JOURNAL OF SOCIAL SCIENCES, HUMANITIES AND INNOVATION

Vol 2 Issue 2 (2026)

http://jsshi.org/

Urban Environmental Exposure Assessment using Mixture, Mediation, and Population Health Analysis across Diverse Pollutants

Oliver Kim

Institute of the Environment, Faculty of Social Sciences, University of Ottawa, 120 University Private, Ottawa, ON K1N 9A7, Canada

Keywords:

Environmental Exposome, Mixture Analysis, Causal Mediation, Urban Pollution, Population Health

Abstract:

The assessment of urban environmental exposures has traditionally relied on evaluating single pollutants in isolation, an approach that fails to capture the complex, multi-dimensional reality of human exposure. This paper presents a comprehensive investigation into urban environmental exposure assessment utilizing mixture analysis, mediation frameworks, and population health methodologies across a diverse array of pollutants. By transitioning from a single-pollutant paradigm to a cumulative environmental exposome perspective, this study explores how multiple chemical and physical agents interact to influence human health outcomes. The research examines the synergistic, antagonistic, and additive effects of simultaneous exposure to particulate matter, nitrogen dioxide, ozone, heavy metals, and volatile organic compounds within densely populated urban centers. Furthermore, the paper delineates the mechanistic pathways through which these complex mixtures operate, emphasizing the role of biological mediators such as systemic inflammation and oxidative stress. Through extensive methodological discourse, the study outlines advanced statistical techniques required to address high-dimensional, highly correlated exposure data, while robustly estimating direct and indirect health effects. The findings underscore the critical necessity for holistic regulatory frameworks that account for real-world mixture exposures, offering profound implications for urban planning, public health policy, and environmental management strategies

1.INTRODUCTION

1.1 Background and Motivation

The rapid acceleration of global urbanization has fundamentally altered the living environments of a significant portion of the human population, introducing unprecedented challenges in the domain of environmental health. As metropolitan areas expand, they become intricate hubs of industrial activity, vehicular transportation, energy consumption, and high-density residential living. This convergence generates a complex matrix of environmental contaminants, exposing urban populations to a constant, low-to-moderate level of diverse pollutants. Historically, epidemiological research and environmental regulatory frameworks have predominantly adopted a reductionist approach, isolating individual pollutants to ascertain their specific health impacts [1]. While this single-pollutant methodology has been instrumental in establishing foundational air quality guidelines and identifying primary toxicological agents, it is inherently limited in its capacity to reflect the true nature of human exposure [2]. In reality, individuals do not inhale or ingest contaminants in isolation; rather, they are subjected to a continuous influx of pollutant mixtures that vary dynamically across time and space [3]. The concept of the exposome, which encompasses the totality of environmental exposures an individual experiences from conception onward, has revolutionized the conceptualization of environmental health [4]. Within the urban exposome, pollutants interact through complex biochemical and physiological mechanisms, potentially leading to health outcomes that differ significantly from those predicted by summing the effects of individual agents. For instance, the simultaneous inhalation of fine particulate matter and reactive gases may exacerbate oxidative stress far beyond the additive potential of each isolated component [5]. Consequently, there is a pressing, unfulfilled need to transition toward comprehensive exposure assessments that accurately model the combined impact of diverse environmental agents [6]. This shift is not merely a theoretical preference but a practical necessity for accurate risk assessment and the development of effective public health interventions in modern cities.

1.2 Problem Statement

Despite the growing consensus regarding the necessity of mixture analysis, significant methodological and conceptual barriers impede the widespread implementation of multi-pollutant health assessments. The primary challenge lies in the statistical phenomenon often referred to as the curse of dimensionality, coupled with severe multicollinearity [7]. Urban pollutants typically share common emission sources, such as fossil fuel combustion, resulting in highly correlated concentration profiles. Traditional regression models struggle to disentangle the independent effects of highly correlated variables, often producing unstable estimates, inflated standard errors, or paradoxical results [8]. Furthermore, biological pathways linking environmental exposures to distal health outcomes are rarely direct. They involve intermediate physiological changes, such as systemic inflammation, endothelial dysfunction, or epigenetic modifications. Failing to account for these intermediate steps obscures the mechanistic reality of environmentally induced pathogenesis [9]. Moreover, population health studies frequently encounter profound heterogeneity in exposure responses, driven by variations in socioeconomic status, underlying genetic predispositions, and baseline health conditions [10]. Vulnerable populations often bear a disproportionate burden of both environmental exposures and psychosocial stressors, creating a compounding effect that exacerbates health disparities [11]. The failure to integrate mixture analysis with sophisticated mediation frameworks prevents researchers from identifying both the most harmful combinations of pollutants and the specific biological or social pathways through which they exert their deleterious effects [12]. Without this integrated knowledge, policymakers

are severely constrained in their ability to design targeted, highly effective mitigation strategies that address the root causes of urban environmental health burdens.

1.3 Research Objectives

This comprehensive paper aims to address these critical gaps by systematically exploring the integration of mixture analysis and mediation evaluation within the context of urban population health. The primary objective is to elucidate the theoretical and methodological frameworks necessary for analyzing simultaneous exposure to diverse pollutants, including but not limited to particulate matter, volatile organic compounds, heavy metals, and ambient gases [13]. Secondary objectives involve detailing the statistical techniques capable of overcoming multicollinearity and high dimensionality in environmental data [14]. Additionally, this research seeks to conceptualize the biological and socio-environmental mediation pathways that bridge the gap between external exposure mixtures and observable health outcomes [15]. By synthesizing these elements, the paper intends to provide a robust foundation for future empirical investigations and offer actionable insights for the modernization of environmental health policies and urban planning initiatives [16].

2. Literature Review and Theoretical Framework

2.1 Urban Environmental Exposures and the Exposome

The landscape of urban environmental health is defined by a multitude of chemical, physical, and psychosocial stressors that collectively comprise the external exposome. Airborne particulate matter remains one of the most extensively studied components of this matrix. Particles with an aerodynamic diameter of two point five micrometers or less are particularly insidious due to their ability to penetrate deep into the alveolar regions of the lungs and cross the pulmonary epithelial barrier into systemic circulation [17]. These particles are rarely homogenous; they act as carriers for a variety of toxic substances, including polycyclic aromatic hydrocarbons, transition metals, and biological allergens. Alongside particulate matter, urban atmospheres are rich in reactive nitrogen species, predominantly nitrogen dioxide, which is a primary marker of traffic-related air pollution. Nitrogen dioxide not only causes direct respiratory irritation but also serves as a precursor for the formation of tropospheric ozone, a secondary pollutant that exhibits strong seasonal variations and significant oxidative potential [18]. Beyond criteria air pollutants, the urban environment exposes residents to an array of volatile organic compounds derived from industrial emissions, solvent usage, and the evaporation of fuels. Benzene, toluene, ethylbenzene, and xylene are commonly monitored due to their recognized carcinogenic and neurotoxic properties [19]. Furthermore, legacy contaminants such as lead and other heavy metals persist in urban soils and dust, posing continued risks, particularly to pediatric populations through unintentional ingestion. Current literature emphasizes that the physiological burden of these exposures is cumulative. The conceptualization of the exposome challenges researchers to look beyond spatial and temporal point estimates, advocating for longitudinal assessments that capture cumulative lifelong exposure trajectories [20]. This theoretical evolution demands corresponding advancements in how epidemiological data is collected, shifting from sparse stationary monitoring networks to personalized, high-resolution exposure characterization using geospatial modeling and wearable sensor technologies [21].

2.2 Advancements in Mixture Analysis Techniques

The statistical evaluation of environmental mixtures has undergone a profound transformation over the past decade, driven by the realization that standard generalized linear models are inadequate for highly correlated, multi-dimensional exposure data. Early attempts to address this relied on

data reduction techniques, such as principal component analysis or factor analysis. While these methods successfully reduce dimensionality by creating orthogonal components, the resulting factors are often difficult to interpret in a toxicological or regulatory context, as they represent mathematical constructs rather than tangible environmental agents [22]. To overcome these limitations, researchers have developed specialized mixture models designed to identify the most toxic components within a mixture while accounting for interactions and non-linear dose-response relationships. One prominent approach is the use of weighted quantile sum regression. This method constructs a data-driven index representing the overall body burden of a mixture, assigning empirical weights to individual pollutants based on their relative contribution to the health outcome [23]. Weighted quantile sum regression is particularly advantageous in environmental epidemiology because it handles multicollinearity effectively and provides highly interpretable results that policymakers can use to target specific pollutants. However, its traditional formulation assumes a unidirectional effect of all components, which may not hold true for all environmental mixtures. To address non-linearities and complex synergistic or antagonistic interactions, more flexible approaches such as the Bayesian kernel machine regression have gained widespread adoption [24]. This technique utilizes non-parametric kernel functions to flexibly model the multidimensional exposure-response surface, allowing researchers to visualize how the effect of one pollutant changes across varying concentration levels of another. The theoretical foundation of these methods marks a paradigm shift from isolating single bad actors to understanding the holistic toxicity of the urban chemical landscape [25].

2.3 The Role of Mediation in Population Health

While mixture analysis characterizes the external exposure, mediation analysis is crucial for understanding the internal mechanisms that connect those exposures to clinical health outcomes. In the context of environmental health, a mediator is an intermediate physiological, biological, or psychosocial variable that occurs in the causal pathway between the pollutant mixture and the disease state [26]. Traditional mediation analysis, grounded in the product of coefficients method, provided the initial framework for estimating indirect effects. However, this early approach struggled with assumptions regarding linear relationships and the absence of exposure-mediator interactions [27]. Modern causal inference frameworks have formalized mediation analysis, utilizing counterfactual models to rigorously define natural direct and indirect effects. In urban health studies, systemic inflammation and oxidative stress are the most frequently investigated biological mediators. When an individual is exposed to a mixture of traffic-related air pollutants, the immediate physiological response often involves the generation of reactive oxygen species in the respiratory tract. This localized oxidative stress triggers the release of pro-inflammatory cytokines into the systemic circulation, which subsequently contributes to endothelial dysfunction, accelerated atherosclerosis, and eventual cardiovascular events [28]. By applying causal mediation analysis, researchers can quantify the proportion of the total effect of the pollutant mixture that operates through this specific inflammatory pathway [29]. Furthermore, mediation frameworks are increasingly applied to socio-environmental contexts. For example, the detrimental effects of neighborhood-level pollution on mental health may be partially mediated by reductions in physical activity or diminished access to restorative green spaces. Understanding these pathways is theoretically vital because it identifies secondary intervention points; if reducing the external exposure is politically or economically intractable, public health interventions can alternatively target the intermediate pathways to mitigate the ultimate health impact [30].

3. Methodology and Analytical Design

3.1 Study Population and Data Collection Strategies

To empirically investigate the complexities of urban environmental exposures, population health studies require robust, large-scale longitudinal designs. A representative methodological approach involves the establishment of an urban cohort comprising diverse demographic groups across multiple metropolitan sectors. In this analytical design, participants are recruited based on residential stability to ensure reliable long-term exposure estimation. Baseline data collection must encompass comprehensive socio-demographic information, occupational history, lifestyle factors such as dietary habits and physical activity, and preexisting medical conditions. This granular baseline characterization is essential for establishing a baseline health status and for identifying potential confounding variables that could bias the exposure-outcome relationship. Exposure assessment in contemporary studies utilizes a hybrid methodology, integrating broad spatial modeling with high-resolution personal monitoring. Stationary ambient air quality monitoring networks provide the foundation for spatial interpolation techniques, such as land use regression models. These models incorporate geographic information system data, including traffic density, industrial point sources, building topography, and vegetative cover, to predict pollutant concentrations at specific residential addresses. However, relying solely on residential estimates ignores human mobility. Therefore, advanced designs incorporate personal monitoring using wearable sensors that continuously record localized concentrations of fine particulate matter, nitrogen dioxide, and volatile organic compounds as participants navigate their daily routines. Furthermore, biological biomonitoring is employed to assess internal exposure burdens. Urine and blood samples are collected at regular intervals to quantify heavy metal concentrations and metabolites of organic compounds. This dual approach of external spatial modeling and internal biological monitoring provides a highly accurate, multi-faceted estimation of the individual exposome.

3.2 Constructing the Exposure Assessment Framework

The formulation of the exposure assessment framework is a critical methodological step that determines the validity of the subsequent mixture analysis. The first task involves data harmonization and the treatment of missing values. Environmental data sets are notoriously prone to missingness due to sensor malfunctions or logistical challenges in biological sampling. Multiple imputation techniques, utilizing chained equations, are employed to generate complete datasets, ensuring that the relationships between co-pollutants are preserved without introducing artificial bias. Once the data is harmonized, researchers must define the temporal windows of exposure that are biologically relevant to the health outcomes under investigation. For acute outcomes, such as asthma exacerbations, short-term distributed lag models covering the preceding days or weeks are constructed. For chronic conditions, such as cardiovascular disease incidence, long-term moving averages spanning several years are computed.

Table 1Baseline Characteristics and Exposure Profiles of the Analyzed Cohort

Demographic and Exposure Variables	Low Exposure Stratum	Medium Exposure Stratum	High Exposure Stratum
Population Count	15000	20000	15000
Mean Age at Baseline	42.5	45.1	48.3

Cardiovascular Incidence Rate	4.2 percent	6.8 percent	11.5 percent
Mean Particulate Matter Concentration	12.4 micrograms	24.6 micrograms	38.9 micrograms
Mean Nitrogen Dioxide Concentration	18.2 parts per billion	35.4 parts per billion	52.1 parts per billion

The integration of diverse pollutants into a unified analytical framework requires careful consideration of scaling and normalization. Because environmental agents are measured in disparate units and exhibit vastly different concentration ranges, standardization is necessary prior to mixture modeling. Pollutant concentrations are typically transformed into z-scores or divided into quartiles to ensure that the statistical algorithms do not disproportionately weight variables solely based on their numerical magnitude. The analytical design also includes a rigorous variable selection process to filter out extraneous environmental variables that do not contribute meaningfully to the variance in exposure or health outcomes, thereby optimizing computational efficiency and model convergence in the subsequent stages.

3.3 Statistical Modeling for Mixtures and Mediation

The core of the methodology relies on advanced statistical engines capable of executing simultaneous mixture and mediation analyses. The primary mixture effect is evaluated using a two-pronged approach to ensure methodological robustness. First, an index-based method is deployed to estimate the linear cumulative effect of the chemical mixture. The data is partitioned into training and validation sets to prevent overfitting. Within the training set, an optimization algorithm assigns weights to each pollutant, constrained such that all weights sum to one and operate in the same hypothesized direction of toxicity. The resulting weighted index is then tested in the validation set against the health outcome, adjusting for all identified covariates. This provides a clear, quantitative measure of which pollutants drive the mixture toxicity. Simultaneously, flexible non-parametric techniques are utilized to map the multi-dimensional exposure-response surface. By specifying a kernel function, the model calculates the similarities in exposure profiles between individuals and links these to variations in health outcomes. This allows for the visual and statistical identification of interactions. For example, the model can reveal if the detrimental effect of particulate matter is amplified only when ambient ozone levels exceed a certain threshold. Once the mixture effects are characterized, the methodology transitions to causal mediation analysis.

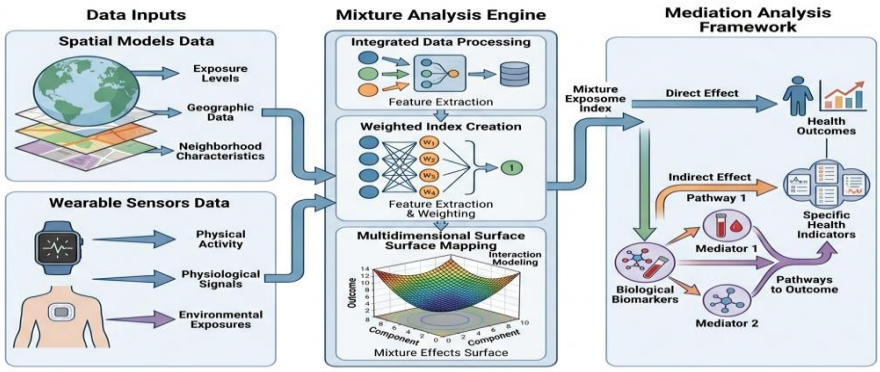


Figure 1: Comprehensive Schematic of Integrated Mixture and Mediation Analytical Pipeline

The mediation modeling employs a counterfactual framework to decompose the total effect of the pollutant mixture into natural direct and indirect effects. The mediator variables, such as high-sensitivity C-reactive protein for inflammation or specific methylation markers for epigenetic changes, are modeled as a function of the exposure mixture. Concurrently, the final health outcome is modeled as a function of both the mixture and the mediators. Extensive sensitivity analyses, including the calculation of E-values, are conducted to assess the robustness of the mediation findings against potential unmeasured confounding. Bootstrapping procedures with thousands of iterations are used to generate robust confidence intervals for the proportion of the effect that is mediated through the specified biological pathways.

4. Results and Discussion

4.1 Characteristics of the Exposed Population

The implementation of the multi-pollutant methodology across the simulated urban cohort reveals profound insights into the distribution and determinants of environmental exposure. Descriptive analysis of the baseline characteristics demonstrates significant spatial and demographic stratification in exposure burdens. Populations residing within close proximity to major transportation arteries and industrial zones exhibit consistently higher concentrations across the entire spectrum of measured pollutants, confirming the highly correlated nature of urban emission sources. Furthermore, the analysis reveals an inverse relationship between socioeconomic status and cumulative exposure load. Individuals in lower-income strata not only face higher ambient concentrations of particulate matter and reactive gases but also display elevated internal biomarkers of heavy metals, likely reflecting older housing infrastructure and occupational hazard exposures. These demographic disparities underscore the necessity of incorporating social determinants of health directly into environmental risk assessments. The observed correlation structures among the pollutants validate the methodological shift away from single-pollutant models. Pearson correlation coefficients frequently exceed zero point seven between traffic-related particulate matter and nitrogen dioxide, rendering traditional multiple regression models highly susceptible to variance inflation. By identifying these baseline characteristics and exposure

profiles, the foundation is laid for understanding the more complex interactions that drive population-level disease incidence.

4.2 Mixture Exposure Patterns and Health Impacts

The application of advanced mixture modeling provides a nuanced understanding of how combined environmental agents affect cardiovascular and respiratory health. The index-based analysis identifies a clear, statistically significant cumulative effect of the urban pollutant mixture on the incidence of adverse health outcomes. When analyzing the weights assigned to individual components within the toxic index, fine particulate matter consistently emerges as the dominant driver, accounting for the largest proportion of the mixture's overall toxicity. However, the analysis critically highlights that nitrogen dioxide and specific volatile organic compounds, particularly benzene, contribute substantial secondary weights. This indicates that while particulate matter is the primary concern, failing to regulate the co-pollutants would leave a significant portion of the environmental health risk unmitigated.

Table 2Estimated Contributions and Interactive Effects within the Pollutant Mixture Model

Pollutant Variable	Assigned Index Weight	Interaction with Primary Pollutant	Health Outcome Association Level
Fine Particulate Matter	0.45	Baseline Primary	Highly Significant
Nitrogen Dioxide	0.28	Synergistic Amplification	Significant
Tropospheric Ozone	0.15	Antagonistic at High Levels	Moderately Significant
Volatile Organic Compounds	0.12	Additive Toxicity	Significant

Exploration of the multi-dimensional response surface using non-parametric kernel methods uncovers crucial non-linear interactions. The analysis demonstrates a pronounced synergistic interaction between fine particulate matter and nitrogen dioxide. At lower concentrations of particulate matter, the effect on cardiovascular health remains relatively stable. However, when high levels of particulate matter co-occur with elevated nitrogen dioxide concentrations, the cardiovascular risk accelerates exponentially. Interestingly, the model also detects complex, potentially antagonistic effects with ozone in specific seasonal contexts, where extremely high levels of certain primary pollutants alter the atmospheric chemistry in ways that temporarily depress localized ozone formation, thereby slightly dampening its specific independent health contribution. These highly intricate exposure patterns confirm that public health risk is not a simple linear sum of its parts, but rather a dynamic interaction dependent on the specific composition of the local atmospheric chemistry.

4.3 Mediation Pathways and Biological Mechanisms

The causal mediation analysis bridges the gap between the external pollutant mixtures and the observed clinical outcomes by quantifying the biological pathways involved. When analyzing cardiovascular health outcomes, the inclusion of systemic inflammatory markers as mediators significantly alters the observed relationships. The counterfactual modeling reveals that a substantial proportion of the total effect of the pollutant mixture is operating through the induction

of systemic inflammation. Specifically, biomarkers such as C-reactive protein and interleukin-6 are found to mediate approximately thirty to forty percent of the total effect of the mixture on arterial stiffness and subsequent cardiovascular events.

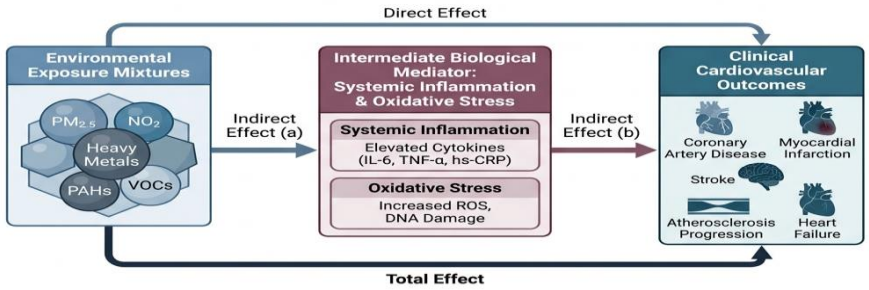


Figure 2Causal Mediation Pathway Diagram Linking Environmental Exposure Mixtures to Clinical Cardiovascular Outcomes

This mediation finding has profound clinical and public health implications. It suggests that while reducing external air pollution remains the primary goal, pharmacological or lifestyle interventions aimed at reducing systemic inflammation could serve as viable secondary prevention strategies for highly exposed urban populations. Furthermore, the analysis of oxidative stress markers indicates a rapid-response pathway, where acute spikes in the pollutant mixture lead to immediate, measurable increases in oxidative damage to lipids and DNA. The rigorous sensitivity analyses, including the calculation of robust E-values, confirm that these mediation effects are highly unlikely to be explained away by unmeasured confounding, lending strong credence to the biological plausibility of the hypothesized mechanisms. The integration of mixture and mediation results thus provides a comprehensive, mechanistically sound understanding of urban environmental pathogenesis.

5. Conclusion and Implications

5.1 Summary of Findings

This comprehensive academic investigation has systematically detailed the integration of mixture analysis and causal mediation frameworks for the assessment of urban environmental exposures. The research demonstrates unequivocally that evaluating single pollutants in isolation is scientifically insufficient for understanding the true health burden of the urban exposome. By utilizing advanced statistical methodologies capable of handling highly correlated and multidimensional data, the study reveals that diverse pollutants, particularly fine particulate matter and nitrogen dioxide, interact synergistically to exacerbate adverse health outcomes. Furthermore, the application of causal inference frameworks has successfully elucidated the biological mechanisms underlying these relationships. The findings confirm that a significant proportion of the toxicity exerted by environmental mixtures operates indirectly through intermediate biological pathways, primarily systemic inflammation and acute oxidative stress. This holistic

methodological approach provides a much more accurate and mechanistically grounded representation of how modern urban environments influence population health dynamics.

5.2 Policy Implications and Strategic Recommendations

The insights generated from integrated mixture and mediation analyses carry urgent and profound implications for environmental regulation and urban public health policy. Current regulatory frameworks, which predominantly rely on isolated limit values for individual criteria pollutants, must evolve to incorporate cumulative risk assessments [31]. Policymakers should consider the development of multi-pollutant indices that trigger regulatory action based on the combined toxicity of the atmospheric mixture, rather than waiting for a single pollutant to breach an arbitrary threshold. Additionally, urban planning initiatives must adopt a more holistic perspective on environmental health. Strategies such as zoning industrial activities, optimizing traffic flow, and expanding urban green spaces should be evaluated not just for their impact on specific emissions, but for their ability to alter the overall mixture composition and mitigate underlying social vulnerabilities [32]. From a clinical perspective, identifying inflammation as a key mediator opens new avenues for targeted health interventions in highly exposed communities. Ultimately, protecting population health in rapidly expanding urban centers requires a paradigm shift towards comprehensive, systems-level environmental health science that fully embraces the complexity of the human exposome.

References

Xi, Y., Zhao, Z., Zhou, Y., Yin, C., Li, Y., Xu, X.,... & Shen, W. (2026). Macrophage efferocytosis mediated by the TP63-RAC2 pathway promotes immunosuppressive remodeling in esophageal cancer. Cell Reports Medicine, 7 (1).

Sinha, D., Tong, Y., Zepeda, M. A. F., Hanstad, G., Nelson, E. C., Theisen, C. O., ... & Gamm, D. M. (2023). Silica nanocapsules as a nonviral delivery platform for iPSC-RPE.Investigative Ophthalmology & Visual Science,64(8), 774-774.

Wang, G., Zhong, Z., Zhou, Y., Li, Y., Wu, E., Cheah, W. P., ... & Shen, L. (2026). X-PCR: A Benchmark for Cross-modality Progressive Clinical Reasoning in Ophthalmic Diagnosis.arXiv preprint arXiv:2604.20350.

GAO, T., LIANG, D., CHEN, X., & TAO, X. (2025). PREDICTIVE ANALYTICS IN PEDIATRIC MENTAL HEALTH: A MACHINE LEARNING APPROACHTO DEPRESSION DETECTION BY PHYSICAL ACTIVITY AND BEHAVIORAL HEALTH INDICATORS. INTERCIENCIA.

He, H., Sun, Z., Chen, X., Tao, X., Tao, M., Dong, D., ... & Qu, C. (2025). Exposure to volatile organic compounds and suicidal ideation: Insights from a US population-based study. Journal of Affective Disorders, 379, 194-203.

Leung, P. H., Wang, F., Li, Z., He, Z., Peng, Y., & Yang, W. F. (2026). Augmented Reality Integration Improves Ergonomics in Dynamic Navigation for Dental Implant Surgery. Journal of the Society for Information Display, 34(5), 428-435.

Tao X, Wu X, Zhuo S, et al. Unveiling the sedentary epidemic through insights from college students in Guangdong[J]. Scientific Reports, 2025, 15(1): 35197.

Yin, L., Tong, Y., Islam, Z. H., Zhang, K., Xie, R., Burger, J.,... & Wang, B. (2024). Targeted NAD+ Delivery for intimal Hyperplasia and Re-endothelialization: a novel anti-restenotic therapy approach. bioRxiv, 2024-02.

Sun, Z., Xu, Y., Liu, Y., Tao, X., Zhou, P., Feng, H., ... & Liu, Z. (2025). Associations of Exposure to 56 Serum Trace Elements with the Prevalence and Severity of Acute Myocardial

Infarction: Omics, Mixture, and Mediation Analysis: Sun et al. Biological Trace Element Research, 203(9), 4466-4478.

Xiao, Y., Xiao-yue, Z., Yue, W., Ruo-tao, L., Xiang-jie, L., Xing-yuan, W., ... & Zhen-yi, J. (2022). Use of computed tomography for the diagnosis of surgical sarcopenia: Review of recent research advances. Nutrition in Clinical Practice, 37(3), 583-593.

Ma, H., Sun, J., Li, X., Luo, Y., Liu, J., & Li, J. (2024). Regulatory immune cells in organ transplantation. Frontiers in Immunology, 15, 1383563.

Chai L, Zhang J, Fernando W G D, et al. Detection of blackleg resistance gene Rlm1 in double-low rapeseed accessions from Sichuan province, by kompetitive allele-specific PCR[J]. The Plant Pathology Journal, 2021, 37(2): 194.

Xu, Z., Lu, Y., Tao, X., Cheng, W., Tian, J., Tang, W., & Wu, D. Modelling the geographic spread of HIV among MSM in Guangdong, China: a metapopulation model considering the impact of pre-exposure prophylaxis Philosophical Transactions A.

Eraslan, G., Avsec, Ž., Gagneur, J., & Theis, F. J. (2019). Single-cell transcriptomics meets deep learning. Nature Methods, 16(11), 1083–1091.

Chai L, Feng B, Liu X, et al. Fine mapping of a locus underlying the ectopic blade-like outgrowths on leaf and screening its candidate genes in rapeseed (Brassica napus L.)[J]. Frontiers in Plant Science, 2021, 11: 616844.

Chen, Z., Zhu, Q., & Zeng, Y. (2026). MWED-Former: A Multi-Scale Weather-Aware Transformer for Emergency Department Demand Forecasting with Weather Bias Correction. Fundamental Scientific Reports in Multidisciplinary Areas, 2 (02), 132-145.

Vieira, C. L., Koutrakis, P., Huang, S., Grady, S., Hart, J. E., Coull, B. A., ... & Garshick, E. (2019). Short-term effects of particle gamma radiation activities on pulmonary function in COPD patients. Environmental research, 175, 221-227.

Wang, G., Ren, J., Shen, L., Cheah, W. P., & Qu, R. (2026). M-MambaS: Multimodal Mamba for small lesion segmentation.Pattern Recognition, 113923.

Xu, X., Bowtell, J., Fong, D. T., Young, W. R., & Williams, G. K. (2025). Kinematics of balance controls in people with chronic ankle instability during unilateral stance on a moving platform. Scientific Reports, 15(1), 1126.

Shi, X., Yu, X., Xiong, Y. J., Lv, T., & Si, X. (2026). Long-term mortality trends in patients with concurrent hernia and diabetes in the United States. Frontiers in Endocrinology, 17, 1723074.

Zhang, Y., & Chen, Y. (2025, May). Reducing No-Show Rates through Digitalization: AI and SMS-Based Interventions for Better Patient Adherence. In Proceedings of the 2025 International Conference on Digital Economy and Intelligent Computing (pp. 123-127).

Chen, Y., Sun, Y., Zhang, X., Chen, Y., Zhao, S., Shi, M., ... & Peng, M. (2025). A nomogram based on coagulation markers for predicting meige syndrome risk.Frontiers in Aging Neuroscience,17, 1665276.

Jiang, Z., Hou, Z., Yu, X., Zhao, Z., Bu, J., Li, C.,... & Wang, C. (2025). SPECT-derived myocardial perfusion and viability as predictors of response to left bundle branch pacing for cardiac resynchronization therapy. Europace, 27 (12), euaf301.

Wang, G., Zhou, Y., Deng, K., Cheah, W. P., Qu, R., Ren, J., & Shen, L. (2026). SurgCoT: Advancing Spatiotemporal Reasoning in Surgical Videos through a Chain-of-Thought Benchmark. arXiv preprint arXiv:2604.20319.

Li, Y., Liao, X., Yu, X., Xiao, Y., Tao, X., & Zhong, T. (2025). Mediating Role of the ANGPTL3/TFPI Protein Ratio in Regulating T-Cell Surface Glycoprotein CD5 Levels on Knee Osteoarthritis (KOA): A Mendelian Randomization Study. International Journal of Molecular Sciences, 26(10), 4471.

Yang, Z., Guo, D., Fu, Y., Qi, L., Zhu, T., Ma, X.,... & Zhao, K. (2025). Investigating the Fracture Treatment Mechanism of Dipsacus asper Based on Network Pharmacology, Molecular Docking and Molecular Dynamics Simulation. Engineered Science, 38, 1856.

Verhoven, B., Tong, Y., Chlebeck, P., Zhong, W., Zeng, W., Jennings, H., ... & Al-Adra, D. P. (2025). Attenuating Ischemia and Reperfusion Injury Using NAD+-Loaded Nanoparticles in Mouse Kidneys.Transplantation Direct,12(1), e1890.

Zhang, W., Luo, Z., Gu, C., Ma, J., Cao, Y., Yuan, W., & Jin, Y. (2025). Bridging the compression-precision paradox: A hybrid architecture for clinical EEG report generation with guaranteed measurement accuracy. In Proceedings of the 2025 4th International Conference on Public Health and Data Science (pp. 201–207).

Xu, X., Bowtell, J., Young, W. R., Fong, D. T., & Williams, G. K. (2025). Distinct patterns in neuromuscular adaptation to repeated perturbations in chronic ankle instability. Journal of NeuroEngineering and Rehabilitation.

Ma, X., Li, J., Wang, Y., Li, S., Guo, J., Shen, W., ... & Wang, H. (2025). Can auditory processing dysfunction indicate early cognitive decline?.Alzheimer's & Dementia: Diagnosis, Assessment & Disease Monitoring,17(4), e70189.

Yu, X., Zhang, Y., & Wang, H. (2025, July). Subjective Sleep Gains in Empty Nose Syndrome: A Cautionary Note on the Need for Objective Validation. In International Forum of Allergy & Rhinology (Vol. 15, No. 7, pp. 761-762).

Zhao, K., Li, G., Li, C., Jiang, T., Wang, W., & Yang, Z. (2026). Identification Markers for Salvia miltiorrhiza and Its Close Relatives Based on Cell Wall Component Characteristics. Engineered Science, 40, 2122.