Reproduction in a polluted world: implications for wildlife

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Abstract

Environmental pollution is an increasing problem for wildlife globally. Animals are confronted with many different forms of pollution, including chemicals, light, noise, and heat, and these can disrupt critical biological processes such as reproduction. Impacts on reproductive processes can dramatically reduce the number and quality of offspring produced by exposed individuals, and this can have further repercussions on the ecology and evolution of affected populations. Here, we illustrate how environmental pollutants can affect various components of reproduction in wildlife, including direct impacts on reproductive physiology and development, consequences for gamete quality and function, as well as effects on sexual communication, sexual selection, and parental care. We follow with a discussion of the broader ecological and evolutionary consequences of these effects on reproduction and suggest future directions that may enable us to better understand and address the effects of environmental pollution.

Reproduction (2020) 160 R13–R23

Introduction

Environments around the globe are changing at an unprecedented pace and scale as a result of pollution from human activities. In this regard, wildlife are confronted with anthropogenic pollution in a wide variety of forms. This includes chemical contaminants that enter the environment from the manufacture, usage, and disposal of myriad products, such as pesticides, pharmaceuticals, and heavy metals (Dixit et al. 2015, Bernhardt et al. 2017). Other pollution sources include widespread anthropogenic light sources that illuminate the night (Longcore & Rich 2004), noise that is propagated throughout environmental landscapes from traffic, machinery, and industrial activities (Barber et al. 2011), and heat pollution, both local and global, that alters thermal niches within ecosystems (Hansen et al. 2006, Yow 2007). These diverse forms of pollution can all have detrimental effects on wildlife and the ecological communities they inhabit.

A particularly concerning consequence of environmental pollution is the disruption of reproductive processes. Pollutants can interfere with a broad range of traits necessary for reproductive success, such as reproductive physiology, gamete function, and organismal behaviour (Table 1). Alteration of these processes can reduce mating success, either directly by decreasing fertilisations or indirectly by disrupting mate attraction and/or encounter rates. These effects can dramatically alter the number and quality of offspring that individuals can contribute to successive generations, a concept commonly referred to as the ‘fitness’ of an individual (Orr 2009). These changes in fitness can then have harmful repercussions at the population and community levels, as well as influence the evolutionary trajectories of affected populations. Despite being essential for understanding how species will be affected by human-induced environmental change, the ecological and evolutionary repercussions of pollutants are rarely considered (but see Saaristo et al. 2018). Additionally, most studies focus on the direct effects of a single pollutant on a single species, limiting our understanding of how biotic and abiotic interactions influence the broader impacts of pollution.

In this review, we illustrate the ways in which reproduction can be impacted by environmental pollution and discuss the ecological and evolutionary consequences of these effects. We begin by outlining the direct impacts of environmental pollution on reproductive physiology and development, such as pollution-induced changes in sex hormones/pheromones, sex differentiation, and reproductive timing. We then consider how pollution can affect gamete production and function, including sperm motility and egg viability,
before going on to examine impacts of pollution on reproductive processes such as sexual communication and mate choice. This is followed by a discussion of the ecological and evolutionary repercussions of these effects on reproduction. We conclude by highlighting future directions and steps necessary to understand and address the realised effects of environmental pollution on reproduction.

### Direct effects of pollution on reproductive physiology and development

Environmental pollution can fundamentally affect reproduction by altering the production or signalling efficacy of sex hormones. A variety of chemical pollutants, for instance, can interact with steroid hormone receptors directly or indirectly, altering levels of blood sex steroids

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<thead>
<tr>
<th>Effect</th>
<th>Pollutant</th>
<th>Source of pollutant</th>
<th>Animal</th>
<th>Paper</th>
</tr>
</thead>
<tbody>
<tr>
<td>Feminisation of males</td>
<td>Ethinylestradiol</td>
<td>Contraceptive pill</td>
<td>Fish (Rutilus rutilus, Pimephales promelas)</td>
<td>Jobling et al. (1998), Kidd et al. (2007), Lange et al. (2009)</td>
</tr>
<tr>
<td></td>
<td>Atrazine</td>
<td>Herbicide</td>
<td>Amphibians (Xenopus laevis, Rana pipiens)</td>
<td>Hayes et al. (2002a,b, 2003, 2006, 2010)</td>
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<td></td>
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<td>Mice (Mus musculus)</td>
<td>Cook et al. (2019), Govers et al. (2019)</td>
</tr>
<tr>
<td>Disruption of gonad development</td>
<td>Neonicotinoids</td>
<td>Insecticide</td>
<td>Bumblebees (Bombus terrestris, B. lucorum, B. pratorum, B. pascuorum)</td>
<td>Baron et al. (2017)</td>
</tr>
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<td></td>
<td>Heat</td>
<td>Global warming, heat islands, factory discharge, and so on</td>
<td>Fish (Rutilus rutilus)</td>
<td>Lukiene and Sandström (1994)</td>
</tr>
<tr>
<td>Biased sex ratios</td>
<td>Heat</td>
<td>Global warming, heat islands, factory discharge, and so on</td>
<td>Sea turtles (various species)</td>
<td>Fuentes et al. (2009)</td>
</tr>
<tr>
<td></td>
<td>Clotrimazole</td>
<td>Antifungal</td>
<td>Zebrafish (Danio rerio)</td>
<td>Brown et al. (2015)</td>
</tr>
<tr>
<td></td>
<td>Dieldrin</td>
<td>Insecticide</td>
<td>Daphnia galeata</td>
<td>Dodson et al. (1999a)</td>
</tr>
<tr>
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<td>Atrazine</td>
<td>Herbicide</td>
<td>Daphnia plicaria</td>
<td>Dodson et al. (1999b)</td>
</tr>
<tr>
<td>Altered reproductive timing</td>
<td>Light</td>
<td>Artificial lighting from streetlights, building exteriors, advertising, and so on</td>
<td>Blackbirds (Turdus merula)</td>
<td>Dominoni et al. (2013a,b)</td>
</tr>
<tr>
<td></td>
<td>Heat</td>
<td>Global warming, heat islands, factory discharge, and so on</td>
<td>Tammar wallabies (Macropus eugenii)</td>
<td>Robert et al. (2015)</td>
</tr>
<tr>
<td>Reduced sperm motility</td>
<td>Bisphenol A (BPA)</td>
<td>Plastic production</td>
<td>Brown trout (Salmo trutta)</td>
<td>Lahnsteiner et al. (2005)</td>
</tr>
<tr>
<td></td>
<td>Air particulates</td>
<td>Smoke, fumes, and so on</td>
<td>Humans</td>
<td>Deng et al. (2016), Jurewicz et al. (2018)</td>
</tr>
<tr>
<td>Decreased fertilisation success of eggs</td>
<td>Titanium dioxide</td>
<td>Personal care products</td>
<td>Bivalves (Tegillarca granosa)</td>
<td>Han et al. (2019)</td>
</tr>
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<td></td>
<td>Heavy metals</td>
<td>Mining, products such as fertilisers, batteries, and so on</td>
<td>Fish (various species)</td>
<td>Jezierska et al. (2009)</td>
</tr>
<tr>
<td>Inhibited pheromone production</td>
<td>Diclofop-methyl</td>
<td>Herbicide</td>
<td>Cotton bollworm moth (Helicoverpa armigera)</td>
<td>Eliyahu et al. (2003)</td>
</tr>
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<td></td>
<td>Light</td>
<td>Artificial lighting from streetlights, building exteriors, advertising, and so on</td>
<td>Cabbage moths (Mamestra brassicae)</td>
<td>van Geffen et al. (2015)</td>
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<td></td>
<td>Endosulphan</td>
<td>Insecticide</td>
<td>Red-spotted newts (Notophthalmus viridescens)</td>
<td>Park et al. (2001)</td>
</tr>
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<td>Masked mating signals</td>
<td>Noise</td>
<td>Traffic, industrial activities, and so on</td>
<td>Fish, amphibians, birds and mammals (various species)</td>
<td>Shannon et al. (2016)</td>
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<tr>
<td></td>
<td>Light</td>
<td>Artificial lighting from streetlights, building exteriors, advertising, and so on</td>
<td>Glow-worms (Lampyris noctiluca)</td>
<td>Bird and Parker (2014)</td>
</tr>
<tr>
<td>Disrupted mate choice</td>
<td>Trebunolone</td>
<td>Agricultural growth-promotant</td>
<td>Guppies (Poecilia reticulata)</td>
<td>Tomkins et al. (2018)</td>
</tr>
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<td></td>
<td>Ethinylestradiol</td>
<td>Contraceptive pill</td>
<td>European Starlings (Sturis vulgaris)</td>
<td>Markman et al. (2008)</td>
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<td>Reduced parental care</td>
<td>Noise, light</td>
<td>Traffic, industrial activities, artificial lighting from streetlights, building exteriors, advertising, and so on</td>
<td>Seabirds (Calonectris diomedea)</td>
<td>Cianchetti-Benedetti et al. (2018)</td>
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**Table 1** Summary table of examples featured in this review to illustrate the different effects environmental pollution can have on reproductive function across diverse taxa.
and disrupting sexual development. Such chemicals are collectively referred to as endocrine-disrupting chemicals (EDCs). For example, ethinylestradiol, an oestrogen commonly used in contraceptive pills, can enter the environment via urine (Johnson & Williams 2004) and has been shown to induce physiological feminisation in wild fish through its interaction with oestrogen receptors (Jobling et al. 1998, Lange et al. 2009). Various other chemical pollutants that mimic endogenous steroids are also known to interact directly with oestrogen receptors (e.g. bisphenol A: Gould et al. 1998; phthalates: Jobling et al. 1995) and androgen receptors (e.g. the livestock growth promoter trenbolone: Sone et al. 2005; various prostate chemotherapeutic agents: Mateo et al. 2014), with downstream consequences for reproductive physiology. Pollutants can also change sex hormone levels via stress-related mechanisms or through effects on other hormonal pathways (Brüning et al. 2016). Indeed, there is evidence that light pollution can act as a chronic stressor, inhibiting the production of sex hormones in birds via the hypothalamic–pituitary–adrenal axis (Russ et al. 2015).

Such changes in sex hormone levels can have repercussions for the development of reproductive anatomy and morphology in organisms. For example, in Lake Apopka, Florida, exposure of American alligators (Alligator mississippiensis) to a mixture of chemicals – including a spilt of dicofol and DDT, agricultural runoff such as pesticides, and sewage treatment outflow – resulted in females exhibiting abnormal ovarian morphology and unusually prominent poliovular follicles, while males developed poorly organised testes and small phalli (Guillette et al. 1994, 1995, 1996). Further, developmental exposure of amphibians to the widespread herbicide atrazine has been shown to induce reproductive malformations due to feminisation of males. This includes the anomalous development of multiple gonads (Hayes et al. 2002a), altered testicular anatomy (Tavera-Mendoza et al. 2002, Hayes et al. 2003), development of ovoestes and testicular oocytes (Hayes et al. 2006, 2010, Murphy et al. 2006) and hermaphroditism (Hayes et al. 2002a,b). Atrazine can also lead to feminisation in mice, resulting in penis abnormalities (Govers et al. 2019) and altered sperm production (Cook et al. 2019). The direct impacts of chemical pollution on reproductive anatomy and morphology have been reported across a diverse array of taxa (e.g. feminisation of fish populations exposed to oestrogenic chemicals: Jobling et al. 1998, 2006; masculinisation of gastropods exposed to anti-fouling paint: Ten Hallers-Tjabbes et al. 1994, Matthiessen & Gibbs 1998; reduced ovary development in neonicotinoid-exposed wild bumblebee queens: Baron et al. 2017), providing evidence that chemical pollution could be a major driver in the reported increases in abnormalities seen in sexual development in humans (discussed in Giwercman et al. 1993, Skakkebæk et al. 2001, Damgaard et al. 2002).

Direct effects of pollution on wildlife reproductive anatomy and morphology are not limited to impacts of chemical pollution. Indeed, thermal pollution from nuclear power plants has been associated with altered gonad growth and asynchronous gonad and oocyte development in fish populations (Luksiene & Sandström 1994). Further, thermal pollution exposure has been shown to result in a greater variation in gonad development, both within and among fish populations (Efimova 1977, Virbickas et al. 1981, Lapina 1991, Luksiene & Sandström 1994). While few studies have investigated the impacts of noise pollution on wildlife reproductive morphology, long-term (60 days) exposure to noise pollution has been shown to cause a decrease in the diameter of seminiferous tubules and the thickness of the germinal epithelium in adult male rats (Farzadinia et al. 2016).

In addition to altering sexual morphology, pollution can disrupt cueing systems for sex determination and/or sexual differentiation, resulting in biased sex ratios. This is best illustrated in species that show temperature-dependent sex determination, including various fish, amphibian, and reptile species (Crews et al. 1994, Baroiller & D’Cotta 2001, Eggert 2004). Shifts in environmental temperatures are occurring globally through climate change (Hansen et al. 2006), as well as more locally through processes such as warm water discharge into rivers and estuaries from power plants (Raptis et al. 2016). An example of the former is seen in sea turtles, where higher nest temperatures are shown to skew sex ratios towards females (Fuentes et al. 2009), and this imbalance is predicted to become more extreme as temperatures increase due to global warming. Additionally, changes in environmental temperature can exacerbate the reproductive effects of certain chemical pollutants that affect sexual differentiation. For example, the antifungal chemical clotrimazole has been shown to skew sex ratios in zebrafish (Danio rerio) towards males by inhibiting production of aromatase, an enzyme that converts testosterone to oestrogen, and this effect is enhanced at higher temperatures (Brown et al. 2015). These imbalances in sex ratios potentially reduce reproductive success by lowering encounter rates between males and females. In facultative sexual species, altered sex ratios may change the rate of sexual reproduction vs asexual reproduction. This has been seen in various Daphnia species, where the production of males can be affected by a variety of pollutants (dieldrin in Daphnia galeata: Dodson et al. 1999a; atrazine in Daphnia pulicaria: Dodson et al. 1999b; dicofol and vinclozolin in Daphnia magna: Haeba et al. 2008), altering the rate of sexual reproduction.

Exposure to environmental pollution can also alter reproductive timing in wildlife. For example, ambient

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Reproduction (2020) 160 R13–R23
light levels, which are used as a cue for the timing of reproductive processes in many animals, can be disrupted by light pollution. A powerful example of this is seen in common blackbirds (Turdus merula), where, after 1 year of exposure to artificial light at night, seasonal development of the birds’ gonads occurred up to 1 month earlier and, after the second year of exposure, gonads did not show seasonal development at all (Dominoni et al. 2013a,b). Further, light pollution has been shown to delay births in tammar wallabies (Macropus eugenii), which is potentially mediated by suppression of melatonin (Robert et al. 2015). Ambient temperature is also used as a cue for biological events in many species, and similar to effects of artificial light pollution on reproductive timing, increased temperatures due to the effect of urban heat islands – or, more broadly, global warming – can result in temporal shifts in reproduction (Visser et al. 2009). Altered reproductive timing can have serious repercussions for offspring production or survival, as the shift may de-synchronise offspring production with peak resource availability. For example, it is predicted that, due to increasing temperatures, forest bird offspring will become increasingly mismatched with peak caterpillar availability (Burgess et al. 2018).

Consequences for gametes
The previously described effects of pollutants on hormones and sexual development can have consequences on gamete function and, hence, fertility. For example, when male fish are feminised via exposure to oestrogenic pollutants, this can result in reduced sperm quality (Jobling et al. 2002a,b). Further, female brown trout (Salmo trutta) exposed to bisphenol A (BPA) prior to, and during, spawning exhibited lower levels and later onset of ovulation, while exposed males showed reductions in sperm density, motility rate, and sperm swimming velocity (Lahnsteiner et al. 2005). These effects were likely due to the oestrogenic activity of BPA. Numerous studies have also shown that human males exposed to particulate air pollution produce sperm that exhibit impaired motility (De Rosa et al. 2003, Hammoud et al. 2010, Huang et al. 2019, see also Deng et al. 2016 for recent meta-analysis and review by Jurewicz et al. 2018) and impaired DNA structure and integrity (Sram et al. 1996, Selevan et al. 2000, Rubes et al. 2005), although the mechanisms for these effects are poorly understood. Similarly, in wildlife such as birds, radiation pollution has been shown to have a range of negative effects on sperm that include increased mutation rates, as well as reduced sperm production, quality, and motility (Møller et al. 2005, 2008, 2014).

In externally fertilising species, pollutants can induce detrimental effects through direct contact with sperm and/or eggs. For instance, in capelin fish (Mallotus villosus), exposure to chemical-dispersing agents can reduce the fertilising capability of sperm (Beirão et al. 2018), while harmful impacts of contaminants on sperm swimming parameters have been reported in both fish (e.g. copper ions: Kowalska-Góralska et al. 2019; heavy metals: Abascaling et al. 2007; EDCs: reviewed by Carnevali et al. 2018) and invertebrates (e.g. xenobiotics: Gallo 2018; nanoparticles: Hollows et al. 2007, Han et al. 2019). Pollutants can also have damaging effects through direct contact with eggs. For example, Han et al. (2019) found that in broadcast-spawning bivalves (Tegillarca granosa), the nanoscale pollutant titanium dioxide can attach to the oocyte surface and damage the plasma membrane, which is thought to contribute to reduced gamete fusion success. Similarly, in fish, exposure of spawned eggs to certain polluting metal compounds can adversely impact fertilisation capacity and general viability (Jezierska et al. 2009).

A particularly concerning aspect of certain environmental pollutants is their ability to induce long-term impacts that may persist across many generations following the initial exposure event. An increasing body of evidence has reported associations between adult exposure to environmental pollutants and reductions in offspring health and fitness, almost certainly via epigenetic mechanisms (Soubry et al. 2014). Much of this evidence comes from studies reporting changes in sperm chromatin structure, changes in sperm small ncRNA (sncRNA) content, and altered DNA methylation patterns in sperm (Evans et al. 2019). Such epigenetic changes in sperm, for example, can occur following exposure to various pollutants, including cigarette smoke (Jenkins et al. 2017), ethanol (Rompala et al. 2018) and the fungicide vinclozolin (Ben Maamar et al. 2018). Fortunately, these effects appear to be at least partially reversibly via exercise and environmental enrichment, depending on the toxicant, effect and specific process affected (Short et al. 2017). Studies have also implicated paternal chronic exposure to EDCs with changes in sperm sncRNAs. For example, in zebrafish, paternal exposure to the synthetic oestrogen 17α-ethinylestradiol (EE2) results in a range of disorders in offspring (e.g. skeletal and cartilage deformations, poor locomotion, etc.), mostly likely due to an up-regulation of microRNA transcripts in the testes and sperm (Valcarce et al. 2017). Such studies showing transgenerational effects suggest that environmental pollutants can impact population health long after the initial exposure and even after contaminants have been removed, which may cause us to underestimate the full scope of the impacts of such exposures.

Effects of pollution on sexual communication, sexual selection, and parental care
Pollutants can impact the ability of animals to locate potential mates by disrupting the production, transmission, and detection of signals important in sexual
An example of this is pollution disrupting the production of pheromones – some of which are steroid hormones, such as glucuronides (Scott & Vermeirssen 1994). For instance, in the cotton bollworm moth (Helicoverpa armigera), exposure to the herbicide diclofop-methyl has been found to inhibit sex pheromone production by reducing fatty acid synthesis (Eliyahu et al. 2003). Similarly, light pollution can disrupt pheromone production, with low-intensity artificial light at night reducing sex pheromone production in female cabbage moths (Mamestra brassicae: van Geffen et al. 2015). As these pheromones are used to indicate reproductive receptivity, inhibited pheromone production will likely reduce mate acquisition and, thus, mating success. Indeed, this has been reported in female red-spotted newts (Notophthalmus viridescens), where exposure to the insecticide endosulfan resulted in the suppression of pheromone production and subsequently reduced mating success (Park et al. 2001).

As well as disrupting pheromone production, environmental pollution can interfere with acoustic or visual signals used for sexual communication. For example, exposure to polychlorinated biphenyls (PCBs) can reduce androgen-dependent growth in cartilage and muscle of the larynx in male African clawed frogs (Xenopus laevis), preventing them from making advertisement calls to females (Qin et al. 2007). Similarly, in species as taxonomically diverse as fish, amphibians, birds, and mammals, anthropogenic noise pollution can mask acoustic signals used by individuals to attract their mates (reviewed in Shannon et al. 2016) or prevent the perception of these mating signals by damaging auditory organs (Jepson et al. 2003, André et al. 2011). Likewise, artificial light pollution is thought to be the primary driver for population declines in glow-worms (Lampyris noctiluca), as light pollution can drastically reduce the ability of males to detect the bioluminescent light signals of reproducitively active females (Bird & Parker 2014).

Environmental pollution can also compromise the ability of animals to select a suitable mate (reviewed in Candolin & Wong 2019). For example, in a freshwater fish, the guppy (Poecilia reticulata), exposure to an agricultural steroid pollutant (17β-trenbolone) disrupted patterns of female preference for male colour traits that are important in signalling male genetic quality to choosy females (Tomkins et al. 2018). Exposure to pollution can also affect the relationship between the expression of sexual traits and the fitness benefits associated with those traits. In European starlings (Sturnus vulgaris), for instance, males exposed to a mixture of synthetic oestrogenic endocrine disruptors showed a reduction in immune function, but developed songs that were longer and more complex and, as a result, were actually preferred by females (Markman et al. 2008). At its most extreme, the disruption of mate selection can even lead to the breakdown of premating reproductive isolation and the loss of biodiversity. For example, in two co-occurring species of swordtail fishes (Xiphophorus birchmanni and Xiphophorus malinche), exposure to sewage effluent and agricultural runoff resulted in the loss of female preference for the odour cues of male conspecifics, leading to females mating indiscriminately with males of both species (Fisher et al. 2006).

After mating, environmental pollution can impact the quality of parental care provided to young, with consequences for the health and survival of offspring (Wong et al. 2012, Suárez-Rodríguez & García 2017, Cianchetti-Benedetti et al. 2018). For example, in seabird nesting colonies (Calonectris diomedea), short-term nocturnal exposure to light and noise pollution has been associated with decreased weight gain of chicks, which is thought to result from a reduction in parental nest attendance and feeding (Cianchetti-Benedetti et al. 2018). Further, human-induced eutrophication and associated algal blooms have been shown to directly impact the quality of nest construction in a fish, the three-spined stickleback (Gasterosteus aculeatus), with nest quality being an important predictor of offspring survival (Wong et al. 2012). A complex example of pollution-induced effects on parental care is seen in urban-dwelling house finches (Carpodacus mexicanus) that have started to incorporate cigarette butts into the lining of their nests (Suárez-Rodríguez & García 2017). In so doing, the chemicals present in the cigarettes reduced the amount of ectoparasites in the nest, although the benefits of this behaviour may be counterbalanced in the long-term by genotoxic damage to offspring (Suárez-Rodríguez & García 2017).

Ecological and evolutionary implications

We have described how environmental pollution can impact reproductive processes in a broad variety of ways, including via direct effects on sexual development and effects on sexual selection processes. These effects on different aspects of reproduction can dramatically alter the fitness of individuals by reducing the quality and/or quantity of offspring produced. By disrupting the overall reproductive success of a population, environmental pollution has many implications for the viability and evolutionary trajectory of affected populations.

Many of the reproductive alterations outlined in this review decrease fitness as a result of reduced mating or fertilisation success. In some cases, pollution-exposed individuals have reduced fertility due to impaired sexual development and/or gamete function. In other cases, individuals are unable to locate a mate due to the disruption of the production, transmission, or detection or sexual communication signals. As population stability depends on new generations of offspring, lowered reproductive success resulting from pollution exposure may lead to population declines or even extinctions.
For example, Kidd et al. (2007) conducted a 7-year whole-lake experiment with chronic exposure of fathead minnows (Pimephales promelas) to low concentrations of 17α-ethinylestradiol (EE2). The resulting feminisation of males and altered oogenesis in females led to a near extinction of this species in the lake, demonstrating the extreme impacts that reproductive impairment due to pollution can have at the population level (see also Desforges et al. 2018).

Effects of pollution on reproductive traits, however, do not always lower organismal fitness. Some alterations in reproductive processes may be minor enough to have negligible effects on fitness. For example, De Jong et al. (2015) reported that exposure to light pollution affected the lay date of great tits, although this did not appear to have any measurable fitness consequences in the offspring. Additionally, where pollutants cause the sex ratio of a population to be skewed towards females, this may not significantly affect the growth of a population in cases where the males are still able to successfully fertilise most of the females (Candolin 2019). In other cases, certain pollutants have even been shown to have potentially positive effects on fitness. For example, male fish exposed to growth-promoting steroids have been found to perform more copulations than unexposed males (Bertram et al. 2015, 2018). Similarly, Daphnia magna have been found to have increased fecundity when exposed to fluoxetine, an ingredient of many antidepressant medications (Campos et al. 2012). Together, the previously mentioned examples highlight how pollution can have negative, negligible, and even potentially positive effects on the average reproductive success of individuals within a population, with clear implications for population persistence over time.

In some cases, populations may be able to adjust or adapt to reproductive challenges posed by pollution, either through plastic or evolved responses. For example, city-dwelling song birds sing at a higher pitch compared to rural conspecifics, so that they can be heard above the low frequency din of urban noise (e.g. great tits, Parus major: Slabekoorn & Peet 2003). Some species are also able to adjust the timing of their calls to avoid the noisiest times of the day, as in frogs (Lithobates clamitans and Lithobates catesbeianus) subjected to road traffic noise (Vargas-Salinas et al. 2014) and birds living adjacent to airports (Gil et al. 2015). There are also numerous cases where females demonstrate a preference for unexposed males compared to those exposed to a pollutant (Gore et al. 2018), although this mechanism is less useful in environments where all males are contaminated. There is evidence to suggest that such changes can impact ecosystem dynamics if some species are able to adapt to pollutants better than others. For example, in New Mexico, bird species that adjust vocalisations in response to noise pollution increased in number, while species unable to do this decreased, altering bird communities and species interactions (Francis et al. 2009). It is also worth noting that the ability of species to adapt to pollutants may be constrained when the pollutants relax sexual selection, as there is evidence that sexual selection may promote evolutionary mechanisms that allow organisms to cope with pollution (Jacomb et al. 2016).

When species are unable to adapt to reproductive challenges caused by pollution, the resulting population declines can have cascading effects on the wider ecosystem due to altered species interactions. For instance, following the decrease in fathead minnows due to EE2 exposure, Kidd et al. (2014) observed an increase in abundance of its prey species, such as zooplankton, chaoborus, and emerging insects, as well as a decrease in biomass of lake trout, a fathead minnow predator. These species were unaffected by direct oestrogen exposure, highlighting the need for research examining broader ecological effects, such as species interactions, when assessing environmental impacts of pollution (see also Windsor et al. 2018). Such cascading effects have also been seen in ecosystems where gastropod populations have declined due to exposure to anti-fouling paint, resulting in disruptions to intertidal community structure and function (Coray & Bard 2007, Roach & Wilson 2009).

**Future directions**

When investigating effects of environmental change, there is a tendency for studies to focus on the direct, short-term effects of a single pollutant on a single species, most often vertebrates (Lewis & Ford 2012). However, this only provides a limited understanding of pollutant impacts and may underestimate broader hazards posed to wildlife and the ecological communities they inhabit. For a more holistic understanding, future studies should consider both biotic and abiotic interactions (discussed in Saaristo et al. 2018). Indeed, the previously mentioned research by Kidd et al. (2014) demonstrates the influence of species interactions on the wider effect of a pollutant, which can easily be overlooked when studying direct effects alone. Furthermore, pollutants can interact with other stressors to cause synergistic effects, where the interaction of multiple stressors causes an effect that is significantly greater than would be expected if the independent effects of each stressor were simply summed together. For example, in zebrafish, exposure to progesterin and high temperatures simultaneously had much more severe negative impacts on female fecundity than either effect in isolation (Cardoso et al. 2017). Additionally, future research should investigate the effects of pollutants over multiple generations. Such research will help to uncover whether species are able to adapt or habituate to environmental changes and will help us understand how pollutants may otherwise impact evolutionary processes (Saaristo et al. 2018, Candolin 2019). More broadly, if we are to more fully understand reproduction...
the impacts of pollutants on reproduction, more focus needs to be given to integrating research across multiple spatial scales at different levels of ecological complexity, from controlled laboratory assays to field-based investigations under more natural settings (Klaminder et al. 2016, Windsor et al. 2018; e.g. mesocosm-based studies on light pollution: Bennie et al. 2015; field-based studies on noise pollution: Simpson et al. 2016; whole-lake experiments on chemical pollution: Kidd et al. 2007, 2014).

Our increasing awareness of the impacts of pollutants on reproduction should be harnessed to implement mitigation strategies to manage many of the deleterious effects outlined in our review. Such actions may not only be beneficial for the sustainability of wildlife populations, but also for human health, with mounting evidence that these pollutants can affect humans in a similar manner to other animals (Damgaard et al. 2002, Deng et al. 2016). Indeed, there has already been some progress towards improved management of pollutants. For example, additional treatment steps, such as ozonation and activated carbon treatment, can play a key role in the removal of EDCs and other contaminants from wastewater (Nowotny et al. 2007, Hollender et al. 2009). In the context of light pollution, limiting the use of artificial light, shielding lights to reduce ‘trespass’ of light into neighbouring areas, and altering the intensity or spectrum of lighting can decrease potential impacts of anthropogenic light at night (Gaston et al. 2012). It has also been suggested that sound barriers and noise curfews, which are already widely used to reduce impacts on human inhabitants, could be effectively employed to alleviate effects of noise pollution on wildlife (Slabbekoorn & Ripmeester 2008). Such initiatives are just some of the many practical outcomes that can be achieved by harnessing research to minimise the detrimental effects of pollutants.

In this review, we highlight that environmental pollution, in its various forms, can interfere with many different aspects of reproduction, including physiology, gamete function, and organismal behaviour. These disruptions often reduce reproductive success of organisms by, for example, interfering with their ability to secure a mate or decreasing their fertility. In addition, pollution can reduce offspring viability through epigenetic effects or by altering reproductive timing (thereby causing a mismatch between ecological resources and offspring), by disrupting process of sexual selection resulting in mating with less suitable partners, and by disrupting parental care. While it is clear that environmental pollution can affect the reproduction of wildlife, the full extent to which these changes are affecting population growth, ecosystem structure, and evolutionary trajectories is less clear. To better understand these higher-level consequences of pollution, it is important that we collect long-term data and employ studies that incorporate increasing levels of ecological complexity through the use of mesocosm- and field-based approaches, as well as continue to implement strategies to mitigate the impacts of pollution on wildlife and humans alike.

Declaration of interest
The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of this review.

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L C A, M G B, J M M and B B M W conceived the idea and developed the structure of the review with input from other coauthors. All authors contributed to the writing of the review.

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R22


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