Chapter 9
Consequences of Developmental Exposure to Pollution: Importance of Stress-Coping Mechanisms

Frédéric Angelier

Abstract Environmental pollution is a global phenomenon that affects all continents and dozens of types of pollutants with highly different properties can be found on Earth. These pollutants may result in detrimental environmental conditions with clear negative effects on fitness, but they can also induce more pernicious and subtle effects by triggering maladaptive responses to environmental conditions. Importantly, the impact of pollutants on organismal systems is often also exacerbated during the developmental stage. Indeed, developmental conditions are known to affect the ontogeny of multiple integrative organismal systems, and notably the ontogeny of stress-coping mechanisms. These mechanisms involve cognition, the fight or flight response and the HPA axis; they are crucial to consider in the context of pollution because they govern the ability of the individual to adjust to the environmental perturbations that may arise from physical pollutants. In addition, they may also be disrupted by chemical pollutants, resulting in a maladaptive response to environmental conditions and in pathologies. In this chapter, we first provide an example of how developmental exposure to a chemical pollutant (lead, Pb) may disrupt stress-coping mechanisms with detrimental consequences later in life. Then, we illustrate the impact of physical pollutants on performance by focusing on the example of noise pollution. We especially aim to highlight the importance of stress-coping mechanisms and their flexibility in determining the ability of individuals to cope with noise pollution. Finally, we propose several avenues of research to better understand how wild species may adapt to this polluted world. We emphasize (1) the importance of considering the cumulative and interactive effects of physical and chemical pollutants on stress-coping mechanisms and performance; (2) the potential importance of priming hormesis in adjusting the functioning and the flexibility of stress-coping mechanisms to a polluted environment; (3) the need to consider microevolution to assess whether selection acts on stress-coping mechanisms and favors specific stress-coping traits that are beneficial in a polluted world.

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9.1 A Polluted World

9.1.1 Human Population Growth, Human Activities, and Global Pollution

Since the establishment of human societies, human activities have been associated with multiple types of pollution and notably with the release of multiple pollutants into the environment (Sauvé and Desrosiers 2014). This phenomenon increased to unprecedented rates during the industrial revolution (18th century) and it has accelerated since that period with the development of new technologies, energies, and agricultural processes (Vane et al. 2011; Hayes et al. 2017). Despite the development of several environmentally friendly policies, pollution is intrinsically and tightly linked to the social and economic needs of the worldwide human population. Therefore, it is expected that this coming century will be associated with an ongoing release of pollutants in the environment as the human population is expected to reach 11 billion by 2100 (Bradshaw and Brook 2014).

Importantly, environmental pollution is a global phenomenon that affects all continents and all ecosystems, although some geographical areas or biotopes are obviously affected to a greater extent than others (Wang et al. 2020). This global contamination mainly results from two concomitant factors. Firstly, humans have settled in all continents and human activities are virtually present everywhere on Earth, and even beyond with the recent multiplication of orbital space debris. Secondly, several pollutants can be transported from their area of emission to other areas through biotic (e.g., transfer of pollutants from one area to another through living organisms, Carbery et al. 2018) and abiotic processes (e.g., global atmospheric and ocean circulations, Zhang et al. 2019). For example, some pollutants have been found sometimes at very high concentrations in remote areas that were thought to be pristine (e.g., the Himalayas, or Polar areas, Wang et al. 2019). Initially, this pollution was restricted to specific locations, but the exponential growth of human populations, urban sprawl, the expansion of human activities to many habitats, and the global circulation of pollutants have led to a global contamination of Earth ecosystems (Bernhardt et al. 2017).

Historically, the detrimental effects of contaminants on non-target species have usually been discovered several years after their use and after the occurrence of specific health problems in humans or drastic population declines in wild species. The best example probably comes from DDT, which was used against mosquitoes worldwide. DDT appeared to affect the reproduction of birds by causing egg-thinning (Cooke 1973). The use of DDT has also been recognized as an important endocrine disruptor in humans, and it has been associated with developmental issues and with the occurrence of multiple pathologies in humans including cancer (reviewed in Hayes et al. 2017). Following these scientific studies and discoveries, strict regulations were set up and DDT was banned in most countries in the 70s. DDT is an excellent example of the delay that often exists between the

commercial use of a molecule and the gathering of robust data to assess the threat it may represent to human health and ecosystems (Sauvé and Desrosiers 2014).

Indeed, the exponential rise of the human population and the development of new technologies and industrial and agricultural processes is currently associated with the production and the release of hundreds of these so-called emerging pollutants into the environment (Sauvé and Desrosiers 2014). These molecules aim to replace older molecules that are no longer effective or that are associated with environmental and health concerns and so are progressively being banned by environmental and health agencies. It is now acknowledged that specific research efforts must be carried out to evaluate how humans and wildlife are exposed to these contaminants (the notion of "exposome," Karlsson et al. 2021), to understand their properties which lead to potential interactions with organisms (Pourchet et al. 2020), and to assess the health and environmental issues that are related to these emerging compounds (Dulio et al. 2018). Importantly, other factors may exacerbate current pollution and its effect on human health, so that it is now essential to study how other perturbations may affect the exposome and potentially exacerbate the impact of contaminants on biodiversity and humans (Karlsson et al. 2021). For example, it is predicted that climate change and heat waves will amplify the negative effects of the emission of air pollutants on human health in cities (Harlan and Ruddell 2011).

9.1.2 A Wide Variety of Pollutants

Because of the complexity and multiple sources of pollution, it would understandably be unrealistic to draw up a comprehensive description of this polluted world. Two wide types of pollutants can however be described: (1) chemical pollutants, which include the release of specific compounds or particles in the environment. These pollutants can be transferred to the environment and can contaminate wild organisms through ingestion, inhalation or cutaneous exposure with associated potential health issues. These pollutants are those focused on earlier in this chapter. They include numerous molecules that are used by humans for multiple activities and that have been released in the environment, sometimes for decades. The most ubiquitous of these organic pollutants belong to the following classes: polychlorinated biphenyls, halogenated hydrocarbons, estrogen analogues, phthalates, dioxins, perfluorinated compounds, and brominated flame retardants (Manzetti et al. 2014). Chemical pollutants also include inorganic molecules (i.e., trace elements) that are naturally present in the environment and are even necessary to allow most living organisms to function (e.g., Fe, Cu, Zn). Human activities are, however, associated with important releases of these trace elements and their environmental concentrations may then reach an upper threshold that is associated with significant toxicological effects. Other trace elements have key detrimental effects on living organisms, even at very low environmental doses (e.g., Pb, Cd, As). Importantly, these inorganic pollutants are persistent in the environment and are not

bio-degradable. This wide variety of inorganic and organic molecules is associated with different chemical and biological properties (e.g., half-life, toxicity, bioaccumulation and biotransformation potentials) that determine the toxicological threat that they represent for the environment and human health; (2) non-chemical or physical pollutants, which are associated with the modification of an individual's environment and with an alteration of environmental cues (Halfwerk and Slabbekoorn 2015). This pollution includes, for example, noise pollution, light pollution or electromagnetic pollution which are all known to create unreliable environmental cues that affect wildlife (Dominoni et al. 2020a, b), and to be associated with health issues in humans (e.g., Goines and Hagler 2007). As for chemical pollution, this physical pollution is complex because it is diverse in nature and intensity and it depends on the emitting sources of pollution. For example, noise and light pollution can vary according to the frequency (noise, Slabbekoorn 2019) or the spectrum (light, Gaston et al. 2012), the intensity, and the duration of the pollution. Indeed, the characteristics of physical pollutants are important determinants on their impact on the physiology and the behavior of vertebrates. For example, recent studies have suggested that the intensity and the spectrum of light can modulate the impact of light pollution on circadian rhythms of wild birds (Ulgezen et al. 2019).

9.1.3 How Can Pollutants Affect Vertebrates?

Historically, toxicological studies have focused on the detrimental effects that chemical and physical pollutants can have on the performance of vertebrates. These studies have aimed to link these pollutants with the occurrence of morbid pathologies in humans (cardiovascular diseases, metabolic syndromes, cancer, neurodegenerative diseases, psychological disorders, e.g., Turner et al. 2017) and they have relied on large-scale epidemiological surveys or on toxicological laboratory experiments in animal models (e.g., Zou et al. 2009). Similarly, ecological and ecotoxicological studies have intended to determine the impact of these pollutants on wild vertebrates (Saaristo et al. 2018). Although experimental ecological studies are very rare for ethical and logistical reasons, correlative studies have demonstrated that some legacy or emerging chemical pollutants are associated with reduced survival or reproductive performance (e.g., Goutte et al. 2014, 2018; Sebastiano et al. 2020). Regarding physical pollutants, such as noise or light pollution, epidemiological studies and laboratory experiments have demonstrated that these pollutants can also be associated with pathologies in humans (sleep disorders, cardiovascular diseases, psychological disorders, retinal degeneration, Contín et al. 2016), and field studies on wild vertebrates have also demonstrated that noise and light pollution can alter the reproductive performance of animals and induce disorders that could be associated with reduced longevity (e.g., sleep disorders, oxidative stress; e.g., Ouyang et al. 2017; Dominoni and Nelson 2018). Beyond these strong effects of pollutants on the health of humans and wild vertebrates, pollutants can also

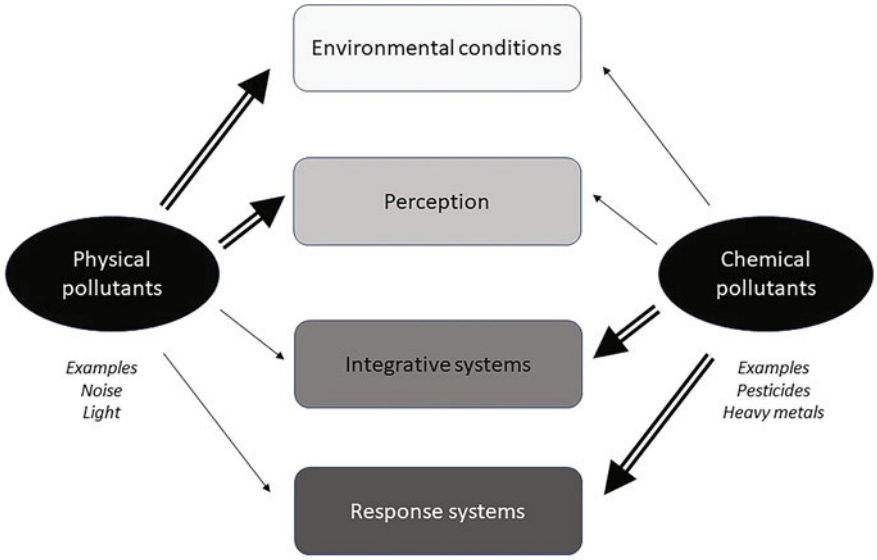

Fig. 9.1 Theoretical influence of chemical and physical pollutants on the functioning of vertebrates. Chemical pollutants are expected to have a strong impact on integrative and response systems through a direct disruption of key central organismal systems ("organismal disrupting pollutant"). Physical pollutants are expected to have a strong impact on environmental conditions and on the perception of the environment ("sensory pollutant," Halfwerk and Slabbekoorn 2015). Chemical pollutants could also affect the environment itself and could also affect sensory systems. Similarly, physical pollutants can also affect the organismal systems (integrative and response systems) through indirect effects (phenotypic plasticity and environmentally driven physiological and neurobiological changes)

have more subtle and pernicious effects. The rest of this chapter will focus on these sublethal effects and will specifically endeavor to evaluate how they can affect the ability of individuals to fulfill their seasonal and life cycles in a polluted environment.

Several modes of actions of pollutants may result in reduced performance in wild vertebrates. Firstly, pollutants may well of course affect the environment itself and result in detrimental environmental conditions. These conditions will subsequently constrain the individuals in terms of resources and they will therefore lead to poor performance during some or even all life-history stages (Fig. 9.1). For example, the release of systemic insecticides in agroecosystems may drastically reduce the quantity of insects in the environment (Cardoso et al. 2020), leading to reduced food abundance for all the insectivorous species, and therefore to poor reproductive and survival performance (Stanton et al. 2018). By drastically affecting environmental conditions, pollutants may also induce maladaptive responses, especially if the pollutants induce environmental conditions that are not within the range of environmental conditions the species has been selected for (Sih et al. 2011). For example, artificial light at night may totally disrupt circadian rhythms and lead to reproduction

impairment in some vertebrates, as recently demonstrated in the Australian budgerigar, *Melopsittacus undulatus* (Malek and Haim 2019). Secondly, pollutants may also induce more pernicious and subtle effects by triggering maladaptive responses to environmental conditions that are within the range of environmental conditions the species has been selected for (Fig. 9.1). Specifically, pollutants could affect the different steps that govern the individual response to environmental cues: (1) the perception of the environmental conditions; (2) the processing of the information by integrative systems; (3) the organismal response to the environmental cues. Classically, chemical pollutants are known to disrupt central organismal systems (Crisp et al. 1998). In the context of these subtle non-lethal effects, they are therefore likely to affect the processing of the information and the organismal response to the environment through their impact on integrative systems (brain and neurological effects, endocrine disruption). They can also affect the environment itself, notably if these chemical pollutants have an effect on other compartments of the ecosystem (e.g., food abundance). The impact of physical pollutants on wild vertebrates is more likely to be mediated through a direct modification of the environment (e.g., noise or light) and through an effect on the perception of the environmental conditions (indeed they are often called "sensory pollutants," Halfwerk and Slabbekoorn 2015; Dominoni et al. 2020a, b). Physical pollutants may also indirectly affect the functioning of the integrative and response systems through phenotypic adjustments, particularly if they occur during the ontogenetic phases of these systems.

9.1.4 Importance of the Developmental Period

In this context, specific attention should be paid to the developmental stage because the ontogeny of multiple integrative organismal systems is mainly determined during this stage (Minelli 2003). As explained earlier, these systems are crucial to process the perceived information and to proceed with a phenotypic response. During development, the plasticity of all these systems is set up and developmental conditions orientate these systems toward a specific function that can usually only be modified to a limited extent during the post-developmental period (de Graaf-Peters and Hadders-Algra 2006). For example, brain structures are mainly determined during the prenatal and postnatal developmental periods. Brain development is under control of gene expression during that period, but prenatal and postnatal environmental conditions also play a crucial role in brain development because they establish and refine neural organization in specific ways that aim to adjust the structure and the functioning of the brain to the conditions in which the organism will live (reviewed in Stiles and Jernigan 2010). Therefore, developmental conditions may have a strong incidence on the ability of individuals to cope with pollutants, or more generally with perturbations, later in life.

In addition, the impact of pollutants on organismal systems is often also exacerbated during the developmental stage. For example, brain development is very vulnerable to pollutants in early life compared to later stages of life (Grandjean

and Landrigan 2014). Indeed, previous experiments have shown that urban air pollution and fine particles result in altered brain development during the prenatal and postnatal periods and this was associated with cognitive issues, reduced neurogenesis and neuropathologies in mice (Allen et al. 2014; Sunyer and Dadvand 2019; Patten et al. 2020). Because of this sensitivity, strict regulations are set up for pregnant women and children in humans, especially regarding the exposure to chemical pollutants. This suggests that the impact of pollutants on the development of key integrative systems may then represent a lifetime burden for the organism with permanent detrimental effects.

9.2 The Relevance to Focus on Stress-Coping Mechanisms in the Context of Pollution

When focusing on the impact of pollutants on wildlife, it is undoubtedly crucial to focus on stress-coping mechanisms because (1) they are required to adjust to the environmental perturbations that may arise from physical pollutants (e.g., noise or light pollution); (2) they may be disrupted by chemical pollutants, resulting in a maladaptive response to environmental conditions and in pathologies. However, surprisingly, stress-coping behavioral and physiological mechanisms have been relatively overlooked in comparison with other organismal systems and endocrine axes.

9.2.1 Stress-Coping Mechanisms: From Behavior to Endocrine Mechanisms

When individuals encounter and/or perceive challenging environmental conditions, a suite of behavioral and physiological changes are activated to allow the organism to maintain a homeostatic state (i.e., the concept of allostasis, McEwen and Wingfield 2003; Romero et al. 2000). These behavioral and physiological responses to perturbations have often been used to define specific coping styles (proactive vs. reactive, Koolhaas et al. 1999). Firstly, this stress response involves the immediate behavioral fight or flight response, which is associated with the sympathetic branch of the autonomic nervous system and the release of catecholamines (Wingfield 2003). This response is typically associated with a rapid increase of heart and respiratory rates, and a vasodilatation of the vessels that supply oxygen to the organs necessary to cope with the stressor (e.g., muscle). It is also associated with the vasoconstriction of the vessels that supply oxygen to facultative functions (not necessary for immediate survival, e.g., digestive organs), and with a rapid conversion of glycogen to glucose to supply the brain and the muscles with energy (McCarty 2016a, b). This response mainly translates into contrasted coping

behavioral strategies (e.g., propensity to adopt an escape behavior). Secondly, this stress response also involves the Hypothalamus-Pituitary-Adrenal (HPA) axis and the regulation of circulating glucocorticoid levels (cortisol or corticosterone, Wingfield 2013). Increased circulating levels of these hormones will in turn modulate the functioning of several organismal systems, such as immunity, metabolism, and reproduction. It will therefore redirect resources from specific functions (e.g., reproduction) toward functions that are essential to immediate survival (Sapolsky et al. 2000). This physiological stress response is thought to prepare the organism to cope with the stressor in case it persists for an extended period of time (the preparative hypothesis, Romero 2002). Importantly, the physiological stress systems also involve the termination of the stress response that allows the organism to avoid the detrimental effect of chronic stress (e.g., Zimmer et al. 2019). Overall, the fight or flight response plays an important role in mobilizing immediate adaptive resources of the body, and the HPA stress response provides for more enduring adjustments to prolonged stress (Frankenhaeuser 1986). Another important way to cope with stress is the development of cognitive processes that are linked to learning and memory (the cognitive buffer hypothesis, Sol 2009). Firstly, learning and memory may help individuals to avoid stressful situations by adjusting their behavior or their physiology. Secondly, they may also help individuals to elicit a stress response that is well adjusted to the stressful situation, and which optimize its benefits (Ursin and Eriksen 2004).

9.2.2 Stress-Coping Mechanisms: The Target of Pollutants

In the context of a polluted world that can affect the life cycle and the seasonal routines of organisms, it is logical to specifically study these mechanisms (Jacobs and Wingfield 2000). They can be the target of the pollutants themselves: pollutants may alter the functioning of these stress-coping mechanisms, leading therefore to maladapted responses to specific environmental conditions (Wingfield and Mukai 2009). This is typically the case for chemical pollutants that disrupt the functioning of physiological systems (e.g., endocrine disruptor chemicals). Indeed, there is increasing evidence that many pollutants can have sublethal effects on vertebrates and can disrupt endocrine and neurological functions, including the HPA axis and the autonomic nervous system (Harvey 2016; Yaglova et al. 2017; Di Lorenzo et al. 2020). In addition, pollutants also seem to detrimentally affect cognition and the development of the brain, therefore impairing learning and memory in humans (Sunyer et al. 2015; Clifford et al. 2016) and wildlife (Jacquin et al. 2020) with potential consequences on their ability to cope with stressors (the cognitive buffer hypothesis, Sol 2009). All these disruptions may impact the stress response in its globality (fight or flight response, HPA axis, and cognition) with potential important fitness costs for wild vertebrates (reduced survival and reduced reproductive performance), and serious pathologies for humans.

9.2.3 *Stress-Coping Mechanisms: How to Adjust to a Polluted Habitat*

Stress-coping mechanisms will determine the response of the organism to the environmental changes that will be associated with the pollutants. As detailed earlier, pollutants may affect not only the functioning of the stress-coping mechanisms (see Sect. 9.1.3), but also the environment or the perception of the environment by individuals (Halfwerk and Slabbekoorn 2015; Dominoni et al. 2020a, b). Poor environmental conditions will activate the stress-coping mechanisms and will lead to phenotypic responses that will be associated with fitness costs and benefits (Wingfield et al. 1998; Wingfield 2013). This stress response aims to help the organism cope with the stressor and it should theoretically allow the organism to cope with the pollutant (Wingfield et al. 1998; Angelier and Wingfield 2013; Schoenle et al. 2018). Individuals may appropriately cope with their new polluted habitat by adopting fresh life-history strategies and by learning how to avoid or cope with the new environmental conditions. The pollutants may, however, induce environmental changes that are incompatible with the requirements of the individual even when the stress response is activated (Angelier and Wingfield 2013). In that scenario, the stress response may lead to the dispersal and the colonization of an alternative habitat (unpolluted). If such dispersal is not possible (there is no appropriate habitat within the dispersal range of the individual), the ineffective stress response will result in null fitness (so no reproduction before death, Fig. 9.2).

When the pollutant affects the perception of the environment, it may lead to inappropriate environmental cues (Halfwerk and Slabbekoorn 2015). In this case, there might be a mismatch between the stress response and the actual environmental conditions, thus the degree of activation of the stress-coping mechanisms will be inappropriate to cope with the actual environmental conditions (Fig. 9.2). As extensively reviewed by Dominoni et al. (2020a, b), these inappropriate environmental cues may result from masking effects of pollutants. In terms of stress response, this inappropriate perception of the environmental situation will translate into a heightened or dampened stress response relative to the optimal stress response. Because the stress response is associated with fitness costs and benefits, this mismatch will respectively increase and decrease these fitness costs and benefits and will jeopardize the persistence of the individual in this polluted habitat (Angelier and Wingfield 2013).

9.2.4 *Flexibility of Stress-Coping Mechanisms: Importance of the Developmental Period*

These stress-coping mechanisms and coping styles are often tightly linked to individual performance and fitness in wild vertebrates. They are indeed thought to mediate life-history trade-offs, and as a consequence, to govern the decisions that

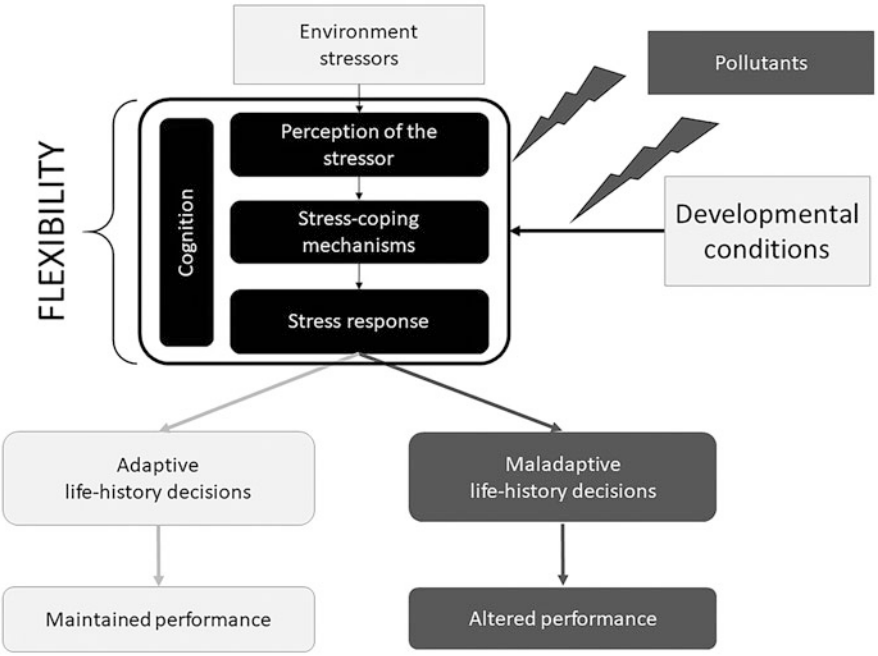

Fig. 9.2 Impact of pollutants on the ability of individuals to adjust the functioning of their stress-coping mechanisms to the environment. Pollutants may primarily disrupt the functioning of these mechanisms by altering their ontogeny during the developmental period, although they can also disrupt them during adulthood. This disruption may reduce the flexibility of these adaptive stress-coping mechanisms and/or may lead to maladaptive responses to environmental stressors with maladaptive life-history decisions, altered performance, and reduced fitness

individuals will adopt when facing specific environmental conditions (Angelier and Wingfield 2013; Taff and Vitousek 2016). In that respect, their study appears relevant to assess whether pollutants lead to maladaptive life-history strategies through their alteration. Importantly, most of these stress-coping mechanisms are heritable and at least partly repeatable, suggesting that they can be under selection and may play a key role in the ability of species to persist in polluted environments. Despite this heritability, these mechanisms are still flexible to some extent and it has been convincingly demonstrated that individuals are able to modulate their stress responses according to their individual state and the environmental conditions they encounter (Wingfield and Sapolsky 2003; Taff and Vitousek 2016). Importantly, this flexibility is certainly crucial in the context of a polluted world because the ability of individuals to adjust their stress response to a wide range of environmental conditions may allow them to complete their life cycle despite the pollution (Saaristo et al. 2018).

The flexibility of these mechanisms is certainly genetically determined and under selection (Hau et al. 2016), but importantly, there is also very strong evidence that prenatal and postnatal developmental conditions can have a strong impact on the

ontogeny of these systems and on their flexibility later in life (Taff and Vitousek 2016). During the developmental period, the ontogeny of multiple systems—including neurological pathways and endocrine axis—is indeed modulated by variations in hormonal levels (Groothuis and Schwabl 2008). For example, there is strong evidence that glucocorticoids are important mediators of ontogenetic transitions in vertebrates and that exposure to glucocorticoids during the developmental period can have important long-lasting effects on the phenotype and life-history strategies (Wada 2008; Marasco et al. 2012; Hau et al. 2016; Dupont et al. 2019). This means that pollutants, as endocrine disruptors, may affect the exposure of the embryo or the offspring to hormones with a potentially strong impact on the ontogeny of these systems, and possibly on their functioning and flexibility later in life (Fig. 9.2). This impact could be mediated by the occurrence of physical and chemical pollutants that alter daily and seasonal endocrine cycles, but also by chemical endocrine disruptors that may affect the degree of exposure of the developing organism to specific hormones (Fig. 9.2).

9.3 Influence of Chemical Pollutants on Stress-Coping Mechanisms: The Example of Lead (Pb)

The Anthropocene is associated with a large panel of anthropogenic activities and the related release of chemical pollutants in the environment. This paragraph aims to illustrate the potential disrupting effects of such pollutants on stress-coping mechanisms by focusing on the historical example of lead (Pb), a well-studied pollutant that is of primary concern for humans, wildlife, and the environment (Levin et al. 2021).

9.3.1 Developmental Impact of Exposure to Lead on Stress-Coping Mechanisms

A large number of studies have focused on the impact of lead exposure on the development of organismal functions (Fig. 9.3a), mainly because lead has been a major concern in child development for decades (Shefa and Héroux 2017). As a result, a great deal of data come from epidemiological studies on human populations. For example, studies of cohorts of children have convincingly shown that lead exposure during development is associated with cognitive disorders (e.g., Kim et al. 2013), increased cortisol secretion in response to stress (e.g., Gump et al. 2008), increased blood pressure (Zhang et al. 2012), and altered brain structure (Marshall et al. 2020).

In animal models, developmental exposure to lead has been associated with the development of cardiovascular issues, such as hypertension (Shvachiy et al. 2020;

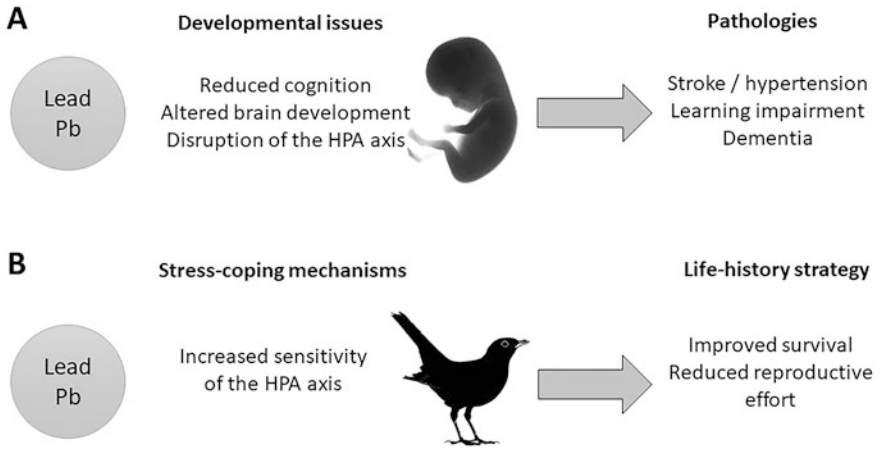

Fig. 9.3 Impact of lead on vertebrates. (**a**) Biomedical studies have demonstrated that developmental exposure to lead is associated with the alteration of the ontogeny of several stress-coping mechanisms, including brain development and the HPA endocrine axis. Such disruption is associated with the occurrence of multiple pathologies in children and adults. (**b**) Ecotoxicological studies have reported that lead contamination is associated with a disruption of the HPA axis and with an increased sensitivity of this endocrine axis to stressors. This modification has been related to a reduced reproductive investment, and as a result to an increased survival in some wild birds. This suggests that exposure to lead may alter life-history strategies in wild vertebrates

Chen et al. 2021). Little data is, however, available on the impact of lead exposure on the sympathetic branch of the autonomic nervous system and the release of catecholamines because most studies have focused on cardiovascular pathologies rather than the fight or flight stress response itself. However, in humans, exposure to lead results in increased heart rate and arterial pressure (Lai et al. 2002), and this could be mediated by a direct effect of lead on the secretion of catecholamines (Carmignani et al. 2000). In addition, developmental lead exposure was associated with a reduced escape behavior in zebrafish larvae, suggesting that lead may affect the fight or flight response (Rice et al. 2011).

Developmental exposure to lead has been reported to have strong effects on several endocrine mechanisms (Doumouchtsis et al. 2009), including the HPA axis. In animal models, exposure to lead results in an increased secretion of cortisol levels (HPA axis) in developing rats (Vyskočil et al. 1990). Importantly, exposure to lead can affect several components of the HPA axis. For example, lead seems to affect not only the secretion of glucocorticoids by the adrenals, but also the glucocorticoid receptors binding (Sobolewski et al. 2018), and the negative feedback system (Rossi-George et al. 2009). Exposure to lead during the developmental period has permanent effects on the HPA axis even when individuals are no longer exposed to lead after the developmental period (Cory-Slechta et al. 2004), and this can lead to psychosocial pathologies (Haider et al. 2013). More specifically, in rats, exposure to lead during the prenatal period results in a modified stress response later in life (Virgolini et al. 2008), suggesting that exposure to lead might disrupt this

stress-coping mechanism and potentially lead to maladaptive behavioral and physiological stress responses (Rossi-George et al. 2011).

Prenatal or postnatal exposure to lead has also been associated with a permanent reduction of memory (Shvachiy et al. 2020) and with learning and cognitive impairments in rats and mice (Rodrigues et al. 1996; Kuhlmann et al. 1997; Morgan et al. 2001). Experimental studies have shown that lead has a strong toxic effect on the development of the brain during the prenatal and the postnatal stages (Toscano and Guilarte 2005; Verstraeten et al. 2008), notably by inducing cell apoptosis in the brain and by resulting in a reduced number of cognition-related proteins (Hossain et al. 2016). Interestingly, similar effects have been found in animal fish and bird models and developmental exposure to lead has been experimentally shown to alter brain structures and to induce cognitive deficit and learning impairments (Xu et al. 2016; Goodchild et al. 2021).

Overall, these studies clearly demonstrate that developmental exposure to lead can affect a wide range of stress-coping mechanisms. Because these epidemiological and biomedical studies aim primarily to understand the pathologies that are linked to developmental exposure to lead, they do not necessarily allow us to assess how exposure to lead may disrupt the ability of individuals to cope with environmental daily and seasonal challenges (Fig. 9.3a). However, they provide crucial information to reliably test how lead functionally affects stress-coping mechanisms, and therefore to understand its potential impact on the ability of organisms to adapt to a changing world.

9.3.2 Wildlife, Exposure to Lead, and Fitness Consequences?

In the wild, several studies have reported that vertebrates can be exposed and contaminated by lead. Such contamination usually occurs because vertebrates exploit some habitats that are polluted by current or past human activities, such as industrial sites (e.g., Scheifler et al. 2006; Fritsch et al. 2012), urbanized areas (e.g., Bichet et al. 2013; Orłowski et al. 2014), or landfills (e.g., de la Casa-Resino et al. 2014). Most data come from urban ecotoxicological studies because cities are characterized by a global lead contamination of the environment, the animals and the humans (Levin et al. 2021). In bird species, lead seems to accumulate in the renal area, the liver, and the brain (Torimoto et al. 2021), suggesting that it may have important effects on cognition (brain) and glucocorticoid regulation by the adrenal glands. Because of the difficulty in studying cognition, the fight or flight response and the release of catecholamines in wild animals, no data is to our knowledge available regarding these stress-coping mechanisms in wild vertebrates (but see Grunst et al. 2020 for problem solving tasks). However, a few studies have examined the impact of lead contamination on the HPA axis of wild birds (Chatelain et al. 2018). In white storks, Baos et al. (2006) found that blood lead levels were positively correlated with stress-induced corticosterone levels (but not baseline corticosterone levels), suggesting that lead may increase the secretion of glucocorticoids in

response to stressors (Fig. 9.3b). Supporting this result further, Meillère et al. (2016) found that in blackbirds feather lead levels were positively correlated with feather corticosterone levels, a proxy of stress-induced corticosterone levels in birds (Bortolotti et al. 2008).

Although the impact of lead on survival has rarely been assessed in wild vertebrates, several studies have suggested that lead and other trace elements may impair reproductive performance (Janssens et al. 2003; Eeva et al. 2009, but see Eeva et al. 2014), even at a rather low level of contamination (Chatelain et al. 2021). Interestingly, two recent correlative studies support the idea that the disruption of stress-coping mechanisms by lead may result in drastic changes in life-history strategies (Guo et al. 2018; Fritsch et al. 2019; Fig. 9.3b). Further supporting this idea, another recent study of an urban songbird found that exposure to lead was clearly associated with a change in territoriality and aggressive behavior, two variables that are tightly linked to life-history strategy (McClelland et al. 2019). In blackbirds, blood lead levels were associated with increased longevity, but with reduced reproductive performance. Overall, lead contamination resulted in reduced lifetime reproductive success in that species (Fritsch et al. 2019). Due to the lack of experimental data, strong evidence is required to conclude that exposure to lead may induce maladaptive stress-coping strategies with detrimental fitness consequences. However, this correlational field data suggests that exposure to lead may lead to maladaptive stress-coping strategies and reduced fitness (Fritsch et al. 2019) through its disrupting effect on major stress endocrine axes (e.g., Baos et al. 2006; Meillère et al. 2016). Future studies should experimentally test whether exposure to lead affects life-history strategies and lifetime reproductive success through changes in the functioning of stress-coping mechanisms, such as the regulation of glucocorticoids (Eeva et al. 2006).

9.4 Influence of Physical Pollutants on Stress-Coping Mechanisms: The Example of Noise Pollution

In addition to the release of chemical pollutants in the environment, human activities are also associated with other types of pollutants, such as light and noise pollution. Here, the intention is to illustrate the impact of such pollutants on performance by focusing specifically on the example of noise pollution, a physical pollutant of global concern for human health and wildlife (Goines and Hagler 2007; Slabbekoorn 2019). We also aim to highlight the importance of stress-coping mechanisms in determining the ability of individuals to cope with such pollutants.

9.4.1 What Are the Effects of Noise Pollution on Performance?

In humans, there is evidence that noise pollution is associated with pathologies, often it seems related to chronic stress. For example, noise pollution has been associated with sleep disorders, more specifically reduced sleep duration and fragmented sleep (reviewed in Halperin 2014). In addition, noise pollution can increase blood pressure, lead to hypertension, and result in a higher risk of cardiovascular failure and stroke (reviewed in Stansfeld and Matheson 2003; Münzel and Daiber 2018). All these health concerns are intrinsically linked to stress regulation because of the clear interconnection between sleep, cardiovascular pathologies, and chronic stress. Indeed, noise pollution activates stress-coping mechanisms and is associated with the release of catecholamines (Borrell et al. 1980) and glucocorticoids (reviewed in Münzel et al. 2021). Animal studies have supported this idea that chronic stress could mediate the detrimental effect of noise pollution on health. They have shown that exposure to noise alters metabolism and impairs immunity and reproduction (Kight and Swaddle 2011), three systems that are functionally related to chronic stress (Dickens and Romero 2013).

Noise pollution is also linked to cognitive deficits in humans and laboratory animals. For example, noise pollution has been associated with learning impairments and reduced memory abilities (Stansfeld and Matheson 2003), and with the development of cognitive pathologies in old age (e.g., dementia, Paul et al. 2019). These effects seem to be at least partly mediated by a direct impact of noise on some brain structures (e.g., hippocampus) that could be linked to stress (Cheng et al. 2011). Indeed, cognition is functionally linked to stress (Lupien et al. 2007; Sandi 2013), and there is increasing evidence that cognitive impairments may be mediated by an effect of noise pollution on stress-coping mechanisms (Jafari et al. 2020). In wild animals, noise pollution has been shown to induce a state of chronic stress in multiple species with reduced body condition, and elevated circulating levels of glucocorticoids (e.g., Tennessen et al. 2014; Kleist et al. 2018; Zollinger et al. 2019; Mills et al. 2020). Importantly, experimental studies have demonstrated that noise pollution can impair memory and spatial learning (Osbrink et al. 2021), reproduction, and survival (Schroeder et al. 2012; Blickley et al. 2012; Kight and Swaddle 2011; Halfwerk and Slabbekoorn 2013; de Jong et al. 2020), further emphasizing the detrimental impact of noise pollution on performance.

Importantly, all these detrimental effects of noise pollution on health seem exacerbated during the developmental period (Gupta et al. 2018). For example, epidemiological studies have suggested that noise pollution impairs cognitive development with non-reversible effects in children (Stansfeld et al. 2005; Klatte et al. 2013). Experimental studies have also shown that exposure to noise during development affects neurogenesis and the ontogeny of spatial memory (Kim et al. 2006). In developing wild animals, noise pollution often translates in altered growth, high oxidative damages, and elevated levels of glucocorticoids through direct effects on the developing individuals (e.g., Meillère et al. 2015a; Raap et al. 2017; Injaian et al.

2018a, b), although these effects may also be partly mediated by indirect effects on parental behavior and parental foraging efficiency (e.g., Luo et al. 2015; Meillère et al. 2015b; Nedelec et al. 2017). Our current knowledge of the long-term consequences of developmental exposure to noise pollution remains limited because of the lack of experimental and correlational data (Stansfeld and Clark 2015). However, noise exposure seems to translate into developmental stress (e.g., elevated levels of glucocorticoids), and it is well-known that such stress has detrimental long-lasting consequences on multiple physiological and behavioral systems (Welberg and Seckl 2001; Cottrell 2009; Spencer 2017).

9.4.2 Importance of Stress-Coping Mechanisms to Adjust to Noise Pollution

Noise pollution is intrinsically linked to stress-coping mechanisms because sounds are used by vertebrates to assess their environment and any potential stressor. Indeed, the auditory system is functionally connected to stress-coping integrative systems, such as the autonomic nervous and the neuroendocrine systems, which govern the fight or flight response and the HPA system (Westman and Walters 1981). Because the activation of stress-coping mechanisms is not only associated with fitness benefits, but also fitness costs (Wingfield 2003, 2013), their degree of activation must be appropriate and adjusted to the environmental situation in order to optimize organismal fitness (Wingfield and Sapolsky 2003; Angelier and Wingfield 2013). In that sense, noise pollution can represent an important challenge because it can alter the direction or the intensity of the link that exists between the perceived environmental conditions and the threat that they actually represent (the concept of "sensory pollution," Halfwerk and Slabbekoorn 2015). For example, in most species, noise pollution may represent a stressful situation either because it produces sounds of high intensity that are perceived as stressors themselves (known as misleading effects, Dominoni et al. 2020a, b) or because it can create a background noise that reduces the ability of the individual to perceive or detect some potential threats (masking or distracting effects, Dominoni et al. 2020a, b). Overall, noise pollution is expected to trigger an activation of stress-coping mechanisms. If these mechanisms are associated with dispersal, the organism will be able to escape the noisy area and will resume its normal daily and seasonal routine. This strategy will, however, be associated with some costs because the organism will have left an environment that appeared, but was not, in fact, detrimental. If these mechanisms do not activate dispersal or if dispersal is not an option (either because the organism has a limited dispersal capacity or because noise pollution is general), their activation will not help to cope with the perceived stressful situation (i.e., noise pollution) and it will lead to a state of chronic stress. This state of chronic stress will in turn lead to poor performance and to pathologies, as described earlier.

To cope with such pollution, individuals need to adjust the functioning of these stress-coping mechanisms to the noisy environment. In other words, the degree of activation of these mechanisms needs to be modulated according to the costs and benefits they provide to the organism (Angelier and Wingfield 2013). In this context, the flexibility of these mechanisms—habituation and sensitization—is certainly a key variable to consider when focusing on the ability to adjust to stressors in general, and noise pollution in particular (Radley et al. 2015; Blumstein 2016). There is indeed evidence that such flexibility is present in animals. For example, repeated experimental exposure to noise was associated with a progressive reduction of the catecholamine and corticosterone stress response in adult rats (de Boer et al. 1988). Similarly, fish reduce their behavioral stress response when exposed to repeated noise stress (Neo et al. 2018). This flexibility may even predict the ability of species to cope with a noisy environment or not (Lowry et al. 2013; Møller 2013). For example, urban birds have been shown to habituate to human disturbance and noise, and as a result, to dampen their behavioral stress response in cities (reduced flight initiation distance, Blumstein 2013). However, flexibility also seems limited under some circumstances, and this may limit the ability of the organism to adjust to noise pollution. For example, recent studies have found that the activation of stress-coping mechanisms are not necessarily modulated according to repeated exposure to noise pollution in adult wild animals (behavioral and physiological stress responses, Injaian et al. 2018c; Mills et al. 2020).

The determinants of this flexibility are complex and linked to the survival optimization system (SOS), which involves integrative systems (central nervous and endocrine systems), and cognitive appraisal and learning systems (Mobbs et al. 2015). The plasticity of this SOS is certainly species-specific and genetically determined, but there is also evidence that it can vary between populations or individuals (Vincze et al. 2016; Grunst et al. 2021), and be affected by previous life experience (McCarty 2016a, b) and notably by early life. Cognition, learning abilities, brain structure, and the ontogeny of most integrative systems (endocrine and nervous systems) are indeed affected by developmental conditions, developmental stress (Welberg and Seckl 2001; McGowan and Matthews 2018), and more specifically noise pollution. For example, the ontogeny of the HPA axis is affected by exposure to noise pollution with a lower sensitivity of this axis to stress (i.e., reduced secretion of corticosterone in response to stress) in several (Kleist et al. 2018), but not all circumstances (Crino et al. 2013; Angelier et al. 2016). There is very little data to test if early-life exposure to noise pollution represents a constraint or if it orientates the SOS toward a functioning that will help the organism to cope with noise pollution later in life (Mariette et al. 2021). Current data suggest that developmental exposure to noise is associated with impaired learning and cognitive abilities (Osbrink et al. 2021), suggesting that early-life exposure to noise pollution may indeed reduce the flexibility of the SOS later in life. However, developmental exposure to noise pollution also seems to reduce the sensitivity of the HPA axis to stress (Kleist et al. 2018), and this may allow the organism to better cope with a stressful noisy environment later in life (e.g., Tennessen et al. 2018). Additional field

and experimental studies are definitely needed to better assess whether adaptive developmental programming occurs in the context of noise pollution.

9.5 Perspectives and Future Research Needs

9.5.1 Cumulative and Interactive Effects of Pollutants on Stress-Coping Mechanisms

The impact of these pollutants on human health and wildlife is now being increasingly studied and the current literature allows scientists and decision makers to assess, at least partly, the risk that each single pollutant represents to humans and biodiversity. Similarly, an increasing number of studies are investigating the impact of the characteristics of a given pollutant on the functioning of vertebrate organismal systems (e.g., the intensity of pollution). However, in the real world these pollutants often covary, and vertebrates are constantly exposed to a combination of them (Karlsson et al. 2021). In this context, there is an important gap in our understanding of the interactive and cumulative effects of these different types of pollutants on living organisms (Vermeulen et al. 2020), especially when referring to stress-coping mechanisms.

Firstly, it is important to understand if the co-occurrence of chemical pollutants and physical pollutants during the developmental period alters stress-coping mechanisms in a complex manner (cumulative, synergic or antagonist effects). Although data is currently lacking in this regard, a few studies suggest that such interactive effects may occur during the development. For example, Cory-Slechta and collaborators have reported that developmental exposure to lead and to developmental stress could indeed have interactive effects on multiple stress-coping mechanisms in rodents (i.e., catecholamines, HPA axis, Cory-Slechta et al. 2004, 2008; Rossi-George et al. 2009) with potential consequences in terms of performance and pathologies later in life (Virgolini et al. 2006).

Secondly, it is also crucial to understand if exposure to a specific pollutant alters the ability of the organism to cope with an additional pollutant. There is indeed evidence that chemical pollutants may disrupt stress-coping mechanisms, and therefore alter the ability of the organism to cope with stress. When the stress-coping mechanisms are not fully solicited, the deleterious impact of such chemical pollutants on fitness might remain limited, even if significant (Fig. 9.4). However, other additional pollutants may alter environmental conditions and their perception by the organism (e.g., physical pollutants). To cope with these pollutants, stress-coping mechanisms are crucial, and their disruption by a chemical pollutant may lead to ineffective and maladaptive responses. In that scenario, the occurrence of a second pollutant may exacerbate the detrimental impact of the first pollutant on health and performance (Fig. 9.4). To the best of our knowledge, there is no data to assess the pertinence of this hypothesis. A few studies have examined the impact of the

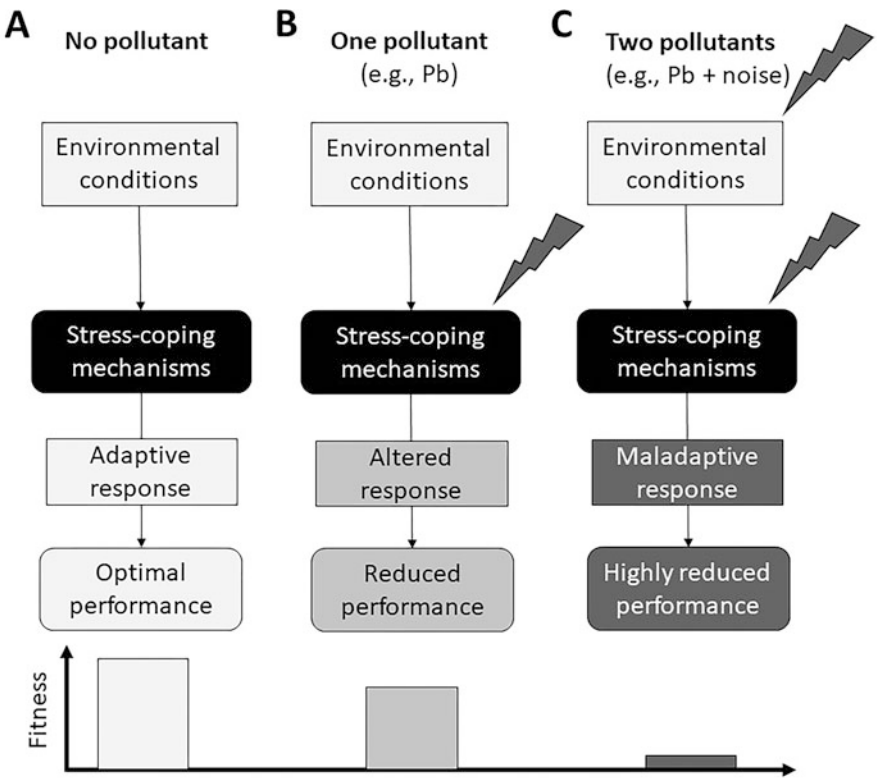

Fig. 9.4 Potential interactive effects of pollutants on stress-coping mechanisms and performance. In this diagram, three scenarios are described and they represent either an absence of pollutant (**a**), the occurrence of one pollutant (e.g., Pb) that disrupts stress-coping mechanisms (**b**), the occurrence of two pollutants (e.g., Pb and noise) that, respectively, disrupt stress-coping mechanisms and induce a stressful environmental situation (**c**). While stress-coping mechanisms are effective to optimize performance when pollutants are absent (**a**), a chemical pollutant (e.g., Pb) could disrupt stress-coping mechanisms (e.g., the HPA axis) and lead to a non-optimal stress response, and therefore to a reduced fitness (**b**). The co-occurrence of a chemical (Pb) and a physical (noise) pollutant could have dramatic effect on fitness, if lead alters the ability of the organismal stress response to cope with noise (**c**)

combination of two pollutants on human health or the performance of wild animals (e.g., Ferraro et al. 2020; Dominoni et al. 2020b), but none of these studies have focused on the importance of stress-coping mechanisms *per se*. For example, lead pollution and noise pollution occur simultaneously in the urban environment, but their potential interactive or cumulative effects on stress-coping mechanisms, human health, and wildlife have to the best of our knowledge never been investigated. We have previously described the importance of cognition and learning in determining the ability of individuals to adjust to noise pollution through habituation and through a better assessment of the costs that noise pollution may entail. However, we have also described the cognitive impairment that results from lead contamination during

the developmental period. Therefore, taken together, these results suggest that exposure to lead may alter the ability of urban individuals to cope with noise pollution through its impact on cognitive processes. Similarly, there is good evidence that exposure to lead can alter the integrative systems that govern the response to stress (fight or flight response and HPA axis), and more particularly it can result in an increased activation of these mechanisms in response to stress. However, a reduced sensitivity to stress may be required to cope with noise pollution to avoid the fitness costs of an overstimulation of stress-coping mechanisms in a context of stressful exposure to noise. Therefore, the impact of lead on these mechanisms may reduce the ability of individuals to adjust to noise pollution. Overall, this suggests that lead pollution could theoretically exacerbate the detrimental impact of noise pollution on health and performance.

Future experimental studies should now explicitly test this hypothesis of cumulative and interactive effects of pollutants on stress-coping mechanisms and fitness. They should specifically examine how the combination of multiple and various pollutants (physical and chemical pollutants) may interact and affect stress-coping mechanisms, individual performance, and health in humans and wild vertebrates.

9.5.2 Hormesis: An Overlooked Mechanism in Wild Vertebrates

This chapter has emphasized the importance of the developmental period to understand not only the impact of pollutants on performance, but also the ability of the organism to adjust to pollutants later in life. Most studies have reported that exposure to chemical pollutants is associated with health issues and reduced fitness, mainly because of the toxicological effects of the pollutants on organismal systems. As a result, the general agreement is that exposure to chemical pollutants will lead to pathologies, to disrupted stress-coping mechanisms, and to a lower ability to cope with pollutants later in life. However, exposure to chemical pollutants may counterintuitively improve the response of the organism to pollutants or stressors later in life by modifying the functioning of organismal systems (see Calabrese 2005 in the context of toxicology; see also Chap. 2). A few studies have shown that such dose-dependent responses to inorganic contaminants can occur in vertebrates. For example, Heinz et al. (2010) showed that prenatal exposure to low doses of methylmercury was associated with benefits in terms of hatching success in a bird species. Similarly, exposure to low levels of lead was associated with increased red blood cells production, while this effect was reversed at high concentrations (i.e., reduced RBC production, Iavicoli et al. 2003). Recently, it has been shown that exposure to a low dose of a mixture of chemical pollutants improved performance in terms of neurobehavioral tests in the rat (Tsatsakis et al. 2019).

In that context, specific attention should be given to the hormesis concept because it is thought to enhance phenotypic plasticity (Costantini et al. 2010), especially during the ontogeny of organismal systems. Indeed, priming hormesis suggests that developmental exposure to a mild stressor could improve the ability of the organism to cope with subsequent exposure to higher levels of that stressor (Costantini 2014a). Because central stress-coping mechanisms govern the response to multiple stressors, priming hormesis may even improve the ability of the individual to cope with other types of stressors. Although there is some evidence that exposure to mild challenging conditions early in life may be associated with better fitness (reviewed in Costantini et al. 2010), little data is available to test whether this effect is mediated by ontogenetic modifications of the functioning of stress-coping mechanisms in vertebrates. Priming hormesis has been extensively studied in the context of immunity and resistance to oxidative stress (Costantini 2014b), but much less in the context of the ontogeny of stress-coping mechanisms (i.e., the HPA axis, the fight or flight stress response). However, glucocorticoids are, for example, potential mediators of priming hormetic effects (Li et al. 2019) because (1) environmental conditions are known to influence the exposure of the developing organism to glucocorticoids and (2) developmental exposure to glucocorticoids often leads to dose-dependent effects on stress-coping mechanisms (Schoech et al. 2011; Crino and Breuner 2015; Eyck et al. 2019). For example, chronic exposure to glucocorticoids has been linked with cognitive impairment, while cognition may be in contrast improved in response to a temporary surge of circulating levels of glucocorticoids (de Kloet et al. 1999; Lupien et al. 2005). Future studies now need to examine whether contaminants induce dose-dependent effects on stress-coping mechanisms in vertebrates, and whether these effects are adaptive.

9.5.3 Microevolution

Finally, the ability of vertebrate populations to cope with a polluted world is not only determined by phenotypic plasticity, but also by evolutionary processes (Swaddle et al. 2015). Selection can drive the fate of vertebrate populations exposed to pollutants by favoring specific stress-coping strategies that are beneficial in response to pollutants. Support for such selective process comes from invertebrates with short generation times, such as mosquitoes that have become resistant to insecticides (Hemingway et al. 2002). In vertebrates, longer generation times may constrain the ability of most vertebrate species to adapt to pollutants, especially when these pollutants cannot be apparated to naturally occurring stressors or chemicals (Hawkins et al. 2019).

In some circumstances, microevolution may lead to resistance to pollutants in vertebrates. For example, resistance to pesticides has recently evolved in sea lampreys, an invasive fish (Christie et al. 2019). Recently, it has also been suggested that artificial light at night and noise pollution may represent a strong evolutionary driver to adapt to urbanization (Swaddle et al. 2015; Miranda 2017; Hopkins et al. 2018).

The importance of stress-coping mechanisms has rarely been highlighted in this context of adaptation to pollutants. However, and importantly, stress-coping mechanisms often show large inter-specific and inter-individual variabilities (e.g., Cockrem 2007; Moller 2010; Tablado et al. 2021), and as a result, they are also the target of microevolution processes (Guindre-Parker 2018). Indeed, comparative studies have shown that urbanization may select specific coping styles in vertebrates (Sadoul et al. 2021; Tablado et al. 2021; but see Iglesias-Carrasco et al. 2020). As explained earlier in this chapter, stress-coping mechanisms can be the target of pollutants. They also mediate the response of the organism to pollution, especially when pollutants can be apparated to stressors (Sect. 9.2). Therefore, stress-coping mechanisms may be key determinants of selective processes and may determine the ability of species to evolve resistance and adaptation to pollutants. Importantly, microevolution can probably select specific phenotypes in terms of stress-coping mechanisms but it can probably also act on developmental plasticity. In other words, selection may favor individuals that are able to mitigate the detrimental impact of pollutants on their development, either by resisting or by avoiding the pollutant, or by selecting developmental strategies that allow individuals to cope better with the pollutants later in life.

To the best of our knowledge, no study has, however, examined whether adaptation to pollutants is mediated by selective processes acting on these stress-coping mechanisms. Therefore, we believe that studying how stress-coping mechanisms evolve in response to pollutants (chemical and physical) represents a promising avenue of research to understand how some species (and not others) may adapt to our polluted world.

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