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REVIEW

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The Urban Exposome: Genomic, Epigenomic, and Evolutionary Consequences Across Species

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Abstract

Urban environments bring together air pollution, heat, noise, artificial light, changing dietary habits, and inequalities in access to green space. This narrative review examines how these exposures relate to molecular changes in humans, microbial communities, and wildlife, presenting an interspecies urban genomics framework.

Findings from human studies mainly concern changes in gene regulation, development, and health across the life course. Responses in microbial communities include shifts in composition and horizontal gene transfer. In some wildlife populations, genetic and experimental evidence support heritable adaptation across generations. Oxidative stress, DNA damage, inflammation, circadian disruption, and epigenetic regulation provide recurring mechanistic links, although their roles vary by exposure and species.

Because most human studies are observational, the molecular associations identified do not, on their own, establish that a particular exposure causes disease. A better understanding of these relationships requires longitudinal cohorts, intervention studies, representative data from low- and middle-income countries, and coordinated monitoring across species. Urbanization also provides health benefits through sanitation, access to services, and well-designed infrastructure. This framework aims to guide research and help policymakers assess these risks and benefits together. Further validation is needed before molecular biomarkers can be used in clinical practice and urban planning.

Keywords: urbanization; urban genomics; urban exposome; environmental epigenomics; microbiome; urban adaptation; One Health

Introduction

The United Nations' 2018 revision projected that 68% of the world's population would live in urban areas by 2050 (United Nations, 2018). Cities concentrate people, infrastructure, and environmental exposures. They also create conditions that can alter physiology, gene regulation, and evolutionary processes in other species (Alberti et al., 2020; Johnson & Munshi-South, 2017).

"Urban" refers to a densely populated and predominantly built-up area characterized by concentrated infrastructure, economic activity, service provision, and interconnected social and environmental systems. Because administrative boundaries alone may not accurately represent urban exposure patterns, this review adopts a functional definition based on population density, built-up land cover, infrastructure intensity, and commuting or mobility linkages (OECD/European Commission, 2020).

Urban heat, pollution, artificial light, noise, habitat fragmentation, and changing dietary and microbial environments form a complex urban exposome (Stingone et al., 2023; Vermeulen et al., 2020). These exposures may influence gene regulation, immune responses, and development across the life course. Persistent effects within an individual should be distinguished from effects transmitted across generations (Renz et al., 2017; Vogli et al., 2025; H. Wu et al., 2023).

Cities serve as new evolutionary laboratories in which processes such as natural selection, gene flow, and genetic drift occur (Alberti et al., 2020; Perry & Göttert, 2024). Urban evolutionary biology has documented rapid, parallel adaptation in a wide variety of taxa responding to common urban stressors. The clearest example is a global survey of white clover (Trifolium repens), which examined 160 cities worldwide and found urban–rural clines in hydrogen cyanide production in 47% of cities, demonstrating recurring but geographically variable evolutionary responses. (M. T. J. Johnson et al., 2018; Santangelo et al., 2022). Similarly, genome-wide parallel adaptation has been observed in urban lizards in numerous cities (Bourgeois et al., 2025; K. M. Winchell et al., 2023). A comprehensive meta-analysis of 167 studies has shown that urbanization alters gene flow or genetic drift. However, the direction of these effects—whether cities act as a barrier or a channel for dispersal varies by species and context (Miles et al., 2019).

Planetary Health connects human health with the integrity of natural systems, while One Health links human, animal, and environmental health, including zoonoses and antimicrobial resistance (Gao, 2021; Logan et 2018; Talukder et al., 2024). Together with urban evolutionary biology, these approaches support the view of cities as interacting social, ecological, and biological systems (Alberti et al., 2020; Roches et al., 2021).

Urbanization can also improve health through better access to healthcare, education, sanitation, social services, and, where equitably distributed, walkable and green infrastructure. These benefits do not negate urban risks; rather, they show that biological outcomes depend on how urban environments are designed, governed, and distributed across populations (Galea & Vlahov, 2005; Vlahov et al., 2007).

This narrative review examines the effects of shared urban exposures on human gene regulation and health, microbial communities, and evolutionary change in wildlife. It brings together findings from different research fields. In evaluating this evidence, it considers whether observed associations support causality and whether biological changes occur within an individual's lifetime or are inherited by subsequent generations. It also identifies research priorities and discusses how reducing exposures and creating more equitable urban living conditions could support health.

The proposed urban genomics framework brings together established research approaches. Exposome science examines environmental exposures that accumulate across the life course. One Health focuses on the connections between human,

animal, and environmental health. Urban evolutionary biology investigates processes such as selection, gene flow, and genetic drift in populations living in cities. The contribution of this review is to connect these fields through shared urban exposures while distinguishing their biological responses. Physiological and molecular changes within a human lifetime, shifts in microbial community composition and horizontal gene transfer, and heritable adaptation across generations in wildlife are treated as distinct processes. The framework allows these findings to be interpreted together while preserving these differences. This approach is a conceptual framework that brings together existing knowledge, rather than a model that establishes causal relationships. However, the breadth and strength of evidence vary across species. Limited longitudinal studies and data from low- and middle-income countries make it difficult to generalize findings to different populations and urban settings.

Review approach and scope

This article is a narrative review integrating evidence from environmental health, epigenomics, microbiome research, and urban evolutionary biology. The literature search was conducted in PubMed and Google Scholar between 1 March and 30 August 2026 using the keywords "urbanization," "urban genomics," "urban exposome," "environmental epigenomics," "microbiome," "urban adaptation," and "One Health." The reference lists of relevant publications were also examined to identify additional studies within the scope of the review. AI-assisted tools, including Elicit and NotebookLM, were used to support literature exploration and information organization. The review provides a critical conceptual synthesis rather than a systematic review or a new quantitative meta-analysis. Study designs and limitations are considered when interpreting exposure–response relationships and claims of heritable adaptation.

2 Urban exposures and molecular consequences

The urban exposome comprises the chemical, physical, biological, social, and behavioral exposures associated with city life. Air pollution, heat, noise, artificial light, dietary change, and reduced access to biodiverse green space often occur together. Shared pathways include oxidative stress, inflammation, neuroendocrine and circadian disruption, and altered gene regulation (Chakif & Furrer, 2026; Daiber & Münzel, 2020; Münzel, Sørensen, et al., 2025; Pandics et al., 2023; Rajagopalan et al., 2024). Figure 1 separates these exposures from molecular responses, individual health outcomes, and population-level evolutionary processes.

2.1 Industrial pollution

Industry and mining contaminate air, soil, water, and sediments with metals, metalloids, petroleum-derived compounds, and other chemicals. Persistent contaminants can accumulate in organisms and sustain exposure in cities and surrounding ecosystems (Frings et al., 2024; H. Wu et al., 2023; Yu et al., 2024).

Heavy metals can alter gene regulation, with reported associations involving neurodevelopmental and neurodegenerative outcomes (Yu et al., 2024). Cadmium has been linked to changes in DNA methylation, histone modifications, and non-coding RNAs (Frings et al., 2024). Experimental work also suggests effects on embryonic stem-cell differentiation (Kim et al., 2024). Oxidative DNA damage, impaired repair, and mitochondrial dysfunction provide additional mechanisms (H. Wu et al., 2023), while metabolic and miRNA changes may modify cellular responses (Xu et al., 2022). These findings support biological plausibility, but their relevance depends on dose, tissue, and study design.

Industrial and consumer-product exposures also include PFAS, phthalates, bisphenols, and flame retardants, often as mixtures (Maddela et al., 2022). Reported molecular responses include altered DNA methylation, chromatin regulation, and non-coding RNAs, although effects depend on the compound, dose, tissue, and study design (S. Kim et al., 2021; Ye et al., 2026). Associations between flame-retardant exposure and sperm methylation raise questions about reproductive effects but do not establish transmission to offspring (Soubry et al., 2017).

A metagenomic meta-analysis found enrichment of some multidrug efflux pumps and mobile genetic elements in chemically polluted environments (Subirats et al., 2023). These findings are consistent with selection or community shifts that favor resistance-associated traits. They do not, by themselves, demonstrate that industrial exposure caused new resistance mutations or horizontal transfer.

Plant-based remediation can complement source-control strategies by sequestering or stabilizing metals and organic pollutants in contaminated urban soils. Some species may also serve as biomonitors of atmospheric pollution, although effectiveness depends on pollutant type, local ecology, and long-term management (Kiran & Prasad, 2017). Heavy-metal exposure also imposes species-specific physiological pressures on urban vegetation; experimental evidence in Plantago species demonstrates differences in germination, seedling growth, and tolerance under Zn, Cu, and Fe exposure (Ltaeif et al., 2024).

2.2 Air pollution

Urban air pollution is a dynamic mixture of particulate matter, gaseous pollutants, metals, and organic compounds whose composition shifts with the local emission profile and the season (Potì et al., 2026) . Fine ($PM_{2.5}$) and ultrafine particles can reach the alveoli and enter systemic circulation, carrying metals such as Fe, Cu, Ni, V, Co, Cr, as well as polycyclic aromatic hydrocarbons (PAHs) (Hamanaka & Mutlu, 2025). Traffic contributes both exhaust gases, particularly NO_2, and non-exhaust particles generated by brake, tire and road-surface wear, which are enriched in redox-active metals (Cu, Fe, Ba, Zn) and now represent the dominant primary traffic-related PM fraction in many European cities (Fussell et al., 2022). Residential heating is a second, strongly seasonal source: in a modeling assessment of 708 European urban areas, the residential sector was one of the main contributors to urban $PM_{2.5}$ in 56% of cities, largely through solid-fuel and wood combustion in domestic

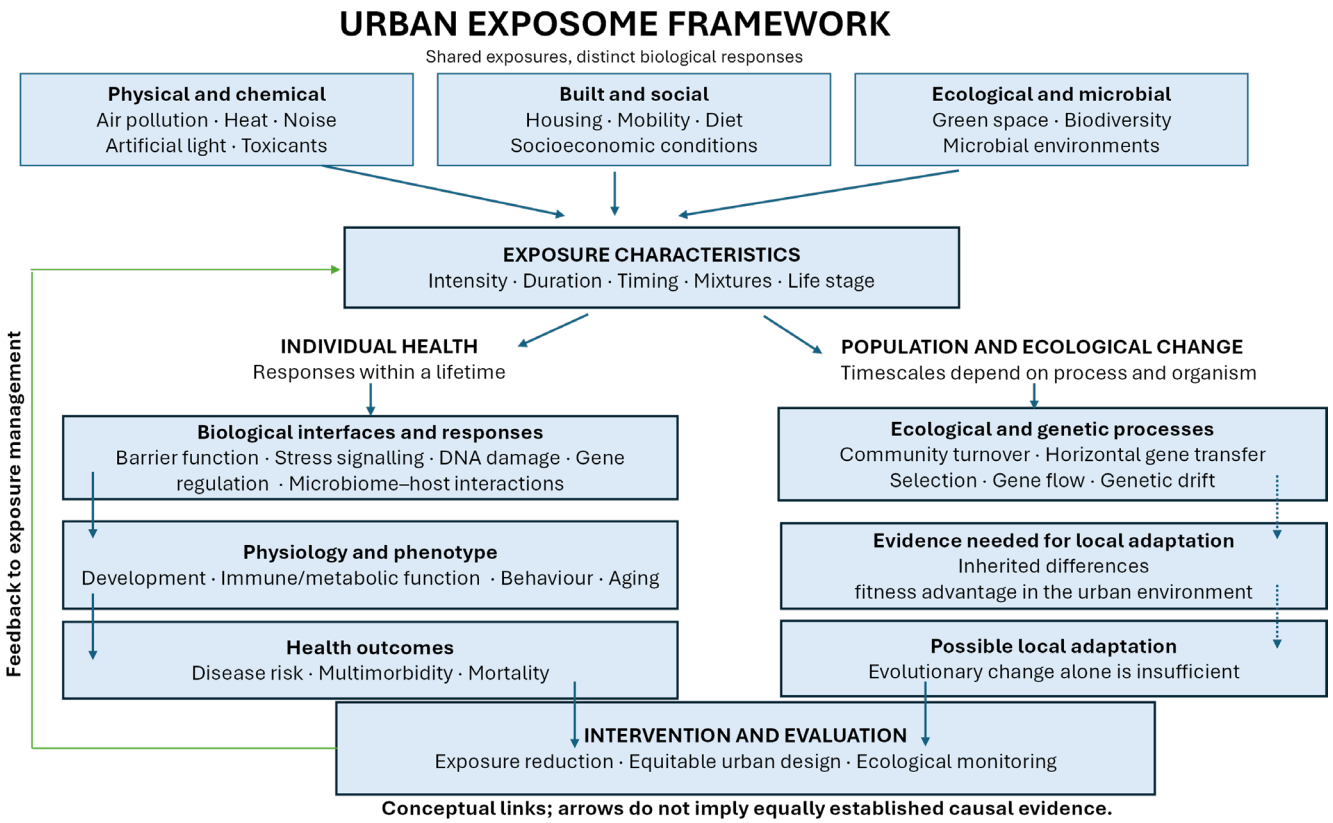


Figure 1. Organization of the urban exposome framework. Urban conditions shape measurable exposures, which act through biological interfaces and molecular response pathways to influence biological states and phenotypes. Individual health outcomes and population-level evolutionary processes are separate branches: a molecular change within an individual does not establish heritable adaptation. Intervention and feedback connect measured outcomes to exposure management. The arrows organize proposed relationships and should not be read as equally established causal links (Alberti et al., 2020; Lambert et al., 2021; Vermeulen et al., 2020; Wild, 2005)

appliances (Zauli-Sajani et al., 2024). Indoor sources add a further contribution, with high-temperature cooking generating reactive compounds such as acrolein. Airborne micro- and nanoplastics (MNPs), released from textiles, tire wear, and the weathering of urban plastic debris, are an emerging component of the urban aerosol: microplastic particles smaller than 5.5 μm have been recovered from human lung tissue at autopsy (Amato-Lourenço et al., 2021), although human exposure estimates and dose–response relationships remain poorly constrained (Gou et al., 2024).

Globally, ambient air pollution is estimated to account for roughly 4.2 million premature deaths each year (World Health Organization [WHO], 2021). Source apportionment shows that the dominant contributors differ by region: residential energy use in South and East Asia, agriculture in Europe and North America, and traffic and power generation elsewhere (Hamanaka & Mutlu, 2025; Lelieveld et al., 2015; Seaman et al., 2009). This matters for urban policy because the intervention that reduces exposure the most is not the same in every city. Air pollutants can cause chemical groups to bind to DNA, forming modifications known as DNA adducts (Montano et al., 2025). They can also disrupt the cell's DNA repair mechanisms (Hartwig et al., 2002), and cause DNA damage and strand breaks through oxidative stress (Uddin, 2025). Dysfunction of mitochondria, which are responsible for cellular energy production, may amplify these harmful effects (Breton et al., 2019). Changes in mitochondrial DNA and in the number of mitochondrial DNA copies within cells may indicate cellular stress. However, their significance should be interpreted in relation to the tissue examined and the characteristics of the exposure (Avilés-Ramírez et al., 2022).

The resulting inflammatory and oxidative burden can also impair vascular function. Excess superoxide reacts with nitric oxide (NO) to form peroxynitrite, impairing endothelial nitric oxide synthase (eNOS) activity and damaging the endothelial barrier (Li et al., 2023). These changes can promote vasoconstriction, hypertension, and atherosclerosis, linking air pollution exposure to broader cardiovascular consequences (Seneviratne & Miller, 2025). A mechanistic review of air pollution and cardiovascular disease places these steps in sequence—pulmonary oxidative stress, systemic inflammation, autonomic imbalance, and endothelial dysfunction—and

concludes that the vascular effects of $PM_{2.5}$ are among the best-established non-pulmonary consequences of urban air pollution (Al-Kindi et al., 2020).

2.3 Urban heat

Urban heat islands arise when vegetation is replaced by heat-retaining surfaces and buildings reduce cooling. Heat intensity varies with city size and background climate (Manoli et al., 2019). Across 13,115 cities, total exposure to extreme heat tripled between 1983 and 2016, reflecting both population growth and warming (Tuholske et al., 2021). Heat can strain cardiovascular, renal, and thermoregulatory systems (Ebi et al., 2021), and exposure is often greater in disadvantaged neighborhoods (Hsu et al., 2021). Surface temperature, ambient temperature, and individual heat exposure are related but distinct measurements.

Heat shock responses

Heat stress disrupts protein folding and activates the heat-shock response. Intracellular heat-shock proteins help maintain protein function and limit injury, whereas extracellular proteins can also stimulate inflammation. Experimental respiratory evidence suggests effects on epithelial barriers and airway responses, but these depend on exposure and model conditions (Celebi Sozener & Hoffmann, 2023; Hu et al., 2022; Iba et al., 2025).

Repeated heat exposure can produce physiological acclimatization and cellular protective responses (Laitano et al., 2025; Lin et al., 2021). Human cohorts with different thermal histories show differences in heat tolerance and autonomic or vascular responses (Lin et al., 2021). These findings concern acclimatization and should not be treated as evidence of heritable human adaptation to cities.

2.4 Environmental noise

Traffic noise can activate physiological stress responses even during sleep or without conscious awareness (Arregi et al., 2024; Yang et al., 2024; Spreng, 2000, 2004). Noise-related stress can activate sympathetic signaling and the hypothalamic–pituitary–adrenal axis, increasing catecholamine and cortisol release. Repeated activation may contribute to sustained cardiovascular stress (Daiber et al., 2020; Yang et al., 2024). Repeated noise exposure can activate stress responses and contribute to vascular oxidative stress and endothelial dysfunction (Hahad et al., 2022; Tabaei et al., 2025). Experimental findings also support neuroinflammatory responses, but clinical neurological consequences require cautious interpretation (Hahad et al., 2022). Meta-analytic estimates associate each 10 dB(A) increase in road traffic noise with an approximately 4–5% increase in the risk of ischemic heart disease, stroke, and heart failure (Münzel et al., 2021; Münzel, Kuntic, et al., 2025). These risk estimates describe population associations rather than a uniform effect in every individual.

2.5 Artificial light at night

Exposure to artificial light at night can disrupt the body's natural day–night rhythm. Light-sensitive cells containing melanopsin in the retina transmit information about environmental light to the brain's central biological clock. Nighttime light exposure can therefore alter sleep and wake timing and reduce melatonin secretion, a hormone normally released at night. These changes may affect cardiovascular health and metabolism. However, the potential effects depend on the timing and duration of exposure, as well as the intensity and wavelength of the light (Korf et al., 2025; Meléndez-Fernández et al., 2023). Assessing these effects requires consideration of the light an individual actually encounters at night; measurements of sky brightness alone are insufficient to determine personal light exposure (Falchi et al., 2016).

2.6 Green space and biodiversity

The Biodiversity and Old Friends hypotheses propose that contact with environmental microorganisms supports immune regulation. Reduced contact with biodiverse environments may alter human microbial communities and immune responses, although effects depend on the organisms and exposure context (Haahtela, 2019; Rook et al., 2014).

In a study of 118 adolescents in eastern Finland, environmental biodiversity and skin microbiota were associated with allergic sensitization (Hanski et al., 2012). This supports a link between environmental conditions, microbial contact, and immune outcomes, but the observational design does not establish causality.

Comparisons of Amish and Hutterite children provide complementary human and experimental evidence. Asthma and allergic sensitization were respectively four and six times less prevalent among Amish children. Amish house-dust extracts protected mice against experimental airway responses, and this protection depended on MyD88/TRIF signaling (Saranz, 2017). Separate experiments implicated the epithelial regulator A20 in the protection conferred by exposure to farm dust and endotoxin (Schuijs et al., 2015). These findings support specific immune mechanisms rather than a protective effect of all microbial exposures.

In Finnish preschool studies, adding forest floor materials to play areas increased the children's skin and gut microbial diversity. This change, accompanied by an increase in Treg frequency, plasma TGF-β1 levels, and the IL-10:IL-17A ratio, suggests that environmental microbial exposure may enhance the immune regulatory balance (Barrable & Robinson, 2026; Roslund et al., 2020, 2022).

Microbial metabolites, including short-chain fatty acids, can support immune regulation and epithelial barriers. The Microbial Peace Signal Hypothesis extends this idea to lifelong host–microbe interactions, but its urban-health implications remain to be tested (Han, 2026; Rook, 2023). Selected probiotic strains also reduce inflammatory signaling in epithelial models; these in vitro results do not establish clinical benefit or mitigation of urban exposures (Boyajian et al., 2024).

Green space is associated with better health through several potential routes, including cooling, reduced noise, physical activity, stress reduction, and microbial contact (Dzhambov et al., 2023; Heo & Bell, 2021; Gorman et al., 2019). Associations with physiological and molecular markers are promising, but their contributions to clinical outcomes and their variation across socioeconomic groups require further study (Arriazu-Ramos et al., 2026; Heimlich-McQuarters et al., 2025; Knobel et al., 2025).

Urban vegetation can reduce heat and some pollutant exposures, but it can also alter pollen abundance, season length, and allergenicity. Warming, elevated CO_2, air pollution, and the selection of highly allergenic species may increase aeroallergen exposure and interact with epithelial barrier dysfunction. Green-infrastructure planning should therefore combine biodiversity and cooling goals with allergen-aware species selection and long-term pollen monitoring (Anderegg et al., 2021; Ziska et al., 2019).

2.7 Shared molecular pathways

Urban stressors do not operate independently. While the circadian clock regulates redox homeostasis, oxidative stress affects clock proteins and antioxidant networks, including NRF2. Thus, air pollution, noise, and artificial nightlight converge within a common biological network in which oxidative stress, neuroendocrine activation, and circadian disruption intersect (Arregi et al., 2024; Ding et al., 2024). The principal pathways and their biological crosstalk are synthesized in Figure 2, while exposure-specific evidence is summarized in Table 1.

Mitochondria connect link energy metabolism to oxidative stress responses. Pollutants and sustained neuroendocrine signaling may alter mitochondrial function, mtDNA integrity, and copy number. Released mtDNA can also activate innate immune pathways (Picard et al., 2022; Picard & Mcewen, 2019; Reddam et al., 2022). Maternal–fetal associations should be interpreted as possible developmental effects, rather than proof of germline inheritance.

2.8 Dietary transition

Urbanization significantly alters dietary habits, access to food, and lifestyle. The prominence of cheap, readily available, and palatable ultra-processed foods (UPFs) in urban food environments can increase the risk of excessive energy intake and obesity (Dicken & Batterham, 2024). In a prospective cohort of 138,076 adults from five continents, consuming at least two servings of UPFs per day was associated with a 28% increased risk of all-cause mortality compared to non-consumers (Dehghan et al., 2023)

Urban–rural comparisons in Xhosa populations linked dietary differences to altered gut microbial composition and metabolic profiles, including bile-acid metabolism (Ramaboli et al., 2024). These associations suggest possible dietary–microbial pathways but cannot isolate diet from other urban exposures.

Urban dietary change may also relate to epigenetic regulation (Cronjé et al., 2020). In an EWAS meta-analysis of 3,152 children, suggestive associations with ultra-processed food consumption did not survive multiple-testing correction (Llauradó-Pont et al., 2025). In 760 LifeLines-DEEP participants, dietary quality was associated with epigenetic aging measures (Bordoni et al., 2024). These findings remain observational and do not establish methylation as a mediator of diet-related disease.

Evidence linking urban exposures to health effects is more convincing when findings from population studies are supported by experiments or studies that assess the effects of reduced exposure. Molecular changes alone do not show how much the risk of disease increases. People are also exposed to multiple urban stressors simultaneously, and their socioeconomic circumstances and daily habits can influence their health. These overlapping factors make it difficult to isolate the contribution of a single exposure.

2.9 Genomic and regulatory responses

Urban exposures can contribute to DNA damage and genomic instability. They can also alter gene regulation through epigenetic mechanisms without changing the underlying DNA sequence (J. Y. Kim & Kim, 2026; Martin & Fry, 2018; Mukherjee et al., 2021; Real et al., 2021; Vu et al., 2025). Non-coding RNAs and developmental programming provide further routes through which exposure histories may influence later health (Abarca-Castro et al., 2025; Alfano et al., 2018; H. Wu et al., 2023). The following sections distinguish these mechanisms from population-level evolutionary change.

2.9.1 DNA damage and genomic instability

Oxidative DNA damage

Experimental and human exposure studies report oxidative DNA lesions, strand breaks, and DNA adducts following particulate exposure (Bräuner et al., 2007; Poetsch, 2020; Quezada-Maldonado et al., 2021). Damage and impaired repair are distinct processes; persistent lesions may contribute to genomic instability when repair capacity is exceeded.

DNA double strand breaks

Replication stress and unrepaired lesions can generate DNA double-strand breaks. ATM-mediated H2AX phosphorylation helps recruit repair and checkpoint proteins, and increased γ-H2AX has been reported in particulate-exposed epithelial models (Bessa et al., 2022; Burhans & Weinberger, 2007; Gao et al., 2016; Maliszewska-Olejniczak et al., 2025; Merlin et al., 2022). Unresolved damage may lead to genomic rearrangements, cell death, or senescence; the outcome depends on the cellular context.

Mutational signatures

The Sherlock-Lung study examined cancer genomes from 871 never-smokers across 28 geographical locations. Higher estimated $PM_{2.5}$ exposure was associated with greater somatic mutation burden, shorter telomeres, and increased contributions from signatures including SBS4 and SBS5 (Díaz-Gay et al., 2025). These associations complement experimental evidence

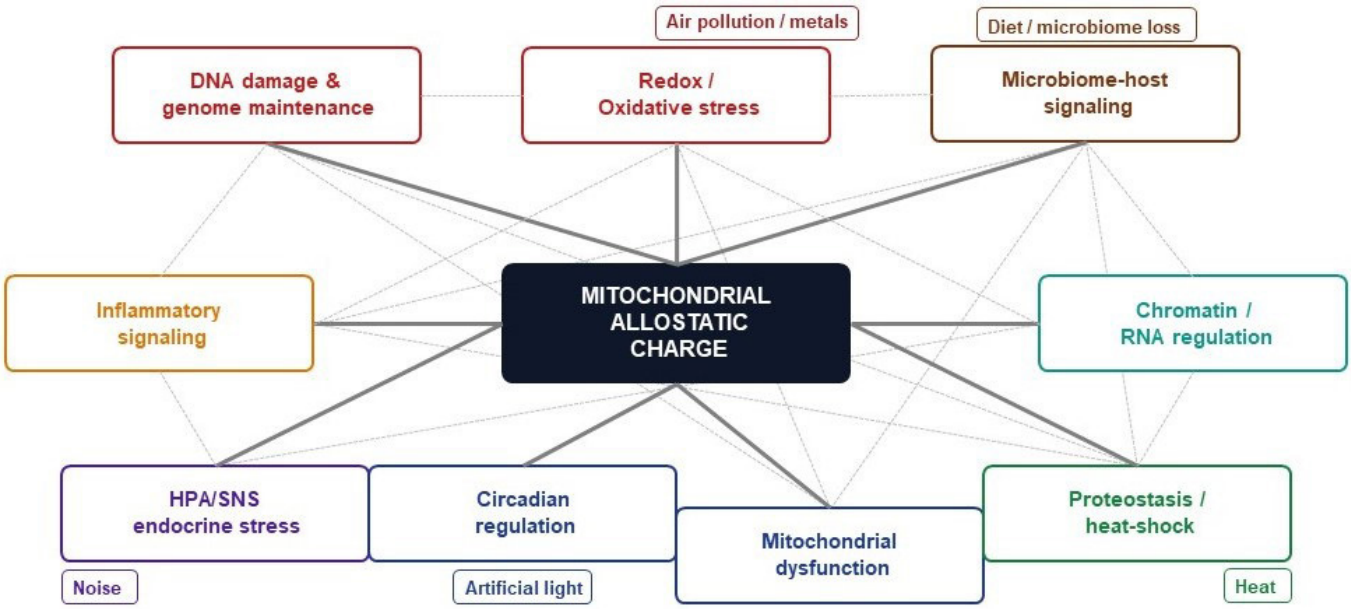


Figure 2. Shared molecular response network. Solid arrows connect illustrative exposure domains with response modules; dashed lines denote proposed interactions between modules and do not specify direction or equal evidence strength. The network includes oxidative stress, DNA damage, inflammatory and endocrine signaling, circadian regulation, mitochondrial dysfunction, proteostasis, chromatin/RNA regulation, and microbiome–host signaling. Mitochondrial allostatic load is an integrative concept, rather than a measured universal mediator. HPA, hypothalamic–pituitary–adrenal axis; SNS, sympathetic nervous system (Anckar & Sistonen, 2011; Fonken & Nelson, 2014; Kelly & Fussell, 2015; Kuntić et al., 2024; Lawal, 2017; Münzel et al., 2017, 2018; Su et al., 2019).

of tumor-promotion but do not identify a single mechanism shared by all pollutants and cancers.

Telomere shortening

Telomeres are vulnerable to oxidative damage, which can impair replication and contribute to shortening (Barnes et al., 2019; Coluzzi et al., 2014; Opresko et al., 2025). Exposure studies report associations with telomere measures, but interpretation depends on tissue and method (Díaz-Gay et al., 2025; Ward-Caviness et al., 2016). Critically damaged telomeres can induce senescence, and senescent cells may produce inflammatory signals through the senescence-associated secretory phenotype (Bessa et al., 2022; Peters et al., 2021).

Occupational and environmental exposures to pollutants and pesticides similarly alter telomere length, reinforcing telomeres as integrative biomarkers of combined genetic susceptibility and environmental burden (Kahl & Silva, 2016; Shoeb et al., 2021; Srinivas et al., 2020).

2.9.2 Epigenetic regulation

Epigenetic regulation changes gene activity without changing the DNA sequence. DNA methylation, histone modifications, and chromatin organization are responsive to environmental conditions (Caizergues et al., 2022; J. Y. Kim & Kim, 2026; Watson et al., 2020). Pollutant-associated differences have been reported across these layers (Kim & Kim, 2026; H. Wu et al., 2023). Such changes may be transient or persistent; they do not, by themselves, establish disease causation or inheritance through the germ line.

DNA methylation

DNA methylation is the most widely studied epigenetic mark in environmental epidemiology (E. M. Martin & Fry, 2018). Pollutant-associated changes can include global or locus-specific hypo- and hypermethylation, but their direction varies with exposure, tissue, and developmental stage (Alfano et al., 2018; Fussell et al., 2024; Luyten et al., 2018; Yauk et al., 2008). A meta-analysis of four European birth cohorts linked prenatal NO_2 exposure to differences in cord-blood methylation of mitochondria-related genes, with independent replication (Gruzieva et al., 2017).

Across seven European birth and adult cohorts, the EXPANSE project identified exposure- and life-stage-specific associations with blood DNA methylation. Enriched pathways included immune responses in children and immune-checkpoint signaling

Table 1. Representative exposure-specific molecular evidence. Study context and interpretation are separated to distinguish measured associations from proposed mechanisms. The examples are illustrative and do not cover every urban exposure. $PM_{2.5}$ denotes particulate matter with an aerodynamic diameter of 2.5 μm or less; NO_2, nitrogen dioxide; CpG, cytosine–phosphate–guanine site; FDR, false discovery rate.

Urban Stressor	Genomic or Epigenomic Consequence	Evidence Context	Interpretation
Traffic-related air pollution ($PM_{2.5}$, NO_2, black carbon, and ultrafine particles)	Widespread alterations in DNA methylation, affecting pathways related to inflammation, cardiometabolic signaling, immune responses, the nervous system, and metabolism (E. M. Martin & Fry, 2018)	In a randomized crossover study of 35 healthy adults, Du et al. (2022) reported significant changes at 68 CpG sites after roadside exposure; 49 sites were hypermethylated and 19 were hypomethylated (X. Du et al., 2022).	Even short-term exposure may elicit an epigenetic response; persistent disease effects and causal mediation require further confirmation (X. Du et al., 2022; Rider & Carlsten, 2019).
Long-term traffic noise	Differentially methylated regions associated with inflammation, cellular immunity, cell growth and proliferation, and cardiovascular, auditory, neurological, and embryonic-development pathways (Eze et al., 2020).	Across 2,542 observations from 1,389 SAPALDIA participants, Eze et al. (2020) reported 14, 48, and 183 differentially methylated regions for aircraft, railway, and road traffic noise, respectively (Eze et al., 2020).	Noise and air pollution may generate independent but partly overlapping epigenetic signatures; additional analyses are needed to clarify mediating mechanisms (Arregi et al., 2024; Eze et al., 2020).
Prenatal NO_2 exposure	Prenatal NO_2 exposure has been associated with cord-blood methylation differences at CpG sites in mitochondria-related genes (Gruzieva et al., 2017).	Gruzieva et al. (2017) used an epigenome-wide approach to examine associations of prenatal NO_2 air pollution exposure with DNA methylation in a mother–child setting (cord blood), and reported FDR-significant CpG associations in mitochondria-related genes (Gruzieva et al., 2017).	These associations do not establish that methylation differences mediate later disease risk.
Residential greenness and green infrastructure	Differentially methylated regions with enrichment for pathways related to allergic sensitization, physical activity, and allostatic load (Jeong et al., 2022).	Jeong et al. (2022) assessed greenness at 30 x 30 m and within a 500-m buffer in 1,938 blood samples from 982 participants, identifying 163 and 56 differentially methylated regions, respectively; no genome-wide significant associations were detected at the individual CpG level (Jeong et al., 2022).	The associations vary with spatial scale; proposed links to immune regulation and behavior require causal testing (Jeong et al., 2022).
Mixtures of urban chemical and social exposures	Multiple molecular changes within the internal exposome, reflecting correlated exposure clusters and their biological responses rather than a single pollutant (Vermeulen et al., 2020)	Urban exposome exposures are heterogeneous, variable, correlated, and occur as mixtures, and any single omics layer may capture only part of their effects (Niedzwiecki et al., 2019; Vermeulen et al., 2020)	Urban genomic outcomes should be interpreted using mixture modeling, repeated measurements, and mediation analyses rather than single-exposure models (Maitre, Guimbaud, et al., 2022; Niedzwiecki et al., 2019)

Note: The examples were compiled for this narrative review. They are not a systematic inventory of evidence.

in adults (Vogli et al., 2025). Greenness-related signatures differed from pollutant-related signatures, but this contrast alone does not establish a protective function.

The Multi-Ethnic Study of Atherosclerosis (MESA) identified specific CpG sites in circulating monocytes that directly correlated with long-term $PM_{2.5}$ exposure (Chi et al., 2016). Prenatal and childhood cohorts further identified differentially methylated positions (DMPs) and regions (DMRs) targeting neural development, behavioral control, and immune pathways (Chi et al., 2016; Ghazi et al., 2021; Luyten et al., 2018).

Histone and chromatin regulation

Histone modifications provide another regulatory layer. Single-cell EpiTOF studies reported $PM_{2.5}$-associated differences in acetylation, ubiquitination, and methylation marks across immune-cell types (Jung et al., 2024). Experimental findings support biological plausibility, while dose, timing, and tissue context remain important for interpretation (J. Y. Kim & Kim, 2026; Klibaner-Schiff et al., 2024).

Experimental studies link particulate exposure to altered histone deacetylase expression and inflammatory transcription (Mukherjee et al., 2021). Changes involving HDAC9, KDM2B, and regulatory regions of thermogenic and antioxidant genes have also been reported in metabolic tissues (Palanivel et al., 2025). Translation of these findings to human urban exposure requires attention to dose and tissue context.

Cellular memory and inheritance

Epigenetic memory describes the persistence of altered gene-regulatory states after a stimulus has diminished. It includes stable cell-identity programs and transcriptional memory, in which previously stimulated genes respond more rapidly to later exposure (D'Urso & Brickner, 2014; Ge & Brickner, 2024). Experimental mechanisms include persistent chromatin marks, nuclear-pore interactions, and poised RNA polymerase II (D'Urso et al., 2016; Sump et al., 2022). Memory-like responses have been demonstrated in interferon-stimulated human cells, epithelial stem cells, and trained innate immunity (Kamada et al., 2018; Larsen et al., 2021; Light et al., 2013; Naik et al., 2017; Netea et al., 2020). These models establish mechanistic possibilities rather than direct evidence for persistent responses to urban exposures.

Repeated pollution peaks, heat waves, or noise could influence responses to later exposures through cellular memory. Direct evidence in human urban-exposure settings remains limited. Testing this hypothesis requires repeated sampling and cell-resolved analyses. Persistence through cell division must be distinguished from inheritance through the germ line.

Epigenetic clocks

Epigenetic clocks use DNA methylation to estimate chronological age, aging-related risk, or the pace of aging. Their targets differ, so age-acceleration and pace measures are not interchangeable. Some measures predict disease or mortality, but interpretation requires validation in the relevant population and tissue (Levy et al., 2024; Y. Zhang et al., 2026).

Cohort studies report associations between air pollution and several epigenetic aging measures, with results varying by exposure and clock (Alfano et al., 2018; Ward-Caviness et al., 2016; Yao et al., 2024). A quasi-experimental study linked short-term personal $PM_{2.5}$ exposure to changes in Horvath, Hannum, and GrimAge acceleration, DunedinPoAm, a mortality-risk score, and epiTOC. Statistical mediation analyses implicated biomarkers, including EC-SOD and sCD40L, but do not establish permanent acceleration of aging (X. Gao et al., 2022). In the Health and Retirement Study, associations with high heat and low $PM_{2.5}$ were stronger on some clocks than those with joint high exposure; this does not establish synergy (Dang et al., 2025). Population and tissue validation remain priorities (Baki Yildirim et al., 2026).

Limits of epigenome wide association studies

Urban EWAS findings are vulnerable to residual confounding, reverse causation, tissue and cell-composition differences, batch effects, multiple testing, and limited replication across populations. Methylation differences may represent exposure biomarkers, mediators, disease consequences, or adaptive responses. Stronger inference requires prespecified analyses, cell-type adjustment, repeated sampling, independent replication, ancestry-diverse cohorts, and integration with genetic instruments or experimental evidence (Heijmans & Mill, 2012; Michels et al., 2013).

2.9.3 Non-coding RNAs

Non-coding RNAs regulate gene expression through effects on mRNA stability, translation, and chromatin organization (Hossam Abdelmonem et al., 2025; Miguel et al., 2020; Y. Zhang et al., 2024). Pollutant-associated changes in miRNA and lncRNA have been reported (Aghaei-Zarch et al., 2023; He et al., 2023; J. Y. Kim & Kim, 2026; Miguel et al., 2020), but their persistence and functional significance require validation in the relevant tissue.

Chronic $PM_{2.5}$ exposure selectively downregulates immunomodulatory miRNAs such as *miR-125b* and *miR-21* in proportion to the oxidative potential of the particulate burden (H. Chen et al., 2020; He et al., 2023). Conversely, *miR-146a-5p* is upregulated up to six-fold in monocytes and bronchial epithelial cells following acute PM exposure, functioning as an endogenous negative-feedback brake on TLR/NF-κB signaling by targeting *IRAK1* and *TRAF6* (Kundu & Basu, 2021; Shang et al., 2021). Because *IRAK1* and *TRAF6* also regulate autophagy, their suppression simultaneously activates protective clearance of internalized particles and damaged mitochondria (Shang et al., 2021). However, when *miR-146a-5p* is overwhelmed or inhibited, IL-8-driven chronic unresolved inflammation ensues (Shang et al., 2021).

Changes in circulating miR-495-3p, miR-223-5p, and miR-194-3p have been reported in a Beijing exposure setting (T. Zhou et al., 2018). These are candidate exposure or injury markers; wider clinical use requires independent replication and validation. $PM_{2.5}$ exposure has been associated with lower circulating levels of several lncRNAs. Statistical mediation analyses suggested

Table 2. Representative epigenetic findings associated with urban exposures. Study population, molecular outcome, and reported pathways are distinguished. Pathway enrichment does not establish functional change or disease mediation. DMR, differentially methylated region; CpG, cytosine–phosphate–guanine site; FDR, false discovery rate.

Exposure	Study and Population	Epigenetic Change	Biological Pathways	Citation
Traffic-related air pollution	Du et al. (2022); 35 healthy adults in Shanghai; randomized crossover roadside-versus-park exposure	Significant changes at 68 CpG sites: 49 hypermethylated and 19 hypomethylated.	Cardiovascular signaling, cytokine signaling, immune responses, nervous-system signaling, and metabolism.	(X. Du et al., 2022)
Aircraft, railway, and road traffic noise	Eze et al. (2020); 1,389 SAPALDIA participants and 2,542 observations	Fourteen, 48, and 183 differentially methylated regions for aircraft, railway, and road traffic noise, respectively; no individual CpG reached FDR < 0.05.	Inflammation, cellular immunity, cell proliferation, and cardiovascular and neurological development pathways.	(Eze et al., 2020)
NO_2 and $PM_{2.5}$	Eze et al. (2020); long-term ambient exposure and a peripheral-blood EWAS (Eze et al., 2020).	Increased methylation at NO_2-associated cg08500171 and decreased methylation at $PM_{2.5}$-associated cg17629796 were replicated; eight DMRs for NO_2 and 71 for $PM_{2.5}$ were reported	Enrichment related to C-reactive protein, body mass index, kidney function, and allostatic load.	(Eze et al., 2020)
Residential greenness	Jeong et al. (2022); 982 SAPALDIA participants and 1,938 blood samples.	A total of 163 differentially methylated regions were associated with greenness at 30 x 30 m and 56 with a 500-m buffer; no genome-wide significance was observed at the individual CpG level	Allergic sensitization, physical activity, and allostatic load; more proximal greenness may show a stronger biological association.	(Jeong et al., 2022)
Urban versus forest habitat	Watson et al. (2020); liver and blood from great tits (Parus major) inhabiting urban and forest habitats	Genome-wide DNA methylation differences between urban and forest birds; in liver, differentially methylated regions were enriched in regulatory genomic regions and expressed genes	Steroid biosynthesis, superoxide and ROS metabolism, cholesterol transport, chylomicron remodeling, and epithelial-cell proliferation	(Watson et al., 2020)
Prenatal NO_2	Epigenome-wide meta-analysis, four European birth cohorts, cord blood	FDR-significant CpG associations, replicated in an independent cohort	Mitochondria-related genes	(Gruzieva et al., 2017)

that these differences could account for part of the association with inflammatory markers, but cannot establish a causal molecular pathway (Diao et al., 2023).

Air pollution exposure has also been associated with alterations in circulating extracellular vesicle-associated microRNA (EV-miRNA) profiles. In the MADRES pregnancy cohort, $PM_{2.5}$ and PM_{10} exposures were associated with multiple plasma EV-miRNAs, particularly during late pregnancy (Foley et al., 2024). However, the detection of exposure-associated miRNAs in EV-containing preparations does not by itself demonstrate their vesicular localization, functional delivery to recipient cells, or subsequent epigenetic effects. Given the het-

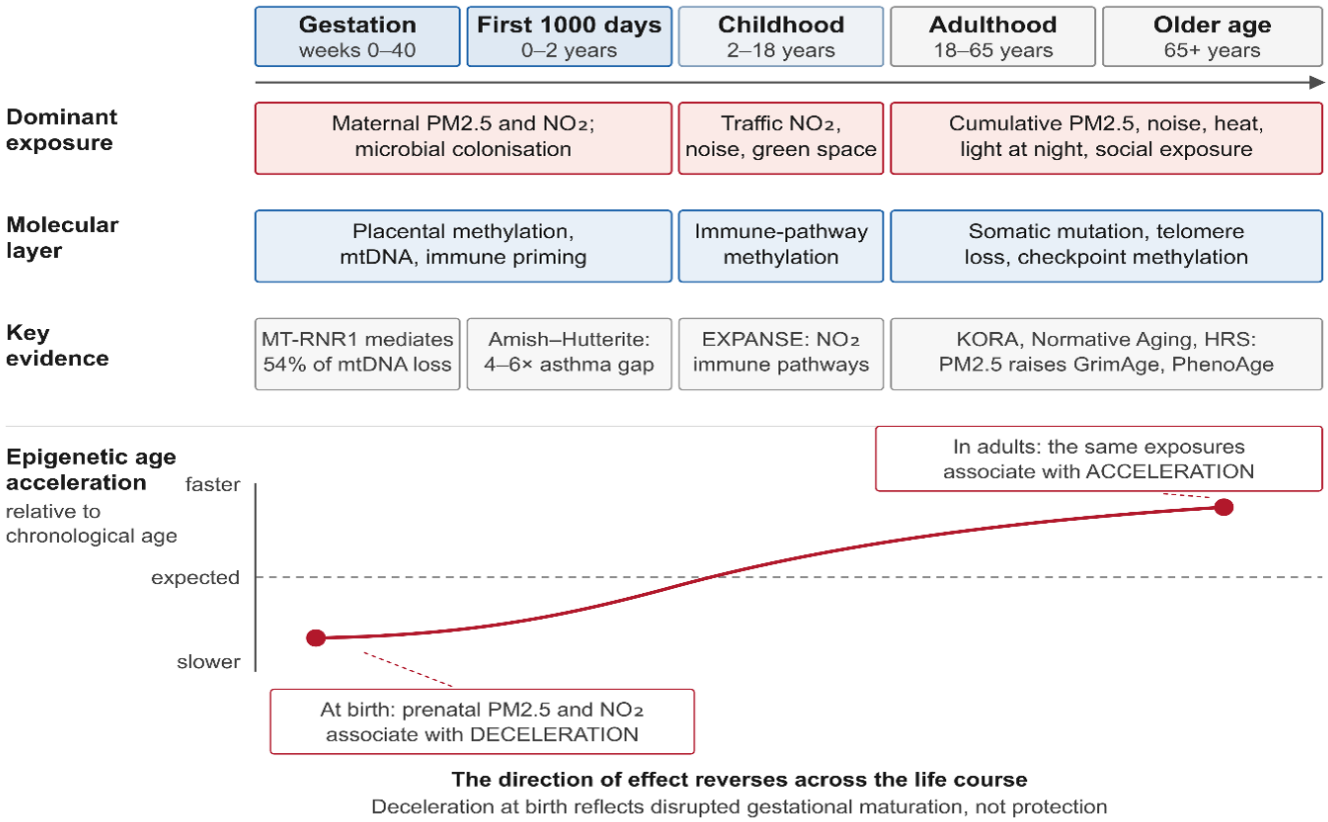


Figure 3. Life-stage-specific interpretation of epigenetic aging. Prenatal studies use gestational-age clocks, whereas adult studies use chronological-age, risk, or pace measures. Song et al. (2022) reported associations of prenatal particulate exposure with lower gestational epigenetic-age estimates, but no significant association with NO_2 (Song et al., 2022). Adult studies report associations between air pollution and higher scores on some aging measures (X. Gao et al., 2022; Ward-Caviness et al., 2016). These findings come from different populations and clocks; they do not demonstrate a within-person reversal across the life course. The clinical meaning of lower gestational estimates requires follow-up.

erogeneity of EV preparations and the potential co-isolation of non-vesicular extracellular particles, EV-specific characterization and functional validation are required before these miRNAs can be interpreted as mediators of persistent regulatory effects (Welsh et al., 2024). Therefore, EV-associated miRNAs should currently be regarded primarily as candidate biomarkers of environmental exposure, while their involvement in durable epigenetic reprogramming remains a testable hypothesis (Rodosthenous et al., 2016).

2.9.4 Prenatal and early life responses

Urban exposure may influence biology before birth (Abarca-Castro et al., 2025; Alum et al., 2025; Parikh & Patel, 2025). The placenta is a highly sensitive 'sensor' of the air-pollution exposome: Inhaled ultrafine particles and heavy metals may reach placental tissues and have been associated with nitrosative stress and epigenetic changes (Ghazi et al., 2021; Saenen et al., 2019). Maternal $PM_{2.5}$ exposure has been associated with global and locus-specific differences in DNA methylation in placental tissue. Reported findings involve several regulatory pathways: (Ghazi et al., 2021; Luyten et al., 2018)

- Global methylation differences have been reported in relation to prenatal exposure, but their interpretation depends on tissue, exposure timing, and measurement method (Maghbooli et al., 2017).
- HSD11B2: Methylation changes in HSD11B2 may affect placental regulation of glucocorticoid exposure, with potential implications for fetal growth and later susceptibility (Ghazi et al., 2021).
- NR3C1 and EMID2: Methylation differences at these

loci have been investigated in relation to developmental stress regulation and neurobehavioral outcomes (Abarca-Castro et al., 2025; Ghazi et al., 2021).

- AHRR, DPP10, HLA-DRB1: Exposure-associated methylation differences at these loci may be relevant to immune development and allergic or respiratory outcomes (Ghazi et al., 2021; Gheissari et al., 2022; Luyten et al., n.d.).
- DNA damage response: Promoter methylation changes involving TP53, APEX1, OGG1, and ERCC4 have been discussed in relation to prenatal toxicant exposure; functional consequences for fetal DNA repair and later cancer risk require further confirmation (Ghazi et al., 2021).

The ENVIRONAGE cohort reported sex-specific associations between maternal $PM_{2.5}$ exposure and placental BDNF and HTR2A promoter methylation (N. Saenen et al., 2015). These findings suggest that fetal sex may modify molecular responses, but do not establish that the methylation differences explain sex differences in later neurodevelopmental disease.

Maternal $PM_{2.5}$ exposure has been associated with placental mitochondrial DNA methylation and content (Janssen et al., 2015). These findings suggest possible links between exposure and mitochondrial regulation, but causal effects on fetal growth cannot be established from these associations alone (Fussell et al., 2024; Janssen et al., 2015; Maghbooli et al., 2017).

Aging measures, therefore, need to be interpreted by life stage and clock target. Prenatal and adult findings should not be plotted on a single continuous trajectory or treated as directly comparable biological ages (Figure 3).

Early-life exposure may influence later susceptibility through developmental, immune, and regulatory pathways. Prenatal air pollution has been associated with adverse birth outcomes and neurodevelopmental differences, but the extent to which particular molecular markers mediate these outcomes remains uncertain (Abarca-Castro et al., 2025; Fussell et al., 2024; Gheissari et al., 2022).

3 Microbial and wildlife responses

Urban life changes contact with soil, vegetation, animals, water, and waste. This section examines human and environmental microbial communities, followed by wildlife responses. Microbial community turnover, horizontal gene transfer, and host population adaptation are related processes with different evidentiary requirements.

3.1 Microbial diversity

Many urban–rural studies report lower gut microbial diversity in industrialized populations, although findings vary across settings and diversity measures (Huang et al., 2023; Lokmer et al., 2020; Peng et al., 2025; Zuo et al., 2018).

Diet, antibiotics, sanitation, and contact with natural environments can alter microbial transmission and community composition. Experimental models suggest that low-fibre diets may cause losses that compound across generations, but this is not an inevitable trajectory for all urban populations (Bosch et al., 2024; Sonnenburg & Sonnenburg, 2019).

Urban–rural studies report shifts in fibre-associated taxa and microbial metabolic capacity, but genera should not be classified as uniformly beneficial or harmful (Y. Du et al., 2021; Peng et al., 2025; Sun et al., 2022). A migration study found declining diversity and a shift from Prevotella towards Bacteroides after relocation to the United States (Vangay et al., 2018). Repeated observations strengthen temporal inference, but simultaneous changes in diet, medications, and other exposures preclude attributing the effect to urbanization alone.

Urbanization and industrialization are not equivalent microbial exposures. Findings from Cameroon illustrate how medication, hygiene, diet, and parasites can shape urban–rural contrasts (Lokmer et al., 2020). Local context should therefore guide interpretation (Tasnim et al., 2017).

3.2 Microbiome and host health

The gut microbiome is a potential interface between changing diets and environments and host immune or metabolic function. Urbanization-related microbial patterns are associated with chronic disease, but the direct mediating role remains incompletely established (Huang et al., 2023).

Proposed mechanisms include altered short-chain fatty-acid production, intestinal permeability, and LPS-related inflammatory signaling. These pathways could contribute to insulin resistance, but their importance varies by microbial function and host context. Changes in a genus alone do not demonstrate loss of a beneficial function or disease causation (Peng et al., 2025; Shukla et al., 2025; Tasnim et al., 2017).

Microbiome–gut–brain mechanisms make the biplogical links to depression biologically plausible, but evidence that urbanization-related microbial changes directly cause depression remains limited (Huang et al., 2023; Peng et al., 2025; Shukla et al., 2025).

3.3 Antimicrobial resistance

Wastewater and built environments connect human, animal, and environmental microbial reservoirs. Nutrients, surfaces, antimicrobial residues, and treatment conditions can influence selection and gene exchange, although detecting resistance genes alone does not establish active transfer (Larsson & Flach, 2022; Manaia et al., 2018).

Urban microbial communities can show enrichment of antimicrobial resistance genes (Bruno et al., 2022; Sun et al., 2022). Built environments and antimicrobial selection influence this pattern (Larsson & Flach, 2022). Resistance must therefore be studied across human, animal, and environmental reservoirs, including bacterial movement and gene transfer.

Soil appears to be a critical nodal point here. Soil microbiomes are reservoirs of both beneficial microorganisms and pathogens for *One Health*; in addition, soil, plant, animal, and human microbiomes are more interconnected than previously assumed through shared microorganisms (Banerjee & van der Heijden, 2023). Therefore, antibiotic resistance in cities is not only a clinical problem but also an ecological circulation

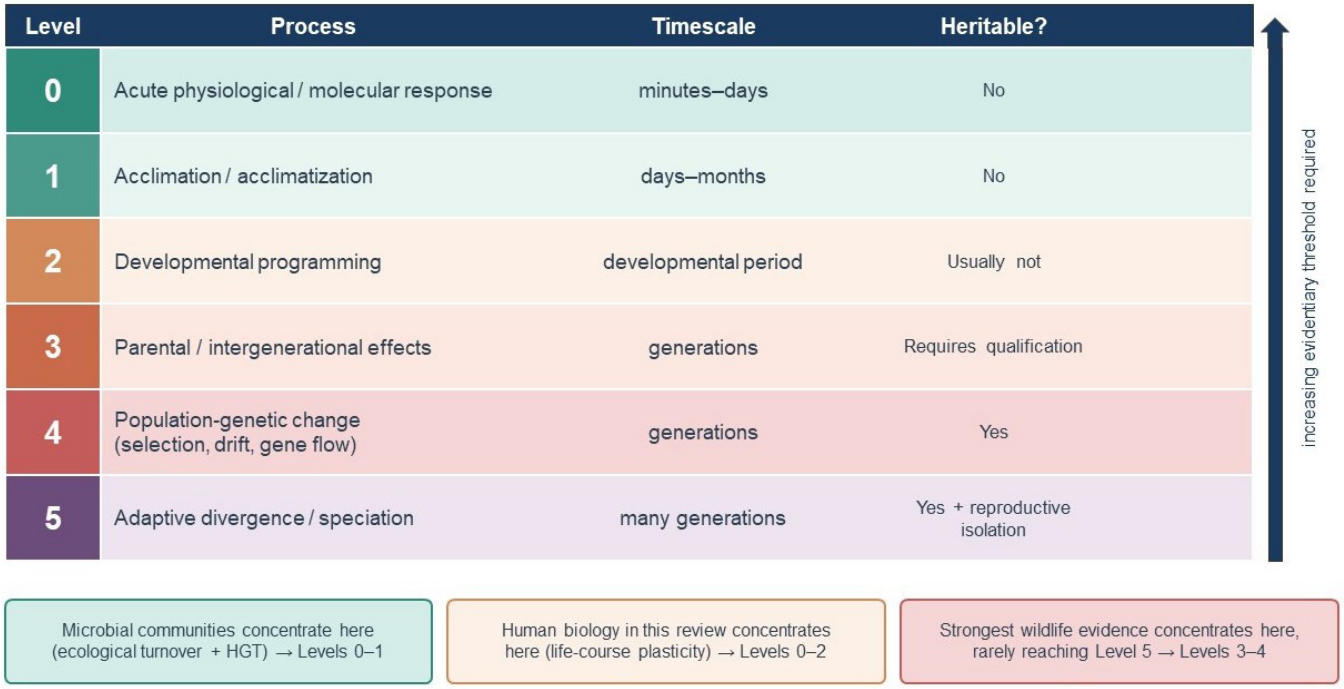


Figure 4. Response processes and inheritance. Acute responses, acclimatization, developmental programming, parental effects, population-genetic change, and adaptive divergence are distinct categories rather than a mandatory progression. Ecological turnover and horizontal gene transfer are separated: acquired microbial genes can be inherited by descendants. Parental effects do not by themselves demonstrate germline inheritance. Selection, drift, and gene flow can change allele frequencies without producing adaptation; local adaptation requires evidence of inherited differences and a fitness advantage. Adaptive divergence does not necessarily imply speciation or reproductive isolation. Timescales overlap, and vary by organism.

problem operating at the human-animal-environment interface (Banerjee & van der Heijden, 2023; Larsson & Flach, 2022) Human-associated diversity, environmental communities, and antimicrobial resistance should be assessed separately. One Health provides a framework for connecting these systems, while longitudinal and intervention studies are needed to test microbial mediation of health outcomes.

3.4 Urban evolution

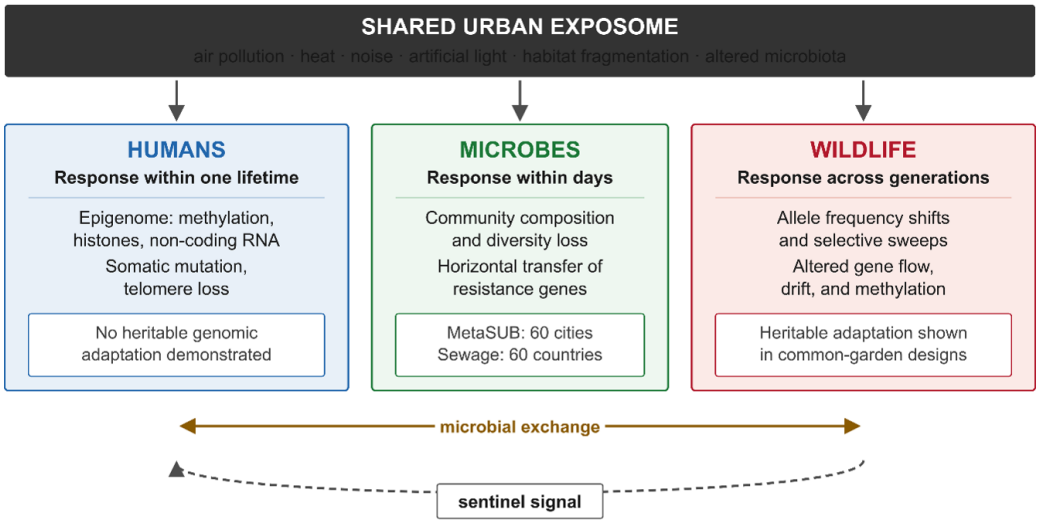
Urbanization can influence both adaptive and non-adaptive evolutionary processes. Faster phenotypic change in cities does not, by itself, establish adaptation (Alberti et al., 2017; M. T. J. Johnson & Munshi-South, 2017). Replicated urban contrasts help test recurring responses, while common-garden and reciprocal-transplant studies can assess inherited differences and local fitness advantages (Lambert et al., 2021; R. A. Martin et al., 2021; Miles et al., 2020; Santangelo et al., 2018). Figure 5 separates these questions from the human life-course and microbial responses.

The critical point is that these changes cannot be reduced to a single mechanism. Urban heat, habitat fragmentation, light and noise pollution, new food sources, and intense human contact together alter selection regimes (Babik et al., 2023; Harris & Munshi-South, 2017; K. Winchell et al., 2023). Therefore, parallel evolution will be strong in some axes and weak in others because cities carry both common and local pressures (Santangelo et al., 2020).

3.4.1 Wildlife phenotypes

Urban wildlife often differs in behavior, resource use, and life history. Reviews report greater boldness, exploration, and activity in some urban populations, with substantial variation in taxonomy and study-design (Burkhard et al., 2026; Lowry et al., 2013; Sol et al., 2013). Such differences can reflect plasticity, sorting, or evolution and should not automatically be labeled as adaptation.

Reviews of urban mammals report changes in space use, vigilance, diet, and activity (Ritzel & Gallo, 2020). Human contact



Evidence standard met, by system

System	Urban stressor	Study design	Adaptation
Killifish, *Fundulus heteroclitus*	industrial pollutants	384 genomes	yes
Water flea, *Daphnia magna*	urban heat	common garden	yes
Anole, *Anolis cristatellus*	urban heat	common garden	yes
White clover, *Trifolium repens*	multiple	160 cities	yes
Great tit, *Parus major*	multiple	observational	not resolved

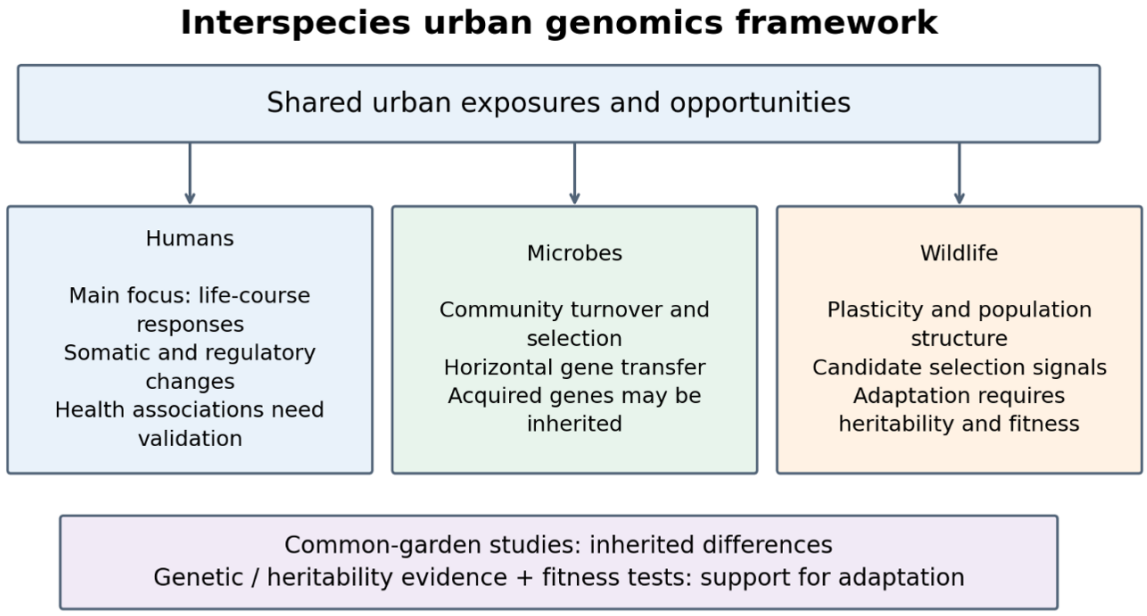


Figure 5. Interspecies urban genomics framework. Shared exposures connect human, microbial, and wildlife systems, but their response timescales overlap, and their inheritance mechanisms differ. Human evidence here mainly concerns life-course molecular and health responses. Microbial change includes community turnover, selection, and horizontal gene transfer; acquired genes may be inherited. Wildlife studies distinguish plasticity and population structure from heritable adaptation. Common-garden studies support inherited differences, while genetic or heritability evidence, together with fitness evidence, support adaptation. Sentinel observations require validation before translation to human risk.

Table 3. Wildlife responses to urban environments and the evidence supporting them. Genomic or epigenomic differentiation is distinguished from demonstrated inherited phenotypes and fitness advantages. Candidate selection signals alone are insufficient to establish adaptation. AHR, aryl hydrocarbon receptor; ROS, reactive oxygen species.

Species or Group	Urban Context	Adaptation Signal	Genomic Evidence	Caveat	Citation
Great tit (Parus major)	Comparison of populations inhabiting urban and forest habitats	Habitat-associated genome-wide DNA methylation differences in liver and blood, with enrichment of metabolic, ROS-processing, and proliferation pathways in liver	Reduced-representation bisulfite sequencing and transcriptomic data indicating a coordinated, regulated response to the urban environment	It remains uncertain whether the methylation differences represent heritable adaptation or phenotypic plasticity	(Watson et al., 2020)
Burrowing owl (Athene cunicularia)	Three replicated urban-rural population comparisons	Parallel selection signals in genes related to synapses, neuronal projections, brain connectivity, and cognitive function	Whole-genome resequencing data from Mueller et al.; Garroway and Schmidt (2020) summarized the evidence for parallel adaptation	The recent origin of urban populations and their proximity to natural populations make population-genetic differentiation difficult to detect	(Garroway & Schmidt, 2020)
White-footed mouse (Peromyscus leucopus)	48 individuals from three urban and three rural populations	Candidate selection signals involving lipid and carbohydrate metabolism	154,770 SNPs obtained from transcriptome data; outlier and landscape-genomic analyses	Selection signatures identify candidate loci but do not, by themselves, establish an urban fitness advantage.	(Harris & Munshi-South, 2017)
Comparative genomic approach to urban fauna	Repeated environmental selection pressures across cities and natural habitats	Potential to test whether urban phenotypic differentiation is heritable and associated with divergent selection	Perrier, Caizergues, and Charmantier (2020) recommend integrating quantitative genetics, field experiments, and next-generation sequencing	Phenotypic differentiation alone does not demonstrate evolutionary adaptation and should be supported by common-garden or reciprocal-transplant experiments	(Perrier et al., 2020)
Atlantic killifish (Fundulus heteroclitus)	Four independently polluted urban estuaries on the US Atlantic coast	Up to several thousand-fold tolerance to lethal industrial pollutants	384 whole genomes; repeated selection on AHR pathway genes, different variants per population	Tolerance heritable and retained in lab-reared offspring—adaptation demonstrated, not inferred	(Reid et al., 2016)
Water flea (Daphnia magna)	Urban versus rural ponds along an urbanization gradient	Higher heat tolerance and smaller body size in urban populations	Common-garden rearing separating genetic change from plasticity	Single region; replication across climates not yet available	(Brans et al., 2017)
Puerto Rican crested anole (Anolis cristatellus)	Multiple independent urban-forest population pairs	Higher heat tolerance in urban lizards, maintained under common-garden conditions	Parallel allele frequency shifts at thermal tolerance loci across cities	Physiological mechanism only partly resolved	(Campbell-Staton et al., 2020)

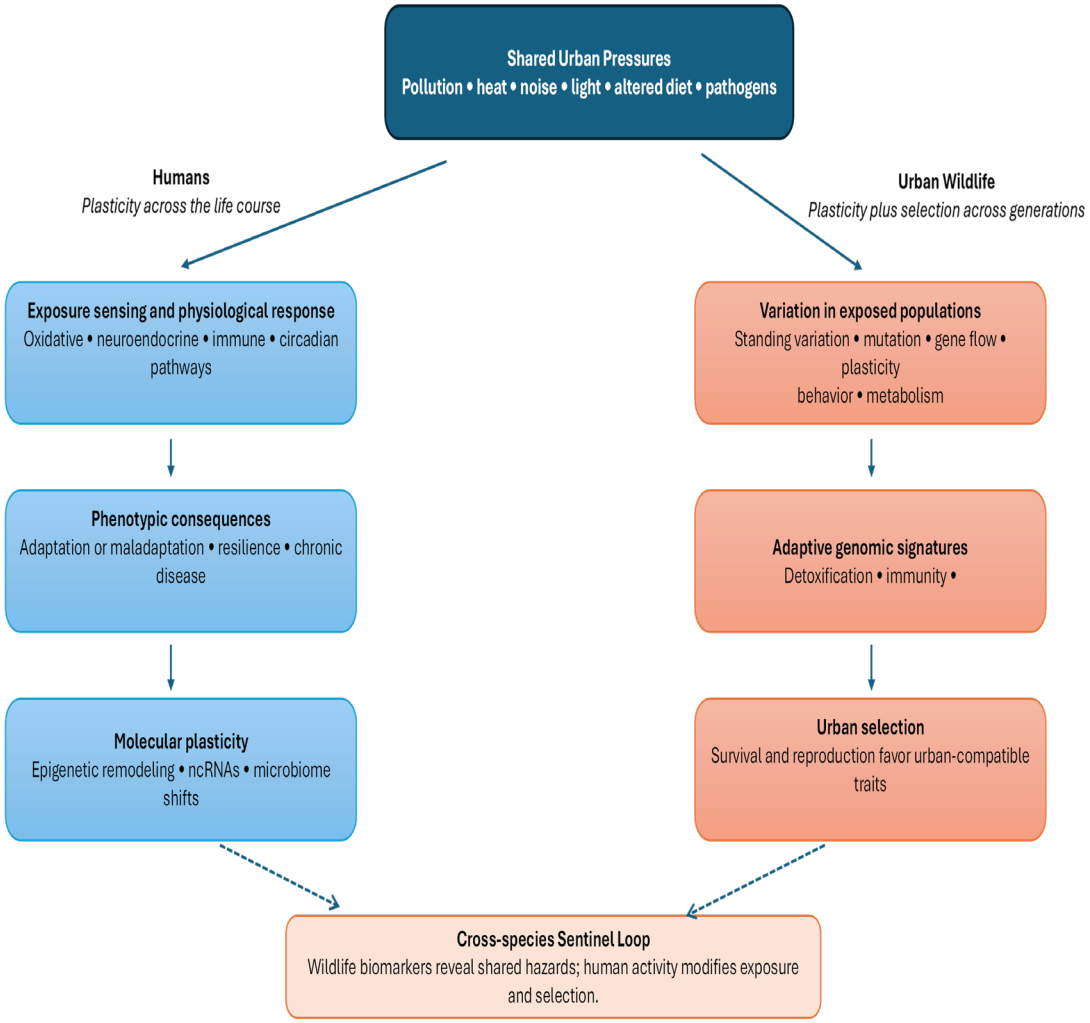


Figure 6. Human and wildlife responses to shared urban pressures. Human responses are primarily examined through the lens of life-course physiological and molecular plasticity. Wildlife research additionally tests population structure, selection, and heritable change across generations. Sentinel biomarkers can identify shared hazards, but translation to human risk requires exposure and species-specific validation (Gavito-Covarrubias et al., 2023; Harris & Munshi-South, 2017; M. T. J. Johnson & Munshi-South, 2017; McEwen, 2007; Merra et al., 2024; Rabinowitz et al., 2005; Rivkin et al., 2019; Salmón et al., 2021).

and management pressures may shape antipredator responses, but similarities to domestication do not establish the same evolutionary process (Geffroy et al., 2020; Schell et al., 2020).

In white-footed mice, candidate selection signals involving lipid and carbohydrate metabolism are consistent with possible responses to urban diets; they do not establish an urban fitness advantage (Harris & Munshi-South, 2017). Pigeon studies link diet, location, coloration, heat, and metal exposure with microbial or physiological differences (Arcila et al., 2025; Jiménez et al., 2023; Schmitt et al., 2025). Greater survival of dark juveniles under lead exposure provides experimental evidence for a context-specific trait advantage, while redox responses alone do not establish evolutionary adaptation (Chatelain et al., 2016; Kurhaluk et al., 2021).

In mosquitoes, rapid adaptation appears to be even more critical from a public health perspective. It has been reviewed that urban adaptation in mosquito strains is shaped particularly along the axes of host preference and insecticide resistance, and that this has consequences on vectorial capacity (Fifer et al., 2025). A strong ecological divergence has been shown between above-ground, bird-feeding and underground, mammal-feeding forms (Haba & McBride, 2022). Differentiated chemosensory genes were identified between two biotypes, consistent with differing preferences for human and chicken odor mixtures (Menon et al., 2025).

Bird studies identify candidate selection loci and associations between urban occurrence and traits such as diet breadth and dispersal (Mascarenhas et al., 2022; Neate-Clegg et al., 2023). These patterns may reflect both environmental filtering and evolutionary change.

3.4.2 Population genetics and adaptation

Urban lizard and great-tit studies report candidate-selection signals and allele-frequency shifts across multiple loci (Salmón et al., 2021; K. Winchell et al., 2023). Their interpretation requires demographic and population-structure controls; genomic

differentiation alone does not prove adaptive function.

Parallel responses need not involve identical variants. Studies of the great-tit and damselfly report limited sharing of individual loci alongside convergence in certain functional pathways (Babik et al., 2023; Caizergues et al., 2022; Salmón et al., 2021). Table 3 distinguishes candidate signals from experimentally supported responses.

Pollution-tolerant killifish provide a strong example of repeated selection involving AHR-related pathways, with different variants across populations (Reid et al., 2016). Urban–forest methylation differences in great tits provide a separate regulatory signal whose heritability and fitness consequences remain unresolved (Caizergues et al., 2022; Watson et al., 2020). Epigenetic differentiation should therefore not be assumed to accelerate genetic adaptation.

Population-genetic signals require joint interpretation of selection, gene flow, drift, and demographic history (Johnson et al., 2018; Miles et al., 2019). Replicated urban–rural comparisons can reveal parallel responses, although similar biological functions may involve different genetic variants across populations (Babik et al., 2023; Salmón et al., 2021). Common-garden studies help distinguish inherited differences from environmental plasticity, while reciprocal-transplant experiments can test whether those differences improve survival or reproduction in urban environments. Genomic differences alone do not demonstrate such a fitness advantage (Lambert et al., 2021; R. A. Martin et al., 2021).

3.5 Wildlife as sentinels

Wildlife can act as sentinels of shared environmental hazards, but species-specific exposure, physiology, and susceptibility limit direct translation to human risk (Hazen et al., 2024). Synanthropic species are particularly useful where their exposure to air, dust, water, or food overlaps with that of people.

Bird and invertebrate studies combine pollutant measurements with markers of oxidative, detoxification, and DNA-damage. Examples include starlings, gull chicks, and terrestrial snails (North et al., 2017; Oliveira et al., 2025; Regoli et al., 2005). These markers support environmental biomonitoring, rather than a direct estimate of human clinical risk.

Pigeons and house sparrows provide examples of metal-exposure biomonitoring (Gillings et al., 2024; Kurhaluk et al., 2021; Novoa et al., 2025). Epigenetic markers may add information about exposure history, but their specificity, persistence, and predictive value require validation (Šrut, 2021; Wade et al., 2024). Findings in wildlife should be compared with, rather than assumed to reproduce, human methylation associations (Vogli et al., 2025).

From an environmental surveillance perspective, urban wildlife can indicate not only pollutants but also antimicrobial resistance and other biological hazards early on. Integrating wildlife into antimicrobial resistance surveillance is critical for monitoring pollution and resistance evolution, assessing ecological impacts, and public health risks. Species such as red foxes, gulls, and waterfowl are among the ideal candidate bioindicators (Doyle et al., 2025). It has also been emphasized that feral pigeons can be reservoirs and vectors for pathogens that threaten human and animal health (Fjodorov et al., 2025).

A central question is how often urban responses are parallel across cities and how often they depend on local conditions. This motivates replicated sampling, demographic controls, and longitudinal research (Section 5.2).

4 Health implications and research methods

4.1 Clinical associations

Urban exposures may influence chronic disease through interacting environmental, social, and biological pathways. Molecular associations provide mechanistic clues, but their contribution to disease varies by exposure and study design (Hoyo et al., 2026; Vogli et al., 2025; H. Wu et al., 2023). Table 4 places disease trends in context without attributing them solely to urbanization.

4.1.1 Cancer

Environmental carcinogenesis can involve mutation, inflammation, and altered gene regulation (Takeshima & Ushijima, 2019). Hill et al. (2023) combined epidemiological observations with experimental models to investigate $PM_{2.5}$-associated lung cancer. In the studied models, particulate exposure promoted the expansion of epithelial cells carrying pre-existing EGFR mutations via macrophage-derived IL-1β, rather than increasing the frequency of driver mutations (Hill et al., 2023). This supports a tumor-promotion mechanism in a defined setting, rather than one mechanism for all pollutants or cancers.

4.1.2 Cardiometabolic disease

Evidence links specific urban exposures, including air pollution, noise, inactivity, and food environments, with cardiometabolic outcomes (Rajagopalan, Vergara-Martel, Zhong, & others, 2024). Ecological analyses associate urbanization with changes in metabolic indicators, while longitudinal studies support associations with walkability (Chandrabose et al., 2018; Goryakin et al., 2017). These designs provide different levels of inference and do not establish a single effect of urbanization.

4.1.3 Neuropsychiatric outcomes

Urban environmental and social characteristics have been associated with affective symptoms and brain-imaging differences (Xu et al., 2023). Studies of urban living and upbringing implicate stress-processing regions (Silva & Steffen, 2019), while youth studies suggest life-stage differences in these associations (Xu et al., 2021). A systematic review and meta-analysis linked $PM_{2.5}$ exposure to depression and suicide outcomes, with weaker or less consistent evidence for several other disorders (Braithwaite et al., 2019). These observational patterns do not establish a single molecular pathway from city living to psychiatric disease.

Associations between urban living and depression or anxiety vary by social and geographic setting (Hoare et al., 2019; Krabbendam et al., 2020; Ventriglio et al., 2020). An ecological

analysis of 191 countries found a nonlinear association between urbanicity and the burden of common, but country-level relationships cannot be assumed to hold for individuals (Van Der Wal et al., 2021).

4.1.4 Immune and inflammatory disease

Barrier dysfunction, altered microbial contact, and immune regulation offer plausible links between changing environments and allergic or autoimmune disease (Akdis, 2021; M. Kumar et al., 2025). Genetic susceptibility and diverse exposures interact; these diseases should not be attributed to urbanization via a single established molecular pathway.

The global prevalence of insufficient physical activity among adults reached 31.3% in 2022, an increase of approximately 5 percentage points since 2010 (Strain et al., 2024). This behavioral shift can also leave molecular traces. In the accelerometer-based Rhineland Study (n=3,567), greater physical activity was associated with slower GrimAge and PhenoAge acceleration and with CpG differences related to activity, although the cross-sectional design prevents causal interpretation (Fox et al., 2023). Sedentary behavior should therefore be presented as a modifiable component of the urban exposome that interacts with inherited susceptibility, diet, pollution, sleep and socioeconomic conditions, rather than as a new evolutionary adaptation

4.1.5 Respiratory disease

Urban environments can affect respiratory health through a combination of outdoor air pollution, poor indoor air quality, occupational exposures and climate-related changes in allergens. Fine particulate matter ($PM_{2.5}$), nitrogen dioxide (NO_2) and ozone have been associated with asthma development and exacerbation, impaired lung growth and declining lung function. Long-term exposure may also contribute to the development and progression of chronic obstructive pulmonary disease (COPD). However, smoking, age, socioeconomic conditions, respiratory infections and occupational history should also be considered when interpreting these associations (Guarnieri & Balmes, 2014; Schikowski et al., 2014).

Air pollution may also influence the epigenetic regulation of genes involved in inflammation, oxidative stress, immune responses and xenobiotic metabolism. Changes in DNA methylation could partly explain how prenatal or early-life exposures influence respiratory health later in life. Nevertheless, many studies have analyzed peripheral blood rather than respiratory tissue and have used cross-sectional designs. It therefore remains unclear whether the reported epigenetic changes directly contribute to disease or simply reflect previous exposure (Rider & Carlsten, 2019).

Indoor dampness and mould are additional respiratory risks, particularly in crowded, poorly ventilated or inadequately maintained buildings. Exposure to fungal spores, allergens and microbial products may damage the epithelial barrier and increase airway inflammation. A systematic review and meta-analysis found that residential dampness and mould were associated with a higher risk of developing asthma (Quansah et al., 2012). Workplace exposure to dusts, fumes, gases, cleaning agents and sensitizing chemicals can also contribute to adult-onset asthma, COPD and other non-malignant respiratory diseases (Blanc et al., 2019).

The COPD and asthma trends presented in Table 4 should therefore be interpreted as the result of several interacting exposures rather than as direct consequences of urbanization alone. Although age-standardised prevalence has declined globally, the absolute burden of COPD has increased, and asthma trends differ considerably among countries and population groups. Longitudinal studies that combine personal exposure measurements, lung-function follow-up, molecular data, housing conditions and occupational history are needed to clarify which urban exposures initiate respiratory disease and which primarily accelerate its progression or trigger exacerbations.

Across these disease groups, molecular findings support several plausible pathways but seldom resolve the complete exposure–molecular change–disease sequence. Longitudinal studies strengthen temporal inference, while intervention, experimental, and other causal approaches are needed to test mediation (Section 5.2).

4.2 Research methods

Urban genomic research increasingly depends on integrating genomic, epigenomic, transcriptomic, proteomic, metabolomic, and spatial data. FAIR data infrastructures and transparent computational workflows are therefore needed to make these heterogeneous datasets interoperable, traceable, and reproducible (Judge et al., 2026).

AI can support exposure mapping, multi-omic integration, and hypothesis generation, but external validation, transparent models, and equitable data governance remain necessary (Ozcelik et al., 2024; Sahin et al., 2026). Table 5 compares complementary research methods and their limitations.

4.2.1 Single cell and spatial omics

Single-cell methods resolve molecular differences between cell types, while spatial omics place these states within tissue neighborhoods. Their integration helps relate responses to cellular context, although many platforms measure only selected molecular layers (Gulati et al., 2025; Jain & Eadon, 2024; Vandereyken et al., 2023).

Experimental air-pollution studies illustrate this value. Particulate exposure produces cell-specific lung transcriptional responses, and ozone models identify dose-dependent macrophage states (Gui et al., 2025; Vo et al., 2026). These findings require validation under realistic human exposure conditions (H. Wu et al., 2023).

4.2.2 Exposomics

Exposomics integrates external measurements, biomonitoring, social conditions, and health outcomes. Analysis must address correlated, time-varying exposures, harmonized definitions, missing data, and reproducibility (Sarigiannis et al., 2025, 2026).

Table 4. Selected conditions with marked frequency changes in recent decades and their relevance to the urban exposome (Values are not directly comparable across rows because surveillance systems, populations, age ranges, and endpoints differ. Trends demonstrate temporal change, not causation by urbanization).

Condition	Comparison period and population	Earlier estimate	Recent estimate	Urban-exposome relevance	Interpretive caution
Obesity	Global, 1990–2022	Adults: women 8.8%; men 4.8%	Women 18.5%; men 14.0% (age-standardized prevalence increased)	Urban food environment, inactivity, sleep/circadian disruption, chemicals and deprivation.	Strong secular rise, but heterogeneous and multifactorial; BMI is an imperfect proxy. (Phelps et al., 2024)
Diabetes	Global adults, 1990–2022	~198 million; prevalence ~7%	~828 million; prevalence ~14% (approximately doubled)	Adiposity, diet, inactivity, pollution, stress and microbiome changes.	Includes type 1 and type 2 diabetes, although most adult cases are type 2. (B. Zhou et al., 2024)
Hypertension	Global adults, 2000–2010	Age-standardized prevalence 25.9%	Absolute increase of 5.2 percentage points, to 31.1% (≈20% relative rise); absolute burden continued to rise thereafter	Noise, $PM_{2.5}$, heat, psychosocial stress, inactivity, obesity and high-sodium diets.	Definitions and measurement vary; ageing and population growth affect case counts. (Mills et al., 2015, 2020).
Chronic kidney disease	Global adults, 1990–2023	~378 million adults	~788 million; age-standardised prevalence 14.2% in 2023, +3.5% relative vs 1990	Diabetes, hypertension, heat stress/dehydration, metals and air pollution.	Large case-count rise is driven substantially by ageing, diabetes and hypertension; urban causality is not isolated. (Mark et al., 2025)
Ischaemic heart disease	Global, 1990–2021	~112 million prevalent cases	~254 million cases (+127%); age-standardised incidence declined ~11%	Air pollution, noise, heat, metabolic risks, inactivity and inequality.	Absolute burden rose while age-standardised incidence fell: population growth and ageing are major drivers. (Jing et al., 2026)
Stroke	Global, 1990–2021	~50.4 million survivors; ~7.0 million incident cases	93.8 million survivors (+86%); 11.9 million incident cases (+70%); standardised rates declined	Air pollution, heat, hypertension, diabetes, inactivity and diet.	Rising counts do not mean rising individual risk; standardised incidence/prevalence declined. (Feigin et al., 2024)
Chronic obstructive pulmonary disease	Global younger population, 1990–2021	20.0 million prevalent cases; ASPR 979/100,000	30.4 million; ASPR 898/100,000	Ambient/household pollution, occupational exposures and smoking.	Case counts increased but age-standardised prevalence declined; smoking remains a dominant confounder. (Zhu et al., 2025)
Asthma	Global, 1990–2021	Higher 1990 age-standardised prevalence	Age-standardised prevalence decreased ~40%	Air pollution, aeroallergens, indoor exposures, biodiversity loss and climate-related pollen changes.	Global decline masks increases in some populations and differences in symptom definitions. (L. Zhang et al., 2025)
Inflammatory bowel disease	Global, 1990–2019	Lower absolute case count; ASPR 73.2/100,000 in 1990	~4.9 million cases; ASPR ~59.3/100,000; age-standardised incidence increased but age-standardised prevalence decreased	Westernised diet, antibiotics, sanitation, microbial diversity loss and pollution.	Incidence is rising in newly industrialised regions, while global standardised prevalence estimates may fall. (Ruan et al., 2025; R. Wang et al., 2023)
Depressive disorders	Global, 1990–2019/2021	~182 million cases in 1990	~290 million in 2019; prevalence/DALY burden further increased through 2021	Social deprivation, noise, pollution, heat, weak social networks, inactivity and circadian disruption.	COVID-19 affected 2020–2021 trends; diagnosis and reporting differ widely. (J. Liu et al., 2024)
Dementia	Global, 1990–2016/2019	20.2 million cases in 1990	43.8 million in 2016; 57.4 million in 2019; age-standardised prevalence changed little	Air pollution, inactivity, diabetes, hypertension, isolation and limited green space are modifiable candidates.	Most growth reflects ageing and population growth; urban living is not a single causal exposure. (Nichols et al., 2019, 2022)
Autism spectrum disorder	US ADDM, age 8, 2000/2002–2022	6.7/1,000 (1 in 150)	32.2/1,000 (1 in 31)	Urban exposures are candidates for G×E and prenatal-programming studies.	Identified prevalence is not biological incidence; diagnostic and surveillance changes are major contributors. (Maenner et al., 2023; Shaw et al., 2025)
Early-onset colorectal cancer	Global, age <50, 1990–2019	3.05/100,000	3.85/100,000; AAPC +1.0%	Obesity, UPFs, inactivity, antibiotics and microbiome shifts.	Ecological associations cannot assign causality; country trends vary. (Z. Wang et al., 2024)

Table 5. Technologies Advancing Urban Genomics Research

Technology	Primary Use	Urban Genomics Value	Main Limit	Citation
Single-cell omics	Resolving cell-specific transcriptomic, epigenomic, proteomic, and multi-omic responses.	Helps identify distinct responses of epithelial, immune, endothelial, or stromal cells to the same environmental exposure.	Tissue sampling, cell loss, cell composition, and clinical interpretation; most studies also measure environmental context only to a limited extent.	(R. Wu et al., 2024)
Spatial omics	Mapping biomolecules together with their tissue locations and intercellular neighborhoods.	Enables assessment of the cellular neighborhoods, barrier regions, and inflammatory niches associated with an exposure within the tissue microenvironment.	Resolution, tissue integrity, multiple cells per spot, and cross-platform standardization.	(Chadeau-Hyam, 2022; Jeong et al., 2022)(Vandereyken et al., 2023)
Exposomics and personal exposure monitoring	Joint profiling of lifetime external exposures, the internal exposome, and molecular responses.	Complements address-based averages with personal time-location patterns, sensor data, and biological samples.	Exposure mixtures, short biomarker half-lives, reverse causation, and incomplete historical records.	(Feimin Yuan et al., 2026; W. M. Kim et al., 2022) (Wild, 2012; Niedzwiecki et al., 2019; Maitre, Guimbaud, et al., 2022)
Environmental DNA (eDNA) and metabarcoding	Molecular detection of single or multiple species in water, soil, and other environmental matrices.	Monitors biodiversity, invasive species, and ecosystem change in urban parks, corridors, rivers, and ponds.	DNA transport and degradation, false negatives, imperfect correspondence between DNA quantity and species abundance, and incomplete reference databases.	
Urban metagenomics and One Health genomic surveillance	Joint monitoring of human, animal, and environmental microbiomes, pathogens, and antimicrobial-resistance genes.	Maps microbial signatures of cities, the distribution of resistance genes, and potential cross-species transmission routes.	Low genome coverage in environmental samples, mismatched sampling across domains, biosafety, and data governance.	(Luan & Daoyu, 2025; Panda et al., 2025; Sillé & Hartung, 2025)(Danko et al., 2021; Djordjevic et al., 2024; Catalano et al., 2024)

Urban exposomics combines spatial exposure mapping with personal measurements and biological sampling (Stingone et al., n.d.; Vlaanderen et al., 2021). Early-life studies report associations across several molecular layers, with patterns varying by life stage (Maitre, Bustamante, et al., 2022; Vogli et al., 2025). DNA methylation may provide exposure-related information (Colwell et al., 2023), but it tis not a complete substitute for measuring exposure histories.

4.2.3 Environmental DNA and ecological genomics

Environmental DNA (eDNA) is genetic material sampled from water, soil, air, or other environmental matrices. It can complement direct surveys through non-invasive biodiversity monitoring (Ruppert et al., 2019; Thomsen & Willerslev, 2015).

Urban studies demonstrate eDNA-based detection of vertebrates in water networks and parks, while rivers can integrate signals from aquatic and terrestrial species (Deiner et al., 2016; Sohn & Song, 2024; S. Zhang et al., 2023). Detection is influenced by transport, persistence, and reference databases, so it does not establish local abundance (Bagley et al., 2019).

Airborne eDNA and metagenomic approaches are further expanding this field. Airborne eDNA has the potential to monitor microbial shifts and pathogen transport related to air pollution. However, while promising for multi-taxon tracking, pathogen

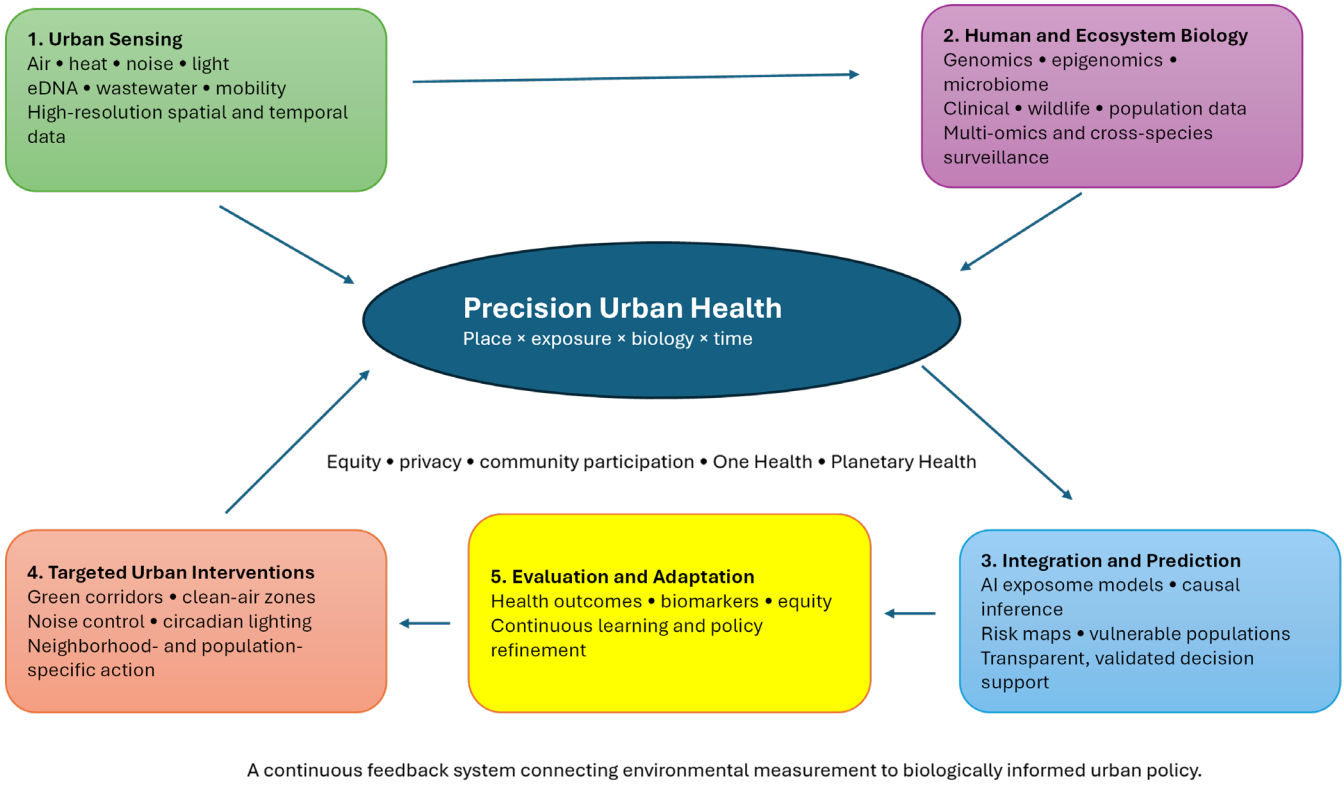


Figure 7. Precision Urban Health Model. A continuous feedback model combines high-resolution urban sensing with human and ecosystem multi-omics, integrates these data through transparent prediction and causal inference, translates results into targeted interventions, and evaluates outcomes to refine policy. Equity, privacy, community participation, One Health, and Planetary Health are cross-cutting principles (Baccarelli et al., 2023; Destoumieux-Garzón et al., 2018; Gago-Ferrero et al., 2026; Giles-Corti et al., 2016; Khoury et al., 2016; Singer et al., 2023; Zielinska-Dabkowska et al., 2023)

surveillance, and the development of standardized monitoring systems, it remains limited by problems such as methodological biases, degradation, and bycatch of human DNA (Q. Liu, 2025; G. Wang et al., 2026; Yoon et al., 2025).

A global atlas sampled 4,728 metagenomes from transport systems in 60 cities, identifying shared and city-associated microbial signatures (Danko et al., 2021). Studies of healthcare and wastewater settings extend this network perspective, but inferred donor relationships require independent evidence before they can betreated as transmission routes (Aminu et al., 2025).

The limitations of the field are also evident. Uncertainty accumulates across the sampling, laboratory, and bioinformatics stages of eDNA workflows; long-term and temporal designs remain scarce (Mathieu et al., 2020). The persistence of extracellular DNA in environmental samples can disrupt community interpretation and quantification and purification strategies are important (Bairoliya et al., 2022).

4.2.4 Computational modeling

AI-assisted urban biology is moving beyond simply identifying exposome data and transforming it into predictions. AI is increasingly used to analyze multiple environmental risks simultaneously and identify patterns associated with cardiometabolic, cardiovascular, respiratory, and neuropsychiatric outcomes; this transformation shift is significant in overcoming the classic single-exposure/single-disease paradigm (Isola et al., 2024). Machine-learning models can combine satellite or street-level imagery, sensors, and demographic information to estimate exposures. Their value depends on independent validation, spatial transferability, and attention to bias and missing data (Iyer et al., 2025; Luan & Daoyu, 2025).

Urban digital twins can enable the comparison of alternative planning scenarios by combining sensor, geographic information system, mobility, environmental exposure, and health data in continuously updated virtual city models. In the context of urban genomics, the implications of these models on epigenetic aging, vulnerable populations, or disease risk are not yet validated clinical tools; they are research hypotheses that need to be tested with experimental and longitudinal data (Argota Sánchez-Vaquerizo, 2025; Lei et al., 2023).

5 Urban action and research priorities

Urban planning can reduce exposures through changes in transport, housing, lighting, and green infrastructure. Precision urban health seeks to combine exposure maps, molecular data, and population vulnerability to guide prevention (Baccarelli et al., 2023; Khoury & Holt, 2021; Roberts et al., 2024). Molecular measures can support evaluation, but their use as routine planning endpoints requires validation and should complement established health and environmental indicators.

5.1 Exposure informed urban design

Exposure-informed urban design uses established environmental and health evidence to reduce harmful exposures. Molecular endpoints may help investigate mechanisms and evaluate interventions, but they should complement validated exposure and health measures (Baccarelli et al., 2023).

Green infrastructure is associated with health benefits, but mechanisms and effects vary with location, access, vegetation, and design (Browning et al., 2022; B.-Y. Yang et al., 2021).

Greenness has been associated with epigenetic aging and prenatal methylation measures, with variation by population and socioeconomic conditions (Alfano et al., 2023; Egorov et al., 2025; K. Kim et al., 2023). These observational markers do not yet define clinical benefit or planning thresholds.

Noise reduction can support sleep and cardiovascular health. Traffic design, quieter surfaces, and night-time restrictions may reduce exposure; electrification alone does not address all noise sources (Münzel, Daiber, et al., 2025; Münzel, Kuntic, et al., 2025). Intervention effects should be assessed with exposure and health outcomes.

Lighting design should consider timing, intensity, spectrum, and duration to limit circadian disruption while meeting safety needs (Bonmati-Carrion et al., 2014; Davis et al., 2023). Associations with chronic disease do not establish that outdoor lighting causes these outcomes via a single pathway (Meléndez-Fernández et al., 2023; Touitou et al., 2017). Municipal circadian metrics remain a proposed approach requiring evaluation (Shu, 2026).

Green infrastructure can be useful for pollution buffering, but the effect is strongly context-dependent. Vegetation can reduce particulate matter by storing, dispersing, and altering the microclimate, but plant selection, spatial arrangement, ventilation, and maintenance are critical (Diener & Mudu, 2021). While green infrastructure may reduce exposure in some cases, it can be ineffective or even harmful in others; therefore, emission reduction and source-receiver distance remain the top priority (Hewitt et al., 2020; P. Kumar et al., 2019).

One Health and equity in implementation

The *One Health* and *Planetary Health* frameworks require consideration of human health alongside animals, microbiomes, ecosystems, and infrastructure. An evidence-based *One Health* framework is proposed that places biodiversity and ecosystem services at the center of healthy and sustainable urban design in urban rehabilitation (Bruno et al., 2024). This framework will be incomplete without an environmental justice perspective in cities, as ecosystem services and burdens are unequally distributed (Murray et al., 2022). The common core of *One Health* and *Planetary Health* highlights the human-animal-environment connection, systems thinking, and cross-sectoral collaboration (Müller et al., 2025; Talukder et al., 2024).

One Health surveillance should monitor not only known pathogens but also the environmental and ecological factors that drive disease outbreaks (Hayman et al., 2023). One Health approaches provide added value, particularly in prevention, preparedness, and early detection (Zinsstag et al., 2023). Measurable indicators, integrated governance, a systems approach, and locally context-sensitive data are needed for urban planning to promote human and planetary health in tandem (Crane et al., 2021; Dyer et al., 2024; Giles-Corti et al., 2022).

Evaluating interventions

Intervention studies can test whether planning changes reduce exposures and improve established health outcomes. Molecular and ecological measures may provide complementary information, but the optimal combinations of interventions, populations, and evaluation timescales remain uncertain (Sarigiannis et al., 2026; Vogli et al., 2025). Figure 7 presents a proposed evaluation cycle.

5.2 Evidence gaps and priorities

The clearest conclusion of this review, based on the current literature, is that evidence on the genomic and epigenomic effects of urbanization is rapidly increasing. However, the field still lacks in robust causality tests, longitudinal human cohorts, representative data from low- and middle-income countries, and non-human genomic surveillance (Carlen et al., 2026; Cronjé et al., 2020; Glover et al., 2024; Ng et al., 2012).

5.2.1 Evidence coverage

Evidence gaps differ across human health, microbial ecology, and wildlife evolution. Common needs include repeated measurements, wider geographic representation, causal testing, and improved genomic resources for non-model species. This review did not formally score or rank evidence coverage across these fields (Carlen et al., 2026; Glover et al., 2024)

Few integrated cohorts track exposures, molecular responses, and clinical outcomes over time. Unequal sample and data infrastructure also limits representation, particularly in low- and middle-income settings (Cronjé et al., 2020; Evertsz et al., 2023; Michalska-Falkowska & Sargsyan, 2026).

5.2.2 Longitudinal and causal evidence

Cross-sectional studies cannot establish temporality or rule out reverse causation. Repeated exposure and biological measurements can strengthen inference, but longitudinal observation alone does not remove confounding or prove causality (Cronjé et al., 2020; Lawrence et al., 2020; Ng et al., 2012).

Studies of discrimination, neighborhood disadvantage, and long-term greenness illustrate how social and environmental histories relate to molecular markers(Del Toro et al., 2024; K. Kim et al., 2023; Reuben et al., 2020). Repeated sampling is needed to establish when these differences emerge and whether

they predict later outcomes.
Mendelian randomization can complement observational analyses when valid instruments and their assumptions are satisfied (Relton & Davey Smith, 2012; Sanderson et al., 2022). Epigenetic applications remain limited and cannot replace interventions or mechanistic experiments (Grau-Perez et al., 2019). Future cohorts should integrate repeated sampling, exposure maps, and health records with prespecified causal analyses (Bick et al., 2024; Glover et al., 2024).

5.2.3 Representation and equitable data infrastructure

One of the most serious structural weaknesses in the field appears to be the concentration of data in high-income countries. A large part of the urban-rural epigenetics literature comes from developed countries; therefore, the generalizability of findings to communities in low- and middle-income countries and to understudied ethnic groups remains uncertain (Cronjé et al., 2020). The lack of diversity in genomic research is also documented on a broader scale; inadequate representation of databases limits both scientific accuracy and clinical applicability (Madden et al., 2024; Tawfik et al., 2023).
This limitation also applies to the present review: much of the mechanistic and cohort evidence synthesized here originates from high-income settings. Consequently, the proposed framework may not fully capture exposure mixtures, infrastructures, genetic diversity, or social conditions in LMIC cities (Bentley et al., 2017; Sirugo et al., 2019).
Equitable research requires both biological sampling and durable digital infrastructure. Local governance and data sovereignty are needed to prevent unequal data-sharing arrangements from reproducing existing disparities (Evertsz et al., 2023; Michalska-Falkowska & Sargsyan, 2026).
Health-system biobanks demonstrate ways to link genomic data with longitudinal records (Bick et al., 2024; R. Johnson et al., 2023). Locally led models, including H3Africa, offer relevant lessons for capacity, governance, and ethical frameworks, rather than one universal institutional template (Sengupta et al., 2025).
The Western Balkans illustrate the need for region-specific evidence. Traffic, residential heating, industry, and energy production create locally distinctive exposure mixtures; the European Environment Agency identifies air pollution as a major regional health concern (EEA, 2023; WHO, 2021). Cohorts in such underrepresented settings could test whether molecular associations reported elsewhere are reproducible under different environmental and social conditions.

5.2.4 Coordinated city cohorts

The global urban metagenome atlas demonstrates the feasibility of coordinated sampling across cities, but does not validate a combined human–wildlife genome atlas or its clinical utility (Danko et al., 2021).
Urbanome and related frameworks could support coordinated city cohorts integrating exposure measurements with human molecular and health data (Glover et al., 2024; Luna-Rivera et al., 2025; Morawska et al., 2022). Biobanks, microbiome surveys, and wildlife monitoring should share compatible spatial and temporal definitions while retaining distinct biological endpoints (X. Liu et al., 2024; Vogli et al., 2025)

5.2.5 Interspecies monitoring

Wildlife monitoring requires broader taxonomic coverage and better reference genomes. Non-model organisms remain difficult to study at consistent genomic resolution (Carlen et al., 2026; M. T. J. Johnson & Munshi-South, 2017).
Lizard, bumblebee, and mouse studies illustrate both candidate selection and changes in population structure (Munshi- South et al., 2016; Theodorou et al., 2018; K. Winchell et al., 2023). Integrating genomic and ecological data can test how habitat connectivity affects these responses (Chau et al., 2023).
One Health sequencing studies can identify shared microbial strains and resistance elements across human, animal, and environmental reservoirs (Hassell et al., 2023; Mourkas et al., 2024; Muloi et al., 2022). Shared sequences alone do not establish the direction of transmission. Coordinated sampling, contact data, and temporal follow-up are needed to interpret these relationships (Martínez-Ortiz et al., 2026).

Conclusion

The urban genomics framework brings together evidence on how shared urban exposures affect human health, microbial communities, and wildlife. It distinguishes changes that occur within a human lifetime, shifts in microbial communities and gene transfer, and inherited changes in wildlife across generations. Demonstrating adaptation in wildlife requires evidence that these changes are heritable and improve survival or reproduction in urban environments. Future research should measure exposures and biological responses repeatedly over time, test interventions, include populations and regions that are currently underrepresented, and use comparable monitoring methods across species. This evidence is needed to determine whether molecular markers can help guide urban planning and public health decisions.
Urbanization is not uniformly harmful. Well-designed cities can improve access to care, sanitation, education, social connection, active transport, and green space. The central challenge is therefore not to reverse urbanization, but to identify and redesign the features that generate unequal biological risk while preserving the health advantages of urban life (Giles-Corti et al., 2016; Vlahov et al., 2007).

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Not applicable

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Declaration of Generative AI and AI-Assisted Tools
During the preparation of this manuscript, the authors used ChatGPT (OpenAI), Claude (Anthropic), Elicit, and NotebookLM (Google) to support literature searching and screening, organize and synthesize relevant information, assist in drafting and restructuring sections of the manuscript, and improve language, clarity, and readability. These tools were not used to generate research data or perform statistical analyses. The authors critically evaluated the scientific interpretations, checked the cited sources against the original publications, and reviewed and revised all AI-assisted text. The authors take full responsibility for the accuracy, integrity, references, and final content of the manuscript.

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