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A BRIEF HISTORY OF INSECTICIDES AND QUAIL

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ABSTRACT

Humans have employed chemical methods of pest control since the earliest days of agriculture and these substances have affected native wildlife, including quail and other gamebirds (Galliformes), to varying degrees. Several quail species have experienced steep population declines over the past several decades and insecticides may be a contributing factor. Quail are also known to use agricultural habitat for nesting and foraging purposes and are therefore likely to encounter elevated levels of insecticidal chemicals in the soil, vegetation, and insect biomass in that environment. The first commercially available insecticides appeared in the early 1900s with the introduction of arsenic-based compounds (arsenicals). Chemical engineering during World War II resulted in arsenicals being replaced with synthetically produced insecticides such as organochlorine, carbamate, and organophosphate compounds over several decades. Many of these substances have been shown to increase mortality rate, alter behavior, and produce severe reproductive complications in quail, both in the lab and the field. Today, the world's most popular insecticides, neonicotinoids, are being reevaluated for environmental safety following reports that they may be affecting nontarget wildlife. This review examines the types of insecticides that have been used in the United States, how quail could be exposed to these substances, and how they may have contributed to declining quail populations.

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Humans have employed chemical methods of pest control since the earliest days of agriculture and different substances have affected native wildlife, including quail and other gamebirds (Galliformes), to varying degrees. It is well-established that several quail species have experienced a steep population decline over the past several decades. Northern bobwhites (*Colinus virginianus*), for example, have decreased by >80% since the 1960s and insecticide use may be a contributing factor. Quail are also known to use agricultural habitat for nesting and foraging purposes and are therefore likely to encounter insecticidal chemicals in the soil, vegetation, and insect biomass in that environment. Here we examine the types of insecticides that have been used in the United States, how quail could have been exposed to these substances, and how their toxic effects may have contributed to diminishing quail populations.

ARSENIC

Arsenic compounds were among the first substances used to control agricultural pests (Bolt 2013). Significant concentrations of arsenic occur naturally in the environment, usually in conjunction with metals such as cobalt, nickel, iron, lead and copper (Chou and De Rosa 2003). Commercial varieties of arsenic pesticides became available in the mid-1800s and were used in the United States for >100 years (USDHHS 2016). The insecticides proved invaluable in controlling destructive pests such as the coddling moth, Colorado potato beetle, boll weevil, and horn worm (Eisler 1988, Stone and Anderson 2009). Despite their effectiveness, human health concerns caused arsenicals to be gradually phased out of agriculture (USDHHS 2016). Today, inorganic arsenic compounds are no longer manufactured or employed as insecticides in the United States.

At the height of their popularity in the late 1920s and 1930s, nearly 20,000 metric tons of arsenic-based pesticides were being applied annually in the United States (Reed et al. 2006). Quail and other game birds likely came into contact with these chemicals because quail have been known to use agricultural habitat for nesting and foraging purposes (Puckett et al. 1995) where they would have access to arsenic-contaminated insects as a food source and be subjected to dermal or inhalation exposure (Khan et al. 2014). Although it is not unusual to detect arsenic in soil from naturally occurring deposits, concentrations on farmland tend to be considerably

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greater than background levels as a result of repeated pesticide applications (Chou and De Rosa 2003). Arsenicals are also highly mobile via wind, surface water, and groundwater pathways (Irwin et al. 1997), meaning they can be transported beyond farmland areas to surrounding habitat and impact wildlife outside the immediate site of application.

Both field and laboratory studies have attempted to gauge the effects of arsenicals on wildlife. Laboratory toxicity tests have demonstrated that inorganic arsenic exposure is connected with stunted growth, weight loss, lethargy, and neurological abnormalities in chickens (*Gallus gallus*)—birds that are physiologically similar to quail (Khan et al. 2014). However, in an experiment designed to mimic field conditions, northern bobwhite hatchlings were fed grasshoppers killed with arsenic trioxide with no detectable impact (Eisler 1988). Arsenicals have also been shown to be quickly metabolized and excreted by gallinaceous birds; in chickens subjected to a diet laced with sodium arsenite, only 2% of the original dose remained in their systems after 60 hours (Eisler 1988). Another study demonstrated that woodpeckers (*Picoides dorsalis, P. villosus*) that fed on arsenic-laced beetle larvae experienced no debilitating effects despite elevated arsenic levels in their blood (Morrissey et al. 2007). Field evidence, coupled with the fact that no large-scale bird mortality events have been attributed to arsenic poisoning, suggests that arsenic-based pesticides may not have been a significant threat to quail even at the height of their use.

It appears unlikely that arsenicals have been a major contributor to declining quail populations. However, the human health impacts of arsenic-based insecticides (cardiac and respiratory disorders, cancers, etc.) meant that they would eventually be replaced. No effective substitute was found until World War II, when fervent interest in chemical engineering produced new classes of synthetic pesticides.

ORGANOCHLORINES

The advent of organochlorines (OCs) marked a new era for insecticides heralded by dichlorodiphenyltrichloroethane (DDT). Dichlorodiphenyltrichloroethane featured broad-spectrum toxicity to insects with the desired low toxicity to mammals and its half-life in the environment was measured in decades (USDHHS 2000, Hoffman et al. 2003). Its persistence, combined with the fact that it was largely insoluble in water and therefore unlikely to be washed away (Delaplane 1996, Muir 2012), reduced the need for reapplication. The insecticide was such a success that its discoverer, Paul Hermann Müller, earned the Nobel Prize in Physiology or Medicine in 1948 (Strandell 2009).

Although DDT and other organochlorines proved effective in controlling a variety of pests, they also had impacts on nontarget wildlife. Birds and mammals experienced neurotoxic effects in the form of uncontrolled muscle contractions and hyperactivity (Bradbury and Coats 1982, Lal and Saxena 1982). The estrogen-mimicking properties of OCs also interfered with avian reproduction by altering the timing of egg laying and producing abnormally thin eggshells. Birds that tried to incubate thin-shelled eggs crushed them in the attempt, resulting in population crashes in several species (Giguere 2008, Muir 2012). Dichlorodiphenyltrichloroethane's primary metabolite, dichlorodiphenyldichloroethylene (DDE), has a tendency to concentrate at higher trophic levels, meaning predatory birds were most severely affected (Connell 1999, Hoffman et al. 2003).

In determining whether quail were also affected, it is first necessary to consider how they might have been exposed. Dichlorodiphenyltrichloroethane was historically used to protect a variety of food crops including cotton, peanuts, and soybeans (NPIC 2000) and quail have been observed in similar agricultural habitat during periods of pesticide application (Palmer et al. 1998). In addition to crop spraying, organochlorines were applied directly to soil and aquatic environments to target specific pests (Lal and Saxena 1982) and detectable levels have remained many decades after use. These residues are subsequently carried by the movement of wind or water to new environments where organisms may be exposed via ingestion, respiratory, or dermal routes (Lal and Saxena 1982, Hoffman et al. 2003). Despite the tendency for quail to eat low on the food chain, organochlorine pesticides have been detected in scaled (*Callipepla squamata*) and bobwhite quail tissues (Baxter et al. 2015).

The effects of OC exposure on quail are varied. The eggshell thinning that was so well-documented in predatory birds appears to be less of a problem for galliforms because Japanese quail (*Coturnix japonica*) and chickens have shown only minor changes in eggshell thickness following exposure to DDT (Bitman et al. 1969, Peakall and Lincer 1996). However, other work has indicated that embryonic exposure to DDE can alter brain structure and interfere with reproduction by accelerating the onset of puberty in female Japanese quail and reducing sexual behaviors in males (Quinn et al. 2008, Mura et al. 2009). Given that quail rely on high rates of reproduction for maintaining populations (Brennan 2014), these changes could have impacts at the landscape level. Organochlorines have also been shown to enhance the toxicity of other substances. Adult male Japanese quail that were pretreated with dietary DDE were more susceptible to subsequent applications of an organophosphate compound, parathion (Ludke 1977). Similarly, pretreatment with the organochlorine endrin has been shown to increase accumulation of another OC, chlordane, in northern bobwhite brain tissue (Hoffman et al. 2003).

Reproductive impacts and synergistic effects with other toxicants make organochlorines a potential factor in the quail decline. Although DDT was effectively removed from the U.S. market in 1972 (USEPA 2016), its residues and effects lingered for decades after the ban. The elimination of organochlorines also left a void to be filled by new types of insecticides.

ORGANOPHOSPHATES AND CARBAMATES

Organophosphate (OP) and carbamate (CB) pesticides are used as insecticides, herbicides, and fungicides on agricultural lands, rangelands, forests, wetlands, and residential and commercial areas (Smith 1987, Glaser 1999). Most widely used OP and CB insecticides are highly toxic but relatively short-lived in the environment (Smith 1987, Glaser 1999, Hill et al. 2012), making them an acceptable replacement for the highly persistent, bioaccumulative organochlorines (Hassall 1982, Smith 1987, Hill 2003). Both OPs and CBs were introduced into commercial use in the 1950s (Kuhr and Dorough 1976, Smith 1987).

Of the 2 classes, OPs are more ubiquitous and comprise more than one-third of the registered pesticides on the world market (Hill 2003). In the United States alone there are approximately 70 organophosphorus pesticides registered as active ingredients in thousands of products, such as chlorpyrifos and methyl parathion (Hill 2003). The OP Glyphosate was the most used pesticide active ingredient in 2007 with 180–185 million pounds applied (USEPA 2016). In contrast, there are approximately 50 registered carbamate-based pesticides available (Hill 2003). Of the 50, only 8 are used for insect control and 3 of the 8—carbofuran, methomyl, and carbaryl—account for >90% of use (Hill 2003).

Common routes of exposure for birds to OP and CB pesticides include consumption of treated seeds, pesticide-coated vegetation, poisoned insects, direct ingestion of pesticide granules, and contaminated water (Dimmick 1992, Glaser 1999). Inhalation and absorption through the skin are also possible (Glaser 1999). Quail are often found in or near agricultural lands, so they are particularly vulnerable to exposure to OPs and CBs used to treat crops (Dimmick 1992). Bobwhite quail chicks may be more susceptible to exposure via consumption of poisoned insects because they feed almost exclusively on insects during the first few weeks posthatch (NRCS 1999). Similarly, adult bobwhites may be at greater risk of exposure by eating pesticide-coated seeds and vegetation because these constitute the majority of their diet (NRCS 1999, Hernández and Guthery 2012).

Birds seem to be particularly sensitive to the effects of OPs and CBs (Grue et al. 1997, Glaser 1999, Hill 2003), which have been shown to produce physiological, behavioral, and reproductive effects in quail and other avian species. Studies have demonstrated an increase in mortality following ingestion of these pesticides in bobwhites (Brewer et al. 1996) and mallard ducks (*Anas platyrhynchos;* Martin 1990). The organophosphate methyl parathion has been known to alter brain chemistry in bobwhites, with subsequent effects on activity level and coordination along with increased predation rates (Galindo et al. 1985, Buerger et al. 1991). Feeding behavior is known to be affected as well: bobwhite chicks dosed with methyl parathion exhibited decreased food-seeking activity and failed to discriminate between treated and untreated food (Bussiere et al. 1989), though other work suggests that they may be able to detect and avoid pesticide contaminated feed when given sufficient alternatives (Bennett 1989). There is also evidence that organophosphates and carbamates may suppress immune system function (Nain et al. 2011), slow hatchling growth and development (Martin et al. 1991), and reduce egg production and hatching rate (Rattner et al. 1982, Stromborg 1986, Kilbride et al. 1992).

Exposure to OP and CB pesticides could therefore contribute to the decline of quail by increasing mortality (directly or through abnormal behavior that increases predation), altering feeding habits, rendering birds immunocompromised or interfering with reproduction. These chemicals are still used today, alongside a new class of insecticides: the neonicotinoids.

NEONICOTINOIDS

When neonicotinoids first entered the market in the early 1990s, they appeared to address the concerns associated with earlier compounds. This new class of insecticides was effective as a form of pest control but possessed a high degree of selectivity to insects (Jeschke et al. 2013), making them safer for human use than the organochlorines, organophosphates, and carbamates. They are most often applied as a seed coating, which is absorbed into plant tissues, localizing the protectant and reducing contamination to the environment (Goulson 2013, Simon-Delso et al. 2014). In the years that followed their introduction, neonicotinoid compounds became the most widely used insecticides in the world (Gibbons et al. 2014).

As the name implies, neonicotinoids behave similarly to nicotine, a chemical that is produced in some plants as a pest deterrent (Gotti and Clementi 2004, Seifert and Stollberg 2005). The toxic effects of nicotine derive from the fact that it mimics the neurotransmitter acetylcholine, overstimulating the nervous system and causing death in insects (Yamamoto and Casida 1999). Neonicotinoids differ in that they are more strongly attracted to receptors in the invertebrate nervous system than the vertebrate one (Millar and Denholm 2007).

The insecticide's ability to translocate into plant tissues would theoretically keep environmental concentrations low and minimize exposure to quail and other wildlife, but data suggest that field concentrations are higher than anticipated. It is estimated that approximately 5% of the pesticide in a seed coating is absorbed by the plant while the rest blows away during sowing or is deposited in the surrounding soil and water (Goulson 2014). The lubricating powder that facilitates sowing can contain active ingredient concentrations up to 15,000 μg/g (Bonmatin et al. 2015) and is often released to the environment after use. Neonicotinoids are purported to be denatured relatively quickly in direct sunlight; however, half-lives have been shown to exceed 1,000 days in soil and water mediums. Several studies have detected environmental neonicotinoid concentrations that far exceed the amounts needed to control pests (Goulson 2013, Gibbons et al. 2014, Hladik et al. 2014). Some seed is frequently spilled during the sowing process or buried

shallowly enough that it can be scratched out (Mineau and Palmer 2013), making the coated seeds themselves a potential source of contact. Plausible routes of exposure for quail therefore include ingestion of coated seeds and environmental residues at greater concentrations than those predicted by normal use.

Despite being engineered for low toxicity to vertebrates, there is evidence of neonicotinoids affecting quail and other birds. There have been reports of avian mortalities in the field attributable to neonicotinoid ingestion as determined by crop and tissue analysis (Berny et al. 1999). Abnormal behaviors, including lethargy, ptiloerection, and impaired locomotor ability, have also been documented in both lab and field investigations following consumption of neonicotinoid coated seeds (Berny et al. 1999, Poppenga and Tawde 2012, Tokumoto et al. 2013). Data suggest that neonicotinoids may decrease clutch size and embryo survival rates in bobwhites (Fernandez-Perea et al. 2009) or produce ovary malformations in laying hens (Hoshi et al. 2014). Neonicotinoids and their metabolites have also been known to exert genotoxic effects, resulting in cancer and DNA fragmentation in germ cells (Casida 2011, Gibbons et al. 2014, Hoshi et al. 2014). By increasing mortality of adult birds through direct toxic effects or increased predation, and by affecting reproductive efforts through decreased egg production, reduced embryo survival, and genetic complications, neonicotinoids become a potential contributor to declining quail populations.

MANAGEMENT AND RESEARCH IMPLICATIONS

The history of insecticide use in the United States makes it clear that they are potential contributors to quail population declines and have important implications for quail management. For many of the compounds described above, the full extent of their impact—field-realistic concentrations, interactions with other potentially toxic substances, effects specific to avian or embryonic physiology—were not understood until after they had been in use for some time. There is, of course, testing that takes place before any pesticide is made commercially available; however, it is difficult to account for all field-realistic variables, every species, and every possible type of effect. The most effective way to minimize the impact of insecticides on quail populations is to limit their use as much as possible by taking advantage of alternative pest control methods. Integrated Pest Management calls for the cultivation of pest-resistant crops; employment of insect pheromones in controlling and monitoring pest species; and the use of predators, pathogens, and parasites to reduce insect damage without insecticides (Abrol 2013). In areas where management goals are more aligned with ranching than crop cultivation, simply maintaining a diverse, native grassland habitat is an effective way to discourage pest species. Nonchemical methods for achieving this include disking, burning, and hoof traffic, which can be used to control for both plant and insect pests (Martin 1983, Hernández and Guthery 2012).

Future research can also help minimize the effects of insecticides on quail populations. Toxicity studies should focus on determining field concentrations based on persistence and real rates of application, which may differ from application instructions. There should also be greater emphasis placed on determining impacts on embryos and chicks, which may be more susceptible to chemical exposure than adult birds. Research that contributes to the improvement of nonpesticide methods of control can also help reduce insecticide use in quail habitat.

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