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Influence of Environmental Pollution on Wildlife Health, Reproductive Performance, and Ecosystem Functioning

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Key Words

Environmental pollution, Wildlife health, Reproductive performance, Ecosystem functioning, Ecotoxicology, Endocrine disruption, Biodiversity decline, Chemical contamination, Wildlife conservation, Ecosystem health.

Abstract

Environmental pollution, such as endocrine-disrupting chemicals and pesticides used in agriculture, among other consequences for invertebrate prey bases, is another pervasive cause of wildlife decline that differs from habitat destruction by its mechanism. The present paper is devoted to reviewing evidence on the ways in which pollutants influence wildlife individuals' health and reproduction and, further on, influence population and ecosystem functioning as well. It was demonstrated through fundamental toxicological studies that a wide range of human-derived chemicals disrupt the hormone function in wildlife species, causing certain reproductive and developmental problems in birds, fish, reptiles, and mammals. Field data from agricultural systems have, however, connected a particular and common family of insecticides with population reductions in insectivorous birds due to surface water pollution; studies on agricultural intensification, on the other hand, demonstrate that the use of pesticides has a negative influence on biodiversity at various trophic levels, even after controlling for other aspects of intensification. Individual and population-level impacts are related to the functioning of ecosystems by severe reductions in the biomass of flying insects found in nature reserves within agricultural landscapes. This suggests that pollution affects the prey base and ecological functioning of even formally protected habitats. In sum, this information demonstrates that pollution represents a multi-level threat to wildlife from individual endocrine disruption to landscape-level reductions in insect biomass, which cannot be dealt with through area-based conservation measures.

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Introduction

As the most thoroughly researched causes of wildlife decline, habitat loss and fragmentation cannot be overlooked; however, environmental pollution is a separate threat that negatively affects the well-being and fertility of wild animals in an otherwise structurally unaltered environment (Hamlin & Guillette Jr., 2010). Pollution can affect wildlife in different ways, including direct contamination, accumulation along food chains, and disturbance of invertebrates on which vertebrates rely. The impacts may manifest themselves in different forms, from physiological effects to population decline and, eventually, alteration of ecosystem function. This review brings together all aspects of biological organization affected by pollution, from the basic toxicology underlying its influence on the endocrine system of wildlife to field studies demonstrating connections between pollution and wildlife decline as well as ecosystem alteration.

Endocrine Disruption and Reproductive Effects

The basic science behind the impact of environmental pollutants on wildlife reproductive health was formulated by the analysis of two decades' worth of studies showing that various man-made compounds can alter the functioning of the endocrine systems in developing individuals, and thus can have quite distinct effects compared to the ones in adult individuals exposed to such contaminants (Acevedo-Whitehouse & Duffus, 2009). Such an analysis of the basic science revealed that endocrine disruptors caused reproductive harm in various wildlife species by causing decreased fertility, developmental anomalies, and disturbances in sexual differentiation in birds, fish, reptiles, and mammals, and further revealed that the critical factor in whether the exposure causes any reproductive harm was not the dose but when during development the exposure occurred (Colborn et al., 1993). The concept of developmentally critical time periods in relation to pollutant-induced harm has been very important for the field of wildlife toxicology, since it means that the concentrations of pollutants deemed safe for adult wildlife can still cause significant harm to reproduction.

Pesticide Exposure and Population-Level Decline

Neonicotinoids and Insectivorous Bird Decline

There is direct field evidence linking a certain class of pesticides to population decline in vertebrates in the study conducted to establish the link between trends in local population sizes of insect-eating farmland birds in the Netherlands with the concentration of surface water imidacloprid, a neonicotinoid insecticide. The results of this study show that negative trends in the local population size of these birds are associated with high concentrations of surface water imidacloprid even after controlling for other land use change factors. This study showed that neonicotinoids have a cascade effect on the ecosystem through depletion of the prey resources needed for reproduction of insect eating birds during their breeding season

(Hallmann et al., 2014). This is a significant finding since it shows that a class of pesticides primarily known to affect pollinating insects has population-level impacts on vertebrates several levels up the food chain.

Agricultural Intensification and Multi-Trophic Pesticide Effects

Additional data on the influence of pesticides on wildlife at different trophic levels can be obtained from the research that uncovers the relative importance of various factors contributing to biodiversity loss within nine European regions. In particular, it was shown in the research that out of the thirteen different factors characterizing intensification of agriculture, the use of insecticides and fungicides negatively influenced the diversity of plant, beetle, and ground-nesting bird species, all being representatives of three different trophic levels, as well as reduced the biocontrol potential of the studied ecosystems with respect to agricultural pest control (Geiger et al., 2010). Notably, in spite of several decades of attempts to decrease the harmful effects of pesticides on wildlife through regulation of their application in Europe, such negative effects still exist.

Ecosystem-Level Consequences: Insect Biomass Decline

The cumulative impact of pollution and agricultural intensification on the functioning of ecosystems is shown through studies of changes in the biomass of flying insects in protected natural areas in Germany over time. In the course of these studies, a reduction of more than 75 percent of the biomass of flying insects was observed over a period of 27 years in protected zones, which means that pollution and inputs from agriculture have led to considerable impairment of ecological functioning even in zones that are formally considered protected (Hallmann et al., 2017). Flying insects provide prey and serve as pollinators for a wide range of species, and, therefore, the reduction of their biomass to this extent means the impairment of ecosystem functioning, regardless of its protection status.

Pathways of Exposure and Bioaccumulation

Trophic Transfer and Biomagnification

Another commonality shared by all of the pollution vectors discussed above is that the contaminants' concentrations and impacts on organisms' physiology are often magnified in the course of food web transfers, a phenomenon referred to as biomagnification. Organisms with high trophic statuses, such as many insectivore and predatory birds examined as part of the neonicotinoid and insect biomass examples (Hallmann et al., 2014; Hallmann et al., 2017), accumulate both through environmental exposure and the consumption of contaminated prey at lower trophic statuses (Guillette Jr. & Guillette, 1996). This trophic magnification accounts for the higher prevalence of apex and near-apex predators in populations demonstrating impacts on reproduction and development caused by environmental pollutants, as observed in fundamental endocrine disruption studies where it was demonstrated that susceptibility to reproductive problems depended

on exposure type and the body burden of the pollutants across species and trophic statuses (Colborn et al., 1993).

Sublethal Effects and Detection Challenges

However, many of the pieces of evidence presented here concern the sublethal effects, low fertility, slow development, and behavioral changes which do not cause immediate death, but weaken the reproduction performance and population growth over time. Sublethal effects are intrinsically more challenging to identify through typical wildlife monitoring because they cannot be seen merely by counting or collecting dead individuals and thus require specific reproductive or physiological assessment. In the case of neonicotinoids and bird populations' decline, the researchers have tried to solve this problem by comparing the population trend data and pollutant concentrations' gradient without direct observation of any toxic effects (Hallmann et al., 2014). As for the long-term insect biomass monitoring, it was made possible only due to the special decades-long sampling program aimed at revealing the gradual change in the insect biomass (Hallmann et al., 2017). This approach gives us a very important insight into wildlife pollution research. Indeed, it is quite possible that the extent of wildlife harm caused by pollution is highly underestimated in many cases where no specific monitoring system to detect sublethal effects has been used.

Policy and Regulatory Considerations

Given the persistence of pesticide-induced biodiversity impacts despite years of EU regulation, one has to question whether the frameworks for the approval and monitoring of chemicals in use may be sufficiently robust to address the impact on wildlife health and reproduction outlined above. Most regulatory systems for chemicals used in agriculture have traditionally been based on the acute toxicity levels, which were derived from the laboratory dose-response testing of a few indicator species, and were therefore ill-suited to addressing the timing-based and low-dose endocrine impacts highlighted in early studies on wildlife toxicology and the trophic-cascade-driven population-level decline in bird populations caused by neonicotinoids, where the causal chain was linked to the decrease in the food base of the affected birds rather than the direct toxicity of the chemical. Moving towards more comprehensive regulatory systems incorporating reproductive and ecological endpoints rather than acute toxicity alone, as well as enhanced post-approval monitoring similar to the insect biomass reduction detected in protected areas, would be the most relevant policy response in light of the evidence outlined above.

Species and Life-History Vulnerability Factors

Not all wildlife species are equally susceptible to health and reproductive risks from pollution; rather, susceptibility is systematically influenced by life-history characteristics and ecological niches. Species that have relatively long lives and produce few offspring each year (e.g., raptors and large mammals) are particularly vulnerable to the sub-lethal reproductive impacts established in pioneering studies on endocrine

disruption because a minor yearly decline in reproductive performance has significant cumulative effects over a long generational period (Colborn et al., 1993). Insectivores experience compound risk routes, as shown in the case of neonicotinoid bird interactions, since these species are exposed to contaminants both directly and through the depletion of their primary food source (Hallmann et al., 2014). Ground-nesting and farmland species also have similar compounded threats, as shown by the evidence that pesticide impact was seen on three different levels of the trophic food chain plants, ground beetles, and ground-nesting birds in the same landscapes (Geiger et al., 2010). Species that live in aquatic and semi-aquatic habitats also have one more route of exposure, namely contamination of the water column and sediments, as expected from their special sensitivity to endocrine disruptors seen in basic toxicological studies (Colborn et al., 1993). Understanding these differences in vulnerability makes it possible to direct efforts of conservation and regulation towards those species that have a compounded threat of pollution.

Discussion

Combining these results reveals a clear chain from molecular-level endocrine disruption to population decline to functional ecosystem decline. The first two pieces of evidence provide the basic biological foundation in terms of understanding how pollutants become biologically damaging in terms of reproduction across wildlife species (Colborn et al., 1993), and the neonicotinoid and agricultural intensification experiments show that, together with depletion of prey resources, this biological effect leads to actual population declines in realistic field conditions (Hallmann et al., 2014; Geiger et al., 2010). The third piece of evidence shows the ultimate outcome of individual and population-level decline across a range of landscapes, in that actual functional collapse occurs, even in nominally protected areas, showing that protection status does not suffice as a protective measure from the adverse impact of pollution on ecosystems (Hallmann et al., 2017). It follows that there is an immediate implication for wildlife conservation, in that since the pollution impacts occur via pathways that include surface water pollution and agricultural runoff, conservation policy has to address both pollution and protected areas. The continued occurrence of biodiversity impacts caused by pesticides, even in the face of the decades-long presence of regulation, also demonstrates the inadequacy of current chemical use policy in light of the impacts that have been shown to be caused, calling for regulatory approaches that will take into account the wildlife reproduction and ecosystem function effects observed rather than merely focusing on toxicity limits.

The evidence on species vulnerability and the exposure pathways involved in pollution also implies that the real price of pollution in terms of population will be systematically underestimated where acute lethality is used to measure impact because of the sub-lethal, trophic amplification, and life history-dependence of the impacts reviewed here (Colborn et al., 1993; Hallmann et al., 2017). For example, the population of a raptor species could show consistent adult survival rates

for years before showing a decrease in numbers caused by a decrease in reproductive capacity due to contamination over the previous years. This delay in detection highlights the need for monitoring programs for wildlife pollution, which would include reproductive or physiological indicators and not just abundances of wildlife populations, especially those species that have been listed as being vulnerable to compounded exposure risk, which are long-lived, insectivorous, and associated with waterbodies. This also indicates that the decline in insect biomass observed at the ecosystem level in the protected areas can serve as an early warning system for reproductive damage in vertebrates that are dependent on insects as a food source.

Conclusion

Pollution of the environment has negative impacts on the health of animals, their reproductive capacity, and also affects the working of the ecosystems due to a chain of mechanisms that starts from disrupting the cellular endocrine system and ends with the reduction of the insect biomass in landscapes. The examples of the neonicotinoid pesticides, as well as the intensification of agriculture, show that there is an impact that causes the decline in the animal populations exposed to pollution, while the studies of the ecosystems show that it results in an important loss of ecological functions that occurs even in the preserved ecosystems.

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