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Urban Ecology: Impact Of Noise And Light Pollution On Local Wildlife

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Abstract

The ecosystems of cities place wildlife at risk of a combination of anthropogenic stressors, where artificial night-time light (ALAN) and man-made noise are two examples of chemically mediated pollutants that have quantifiable biochemical effects on fauna. In this paper, we will discuss, in a chemistry way the science behind how light and noise pollution dysregulate endocrine signalling, favor oxidative stress and inhibit immune competence in urban wildlife. Light pollution interferes with the synthesis of melatonin by the pineal gland involving the action of N-acetyltransferase, phase-shifts the expression of circadian clock genes (BMAL1, CLOCK, Per2, Cry1) and increases reactive oxygen species (ROS) by mitochondrial uncoupling. The chronic effect of noise pollution is the activation of the hypothalamic-pituitary-adrenal (HPA) axis, which increases the levels of glucocorticoids and inhibits the pulsatility of the gonadotropin-releasing hormone (GnRH) and stimulates lipid peroxidation. Symbiotic environmental pollutants, such as polycyclic aromatic hydrocarbons (PAHs), volatile organic compounds (VOCs) and nitrogen oxides (NOx) amplify these biochemical insults by stimulating the cytochrome P450 enzymes and producing the actions of secondary oxidative intermediates. An overview of published biochemical and Eco physiological evidence in avian, amphibian, and mammalian taxa has shown convergent evidence of hormonal perturbation and genotoxic stress. The results of this study demonstrate that incorporating environmental chemistry within urban wildlife management is critical, and such biomarkers as urinary 8-hydroxy-2'-deoxyguanosine (8-OHdG) in a 2-hydroxydeoxyuridine (H2O) form, plasma malondialdehyde (MDA), and faecal corticosterone metabolites are logical as routine surveillance tools of urban wildlife health.

Keywords: Melatonin, Cortisol, Circadian Disruption, Oxidative stress, HPA Axis,

1. Introduction

Urbanisation is the most intensively spatial manifestation of environmental changes, which transform natural habitats into a high-density infrastructure with high acoustic energy, constant artificial light, chemical chemical loading of the atmosphere, and thermal anomalies (Gaston et al., 2013). Over half of the population of the whole world is already living in cities, a number that is set to grow to 68% by 2050, which means that the wildlife population is one that is now inhabiting the habitats that are characterized

by anthropogenic environmental chemistry as opposed to natural biogeochemical cycles (Buxton et al., 2017).

Chemically, there are a number of characteristics that define urban environments which include the consistent release of physical pollutants such as light and sound that interacts with the endocrine and neural chemistry of living organisms in radically disruptive fashions. Artificial light at night (ALAN) excites photoreceptors of the blue wavelength of the vertebrate retina, triggering neuroendocrine events that focus on the inhibition of melatonin (N-acetyl-5-methoxytryptamine), a pineal indoleamine which is the master chronobiological signal (Reiter et al., 2010). Conversely, noise pollution uses the hypothalamic-pituitary-adrenal (HPA) axis and ultimately causes chronic stimulation of glucocorticoid hormones, which have catabolic and immunosuppressive effects that are well-established in mammalian and avian physiology (Partecke et al., 2006). The mechanistic connection between the light and circadian disturbance was made with the breakthrough studies indicating that the light in short-wavelength (blue) spectral range (~ 460-480 nm) is the most powerful in activating the melanopsin-containing intrinsically photosensitive retinal ganglion cells (ipRGCs) (Brainard et al., 2001). Melanopsin (OPN4) is a structurally related photopigment and has a signalling cascade involving Gq/11-coupled phospholipase C which produces inositol trisphosphate (IP3) and calcium-mediated depolarisation (Gaston et al., 2013).

More importantly, light as well as noise pollution do not exist in isolation but coexist with other chemical pollutants polycyclic aromatic hydrocarbons (PAHs), nitrogen oxides (NOx), particulate matter (PM2.5), volatile organic compounds (VOCs), and ozone (O3), which are inherent to urban combustion chemistry. These co-stressors are biochemically active; they trigger the action of cytochrome P450 (CYP) enzyme systems, give rise to reactive oxygen species (ROS), create DNA adducts, and interfere with the chemistry of olfactory receptors, which only enhances the impact of physical pollutants (Jones et al., 2015). The interactive chemistry of assemblages of urban pollutants, therefore, comprises a multi-target biochemical insult to wildlife physiology, which is inexplicable solely through the study of individual stressors.

This paper aims to accomplish the following: (i) to systematically describe the chemical and biochemical pathways through which urban light and noise pollution impact wildlife physiology; (ii) to critically assess evidence that hormonal disruption, oxidative stress, and changes in circadian chemistry takes place across taxa (iii) synthesise evidence through a single unified environmental chemistry approach; and (iv) to identify molecular biomarkers that could be used to monitor how urban wildlife is biochemically stressed by these pollutants. Tables 1, 2, Figures 1, 2, are assembled overviews of the categories of pollutants, influenced pathways, and species level biochemical results.

2. Methodology

2.1 Research Design

This study adopts a secondary research approach based on a systematic review of peer-reviewed literature from Web of Science, Scopus, and PubMed. Relevant studies (2000–2024) were identified using keywords related to artificial light at night (ALAN), noise pollution, urban pollutants, and their biochemical effects on wildlife.

2.2 Inclusion and Exclusion Criteria

Studies were included if they reported quantitative biochemical or physiological effects of urban pollutants on wildlife. Human-only and domestic animal studies were excluded. Preference was given to research focusing on molecular biomarkers such as melatonin, corticosterone, ROS, and enzyme activity. Methodological quality and ecological relevance were also assessed.

2.3 Conceptual Framework

The framework links three pollutant types (light, noise, chemical) to biochemical responses (hormonal disruption, oxidative stress, genotoxicity) and ecological outcomes (individual fitness and population dynamics), guiding the synthesis and development of Figures 1 and 2.

4. Results and Discussion

4.1 Circadian chemistry and melatonin suppression: Empirical evidence

There is strong empirical support of melatonin inhibition induced by ALAN in wildlife across a variety of vertebrates. Nocturnal light at ecologically significant levels (0.5-5 lux, which is close to exposure of urban streetlights) decreased plasma melatonin by 30-70 percent in 24 hours of exposure, and the recovery took 5-7 nights of darkness (Visser et al., 2006). The biochemical action entails inhibition of the rate-limiting enzyme in melatonin production, arylalkylamine N-acetyltransferase (AANAT) which is involved in the transformation of serotonin to N-acetylserotonin the immediate form of melatonin precursor (**see Figure 1**). More importantly, melatonin depression does not have only chronobiological effects. Melatonin is a strong free radical scavenger, and its molar reactivity is higher than that of glutathione with hydroxyl radical (OH.) (Reiter et al., 2010). Its inhibition thus limits antioxidant ability, and allows ROS to build up and cause oxidative injury in the downstream analysis. Plasma malondialdehyde (MDA, or end product of lipid peroxidation) and other oxidative stress biomarkers, such as 8-OHdG (or marker of oxidative DNA damage, in the urine), are more increased in the ALAN-exposed songbirds, and thus, a direct chemical relationship between light pollution and genotoxicity is established (Sands et al., 2021). Loss of phase-shifting of clock genes, is a second-level of chemical perturbation. In city dwelling robins of Europe (Erithacus rubecula), ALAN exposure accelerated maximums of Per2 expression up to 4 hours compared to the natural photoperiod control (Fuller et al., 2007). Per 2 codes transcriptional repressor, which heterodimers with CRY proteins; the rhythms of mistimed accumulation of Per 2 misaligns the downstream gene regulatory network, not only with reproduction, but also with immune functioning, lipid metabolism and xenobiotic detoxification rhythms. This shows the chemical biology of circadian disruption is not a pathway level insult but rather a systems-level insult.

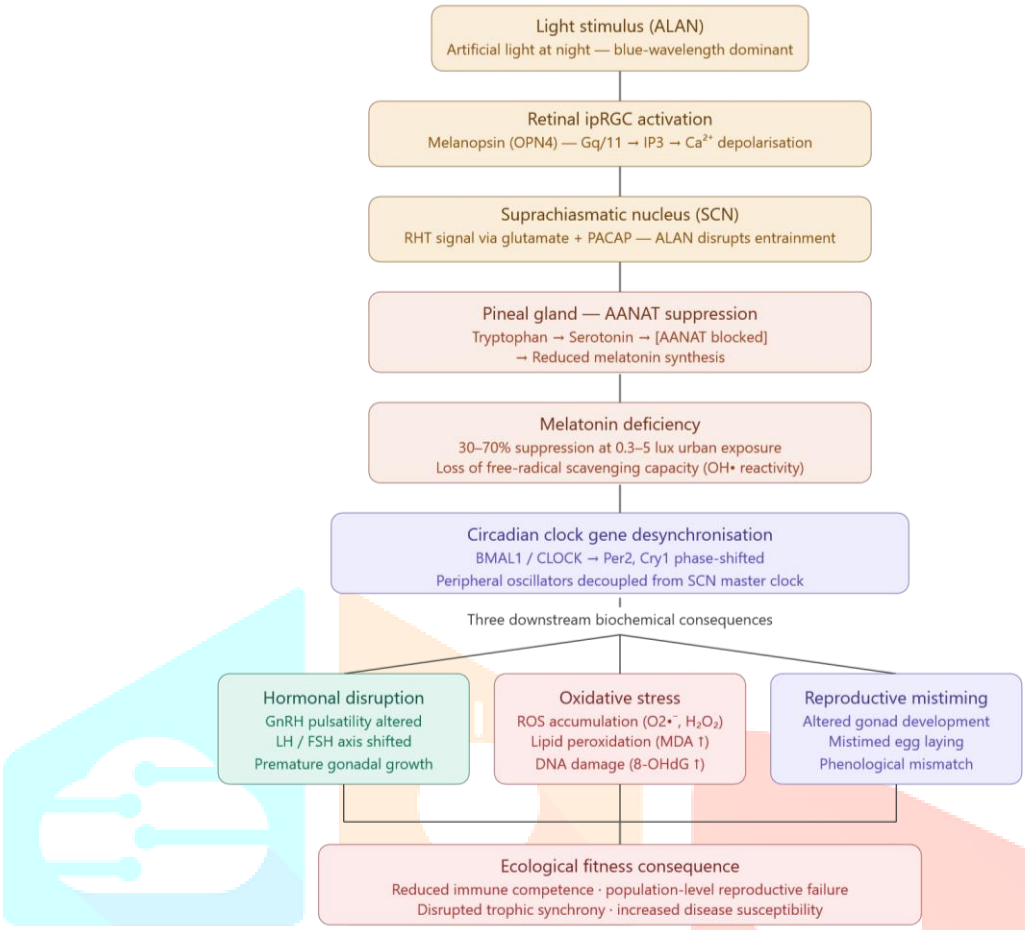


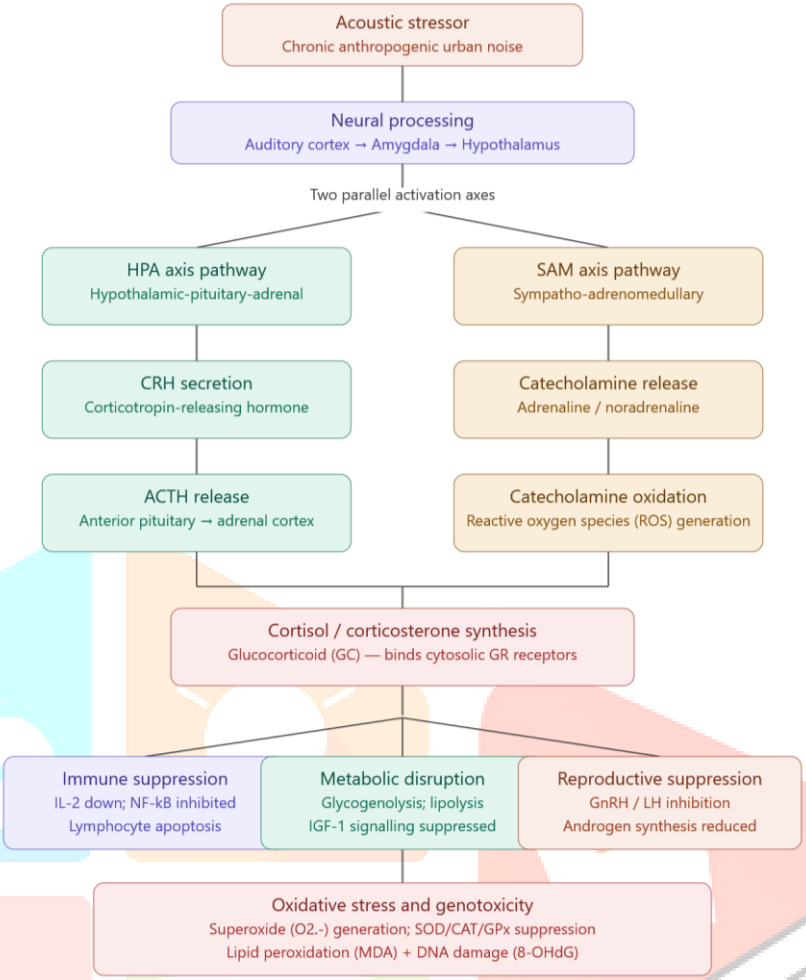
Figure 1. Mechanistic pathway linking artificial light at night (ALAN) to circadian chemistry disruption and downstream biochemical consequences in wildlife.

4.2 HPA Axis Activation by Noise Pollution: Biochemical Cascades

The mechanisms of noise-induced glucocorticoid elevation have been described in birds, amphibians, and mammals in successively greater detail. Francis et al. (2011) established that white-crowned sparrows (Zonotrichia leucophrys) nesting in the noisy habitat had glucocorticoid levels substantially higher than conspecifics nesting in quiet habitat with parallel decreases in nestling body mass which could be ascribed to a GC-mediated suppression of insulin-like growth factor 1 (IGF-1) signalling. The inhibition of IGF-1 is an expression of more global GC-initiated catabolic programming glycogenolysis, lipolysis and protein catabolism - depleting somatic resources during a vulnerable developmental phase. This molecular mechanism can be explained by the fact that chronically high levels of corticosterone disrupt the negative feedback sensitivity of GR in the hippocampus which is achieved through the down-regulation of GR by phosphorylation of GC-receptor proteins. This desensitization leads to HPA axis dysregulation that is maintained by GC production despite the lack of acute stressors a chemical signature of allostatic overload reported in urban birds' populations (Partecke et al., 2006). The oxidative chemistry of continuous GC increases in the downstream is depicted in **Figure 2**: mitochondrial production of ROS, decreased activity of SOD and CAT, increased lipid peroxidation isoforms, and increased accumulation of DNA strand breaks altogether represent a chemically consistent toxicological signature.

Noise-mediated biochemical disruption of aquatic systems is not an exception. It has been demonstrated that traffic noise transferred via soil to pond systems increases corticosterone in Bufo bufo embryos and larvae, which have an impact on metamorphic time and limb development (Halfwerk and Slabbekoorn, 2009). The biochemical pathway is the GC-mediated down-regulation of thyroid hormone receptor beta

(TRbeta) expression which is a major agonist of amphibian metamorphosis as an example of cross-system endocrine interference of anthropogenic acoustic pollution.



Note: HPA = Hypothalamic-Pituitary-Adrenal; SAM = Sympatho-Adrenomedullary; CRH = Corticotropin-Releasing Hormone; ACTH = Adrenocorticotrophic Hormone; GnRH = Gonadotropin-Releasing Hormone.

Figure 2. Biochemical cascade initiated by chronic anthropogenic noise exposure in wildlife

4.3 Oxidative Stress Chemistry: A Common Biochemical Endpoint

One of the findings which are of crucial importance throughout the literature on ALAN and noise pollution is the fact that they all converge at one biochemical endpoint: oxidative stress. Light inhibition of melatonin and noise stimulation of the HPA generate similar conditions that are conducive to the build-up of ROS, namely superoxide (O2-), hydrogen peroxide (H2O2), and hydroxyl radicals (OH). This overlap is not accidental and represents the dominant position of the mitochondrial electron transport chain (ETC) as a major source of cellular ROS in endocrine perturbed conditions (Valko et al., 2007).

The Fenton reaction:

$Fe^{2+} + H_2O_2 = Fe^{3+} + 2OH-$ is specifically applicable in city wildlife that is subjected to metal-enriched PM2.5, which transports bioavailable iron and copper into the pulmonary and systemic systems. These transition metals induce the H2O2 formation by the stressed mitochondria which both catalyse oxidative damage to the DNA of lung epithelium and lymphocytes in circulation. Combined genotoxic stress, in this way, the chemistry of urban pollutant mixtures synergistically increases the oxidative effects of ALAN and noise exposure. Oxidative stress indices that can be detected in wildlife are plasma MDA (measured

using thiobarbituric acid reactive substances [TBARS] assay), 8-OHdG in urine (measured by ELISA or HPLC-MS/MS), and erythrocyte catalase activity. They are non-lethal, non-radioactive, and chemically specific biomarkers of oxidative burden that have been confirmed in multiple urban birds (Sands et al., 2021), indicating that they can be used as sentinel quality indicators of urban environments (Table 1).

Table 1. Types, sources, affected wildlife groups, and primary biochemical mechanisms of urban pollutants relevant to local wildlife.

Pollutant Type	Primary Urban Sources	Affected Wildlife Groups	Key Chemical/Biochemical Mechanism
Artificial Light at Night (ALAN)	LED streetlights, signage, vehicles	Vertebrates, insects, birds	Melatonin suppression, circadian disruption, phototaxis alteration
Anthropogenic Noise	Traffic, construction, aviation, industry	Birds, amphibians, mammals	Cortisol elevation, HPA axis activation, masking of bioacoustic signals
Nitrogen Oxides (NOx)	Combustion engines, power plants	Plants, invertebrates, birds	Nitrosative stress, peroxynitrite formation, olfactory receptor damage
Volatile Organic Compounds (VOCs)	Fuels, solvents, industrial emissions	Insects (pollinators), birds	Floral scent masking, olfactory pathway disruption
Ozone (O_3)	Photochemical smog (secondary pollutant)	All urban wildlife	Lipid peroxidation, pulmonary oxidative injury
Particulate Matter (PM2.5)	Combustion, road dust, industry	Avian, mammalian respiratory systems	ROS generation, mitochondrial dysfunction, inflammation

4.4 Chemical Co-Pollutants and Endocrine Interaction

ALAN and noise are further hormonally influenced by the endocrine distorting chemistry of the overall urban pollution matrix. The PAHs, especially benzo[a]pyrene (BaP) and naphthalene are AhR ligands (stimulating the expression of CYP1A) and oestrogen receptor (ER) agonists or antagonists, depending on hydroxylated metabolite profiles (Incardona et al., 2004). The resultant hormonal chemistry is very complicated: PAH-stimulated AhR-activation represses oestrogen production by upregulating oestradiol breakage by CYP1B1, and concomitantly produces reactive adducts of BAe, Ad, of BaP-7, 8-dihydrodiol-9, 10-epoxide (BPDE), which damages oocyte and gonadal tissue DNA.

Specific attention should be paid to VOC-mediated interference of the olfactory chemistry. The ozone concentration (O3, 40-100 ppb) in cities is high enough to oxidatively break down floral volatiles based on terpene compounds even prior to reaching the antennae of pollinators thus decreasing the strength of potential olfactory cues by as much as 90 percent, when modelled (Knop et al., 2017). NOx in avian reactants with volatile olfactory cues present in the atmosphere as a signal of mate choice and territory marking, and changes the chemical topography of social communication. The planetary oxidation of

terpene double bonds to produce oxidized products with unfavorable odors and the oxidation of thiol-based odorants by NO2 through radical mediators mediate these chemical ecology upsets which have an ecological impact extending via the mating systems and population dynamics.

4.5 Immune Chemistry and Ecological Fitness Consequences

Among the ecologically significant biochemical changes of chronic noise and light stress, glucocorticoid-mediated suppression of the immune system should be listed. Cortisol and corticosterone can interact with cytosolic GRs in lymphocytes at the molecular level and trigger apoptosis and the downregulation of interleukin-2 (IL-2) production, which is the main T-cell proliferative cytokine. The inhibition of nuclear factor-kappa B (NF-kB) which is a master transcription factor in the expression of pro-inflammatory genes by GC further decreases immunocompetence through the inhibition of tumour necrosis factor alpha (TNF-alpha), interferon-gamma (IFN-gamma), and antimicrobial peptide genes expression (Valko et al., 2007).

This immune chemistry has ecological implications, including higher parasite loads in the urban bird population living in the noise-polluted areas (Francis et al., 2012), and lower vaccine antibody titres in amphibians subjected to noise pollution in laboratory tests. The physiological trade-off between maintaining stress responses and immune functionality is an archetypal life-history chemical conflict: same glucocorticoid signalling molecules mobilising energy in acute stress response are themselves anti-immune, generating a biochemically explicable fitness cost of being exposed to urban pollution.

5. Conclusion

As has been shown in this review, the effects of noise and light pollution on the urban wildlife are essentially issues of environmental chemistry in which the disruptive effect of endocrine signalling molecules, interference with intracellular redox chemistry and chemical ecology of bioacoustic and olfactory communication systems play central roles. The biochemical data are drawn together to three key findings. To start with, light at night at night suppresses the production of melatonin by blocking the synthesis of AANAT, which is a biochemical intervention with ramifications that run the entire gamut of circadian gene regulation to reproductive endocrinology. Second, HPA axis becomes activated chronically by anthropogenic noise leading to the production of glucocorticoid levels that suppress immune activity, stop the production of reproductive hormones, and induce oxidative stress by uncoupling the mitochondrion. Third, these effects are supported by the urban chemical matrix of PAHs, VOCs, NOx, O3 and PM 2.5 via induction of CYP enzyme, Fenton chemistry and chemical ecology disruption, forming a multi-target biochemical insult. The implication to the policy is noteworthy. Switching city lighting to amber LEDs (reduced melanopsin stimulation), noise mitigation areas around vulnerable zones of wildlife, and stricter urban air quality would, according to the biochemical evidence that has been synthesised in this paper, decrease the endocrine and oxidative stress load on urban wildlife populations. Non-invasive biochemical biomarker-based policing such as faecal corticosterone metabolite and urinary 8-OHdG and erythrocyte antioxidant enzyme profiles provide useful mechanisms of monitoring wildlife biochemical health in cities. Subsequent studies need to focus on characterisation of pollutant mixture interactions at receptor and enzyme levels, city-specific toxicokinetic mechanisms in urban wildlife and the combination of metabolomic and epigenomic techniques to reflect the complete chemical signature of urban environmental stress upon wildlife physiology.

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