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Light, Noise, Chemical Signals, and Thermal Conditions as Interacting Drivers of Insect Behavior in Anthropogenic Sensory Environments

Elena Petrova^{1*}, Ivan Georgiev¹, Nikolay Stoyanov², Petar Kolev³

¹Department of Anthropogenic Sensory Environments and Behavior, Faculty of Life Sciences, Sofia University, Sofia, Bulgaria.

²Department of Light, Noise, and Chemical Drivers, Faculty of Biosciences, University of Plovdiv, Plovdiv, Bulgaria.

³Department of Thermal Conditions and Behavioral Responses, Faculty of Sciences, University of Ruse, Ruse, Bulgaria.

*E-mail ✉ elena.petrova@uni-sofia.bg

ABSTRACT

Anthropogenic environments simultaneously modify illumination, acoustic conditions, atmospheric chemistry, and temperature, yet insect responses are commonly interpreted within single-driver literatures. This separation risks conflating changes in environmental information with changes in sensory detection, behavioral execution, life-history performance, and ecological consequence. Evidence published from 2017–2026 was evaluated using an article-specific coding scheme that distinguished environmental driver, sensory modality, direct versus signal-mediated exposure, life stage, experimental setting, behavioral endpoint, downstream consequence, and major inference boundary. Laboratory, semi-natural, field, observational, mechanistic, and factorial evidence were kept analytically distinct. Artificial light altered flight orientation, attraction, temporal activity, mate localization, development, and local abundance, with effects conditional on spectrum, intensity, temporal waveform, taxon, and endpoint. Noise and vibration affected mate searching, development, life history, signaling-associated traits, and reproductive outcomes, but realistic traffic noise, generic noise, and substrate vibration were not equivalent exposures. Chemical pollutants acted both by transforming external odor signals and by altering insect sensory or behavioral function. Temperature changed locomotion and thermoregulation directly and also modified plant-derived information used by insects. Across domains, behavioral change did not consistently imply fitness or population decline. The evidence supports treating anthropogenic sensory environments as interacting but biologically non-equivalent information regimes. A proposed synthesis therefore separates modification of environmental signals, sensory acquisition and processing, behavioral response, and subsequent ecological consequences, while requiring direct co-exposure evidence before claiming multimodal interaction.

Keywords: Artificial light at night, Anthropogenic noise, Chemical ecology, Thermal modification, Insect behavior, Multimodal sensory environments.

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Introduction

Urbanization and other forms of anthropogenic environmental change expose insects to simultaneous alterations in illumination, temperature, atmospheric chemistry, sound, vibration, resources, and habitat structure. Responses vary substantially among taxa and traits, making a single undifferentiated concept of “urban disturbance” inadequate for interpreting insect behavior [1]. Air-pollution research likewise identifies several possible routes from altered atmospheric chemistry to changed pollinator behavior rather than one uniform pollutant effect [2]. By contrast, the insect literature on anthropogenic noise and vibration remains comparatively restricted and

strongly dependent on communication modality, signal structure, and taxonomic context [3]. Chemical pollution adds a further complication because pollutants can transform volatile information in the environment before an insect encounters it, independently of any direct toxicological action on the receiver [4].

The inferential problem is therefore not simply whether light, noise, chemicals, or temperature affect insects. It is where in the information-to-behavior sequence an effect occurs, whether different drivers act through comparable biological routes, and when an observed behavioral change warrants ecological interpretation. Light may perturb flight control, alter circadian organization, or interfere with mate localization. Acoustic disturbance may mask airborne signals, modify developmental experience, or operate through substrate vibration. Air chemistry may degrade odor plumes, change source emissions, or influence sensory and cognitive function directly. Temperature may alter insect performance itself or change the visual and chemical phenotype of the resources insects evaluate. This review consequently treats anthropogenic sensory environments as composed of separable but potentially interacting channels rather than as a cumulative unitary stress load. Direct observations are separated from inferred mechanisms; laboratory responses from field transfer; sensory detection from behavioral choice; behavioral or physiological traits from fitness; and local ecological responses from population persistence. The cross-driver architecture developed later is a proposed synthesis, not an empirically validated universal mechanism. Its purpose is to identify where evidence from different sensory domains can legitimately be integrated and where differences in taxon, life stage, exposure history, physical setting, measurement, or analytical scale require them to remain separate.

Materials and Methods

Systematic-review reporting was structured according to PRISMA 2020 principles, particularly the requirement that identification, screening, eligibility, exclusion, and inclusion stages be transparently distinguishable [5]. The search logic combined insect and arthropod terms with four principal environmental-driver clusters: artificial light and light pollution; anthropogenic airborne noise and substrate vibration; air pollution, altered odor environments, and chemically mediated sensory disruption; and warming, heat, or urban thermal modification. These were combined with behavioral and ecological outcome terms including orientation, attraction, locomotion, flight, activity, communication, mating, foraging, circadian behavior, phenology, development, reproduction, survival, and pollination. A supplementary interaction logic targeted explicit co-exposure combinations rather than assuming interaction from separate single-driver studies.

Eligibility was restricted to peer-reviewed evidence within the defined publication period that made an interpretable contribution to insect sensory exposure, behavior, life history, ecological interaction, methodological interpretation, or an essential review-method function. Evidence was retained only when the measured exposure, organism, endpoint, and inference could be identified sufficiently to determine what the study did and did not support. Reviews were used for evidence boundaries and synthesis where appropriate, whereas mechanistic or behavioral claims were preferentially anchored in direct empirical studies.

Multisensory exposure coding was proposed for this review to prevent four recurrent forms of inferential collapse. First, environmental-signal modification was separated from direct exposure of the insect. Second, sensory detection was separated from subsequent processing, motivation, orientation, or behavioral execution. Third, immediate behavioral responses were separated from developmental, life-history, fitness, and population consequences. Fourth, co-occurrence of two anthropogenic drivers was separated from statistically or experimentally demonstrated interaction. Exposure properties such as intensity, spectrum, temporal pattern, atmospheric transformation, distance, acoustic structure, substrate transmission, temperature history, life stage, sex, and field versus laboratory context were retained whenever reported.

Quality interpretation was design-sensitive rather than converted into an unsupported numerical score. Controlled experiments were assessed primarily for treatment realism, comparator adequacy, exposure definition, and endpoint specificity; observational studies for confounding and scale limits; field manipulations for environmental realism and control of co-varying conditions; and cross-level studies for whether signal, sensory, behavioral, and ecological measurements actually supported the proposed pathway. Null and contradictory results were retained because they define when a claimed generality fails.

Complete source-by-source retrieval, deduplication, screening, full-text exclusion, and final inclusion counts were not recoverable from the retained review record and therefore are not reconstructed here. No numerical review-flow values are inferred from the citation set.

Results and Discussion

Artificial light affects insect behavior through more than attraction alone. Three-dimensional measurements of free flight show that insects near artificial light can orient the dorsal body surface toward the light source, producing orbiting, stalling, or inversion and thereby destabilizing normal flight control [6]. This mechanism is directly supported for near-source flight behavior but should not be treated as a universal explanation for all responses to artificial light at night (ALAN).

Field mitigation experiments further show that the physical properties of lighting matter. Tailored spectra and shielding can reduce measured attraction of nocturnal insects to road lighting [7]. Such reduction is evidence for behavioral mitigation, not proof of increased survival or population recovery. Temporal organization is also sensitive to ALAN: under semi-natural conditions, light intensity altered daily behavioral rhythmicity in male *Gryllus bimaculatus*, with responses varying across exposure levels [8]. Artificial lighting therefore cannot be coded adequately as simply present or absent.

Temporal waveform provides another exposure dimension. Pulsed ALAN altered the flight behavior of wild moths across numerous species, indicating that temporal modulation can influence visually guided behavior independently of nominal illumination alone [9]. Spectral composition and intensity likewise interact with taxonomic identity. In forest experiments, increasing intensity generally increased insect attraction, whereas color responses differed among taxa [10]. These findings argue against a universally insect-safe wavelength and instead favor taxon- and context-specific lighting assessment.

Behavioral endpoint is equally important. Streetlighting reduced successful male approach to calling female *Sphinx ligustri*, yet intensity effects were not uniformly significant across the measured flight variables [11]. Attraction to a lamp and successful localization of a mate are therefore not interchangeable response variables. Urban dimming experiments add a conservation-relevant but similarly bounded result: lower streetlight levels reduced insect attraction, while responses of pipistrelle bats differed under darker conditions [12]. A reduction in one exposure-related insect behavior can thus coexist with different consequences at another trophic level.

ALAN effects also extend beyond immediate adult flight. Developmental exposure in *Arctia caja* reduced larval survival and constrained female mass, demonstrating that night lighting can generate cross-stage life-history consequences in at least some moths [13]. Stronger ecological evidence comes from matched roadside studies in which lit sites supported lower local caterpillar abundance, accompanied by experimental evidence of altered feeding [14]. These results justify concern about local ecological consequences while remaining insufficient to attribute broad-scale insect decline solely to artificial lighting.

Overall, the light literature supports a multidimensional exposure description incorporating intensity, spectral composition, temporal pattern, life stage, behavioral task, and ecological context. It also illustrates the review's central inference boundary: evidence for altered orientation, attraction, timing, feeding, or development becomes progressively less direct when translated into fitness or population persistence.

Anthropogenic noise and vibration

Anthropogenic acoustic disturbance is heterogeneous in both physical form and biological route. Airborne traffic noise, broadband laboratory noise, signaling interference, and substrate-borne vibration should therefore not be merged into a single exposure class. Contemporary field evidence illustrates the danger of causal overinterpretation. Male *Glyptobothrus maritimus* from noisier roadside habitats showed greater stridulatory tooth density in one sampling year, but the association was not consistently recovered subsequently and did not translate straightforwardly into signal-frequency change [15]. The pattern supports environmental association, not demonstrated evolutionary adaptation.

Developmental experience can alter adult acoustic behavior. Female *Teleogryllus oceanicus* reared under masking noise later required more time to locate signaling males, and prior noise exposure did not confer compensatory performance when females were tested in noise [16]. Yet the properties of the acoustic treatment itself are critical. In *Gryllus bimaculatus*, traffic noise disrupted mate searching whereas white noise did not generate the same effect [17]. Generic broadband noise is therefore a poor substitute for realistic anthropogenic acoustics when the spectral and temporal relationship between noise and biological signal matters.

Life-history outcomes further demonstrate why behavioral impairment cannot be translated automatically into fitness loss. Developmental masking noise in *T. oceanicus* lengthened juvenile development and shortened adult lifespan, but lifetime reproductive output was not detectably reduced [18]. Intermediate costs and realized

reproductive output can consequently diverge. In addition, acoustic disturbance need not be airborne. Substrate-borne seismic noise altered brood-size dynamics in the burying beetle *Nicrophorus marginatus*, demonstrating a distinct vibration-mediated route into reproductive performance [19].

These findings create an evidence-conflict problem that prose alone tends to obscure: apparently similar “noise effects” differ according to modality, comparator realism, developmental timing, endpoint, and strength of evolutionary or fitness inference. The distinctions are reconciled in **Table 1**.

Table 1. When “anthropogenic noise” is not one exposure: reconciling acoustic modality, developmental history, endpoint discordance, and inference strength

Exposure pattern and scope	Measured construct	Main evidence pattern	Quality boundary	Synthesis consequence
Airborne acoustic exposure and developmental history				
Noise/vibration evidence pattern Roadside acoustic gradient and signaling morphology Taxonomic or methodological scope Adult male grasshopper; repeated field comparison	Stridulatory morphology and habitat acoustics	Morphological association present in one sampling period but not consistently reproduced	Observational association; developmental plasticity and selection unresolved	environmental association should remain separate from evolutionary adaptation
Noise/vibration evidence pattern Developmental masking exposure Taxonomic or methodological scope Female field cricket; developmental-to-adult experiment	Adult mate localization after juvenile acoustic treatment	Early masking experience impaired later mate finding rather than producing compensatory performance	Single species and controlled acoustic regimes	Exposure history should be retained as part of the behavioral context
Noise/vibration evidence pattern Traffic noise versus white noise Taxonomic or methodological scope Female field cricket; playback experiment	Phonotactic mate searching	Realistic traffic noise disrupted search whereas white noise did not produce the same response	Laboratory task and species-specific signal ecology	Noise spectrum and temporal structure are analytically relevant, not interchangeable nuisance details
Noise/vibration evidence pattern Life-history cost with reproductive-output null Taxonomic or methodological scope Field cricket; longitudinal developmental experiment	Development time, adult lifespan, lifetime reproduction	Development and lifespan costs occurred without detected reduction in lifetime reproductive output	Laboratory environment; compensation mechanism unresolved	Trait costs should not automatically be translated into realized fitness loss
Substrate-borne disturbance				
Noise/vibration evidence pattern Substrate-borne disturbance Taxonomic or methodological scope Burying beetle; reproductive experiment	Brood-size dynamics	Seismic noise altered reproductive outcome through a vibration-mediated context	Mechanistic pathway within parental care remains incompletely resolved	Substrate vibration should remain distinct from airborne acoustic masking

Chemical pollution and novel odors

Chemical pollution can disrupt insect behavior before any pollutant contacts the sensory organ. Nitrate-radical chemistry can degrade floral monoterpenes in nocturnal odor plumes, reducing the integrity of floral information and moth visitation in the studied pollination system [20]. This provides unusually direct evidence for an external signal-transformation pathway: atmospheric chemistry changes the information available to the insect.

Ozone research shows why the location of that transformation matters. In the Mediterranean fig–fig-wasp system, direct ozone exposure of the plant did not reproduce the same response as atmospheric reaction of ozone with the emitted odor blend; plume chemistry altered the signal reaching the pollinator [21]. Plant exposure, atmospheric transmission, and insect exposure therefore represent different causal stages. Field experiments with diesel exhaust and ozone likewise found reduced activity or visitation among pollinator and parasitoid components of

flying-insect communities [22]. These are ecologically relevant field responses, but activity and visitation remain different evidence levels from long-term population change.

Direct chemical exposure of the insect can produce a different pattern. In *Bombus terrestris*, chronic exposure to an insecticide formulation altered chemosensory orientation while measured antennal detection of floral scent remained unchanged [23]. Behavioral impairment therefore need not imply failure of peripheral odor detection. More generally, volatile-mediated signaling can be disturbed through altered cue production, atmospheric transformation, receptor-level effects, learning, or memory, and these stages should not be collapsed into one olfactory mechanism [24].

Other studies demonstrate direct sensory and cognitive effects. Acute ozone exposure altered antennal responses and impaired floral-odor learning or recall in honey bees under laboratory conditions [25]. Atmospheric studies, however, show that pollutant concentration alone is not an adequate descriptor of olfactory exposure: ozone-driven degradation interacts with plume mixing, reaction time, and distance to determine the odor information remaining available for recognition [26]. Semi-field experiments further indicate that behavioral plasticity matters, because *B. terrestris* altered foraging in ozone-modified odor environments while retaining an ability to learn altered odor cues [27].

Taken together, the chemical evidence requires at least four analytically separate locations of disruption: the signal-emitting organism, the atmospheric transmission pathway, the insect sensory system, and the behavioral or learned interpretation of the signal. These locations can interact, but demonstrating one does not establish the others. This distinction is essential when comparing chemical pollution with artificial light or noise, because an apparently similar endpoint—failed orientation, reduced visitation, or delayed search—may arise at a different stage of information processing.

Thermal modification

Temperature modifies insect sensory environments through both direct and indirect pathways. In experimental bumblebees, elevated temperature altered flight speed and foraging allocation: some activity measures increased while trips per worker declined [28]. Increased locomotor rate is therefore not synonymous with improved foraging performance. At the colony level, *Bombus impatiens* increased fanning under high temperature, but thermoregulatory compensation became insufficient at the hottest treatment, while some foraging and adult-emergence endpoints remained unchanged [29]. Heat responses can thus combine compensation, physiological constraint, and endpoint-specific null effects.

Temperature also changes the information insects receive from other organisms. Plants grown under hotter conditions altered multiple visual and chemical traits, which in turn changed pollinator and herbivore decisions and affected plant reproductive output [30]. Such responses should be distinguished from direct thermal effects on the insect because temperature acts first on the signal source. Field evidence adds further heterogeneity: urban moth phenology did not show one uniform response across species but varied with traits such as temperature niche and body size [31]. Urban environmental association therefore cannot be reduced to a universal warming response.

Finally, developmental plasticity must remain separate from evolutionary adaptation. Common-garden work with *Pieris rapae* found plastic changes in thermal limits after developmental temperature exposure but no detectable urban–rural evolutionary divergence in those limits [32]. The result supports plasticity while explicitly failing to establish evolved urban thermal adaptation.

The principal thermal distinction is consequently not a simple gradient from benign to stressful temperature. Thermal modification can act on the insect’s current performance, evoke compensatory behavior, restructure developmental physiology, alter the phenotype of a signal-producing organism, or covary with broader urban conditions. **Table 2** separates these stages because each supports a different interpretation and a different validation requirement.

Table 2. Thermal modification changes the location of the behavioral cause: distinguishing direct performance, compensation, source-mediated cues, phenology, and developmental plasticity

Direct locomotor/foraging response		
Adult bumblebee workers under contrasting temperatures	Flight speed, visit dynamics, trips per worker	Different behavioral components can shift in opposite directions

Faster behavior is not automatically improved performance	Measure multiple behavioral outputs together with energetic or resource consequences	Evidence-supported distinction
Compensatory thermoregulation under heat load		
Bumblebee colonies	Increased fanning, colony thermal control, additional colony outcomes	Behavioral compensation may precede incomplete thermoregulation at higher heat
Some endpoints can remain unchanged during the same exposure	Separate compensatory effort from successful regulation and downstream colony effects	Evidence-supported distinction
Signal-source remodeling		
Heat-exposed plants interacting with pollinators/herbivores	Changed plant visual/chemical traits and altered insect choice	Temperature can modify behavior indirectly by changing the information source
Not evidence of direct thermal sensing by the insect	Partition source-mediated and receiver-mediated thermal pathways experimentally	Evidence-supported distinction; cross-driver use is proposed
Trait-conditioned urban phenology		
Larval and adult moth assemblage	Species-specific phenological associations along an urban gradient	Aggregate responses can conceal species-level heterogeneity
Urban variables covary and cannot be assigned solely to temperature	Couple microclimate measurement with species traits and repeated temporal sampling	Evidence-supported boundary
Developmental plasticity without detected divergence		
Urban/rural-source butterfly offspring in common garden	Thermal-limit response to developmental temperature with no source-population divergence	Plastic thermal responses need not indicate evolutionary change
Population, trait and experimental-power limits remain	Test plasticity and inherited divergence with designs that explicitly separate environmental history from population origin	Evidence-supported distinction; adaptation inference rejected

Multimodal interactions

Direct tests of interacting anthropogenic drivers remain much narrower than the single-driver literature. In female *Gryllus veletis*, factorial manipulation of ALAN and anthropogenic noise altered life-history traits, but combined exposure was not consistently more severe than either driver alone [33]. A separate factorial experiment in *Teleogryllus oceanicus* found stronger temperature effects across several developmental and mating-related endpoints than ALAN effects, with limited statistical interaction between the two exposures [34].

Together, these experiments reject a default assumption that coincident sensory alterations necessarily generate additive or synergistic biological effects [33, 34]. The proposed interaction rule used here is therefore conservative; multimodal interaction requires explicit co-exposure evidence, and its form may include dominance, nonadditivity, compensation, antagonism, or endpoint-specific independence.

The distinction is compressed in **Table 3** because interaction topology is more informative than simply recording whether two drivers were present.

Table 3. Testing interaction rather than assuming cumulative sensory load

Interaction dimension	Driver-specific effects	Driver dominance	Nonadditive outcome
Biological context	Factorial light–noise exposure	Factorial light–temperature exposure	Both factorial systems
Observable basis	Separate main effects across life-history traits	Temperature stronger across several endpoints	Combined response not consistently proportional to single-driver responses
Interpretation	Co-occurrence does not itself demonstrate interaction	One driver can structure the response more strongly	Simple cumulative-load models are insufficient
Boundary	One cricket system	Species and treatment specific	Direct evidence taxonomically narrow

Required test	Explicit factorial contrasts	Compare standardized exposure gradients	Replicate across orders, life stages and field settings
Status	Evidence-supported	Evidence-supported	Proposed cross-system inference

Behavioral domains affected

Across drivers, similar behavioral labels can conceal different biological processes. Artificial light can alter flight control itself [6], whereas pulsed illumination modifies flight under different temporal visual conditions [9]. Chemical exposure provides an especially clear boundary: altered odor-oriented behavior may occur despite unchanged peripheral scent detection [23]. “Orientation” should therefore not be treated as a single mechanism. Mating and communication responses also depend on exposure history and task. ALAN alters behavioral timing [8]; streetlighting can interfere with moth mate localization [11]; and developmental acoustic exposure can impair later phonotaxis [16]. Foraging responses span still different pathways: atmospheric chemistry can degrade floral information before detection [20], while elevated temperature can alter insect locomotor allocation directly [28] or remodel plant traits that insects subsequently evaluate [30].

The synthesis therefore organizes behavioral effects temporally—from information available in the environment, through acquisition and interpretation, to behavioral execution and longer-term consequences—while retaining modality-specific routes. The resulting architecture is explicitly proposed, because no approved study validates the complete cross-driver system.

This separation is needed visually because the same observed endpoint can originate at different positions in the information pathway and is shown in **Figure 1**.

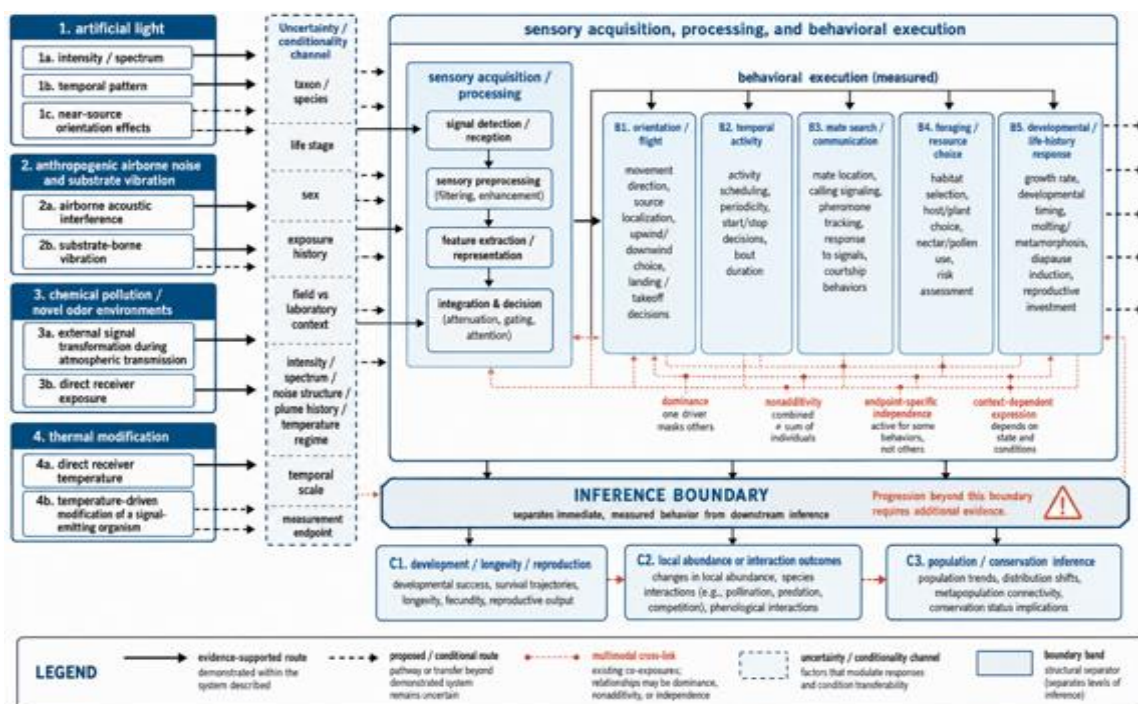


Figure 1. Where anthropogenic sensory change enters insect behavior determines what can legitimately be inferred

Population and conservation consequences

The principal conservation boundary is the difference between a changed behavior and a changed population trajectory. Streetlighting has been associated with lower local caterpillar abundance in matched habitats [14], yet developmental noise can alter growth and lifespan without reducing measured lifetime reproductive output [18]. Pollution experiments can likewise reduce insect activity or visitation without directly measuring population change [22].

Mitigation must be interpreted at the endpoint actually tested. Shielded or spectrally modified road lighting reduces attraction [7], and urban dimming likewise lowers measured attraction [12]; neither result alone

demonstrates population recovery. Thermal experiments similarly span different levels, from colony thermoregulation [29] to plant reproductive consequences mediated through altered insect choice [30].

Table 4 therefore treats conservation interpretation as an evidence-transfer decision rather than a binary harmful/beneficial classification.

Table 4. Evidence thresholds for translating sensory-environment responses into conservation inference

Evidence threshold	Observable basis	Permitted interpretation	Inference boundary	Required validation	Evidential state
Immediate behavioral change	Orientation, activity, mating or foraging response	Exposure alters measured behavior	Fitness consequence unmeasured	Link behavior to survival or reproduction	Evidence-supported
Local ecological response	Local abundance or interaction change	Site-level ecological consequence	Regional persistence not established	Replicated demographic monitoring	Evidence-supported
Mitigation response	Reduced attraction after shielding or dimming	Intervention changes exposure-related behavior	Population recovery untested	Before–after demographic validation	Evidence-supported
Cross-level consequence	Colony or plant reproductive outcome	Downstream biological consequence in tested system	Beneficiary and scale differ among studies	Match endpoints to target conservation unit	Evidence-supported
Highest translation threshold					
Conservation translation	Multiple linked levels measured prospectively	Population relevance becomes testable	Transfer remains taxon- and context-dependent	Demographic and multi-site validation	Proposed decision criterion

The evidence does not support a single generalized “sensory pollution” mechanism. Chemical research explicitly spans external signal degradation and receiver-level effects [2], whereas the acoustic literature remains smaller and highly sensitive to modality and signal structure [3]. Light offers direct sensorimotor evidence near artificial sources [6], while atmospheric chemistry demonstrates that information can be altered before reaching the receiver [20]. Behavioral change can also occur without a corresponding change in a peripheral detection measure [23].

The central contribution of this review is therefore a proposed location-of-effect architecture. Anthropogenic change may act on the information source, transmission environment, receiver, behavioral decision, or downstream life-history process. Which location is demonstrated determines which inference is justified. Taxon dependence in spectral responses [10], temporally inconsistent roadside acoustic associations [15], trait-conditioned urban phenology [31], and thermal plasticity without detected evolutionary divergence [32] all show why heterogeneity should be preserved rather than averaged into universal statements.

Direct multimodal evidence is particularly restrictive. The reviewed factorial experiments demonstrate that co-exposure can produce endpoint-specific, nonadditive, or driver-dominated outcomes, but their taxonomic concentration in crickets limits broader inference. Future work should prioritize field-realistic factorial exposures, explicit separation of direct receiver effects from signal-source or transmission effects, cross-stage designs, and demographic validation of behavioral mitigation. Such studies would test—not merely illustrate—the proposed architecture and determine when altered sensory environments scale from individual decisions to conservation-relevant population change.

A second priority is measurement alignment. Sensory detection, orientation, activity, successful mate localization, reproductive output, local abundance, and population persistence are different evidential objects. Treating them as interchangeable produces apparently coherent narratives while obscuring compensation, null results, and alternative mechanisms. Conservation recommendations should consequently be attached to the endpoint demonstrated and expanded only when additional evidence closes the relevant scale-transfer boundary.

Conclusion

Anthropogenic light, noise, chemical environments, and thermal conditions alter insect behavior through biologically distinct and sometimes interacting routes. Their effects depend on where environmental change enters the information-to-behavior sequence, on taxon and life history, and on the endpoint measured. The proposed

synthesis replaces an assumption of cumulative sensory load with a bounded architecture separating signal modification, receiver processes, behavior, and downstream consequence. Direct multimodal evidence remains limited, and behavioral disruption should not be equated automatically with fitness loss, population decline, adaptation, or conservation benefit. Progress therefore depends on factorial field tests, cross-stage designs, and explicit validation across behavioral, demographic, and ecological scales.

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