollinators, and higher trophic levels. Lichen communities are particularly useful bioindicators of chronic pollution history. Biogeochemically, chronic nitrogen deposition from NO_X initially fertilizes nitrogen-limited ecosystems before causing nitrogen saturation, soil acidification, and dominance of nitrophilic species (Bobbink *et al.*, 2010). Combined nitrogen and sulfur inputs produce interactive acidification effects on soil chemistry and aquatic ecosystems that neither pollutant generates alone.

8.3 Climate Change Interactions

Climate change and air pollution interact synergistically. Rising temperatures accelerate photochemical ozone and secondary aerosol formation, potentially worsening air quality even where

primary emission reductions are achieved (Tai *et al*., 2012). Heat waves compound pollutant health effects, with the combination of extreme heat and elevated ozone and PM doubling or tripling mortality rates relative to either stressor alone (Kinney and Pinkerton, 2022; Nandiyanto *et al*., 2025). Extended pollen seasons and increased aeroallergen production interact with air pollutants to worsen allergic respiratory disease (D'Amato et al., 2016). Climate-driven wildfire increases generate massive PM pulses that synergize with urban pollution. Despite growing public awareness of these climate-pollution linkages through digital platforms, online environmental information remains unevenly distributed across social class and rural-urban divides, limiting its reach to communities most exposed to pollutant mixtures. Translating this awareness into sustained emission controls and adaptation policies remains the central governance challenge (Saini and Rana, 2025). These climate-pollution feedbacks complicate projections of future health and ecosystem burdens, requiring climate adaptation strategies that explicitly incorporate air quality management and equitable access to environmental health information.

9. Regulatory Approaches and Air Quality Management

9.1 Current Air Quality Standards

Most jurisdictions regulate pollutants individually. The US EPA National Ambient Air Quality Standards specify NO_2 annual mean limits of 53 ppb, SO_2 1-hour limits of 75 ppb, and $PM_{2.5}$ annual mean limits of 12 μg/m³ (US EPA, 2020). WHO guidelines are substantially stricter. The Air Quality Index systems used to communicate health risk to the public report the single worst pollutant rather than accounting for mixture effects. Critical loads for acidifying sulfur and nitrogen compounds have been applied to protect European and North American ecosystems, but these frameworks address individual or closely related pollutant groups rather than diverse multi-pollutant mixtures (Grennfelt *et al*., 2020).

9.2 Challenges in Regulating Pollutant Mixtures

Establishing mixture-based standards faces challenges from collinearity between pollutants, spatial and temporal variability in pollutant ratios, and limited statistical power to detect interaction terms in epidemiological data. Options include multi-pollutant indices with weighting factors reflecting synergistic interactions, or composite standards limiting total oxidant or reactive PM burden (Mainka and Żak, 2022; Hwang *et al*., 2025). Risk assessment methodologies are evolving to incorporate mixture effects through hazard index calculations and interaction models, though these require extensive dose-response data for mixtures that remain limited. Applying greater uncertainty factors to account for potential synergism is a conservative option, but may not capture actual interaction magnitudes.

9.3 Emission Control Strategies

Vehicle emission standards, catalytic converters, diesel particulate filters, and low-sulfur fuels have driven substantial NO_2 and PM reductions in developed nations. Industrial flue gas desulfurization, selective catalytic reduction, and electrostatic precipitators have achieved major SO_2 and PM reductions from stationary sources (Chestnut and Mills, 2005). The transition to electric vehicles and renewable energy simultaneously reduces both air pollutant emissions and greenhouse gas output, illustrating the co-benefits potential of integrated environmental policies. Urban planning strategies, including compact development and expanded public transit, reduce vehicle travel and associated co-emitted pollutants.

9.4 Future Directions in Air Quality Management

Progress requires improved air quality models that simulate pollutant interactions and secondary formation, expanded real-time monitoring of multiple pollutants simultaneously, and satellite remote sensing to characterize mixture exposures over large regions. Health impact assessment methods should incorporate mixture interaction terms to accurately quantify the public health benefits of emission reductions. Portable air cleaners, high-efficiency HVAC filtration, and air quality forecasting systems can reduce individual exposure in the interim. Education programs that help vulnerable populations act during high-pollution episodes complement regulatory approaches.

10. Knowledge Gaps and Research Priorities

Despite substantial progress, key mechanistic questions remain. The specific molecular interactions through which pollutant combinations breach synergistic thresholds are not fully defined. Systems biology approaches, including transcriptomics, proteomics, and metabolomics applied to combined-pollutant exposures, can provide comprehensive cellular response maps that single-pollutant studies cannot. Long-term prospective cohort studies explicitly designed to evaluate mixture effects, with detailed exposure assessment capturing spatial and temporal variation in pollutant ratios, are needed to strengthen causal inference for chronic health outcomes.

Ultrafine particles (<0.1 μm) represent an emerging priority. Their large surface area, deep lung penetration, potential systemic translocation, and association with high-temperature combustion products that coat surfaces with reactive organics may make them disproportionately toxic in mixtures (Kumar *et al.*, 2010). Routine ultrafine particle monitoring is absent in most regulatory networks, limiting exposure characterization. Plant research should extend from agricultural crops to natural vegetation assemblages and entire ecosystems, using manipulative field experiments combined with long-term monitoring to distinguish pollution effects from climate change and nitrogen deposition drivers.

Research addressing social disparities in pollutant mixture exposure and health effects is essential for environmental justice. Understanding how socioeconomic factors interact with genetic susceptibility under multi-pollutant conditions can identify populations at highest cumulative risk and inform targeted protective interventions (Rana and Saini, 2025a, 2026). Interdisciplinary collaboration spanning atmospheric chemistry, plant biology, toxicology, epidemiology, and public policy is necessary to translate mechanistic insights into effective regulatory tools.

11. Conclusion

The evidence reviewed here establishes that co-exposure to NO_2, SO_2, and PM produces synergistic biological effects that single-pollutant risk models substantially underestimate. In plants, interaction coefficients exceeding 1.2 are consistently documented for photosynthetic inhibition, oxidative stress, stomatal dysfunction, and reproductive impairment, with yield losses in highly polluted regions reaching 20 to 40%. In humans, synergistic activation of NF-κB, MAPK, and inflammasome pathways amplifies inflammatory responses 2 to 3-fold beyond individual pollutant predictions, worsening asthma, COPD, lung function decline, and cardiovascular outcomes, with interaction coefficients of 1.3 to 2.1 documented in epidemiological studies.

Four converging molecular mechanisms account for this synergism: (1) multi-pathway ROS/RNS generation that collectively overwhelms antioxidant defenses; (2) simultaneous activation of multiple inflammatory cascades; (3) progressive epithelial barrier and mucociliary clearance compromise; and (4) epigenetic reprogramming that extends pathological consequences beyond the exposure period

and potentially across generations. Environmental modifiers including temperature, humidity, and solar radiation, together with genetic polymorphisms and socioeconomic determinants of exposure and susceptibility, create variable but consistently elevated risks in vulnerable populations.

Effective protection of human and ecosystem health demands movement beyond single-pollutant regulatory frameworks. Emission controls targeting major co-emitting sources, integrated multi-pollutant standards informed by synergistic dose-response data, and co-benefits approaches that simultaneously address air quality and climate change are the most promising policy pathways. Filling the identified research gaps in mixture toxicology, ultrafine particle characterization, ecosystem-level assessment, and environmental justice will be essential to quantify mixture risks accurately and design proportionate regulatory responses.

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