Effect of Acute Exposure to Malathion and Lead on Sprint Performance of the Western Fence Lizard (*Sceloporus occidentalis*)

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Abstract. There are few ecotoxicological studies involving reptiles, despite the fact that anthropogenic pollutants have been identified as a major threat to reptile populations worldwide. Particularly lacking are effects-based studies in reptiles exposed to known concentrations of contaminants. We hypothesized that acute exposure to neurotoxic metals and pesticides could influence locomotor performance of reptiles. To test this hypothesis, we exposed western fence lizards (*Sceloporus occidentalis*) to two common and widely studied neurotoxic contaminants, malathion and lead (Pb). Single doses were administered via oral gavage at order-of-magnitude levels ranging from 0.2 to 200 and 1.0 to 1,000 mg/kg (body weight basis) for malathion and Pb, respectively. Lizard sprint velocity was determined using a 2.3-m sprint track interfaced with a laptop computer 24 hrs prior to dosing and again at 4, 24, 120, and 312 hrs post-dose. Twenty percent and 30% mortality occurred at the highest malathion and Pb dose levels (200 and 1000 mg/kg) and 70% of the lizards exposed to 200 mg/kg malathion exhibited clinical symptoms of organophosphate poisoning. Contrary to our predictions, exposure to Pb had no effect on locomotor performance, and exposure to the highest concentration of malathion increased sprint velocity. Based on the fact that the lower and most ecologically relevant concentrations of Pb and malathion had no effect on sprint velocity, we suggest that other performance parameters that require fine locomotor skills (e.g., climbing ability) may be more sensitive metrics of acute neurotoxicity and warrant further study.

Anthropogenic pollutants have been identified as one of the major contributing factors in the global decline of reptiles (Gibbons *et al.* 2000), yet reptiles remain the least studied group of vertebrates in ecotoxicology (Hopkins 2000). Most of the existing reptile toxicology data are focused on chelonians (Sparling *et al.* 2000), which comprise less than 4% of the

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world's 7100 + reptile species (Gibbons *et al.* 2000). Of the few ecotoxicological studies involving squamates (lizards and snakes), even fewer quantify effects resulting from exposure to known concentrations of contaminants. Effects-based studies involving representatives from all reptile orders and major classes of environmental contaminants are necessary to provide a foundation for informed management and remediation decisions.

In reptiles, locomotor performance often plays a critical role in the completion of tasks such as mate selection, foraging, and predator evasion that may influence survival and reproduction, and is therefore believed to be linked to fitness. The relationship between locomotor performance and fitness in lizards was illustrated in a mark-recapture study by Miles (2004), who concluded that fast juvenile lizards (*Urosaurus ornatus*) realized a survival advantage over their slower but similarly sized counterparts. Because of its potential to influence fitness, locomotor performance in lizards has been widely studied. Many studies of the locomotor performance of lizards have concluded that measures such as sprint velocity and endurance are strongly repeatable in a laboratory setting (Huey and Dunham 1987; Van Berkum *et al.* 1989), and factors that affect locomotor performance such as temperature (Angilletta Jr *et al.* 2002; Bauwens *et al.* 1995; Bennett 1980; Crowley 1985; Huey and Bennett 1987; Huey *et al.* 1989; Van Berkum 1986; Van Damme *et al.* 1992), morphology (Bonine and Garland Jr 1999; Martín and López 2001; Sinervo and Losos 1989), phylogenetic history (Sorci *et al.* 1995; Van Damme and Vanhooydonck 2001), reproductive status (Sinervo and Hedges 1991), and parasitism (Sorci *et al.* 1994) are well documented. Previous studies on other vertebrate taxa suggest that performance can also be altered by exposure to neurotoxic contaminants (Beauvais *et al.* 2000; Beauvais *et al.* 2001; Hopkins *et al.* 2003; Hopkins and Winne in press; Hopkins *et al.* 2005b; Walker 2003). However, the effect of environmental contaminants on the locomotor performance of lizards is unknown.

To determine whether environmental contaminants can impact locomotor performance in lizards, we exposed western fence lizards (*Sceloporus occidentalis*) to two common and widely studied contaminants, malathion and lead (Pb).

Malathion (S-[1,2-Bis(ethoxycarbonyl)ethyl] *O,O*-dimethyl phosphorodithioate) is a broad-spectrum organophosphate (OP) insecticide that acts via cholinesterase inhibition. It is most commonly applied to control crop-destroying and disease-carrying insects such as the boll weevil (*Anthonomus grandis grandis*), Mediterranean fruit fly (*Ceratitis capitata*), and mosquito (family Culicidae). The United States Environmental Protection Agency estimates that 20–25 million pounds (active ingredient) of malathion was applied to U.S. crops in 2001 alone, the most recent year for which data were available (Donaldson *et al.* 2004). Lead is the most prolific heavy metal found in the earth's crust (among metals with atomic number >60) and is a toxicant that inhibits the activities of enzymes necessary for normal biological function (Pattee and Pain 2003). It has been shown to significantly affect many aspects of the central nervous system of vertebrates, most notably infants and children (ATSDR 1999). Insectivorous lizards such as *S. occidentalis* could be exposed to malathion or Pb via ingestion of contaminated prey items because nontarget invertebrates preyed upon by *S. occidentalis* (e.g., ants, spiders) have been shown to accumulate Pb (Clausen 1984; Devkota and Schmidt 2000; Eeva *et al.* 2004; Rabitsch 1997) and will likely be exposed to malathion due to broad-scale applications (e.g., aerial spraying). The goal of the current study was to determine whether a single dose of malathion or Pb affects the locomotor performance, specifically the maximum sprint velocity, of *S. occidentalis*.

Materials and Methods

Experimental Animals

Sceloporus occidentalis is a small (12–20-cm total length) diurnal lizard that occurs in many habitat types throughout the western United States. It is an opportunistic predator, frequently preying upon ants, beetles, termites, and other invertebrates (Rose 1976). *Sceloporus occidentalis* was selected as a model for toxicological study based on its role as an active consumer in the terrestrial food web, capacity to occupy diverse habitats, and life history traits that make it desirable for laboratory studies (e.g., rapid maturation, high survival rate (Talent *et al.* 2002)).

Juvenile lizards were obtained from a breeding colony at Oklahoma State University and shipped to the Savannah River Ecology Laboratory (South Carolina, USA). The parental stock of lizards used to establish the breeding colony originated from the San Joaquin Valley, California, USA (Talent *et al.* 2002). Lab-reared individuals were selected over field-captured individuals to avoid any previous contaminant exposure, ensure that all experimental lizards were disease free, and to promote the use of captive colonies of reptiles as a conservation-minded approach to toxicological testing. Juvenile lizards were used so that potential effects were assessed at a sensitive life stage. Lizard husbandry was identical to Hopkins *et al.* (2005a), with the following notable exceptions: a 10 hr:14 hr (light:dark) photoperiod, a daytime temperature gradient of ~28–40°C, and a diet consisting of five crickets (~1.0 cm each) per day.

Sprint Velocity

Lizard sprint velocities were measured using a 2.3-m sprint track (Columbus Instruments, Columbus, Ohio, USA) interfaced with a laptop computer. Pairs of photocells projecting infrared beams lined the sprint track at 10-cm intervals. As lizards advanced down the track, photocell beams were interrupted and elapsed time between photocells was recorded by computer software. Velocities were calculated over each 0.2-m interval. Each lizard was raced three times in succession, and the single fastest 0.2-m interval from each sprint was recorded.

Because body temperature influences locomotor performance in lizards (Bennett 1980; Crowley 1985; Huey and Bennett 1987), the lizards were maintained at 34°C, their optimum activity temperature (Adolph 1987; Brattstrom 1965), and body temperature were recorded before and immediately after each sprint using a quick-reading Schultheis cloacal thermometer. Prior to beginning a sprint, individual lizards were placed in a heated box attached to the starting point of the track and left undisturbed for 2–4 min. A gate separating the start box from the sprint track was then lifted, and lizards were chased by hand down the track (Huey *et al.* 1989). Further motivation was provided by attaching a darkened rectangular cardboard shelter to the opposite end of the track (Bennett 1980).

Vehicle Experiments

We initially sought to determine whether administration of the vehicles (distilled water and corn oil) would influence maximum sprint velocity of lizards. Vehicle experiments were conducted in May (corn oil) and December (distilled water) 2004, respectively. In both experiments, 16 juvenile lizards were randomly assigned to two treatment groups (*n* = 8 per group/equal sex distribution). Individuals in one treatment group received a single dose of either corn oil or distilled water via oral gavage. The remaining treatment groups served as controls and received no gavage but were otherwise handled in a similar fashion. All lizards were immediately returned to their cages after gavage or handling. Dose volumes were regulated using a 2–20 µl Eppendorf Reference pipette and varied according to lizard mass; volumes ranged from 7.30 to 9.94 and 10.72 to 13.90 µl for corn oil and distilled water, respectively. The maximum velocity of each lizard was measured 24 hrs prior to dosing, and again at 4, 24, and 96 hrs post-dose. The snout-to-vent length (SVL, measured in mm) and mass (g) of each lizard were recorded at the beginning and end of the 5-day trial. Because our vehicle experiments demonstrated that gavage of distilled water or corn oil did not influence sprint velocity (see Results section), only vehicle controls were used in subsequent experiments.

Exposure to Malathion and Pb

Malathion and Pb experiments were conducted in June 2004 and January 2005, respectively. In each experiment, 50 different lizards were randomly assigned to five treatment groups (*n* = 10 per treatment group). In the first experiment, lizards in four treatment groups received a single dose of malathion at order-of-magnitude concentrations ranging from 0.2 to 200 mg/kg body weight (bw). The fifth treatment group received a single dose of corn oil (vehicle control). In the second experiment, four treatment groups received a single dose of Pb at order-of-magnitude concentrations ranging from 1.0 to 1000 mg/kg (bw). The fifth treatment group received a single dose of distilled water (vehicle control). All doses were administered via oral gavage, and total handling time during dose administration was generally less than 60 s. Dose volumes ranged from 10.26 to 13.92 and 13.71 to 24.78 µl for malathion and Pb, respectively. Dosing solutions of malathion and Pb were prepared by adding Fyfanon Ultra Low Volume (96.5% malathion; Cheminova, Lemvig, Denmark) and Pb acetate trihydrate (Sigma Aldrich, St. Louis, Missouri, USA) to corn oil and distilled water, respectively. Solutions were prepared within 1 hr of dose administration.

The concentrations of malathion and Pb encountered by lizards in the field have not been adequately assessed. Therefore, malathion doses were selected based on compiled pesticide residue levels in Bishop *et al.* (2000) and malathion-specific application rates. Using residues from invertebrates collected in the field and an application rate of 1.0 kg active ingredient/ha, Bishop *et al.* 2000 calculated a mean pesticide residue level of 27.9 μg/g. At reported malathion application rates of 0.85–1.36 kg active ingredient/ha (Tillman and Mulrooney 2001), lizards could ingest 23.7–36.3 μg malathion for every gram prey consumed. For selection of Pb doses, we relied upon concentrations of Pb in terrestrial invertebrates retrieved from the gut of geckos inhabiting a heavily contaminated region of Spain (Fletcher and Hopkins, in press). Based on these concentrations, lizards could ingest as much as 55 μg Pb per gram of prey if foraging in a heavily contaminated site. Thus, we selected two ecologically relevant doses of malathion and Pb and two additional doses of each contaminant that may be greater than expected concentrations in most field conditions, but less than the probable LD50s for these pollutants (Hall and Clark Jr. 1982; Özelmas and Akay 1995; Salice *et al.* 2003).

The maximum sprint velocity of each lizard was measured 24 hrs prior to dosing and again at 4, 24, 120, and 312 hrs post-dose. Lizards were fasted 12–24 hrs before each sprint interval and were given five crickets (∼1.0 cm each) per day on all other days. The SVL (mm) of all lizards was recorded 24 hrs prior to dosing and at 120 and 312 hrs post-dose. Lizard mass (g) was recorded before each sprint interval.

Statistical Analysis

Prior to statistical analysis, data were tested for normality and homoscedasticity using Ryan Joiner and Bartlett's tests, respectively. All sprint velocity and body size data were normally distributed, and variance was similar among treatments. Lizard survival was compared among treatment groups using a Fisher exact test. In the malathion experiment, two lizards died and two additional lizards were severely incapacitated (i.e., could not complete the 4-hr post-dose sprint trial) after receiving 200 mg/kg malathion (decreasing the sample sizes in the 200 mg/kg treatment to $n = 6$). In addition, one lizard from the 2.0 mg/kg malathion treatment was injured attempting to escape from its cage ($n = 9$). In the Pb experiment, three lizards died after receiving 1000 mg/kg Pb ($n = 7$). Because repeated-measures analysis requires no missing values, these lizards were omitted from all statistical comparisons and figures. For the final data analysis, we calculated the single fastest velocity and the mean of the three fastest velocities (one from each sprint) to generate estimates of maximum sprint velocity and mean maximum sprint velocity, respectively. Although the former metric is the most widely applied indication of sprint performance capacity in lizards, the latter metric better captures the intraindividual variation exhibited by lizards (e.g., some lizards were poor sprinters overall, but could achieve a single fast velocity over a 0.2-m segment of the track). For each experiment, we then compared maximum sprint velocity and mean maximum sprint velocity among treatment groups using repeated-measures analysis of covariance with time as the repeated variable and initial lizard SVL as the covariate.

Results

Vehicle Experiments

Sprint velocities of lizards receiving a single gavage of distilled water did not differ significantly from control lizards (data not shown; treatment: $F_{1,13} = 0.52$, $p = 0.483$; time: $F_{3,39} = 0.75$, $p = 0.527$; treatment X time: $F_{3,39} = 0.13$, $p = 0.939$). Similarly, a single gavage of corn oil had no effect on lizard sprint performance (data not shown; treatment:
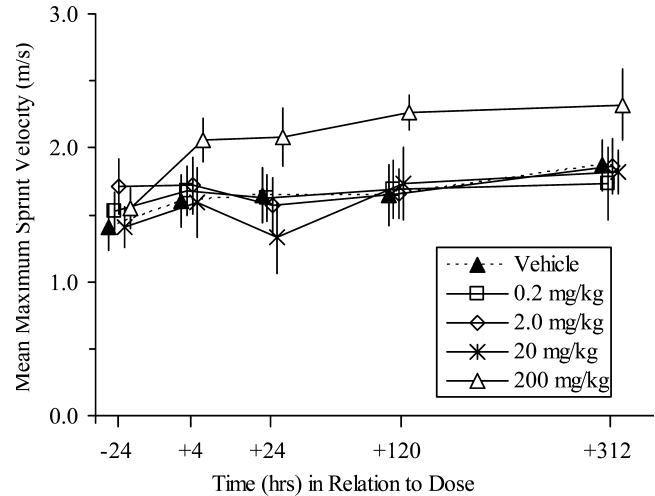
Fig. 1. Mean maximum sprint velocities (m/s) in fence lizards before and after oral administration of malathion on a mg/kg, body weight basis. Three 2.3-m sprints were conducted per time period, and mean maximum velocity equaled the average of the fastest 0.2-m section from each sprint. Error bars are ±1 standard error of the mean. Treatment means are offset slightly on the x-axis for clarity. $N = 10$ for vehicle control, 0.2, and 20 mg/kg groups; $N = 9$ for the 2.0 mg/kg group due to injury, and $N = 6$ for 200 mg/kg group due to mortality and incapacitation.

$F_{1,13} = 0.29$, $p = 0.601$; time: $F_{3,39} = 0.08$, $p = 0.970$; treatment X time: $F_{3,39} = 0.52$, $p = 0.673$).

Malathion

Lizard survival was not significantly influenced by malathion exposure ($p = 0.184$). No lizards died in the vehicle control, 0.2, 2.0, and 20 mg/kg groups, but 20% (2/10) mortality was observed in the highest dose group (200 mg/kg). Despite a noteworthy stimulatory trend in the 200 mg/kg dose group, maximum sprint velocities did not differ significantly among treatment groups (data not shown; treatment: $F_{4,39} = 0.66$, $p = 0.620$; time: $F_{4,156} = 1.02$, $p = 0.400$; treatment X time: $F_{16,156} = 1.33$, $p = 0.186$). However, mean maximum sprint velocities were significantly influenced by exposure to malathion (Fig. 1; treatment: $F_{4,39} = 1.02$, $p = 0.409$; time: $F_{4,156} = 1.23$, $p = 0.299$; treatment X time: $F_{16,156} = 2.00$, $p = 0.016$). Although sprint velocities of lizards receiving single 0.2, 2.0, and 20 mg/kg doses of malathion were clearly similar to vehicle control lizards, lizards at the 200 mg/kg dose level exhibited, on average, a 23% increase in sprint velocity after exposure to malathion (Fig. 1). Initial mass (range of treatment means ± 1 SE: 6.78 ± 0.142–7.09 ± 0.208) and SVL (range of treatment means ± 1 SE: 55.88 ± 0.435–57.22 ± 0.945) were similar among treatments, and decreased 2.7–4.8% throughout the experiment.

Lead

Lizard survival was significantly influenced by Pb exposure ($p = 0.031$). No deaths occurred in the vehicle control, 1.0, 10,
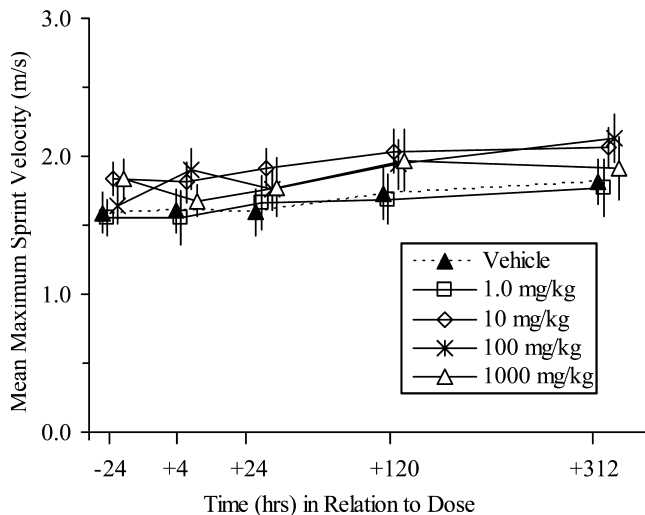
Fig. 2. Mean maximum sprint velocities (m/s) in fence lizards before and after oral administration of Pb on a mg/kg, body weight basis. Three 2.3-m sprints were conducted per time period, and mean maximum velocity equaled the average of the fastest 0.2-m section from each sprint. Error bars are ±1 standard error of the mean. Treatment means are offset slightly on the x-axis for clarity. $N = 10$ for vehicle control, 1.0, 10, and 100 mg/kg groups, and $N = 7$ for 1000 mg/kg group due to mortality.

and 100 mg/kg groups, but 30% (3/10) mortality was observed in the highest dose group (1000 mg/kg). Maximum sprint velocities did not differ among treatment groups (data not shown; treatment: $F_{4,41} = 0.41$, $p = 0.803$; time: $F_{4,164} = 0.26$, $p = 0.902$; treatment X time: $F_{16,164} = 1.31$, $p = 0.195$). Likewise, mean maximum sprint velocities were not influenced by exposure to Pb (Fig. 2; treatment: $F_{4,41} = 0.44$, $p = 0.780$; time: $F_{4,164} = 0.38$, $p = 0.820$; treatment X time: $F_{16,164} = 0.72$, $p = 0.775$). Initial mass (range of treatment means ± 1 SE: 6.06 ± 0. 150–6.53 ± 0.310) and SVL (range of treatment means ± 1 SE: 54.10 ± 0.670–56.09 ± 0.919) did not differ significantly among treatments at the beginning of the experiment, and decreased 3.9–9.3% throughout the experiment.

Discussion

Contrary to our predictions, acute exposure to Pb and malathion did not reduce the sprint velocity of *S. occidentalis*. In the malathion experiment, 70% (7/10) of the lizards that received a 200 mg/kg malathion dose exhibited clinical symptoms of OP poisoning (e.g., body/limb tremors, twitching); two of these individuals died within 24 hrs and two others were temporarily incapacitated. Symptoms manifested themselves within 4 hrs of dose administration and subsided within 24 hrs in the surviving lizards. Despite the fact that lizards were clearly intoxicated at this high dose of malathion, they actually performed significantly better than lizards in all other treatments and their increased performance was sustained for at least 13 days. We are unaware of other instances of improved performance after exposure to near lethal levels of cholinesterase inhibitors. However, in some organisms, exposure to cholinesterase

inhibitors results in hyperactivity (Beauvais *et al.* 2000) and an increase in fasciculation (ATSDR 2003), which could influence sprint velocity. In the Pb experiment, 30% of lizards exposed to 1000 mg/kg Pb died within 24 hrs and 50% of the surviving lizards in this treatment exhibited a significant increase in skin pigmentation (darkened) but showed no other overt signs of poisoning. Changes in skin coloration are a classic symptom of stress in lizards and are primarily mediated by chromoactive hormones (e.g., epinephrine, norepinephrine, and melanotropin; reviewed in Greenberg 2002). Despite showing these symptoms of stress at near lethal doses of Pb, sprint velocity of these lizards was not impaired.

Our findings were not consistent with those from most other studies that documented the negative effects of cholinesterase inhibitors on behavior and performance of other herpetofauna. Hopkins *et al.* (2005b) and Hopkins and Winne (in press) described reduced swim velocities of four species of natricine snakes after exposure to carbaryl, another cholinesterase-inhibiting pesticide. To our knowledge, these are the only other studies investigating effects of neurotoxicants on reptilian performance parameters. However, differences such as exposure pathway (snake: 48-hr immersion vs. lizard: oral gavage), locomotion type (swimming vs. sprinting), and compound (carbamate vs. OP), make direct comparisons of these studies challenging. In an amphibian study, exposure to malathion impaired the ability of bullfrog (*Rana catesbeiana*) tadpoles to maintain equilibrium posture (Fordham *et al.* 2001). Fish (Beauvais *et al.* 2000; Sandahl *et al.* 2005; Scholz *et al.* 2000), birds (Fryday *et al.* 1996; Grue *et al.* 1997), and terrestrial isopods (Engenheiro 2005) also exhibited impaired behaviors and/or locomotor function after exposure to cholinesterase-inhibiting pesticides. In contrast, Baker (1985) described no significant changes in feeding, endurance, and coordination in two species of woodland salamanders (*Plethodon glutinosus* and *P. cinereus*) after exposure to malathion-treated substrates. Combined with findings of the current study, these data suggest that the effects of malathion on behavior and performance can vary widely and highlight the need for additional study across multiple taxa.

Although much is known regarding the neurotoxic effects of Pb in humans and wildlife, little is known about the effects of Pb (or any other metal) in lizards (Burger *et al.* 2004; Linder and Grillitsch 2000). In a review of lizard contaminant data, Campbell and Campbell (2000) reported only five studies that focused on Pb in lizards. All of these studies reported Pb concentrations in tissue and did not quantify biological effects of Pb exposure. Pb has been shown to impair neurobehavioral development in birds (Burger and Gochfeld 1994, 1997), decrease the fright response of Columbia spotted frog tadpoles (*Rana luteiventris*) (Lefcort *et al.* 1998), and induce hypoxia-like behavioral responses in bullfrog larvae (Rice *et al.* 1999). Additionally, songbirds residing in a heavy-metal (lead, arsenic, copper, cadmium, and zinc) contaminated habitat exhibited increased aggressive behavior compared to birds in a reference site (Janssens *et al.* 2003).

Although our study was not designed to examine lethal limits of Pb and malathion, our observations of mortality are valuable given the limited amount of reptilian toxicity data available. Three studies have estimated lizard LD_{50}'s (dose level that results in 50% mortality) or lethal levels for malathion or Pb. Özelmas (1993) calculated an LD_{50} of 169.8

mg/kg malathion for dwarf lizards (*Lacerta parva)*, whereas Hall and Clark (1982) predicted 2324 mg/kg for anoles (*Anolis carolinensis*). In the only previous study to examine the effect of Pb on lizard survival, Salice *et al.* (2003) reported a lethal dose level of 2,000 mg/kg Pb for *S. occidentalis* adults. Our observations of 20% and 30% mortality after exposure to 200 mg/kg malathion and 1000 mg/kg Pb, respectively, support the findings of the abovementioned studies. Reported mammal and avian LD$_{50}$'s for malathion were 1000 to 10,000 mg/kg (rat); 400 to <4000 mg/kg (mouse); and 167 and 1485 mg/kg for pheasants and mallards, respectively (Kamrin 1997). Reported LD$_{50}$'s for Pb range from 35 to 2000 mg/kg in rats (Luckey and Venugopal 1978) and generally <1000 mg/kg in birds (Burton Jr. *et al.* 2002). These data suggest that *S. occidentalis* is equally sensitive to acute exposure to malathion and Pb as other terrestrial vertebrates.

In conclusion, our study represents the first to examine the effects of neurotoxicants on the locomotor performance of lizards. Contrary to our predictions, maximum sprint speed of *S. occidentalis* was not influenced by Pb, and significantly increased after malathion exposure. However, lizards in the wild probably endure longer exposures and must contend with multiple contaminants that could have interactive effects. Thus, exposure to one contaminant via a single bolus, although precise and informative, may not be representative of exposures in the field. Additionally, other performance parameters that require the use of fine motor skills may be more sensitive endpoints than highly repetitive motor behaviors such as sprinting. For example, many lizards (including populations of *S. occidentalis*) are arboreal and navigate complex environments; therefore, assessment of climbing ability across inclined planes or complex substrates may highlight additional effects. Given these factors, future study is necessary to fully evaluate the effects of neurotoxicants on locomotor and behavioral characteristics important to the fitness of lizards such as *S. occidentalis*.

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