Environment International 63 (2014) 40–52

Contents lists available at ScienceDirect

Environment International

journal homepage: www.elsevier.com/locate/envint

Review

Organic contaminants in bats: Trends and new issues

Sara Bayat [a,*], Fritz Geiser [b], Paul Kristiansen [a], Susan C. Wilson [a]

[a] School of Environmental and Rural Sciences, University of New England, Armidale, NSW 2351, Australia
[b] Centre for Behavioral and Physiological Ecology, Zoology, University of New England, Armidale, NSW 2351, Australia

ARTICLE INFO

Article history:
Received 8 June 2013
Accepted 18 October 2013
Available online 19 November 2013

Keywords:
Organic contaminants
Pesticides
Mortality
Bats

ABSTRACT

Exposure to contaminants, often pesticides, has been implicated as a major factor contributing to decreases in bat populations. Bats provide essential ecosystem services and a sustained, thriving population is vital for ecosystem health. Understanding issues threatening their survival is crucial for their protection and conservation. This paper provides the first review for 12 years on organic pollutants in bats and aims to investigate trends and any new issues impacting bat resilience. Organochlorine (OC) pesticides have been reported most often, especially in the older literature, with the dichlorodiphenyltrichloroethane (DDT) metabolite, dichlorodiphenyldichloroethylene (DDE), present at highest concentrations in tissues analyzed. The OC pesticide concentrations reported in bat tissues have declined significantly since the late 1970s, presumably as a result of restrictions in use. For example, DDE study mean concentrations over time periods 1970–1980, 1981–1999 and 2000–2013 ranged from 2.6-62, 0.05–2.31, 0.08–0.19 ppm wet weight, respectively. Exposure, however, still occurs from remaining residues, many years after the compounds have been actively used. In recent years (2000–2013), a range of other organic chemicals have been reported in bat tissues including brominated flame retardants (polybrominated diphenyl ether at a mean concentration of 2.9 ppm lipid weight) and perfluorinated compounds (perfluorooctanyl sulfonate at a mean concentration 0.09 ppm wet weight). The persistent organic compounds concentrate in tissues with higher fat content notably back-depot fat. Numerous factors influence exposure, residues detected and concentrations in different individuals, species and tissues which must be understood to provide meaningful assessment of the impacts of exposure. Exposure can lead to not only acute and lethal impacts, but also physiological sub-lethal and chronic effects, often linked to the annual cycle of fat deposition and withdrawal. Current challenges for bat conservation include collation of a more extensive and standardized database of bat exposure, especially to current use pesticides and emerging contaminants, and better prediction and definition of toxicity end points notably for the sub-lethal effects. Understanding sub-lethal effects will be of greater importance for sustaining populations in the longer-term.

Contents

* Corresponding author. Tel.: +61 2 67732849.
E-mail address: sbayat@myune.edu.au (S. Bayat).

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http://dx.doi.org/10.1016/j.envint.2013.10.009

1. Introduction

Bats comprise about 20% of all mammals and have a vital ecological function due to wide taxonomic and functional diversity, position in the food chain, feeding patterns and habits and relative longevity. They provide a range of essential ecosystem services including pollination, seed dispersal, and insect moderation (Brigham, 2007; Fenton, 1983; Kalcounis-Rueppell et al., 2007; Wickramasinghe et al., 2003). A sustained, thriving population provides a good representation of ecosystem health (Jenssen, 2006). Many studies report locally decreasing populations worldwide with anthropogenic influences often cited as a likely cause (Clarke et al., 2005a,b; Estrada and Coates-Estrada, 2001a, b; Estrada et al., 1993; Fenton et al., 1992; Hayes and Loeb, 2007; Kunz et al., 2007; Medellín et al., 2000; Moreno and Halffter, 2000, 2001). For example in 2007, Elliot reported that since 1979 the population of Indiana bats (*Myotis sodalis*) in Missouri, US has declined by 95%. Low reproduction rates with slow recovery of population losses make any decrease in bat population a concern (Barclay et al., 2004; Clark et al., 1998; Jones et al., 2009). In recent years, the ecological value of bat conservation has been further amplified by the threat to the species from white-nose syndrome, an important novel pathogen (Warnecke et al., 2012) which has resulted in mass mortality and population decline in North America (Frick et al., 2010) with significant impacts on ecosystem integrity. Consequently, it has become even more crucial to identify possible threats to bat populations to ensure the survival of these vital ecosystem members.

Exposure to organic contaminants, often pesticides, has been identified as one possible cause of declining bat populations (Braaksma and Van der Drift, 1972; Clark, 1981; Clark et al., 1978a,b; Dalquest, 1953; Humphry and Cope, 1976; Mitchell-Jones et al., 1989; Ransome, 1989; Stebbings and Griffiths, 1986) and also their susceptibility to white-nose syndrome (Kannan et al., 1995). Hundreds of different organic chemicals are and have been used by society. Bat exposure can occur through a range of different routes but exposure to pesticides can be especially significant because they are typically applied in agricultural areas near dusk or dawn coinciding with times of increased bat mobility, and target the insectivorous diets of many species. Exposure to residues of the now banned persistent organochlorine (OC) pesticides still remaining in the environment (Clark, 1988), and other more recently used persistent organic compounds such as polybrominated diphenyl ethers (PBDEs) present as a result of intentional or accidental releases, is also possible (Kannan et al., 2010). Many bat species use house roof spaces for breeding and can be vulnerable to exposure from chemicals in wood preservatives including lindane, pentachlorophenol (PCP) and more recently the pyrethroids (Bennett and Thies, 2007; Boyd and Myhill, 1988; Mitchell-Jones et al., 1989; Racey and Swift, 1986; Shore et al., 1990, 1991).

Risks associated with exposure to persistent compounds include food chain transfers and bioaccumulation. Exposure to these and other organic chemicals can manifest in acute and lethal impacts, but sub-lethal and chronic effects such as immune suppression are also of concern for the long-term survival of populations (Clark and Shore, 2001; Corraoa et al., 1985; Geluso et al., 1976; Reidinger and Cockrum, 1978; Warnecke et al., 2012). Many organic compounds, including certain PCBs, OC pesticides and brominated flame retardants, possess the ability to inhibit, interfere with or disrupt the action of the endocrine system (O'Shea and Clark, 2002). These compounds have been implicated as the cause of increasing incidence of reproductive disorders and abnormal developmental in some organisms (Shore and Rattner, 2001) and may contribute to decreased bat population density and survival resilience.

Effects of contaminants on bats, however, are little understood, particularly the sub-lethal effects, due often to difficulties in sampling populations, monitoring exposures and relating exposure to effect. Clark and Shore (2001) reviewed data on organic contaminant residue concentrations (mainly OC pesticides and PCBs) in bats available at that time (pre 1997) and any evidence for effects. The aim of our review is to revisit the issue now, more than three decades after many of the organochlorines were banned, building on the work of Clark and Shore (2001). We examine temporal trends in organic contaminant concentrations in bats, and assess whether new issues are emerging and the longevity of the organochlorine contaminant legacy. We also examine the new knowledge for associated lethal and sub lethal impacts to fully understand potential threats to populations now and where to prioritize research.

2. Contaminants, concentrations and trends

Studies reporting organic contaminant concentrations in bats, their organs and guano from the 1970s are summarized in chronological order in Table 1. All tissue concentrations are presented as ppm wet weight unless indicated otherwise throughout this manuscript. Data were collected from the primary literature. Most work reported in Table 1 is from the US and, pre-1997, includes many of the studies cited in the review of Clark and Shore (2001) available in the primary literature. This is, however, further supplemented by numerous post-1997 studies reporting a range of compounds in addition to the organochlorine pesticides and PCBs. It is important to understand that organic contaminant studies have historically suffered from difficulties with compound identification, method variability and interferences. The development, however, of more sophisticated instrumentation and analysis in more recent years has allowed detection of a wider range of compounds and more confidence in the results reported in the later studies reported in Table 1.

The compounds detected, their concentrations and any temporal trends have been discussed for individual compound groups below where possible. Many difficulties are presented when trying to statistically assess trends using data available on bats. Spatially studies are very limited with most work reported in the US and studies in other countries often restricted to one report. Data lack consistency and standardization. For example, concentration may be reported as wet, dry or lipid weight, as median or mean, are often presented for a range of different body components, and carcasses may either include whole body parts or some thereof. As a consequence we have only included data for time trend analysis from the US where sufficient data has been published to allow for reasonable statistical analysis. We also include only the body tissue data reported in wet weight. Statistical analyses were carried out using R-software version 3.0.0 (R Core Team, 2013).

2.1. Organochlorine pesticides and PCBs

The OC pesticides, introduced in the 1940s, were used ubiquitously in agriculture for pest control until concerns regarding their now well known persistence and toxicity led to restrictions and bans in the 1970s and 80s. Residues, however, still persist in the environment today and, indeed, use continues in some developing countries (Senthilkumar et al., 2001; Van den Berg et al., 2013). PCBs were widely used as dielectric fluids and coolants until they were banned in 1979, but incidental and accidental releases have resulted in residues still remaining in the environment. Both groups of compounds are included on the Stockholm Convention on Persistent Organic Pollutants (Colles et al., 2008; UNEP, 2009).

Table 1 shows that OC pesticides not only have been reported frequently in bat tissues historically (as also evidenced by Clark and Shore, 2001) but also in recent samples collected in the last 10 years, decades after compound use was restricted (Clark, 1981; Clark and Krynitsky, 1983; Clark and Lamont, 1976; Geluso et al., 1976; Kannan et al., 2010; Thies and McBee, 1994). A range of different OC pesticides have been detected including, DDT, dichlorodiphenyldichloroethane (DDD) and DDE, toxaphene, chlordane (CHLs), dieldrin and endrin (Table 1), but the main and most persistent DDT metabolite, DDE (a potent androgen receptor antagonist; Kelce et al., 1995), occurs most

Table 1
A review of key studies on organic contaminant concentrations and impacts in different bat species in chronological order [in ppm].

Bat species	Contaminant	Mean concentration (ppm)	Maximum concentration (ppm)	DDE/∑DDT	Body part	Country	Impacts	Reference (study year)
Forest bats (n = 11) (*Eptesicus/Vespadelus pumilus*)	DDT, DDD, DDE, dieldrin, lindane, HCB	0.25, ND, 1.82, 4.03, 0.03, 0.06	NR	0.87	Whole body (wet weight)	Australia	NR	Best (1973) (1970–71)
Free-tailed bat (n = 4) (*Taphozous georgianus*)	DDT, DDD, DDE dieldrin, lindane,	ND, ND, 0.06, 0.05, ND		1	Intraperitoneal fat			
Black flying fox (*Pteropus alecto gouldii*)	DDT, DDD, DDE, lindane	0.1, ND, 0.01, 0.1		0.09	Subcutaneous fat			
Five bat species (n = 30)	DDT, DDE, dieldrin	4.6, 9.96, 0.26	28.6, 26.2, 1.4		Liver (wet weight)	UK (East Angelia)	9 bats found dead or badly injured	Jefferies (1972) (1963–1970)
Pipistrelle bat (n = 3) (*Pipistrellus pipistrellus*)		9.5, 16.9, 3.9	14.9, 29, 7.9		Fat (wet weight)			
Four bat species (n = 7)		2.18, 2.7, 0.24	5.9, 7.3, 0.5		Carcass minus liver (wet weight)			
Big brown bats (n = 11) (*Eptesicus fuscus*)	DDE, DDT, Oxy, PCB (Aroclor 1260)	2.55, 0.23, 0.35, 1.96	3.3, 0.41, 0.6, 3.6		Carcasses (wet weight)	US	NR	Clark and Lamont (1976) (1974)
Free-tailed bats (n = 8) (*Tadarida brasiliensis*)	DDE, DDD, DDT, dieldrin	Median (3.7, ND, ND, 0.02)	Median (17, 0.03, 0.03, 0.03)		Brain	US	Symptoms of poisoning after fat mobilization during migration, decline in bat populations	Geluso et al. (1976) (1976)
		Median (92, 0.21, 0.45, 0.19)	Median (300, 0.36, 0.91, 0.41)	0.99	Carcasses			
Pallid bat (n = 4) (*Antrozous pallidus*)	*p,p′*-DDE, *p,p′*-DDD, *p,p′*-DDT	Median (5.8, 0.1, 1.8)	8.2, 0.3, 3.8	0.75	Carcasses (wet weight)	US, collected from cities	NR	Reidinger (1976) (1976)
E. fuscus (n = 5)		Median (120, 0.5, 1)	160, 0.5, 1.8	0.98				
T. brasiliensis (n = 5)		Median (25, 23, 120)	38, 68, 550	0.14			3 of the five died in or near human dwellings	
Long-nosed bat (n = 5) (*Leptonycteris sanborni*)		Median (0.2, <DL, 0.2)	0.5, 0.1, 0.7	0.50		US, collected from caves	NR	
Leaf-nosed bat (n = 2) (*Macrotus waterhousii*)		Median (1.8, <DL, 0.2)	3, <DL, 0.4	0.90				
E. fuscus (n = 6)		Median (5, <DL, 0.2)	9.6, 0.4, 0.6	0.96				
Pipistrellus hesperus (n = 6)		Median (1.4, 0.1, 0.1)	4.4, 0.1, 0.4	0.87				
T. brasiliensis (n = 17)		Median (3.3, ND, 0.1)	27, <DL, 0.4	0.97				
6 bat species (n = 50)		Median (4.3, <DL, 0.2)	160, 68, 550	0.95		US		
Gray bat (*Myotis grisescens*)	DDE, DDD, dieldrin, HEPX, t-nonachlor, cis-nonachlor, PCBs	3.4, 1.1, 0.14, 0.41, 0.17, 0.05, 2.0	NR		Guano (dry weight)	US	Bats found dead or dying in caves but no equivocal evidence for bat mortality	Clark et al. (1998) (1976)
	DDE, DDD	2.0, 0.06						Clark et al. (1998) (1985)
	DDE, DDD, endrin, toxaphene, PCBs	25, 4.9, 4.8, 1.1, 3.4			Milk (wet weight)			Clark et al. (1998) (1978)
	DDE, DDD, DDT	16, 2.5, 0.99		0.82				Clark et al. (1998) (1986)
Juvenile (*M. grisescens*)	DDE, DDD, DDT, dieldrin, Oxy, PCBs	9.1, 3.8, 0.28, 0.26, 0.29, 1.8	27, 29, 0.81, 0.68, 0.88, 6.9	0.69	Brain (wet weight)			Clark et al. (1998) (1978)
Juvenile (*M. grisescens*)	DDE, DDD, Oxy, PCBs	7.5, 3.5, 0.17, 1.6	NR					Clark et al. (1998) (1979)
Juvenile (*M. grisescens*)	DDE, DDD, DDT, dieldrin, HEPX, Oxy, t-nonachlor, cis-nonachlor, toxaphene, mirex, PCBs	30, 7.2, 0.75, 0.47, 0.38, 0.67, 0.24, 0.11, 0.19, 0.11, 3.1	31, 8.5, 1.2, 0.61, 0.98, 1.5, 0.6, 0.12, 0.37, 0.12, 3.5	0.79				Clark et al. (1998) (1980)
Juvenile (*M. grisescens*)		24, 9.9, 0.36, 0.45, 0.3, 0.80, 0.47, 0.15, 0.16, 0.11, 9.8	35, 21, 0.53, 0.84, 0.62, 1.5, 0.86, 0.49, 0.29, 0.17, 17	0.70	Carcasses (wet weight)			Clark et al. (1998) (1978)

Table 1 (*continued*)

Bat species	Contaminant	Mean concentration (ppm)	Maximum concentration (ppm)	DDE/∑DDT	Body part	Country	Impacts	Reference (study year)
Juvenile (*M. grisescens*)	DDE, DDD, DDT, dieldrin, HEPX, Oxy, endrin, mirex, PCBs	34, 12, 0.34, 0.64, 0.46, 0.91, 0.24, 0.20, 7.5	63, 47, 1.1, 1.4, 0.85, 1.9, 0.77, 0.36, 14	0.73				Clark et al. (1998) (1979)
Juvenile (*M. grisescens*)	DDE, DDD, DDT, dieldrin, HEPX, Oxy, t-nonachlor, cis-nonachlor, toxaphene, mirex, PCBs	62, 15, 1.1, 0.86, 0.42, 0.99, 0.40, 0.13, 0.62, 0.40, 12	69, 20, 1.1, 0.92, 0.55, 1.8, 0.70, 0.16, 1.9, 0.63, 14	0.79				Clark et al. (1998) (1980)
M. grisescens	*p,p′*-DDE, *o,p′*-DDE, *p,p′*-DDD, *o,p′*-DDD, dieldrin, Oxy, HEPX, t-nonachlor	1.3, 0.05, 0.07, 0.13, 0.10, 0.40, 0.20, 0.13	NR		Carcasses (dry weight)	US	70–80% population decline between 1930 and 1980	Martin (1992) (1990)
Schreiber's bat (n = 20) (*Miniopterus schreibersii*)	*p,p′*-DDE, *β*-HCH, lindane, dieldrin, HEPX, PCBs	9.62, 0.04, 0.05, 0.1, 0.09, 0.66	17.98, 0.18, 0.11, 5.71, 0.68, 1.43		Carcasses (wet weight)	Spain	OC levels are much lower than lethal levels	Hernandez et al. (1993) (1989–1990)
Horseshoe bat (n = 19) (*Rhinolophus ferrumequinum*)		0.06, 0.03, 0.03, 0.02, 0.04, 0.43	0.25, 0.11, 0.08, 0.12, 0.27, 0.94					
T. brasiliensis	*p,p′*-DDE, *p,p′*-DDT, *o,p′*-DDT, *p,p′*-DDD, dieldrin, *β*-HCH, Oxy, HEPX, t-nonachlor	0.51, 0.02, 0.001, 0.001, 0.001, 0.001, 0.006, 0.006, 0.003	0.99, 0.06, 0.01, 0.01, 0.01, 0.01, 0.03, 0.04, 0.01	0.95	Guano (dry weight)	US	NR	Clark et al. (1995) (1991)
T. brasiliensis (n = 18)	*p,p′*-DDE	1.21	2.33		Carcass (wet weight)	US (Vicery Cave), Oklahoma		Thies et al. (1996) (1990–91)
		0.01	0.02		Brain (wet weight)			
		0.5	1.2		Liver (wet weight)			
		0.21	0.4		Kidney (wet weight)			
		0.07	0.17		Spleen (wet weight)			
		6.90	10.48		Carcass (wet weight)	US (Carlsbad Caverns), New Mexico		
		0.01	0.02		Brain (wet weight)			
		1.78	1.89		Liver (wet weight)			
		0.63	0.65		Kidney (wet weight)			
		0.18	0.19		Spleen (wet weight)			
T. brasiliensis (n = 10)	*p,p′*-DDE	2.31, 0.18	NR		Carcasses brain (wet weight)	US	NR	Thies and Thies (1997) (1997)
Cave Myotis (n = 12) (*Myotis velifer*)		0.19 0.09						
Indiana bat (*Myotis sodalis*)	Permethrin	0.84	NR		Carcasses (wet weight)	US	Impaired fly ability at medium dose	McFarland (1998)
	Esfenvalerate	0.19						
T. brasiliensis	Cis-permethrin	0.06	NR		Guano (dry weight)	US	NR	Sandel (1999)
	Trans-permethrin	0.05						
Flying fox (n = 1) (*Pteropus marianus*)	HCHs, DDTs, CHLs HCB, PCBs	0.058, 0.024, 0.0001, 0.0004, 0.008	NR		Whole body (wet weight)	India	NR	Senthilkumar et al. (2001) (1995)
(*P. pipistrellus*) (n = 4)		0.140, 0.39, 0.0014, 0.0034, 0.13	0.15, 0.67, 0.002, 0.005, 0.23					
Juvenile (*P. pipistrellus*) (n = 5)		0.029, 0.016, <DL, <DL, 0.005	0.038, 0.021, <DL, <DL, 0.006					
Adult (*P. pipistrellus*) (n = 2)		0.33, 0.03, 0.0008, 0.0005, 0.13	0.37, 0.045, 0.001, 0.0005, 0.21					
Short-nosed fruit bat (n = 3) (*Cyanopterus sphinx*)		0.062, 0.0037, 0.0002, 0.0013, 0.019	0.15, 0.01, 0.0003, 0.0002, 0.033					
Male (*E. fuscus*) (n = 23)	Dieldrin, DDT, DDE	0.13, 0.04, 1.2	2.7, 0.28, 16.0		Carcasses (wet weight)	US	NR	O'Shea et al. (2001) (1998)
Females (*E. fuscus*) (n = 16)		0.06, 0.02, 0.25	2.6, 0.09, 2.9					
Juvenile (*E. fuscus*) (n = 12)		0.22, 0.04, 1.1	1.4, 0.73, 3.3					

(*continued on next page*)

Table 1 (*continued*)

Bat species	Contaminant	Mean concentration (ppm)	Maximum concentration (ppm)	DDE/∑DDT	Body part	Country	Impacts	Reference (study year)
Males (*E. fuscus*) (n = 23)	Dieldrin, DDE	0.12, 0.23	0.5, 1.6		Brain (wet weight)			
Females (*E. fuscus*) (n = 16)		0.09, 0.10	0.7, 1.4					
Juvenile (*E. fuscus*) (n = 12)		0.08, 0.15	0.23, 0.67					
Juvenile (*E. fuscus*) (n = 6)	Dieldrin, DDT, DDE	0.07, 0.075, 0.03	0.21, 0.1, 0.31		Guano (wet weight)			
E. fuscus	HEPX, Oxy, cis-nonachlor, T-nonachlor, dieldrin	0.62, 0.87, 0.18, 0.6, 0.2	4.6, 4.8, 1.0, 4.10, 1.3		Brain (wet weight)	US	Caused debility or abnormal behavior in three of the bats	Stansley et al. (2001) (1998–99)
Little brown bat (*Myotis lucifugus*)		1.13, 1.93, 0.42, 1.28, 0.38	3.7, 5.03, 1.32, 3.65, 0.93					
Male Southern bent-wing bat (n = 5) (*Miniopterus schreibersii bassanii*)	DDE	0.651	1.94		Liver (wet weight)	Starlight Cave, south-eastern Australia	Stable population	Mispagel et al. (2004) (2002)
Female (*M. s. bassanii*) (n = 5)		0.257	0.40					
Male (*M. s. bassanii*) (n = 5)		0.94	2.02		Muscle (wet weight)			
Female (*M. s. bassanii*) (n = 4)		0.86	1.48					
Male (*M. s. bassanii*) (n = 2)		16.86	24.200		Back-depot fat tissues wet weight)			
Female (*M. s. bassanii*) (n = 2)		ND	ND					
Male (*M. s. bassanii*) (n = 5)		0.076	0.28		Brain (wet weight)			
Female (*M. s. bassanii*) (n = 5)		0.035	0.10					
Male (*M. s. bassanii*) (n = 5)	DDE	0.013	0.033		Liver (wet weight)	Bat Cave, Southest part of South Australia	Population decline	
Female (*M. s. bassanii*) (n = 5)		0.011	0.057					
Male (*M. s. bassanii*) (n = 5)		0.188	0.3		Muscle (wet weight)			
Female (*M. s. bassanii*) (n = 5)		0.151	0.28					
Male (*M. s. bassanii*) (n = 5)		1.44	2.32		Back-depot fat tissues (wet weight)			
Female (*M. s. bassanii*) (n = 3)		0.97	2.27					
Male (*M. s. bassanii*) (n = 5)		<DL	–		Brain (wet weight)			
Female (*M. s. bassanii*) (n = 5)		<DL	–					
M. grisescens (n = 4)	DDE, dieldrin	0.034, 0.013	0.057, 0.014		Guano (dry weight)	US	Fatal impacts	Sasse (2005) (2004)
Eptesicus serotinus (n = 10)	∑DDT, HCB, lindane, PCBs	1.12, 0.01, 0.01, 0.56	4.02, 0.08, 0.03, 3.56		Liver (wet weight)	Austria	Inflammatory process in lungs as signs of immune system disruption in dead or injured bats	Luftl et al. (2005) (1996–2000)
Myotis emarginatus (n = 11)		0.27, 0.018, 0.1, 0.33	2.34, 0.18, 0.72, 1.09					
Myotis mystacinus (n = 26)		0.46, 0.008, 0.038, 1.56	5.83, 0.08, 0.02, 21.58					
Nyctalus noctula (n = 5)		0.84, 0.02, 0.004, 0.46	0.97, 0.09, 0.007, 1.68					
Kuhl's pipistrelle bat (n = 23) (*Pipistrellus kuhlii*)		0.68, 0.01, 0.02, 2.07	4.90, 0.04, 0.07, 9.53					
P. pipistrellus (n = 43)		0.51, 0.12, 0.02, 1.66	8.73, 4.76, 0.13, 10.91					

Table 1 (*continued*)

Bat species	Contaminant	Mean concentration (ppm)	Maximum concentration (ppm)	DDE/∑DDT	Body part	Country	Impacts	Reference (study year)
Horse shoe bat (n = 5) (*Rhinolophus hipposiderus*)		0.47, 0.01, 0.09, 1.93	1.31, 0.07, 0.35, 4.04					
Particolored bat (n = 6) (*Vespertilio murinus*)		0.05, 0.008, 0.01, 0.44	0.11, 0.01, 0.04, 2.04					
Female (*M. s. bassanii*)	*p,p′*-DDE, *p,p′*-DDD, *p,p′*-DDT, DDT, CHLs, HCB, HCHs, HEPX, TCPMe, TCPMOH, PCB	25, 0.15, 0.61, 26, 0.014, 0.005, 0.002, 0.008, 0.0005, 0.009, 0.075	NR	0.97	Carcasses minus wings, fur and liver (lipid weight)	Australia, Starlight Cave, south-eastern of Australia	Stable population	Allinson et al. (2006) (2003)
Male (*M. s. bassanii*)		24, 0.11, 0.46, 25, 0.023, 0.004, 0.002, 0.055, 0.0005, 0.009, 0.11		0.97				
Female (*M. s. bassanii*)		25, 0.16, 0.58, 26, 0.088, 0.054, 0.004, 0.051, 0.0008, 0.02, 0.14		0.97		Australia, Bat Cave, Southest part of South Australia	Population decline was more suspicious to contribute to human disturbance and visitation	
Male (*M. s. bassanii*)		23, 0.3, 0.78, 25, 65, 0.07, 0.004, 0.044, 0.001, 0.02, 0.16		0.95				
M. sodalis	*p,p′*-DDE, dieldrin, HEPX, Oxy, diazinon, methyl parathion	0.19, 0.086, 0.016, 0.14, 0.03, 0.02	0.3, 0.13, 0.14, 0.21, 0.03, 0.02		Carcasses minus brain (wet weight)	US	OPs caused impair echolocation, coordination and response time even with low doses exposure	Eidels and Whitaker (2007) (2000)
	Chloropyrifos, dichlorovos	0.004, 0.023	0.001, 0.086		Guano (wet weight)			
	Chloropyrifos	0.002	0.004		Carcasses (wet weight)			
Myotis septentrionalis	*p,p′*-DDE, dieldrin, HEPX, Oxy, chloropyrifos	0.08, 0.13, 0.16, 0.16, 0.18	0.16, 0.23, 0.11, 0.28, 0.18		Carcasses minus brain (wet weight)			
T. brasiliensis	*p,p′*-DDE	Range (0.001–0.015)	0.015		Guano (DRY weight)	US	Population decline	Bennett and Thies (2007) (1998–99)
Diseased males (*M. lucifugus*) (n = 15)	PCBs, PBB, PBDE, DDT, CHL, HCH, HCB	3.26, 5.97, 0.68, 2.4, 0.28, 0.0009, 0.035	5.58, 23.9, 1.15, 4.0, 0.35, 0.002, 0.067		Fat tissues (lipid weight)	US, New York	NR	Kannan et al. (2010) (2008)
Diseased females (*M. lucifugus*) (n = 13)		2.57, 0.005, 0.62, 2.36, 0.46, 0.001, 0.65	5.78, 0.01, 1.49, 5.34, 1.27, 0.003, 0.12					
Diseased males (*M. lucifugus*) (n = 15)	PFOS, PFDS, ∑PFCs	0.027, 0.009, 0.048	0.046, 0.021, 0.63		Liver (wet weight)	US, Kentucky		
Diseased females (*M. lucifugus*) (n = 13)		0.1, 0.008, 0.13	0.23, 0.013, 0.25					
Reference males (*M. lucifugus*) (n = 5)	PCBs, PBB, PBDE, DDT, CHL, HCH, HCB	8.01, 0.038, 2.04, 1.14, 1.55, 0.0002, 0.05	NR		Fat tissues (lipid weight)			
Reference females (n = 6)		11.7, 035, 8.29, 2.37, 3.39, 0.0007, 1.49						
Reference males (*M. lucifugus*) (n = 5)	PFOS, PFNA, ∑PFCs	0.1, 0.078, 0.21			Liver (wet weight)			
Reference females (*M. lucifugus*) (n = 6) Male (*P. pipistrellus*) Female (*P. pipistrellus*)	PCBs	0.142, 0.035, 0.20 0.33 0.19	NR		Whole body (wet weight)	India		Senthilkumar et al. (2010)
Myotis daubentonii (n = 166)	DBT	6.39	33.34		Fur (dry weight)	Finland		Lilley et al. (2013)

DDT = Dichlorodiphenyltrichloroethane, DDD = Dichlorodiphenyldichloroethane, DDE = Dichlorodiphenyldichloroethylene, HCB = Hexachlorobenzene, PCBs = Poly Chlorinated Biphenyls, HCH = Hexachlorocyclohexane, Oxy = Oxychlordane, HEPX = Heptachlor epoxide, CHLs = Chlordanes, TCPMe = Tris(4-chlorophenyl)methane, TCPMOH = Tris(4-Chlorophenyl)Methanol, PBDEs = Polybrominated diphenyl ethers, PBBs = Polybrominated biphenyls, PFOS = Perfluorooctanyl sulfonate, PFDS = Perfluorodecanesulfonic acid, PFNA = Perfluorononanoic acid, PFCs = Perfluorinated compounds, DBT = Dibutyltin.
ND = not detected, NR = not reported, <DL = below detection limit, n = number of bats.

frequently and at greatest concentrations in the literature reviewed, in both historical and more recent samples. For example, in the 1970s in the US, where most studies have occurred, Geluso et al. (1976) reported median DDE concentrations of 3.7 ppm in brain and 92 ppm in carcasses of free-tailed bats (*Tadarida brasiliensis*), whereas DDD, DDT and dieldrin were not detected in brain tissue, but occurred at a much lower median concentration of ≤0.45 ppm in carcasses. In the same year, Clark and Lamont (1976) also reported a mean of 2.55 ppm DDE in carcasses of big brown bats (*Eptesicus fuscus*) in Maryland, US, compared to ≤0.35 ppm DDT and oxychlordane. Reidinger (1976) reported a median concentration of 4.3 ppm *p,p′*-DDE in fifty bats from six different species (range 0.1–160 ppm) higher than the median for *p,p′*-DDT of 0.2 ppm (range <0.1–550 ppm) and *p,p′*-DDD with a median of <detection limit (range ND–68 ppm). In a 10 year US study by Clark et al. (1998), high residue mean concentrations (62 ppm DDE, 15 ppm DDD) were detected in carcasses of *Myotis grisescens* located close to a Tennessee River site contaminated from a former DDT manufacturing plant. The residues in the bats were significantly higher than those found in carcasses of red-wing blackbirds (*Agelaius phoenicus*) (7.7 ppm DDE, 0.82 ppm DDD) from the same site indicating that the gray bats seemed to be a sensitive indicator of both the level and geographic extent of the contamination in an area (Clark et al., 1998).

In more recent US samples, organochlorine pesticides are still reported and again DDE occurs usually at the highest concentrations. For example, Eidels and Whitaker (2007) tested nine insectivorous bats, five Indiana myotis *M. sodalis* and four northern myotis (*Myotis septentrionalis*), and reported OC pesticides in all samples with *p,p′*-DDE occurring at greatest mean concentration (0.19 ppm) in *M. sodalis* while oxychlordane (Oxy) was present at greatest mean concentration (0.16 ppm) in *M. septentrionalis*. As recently as 2010, Kannan et al. (2010) reported significant concentrations in fat tissues of males and females of little brown bats (*Myotis lucifugus*) (DDT maximum 4.0 and 5.3 ppm; chlordanes maximum 0.35 and 1.27 ppm; hexachlorobenzene (HCB) maximum 0.067 and 0.12 ppm (all concentrations lipid weight)) suffering from white nose syndrome in New York.

Residues and the same pattern of occurrence are reported in countries outside the US with recent samples showing evidence of continuing exposure worldwide. In Spain, for example, Hernandez et al. (1993) reported DDE as the predominant residue detected in carcasses of two bat species (*Miniopterus schreibersii*) (mean concentration 9.62 ppm) and horseshoe bats (*Rhinolophus ferrumequinum*) (0.06 ppm). They also reported β-hexachlorocyclohexane (β-HCH), δ-hexachlorocyclohexane (δ-HCH), dieldrin, and heptachlor epoxide (HEPX) in both species. In India, Senthilkumar et al. (2001) reported hexachlorocyclohexanes (HCH), DDT, CHLs and HCB in three bat species including a flying fox (*Pteropus marianus*), an Indian pipistrelle (*Pipistrellus pipistrellus*) and short-nosed fruit (*Cyanopterus sphinx*) bats collected between 1995 and 1998. HCH and DDT and its metabolites were the most abundant pollutants (Singh et al., 1988). Luftl et al. (2005) reported ∑DDT, HCB and lindane in liver samples of 8 bat species collected from Austria with again ∑DDT at the maximum concentration of 8.73 ppm in *P. pipistrellus*.

Best (1973) in one of the first studies on OC pesticide residues in wildlife in the Northern Territory, Australia, reported that DDT, DDE, dieldrin, lindane and HCB were detected in carcasses of forest bats (*Eptesicus/Vespadelus pumilus*), DDE and lindane in free-tailed bats (*Taphozous georgianus*), and DDT, DDE and lindane in subcutaneous fat samples of frugivorous black flying foxes (*Pteropus alecto gouldii*). Again DDE was present at greatest concentrations. In more recent (2006) carcass samples from Australia, a range of OC pesticides (*p,p′*-DDE 11–59 ppm, *p,p′*-DDD 0.035–0.62 ppm, CHLs 0.007–0.027 ppm, HEPX 0.003–0.023 ppm, HCB 0.001–0.12 ppm, ∑HCHs 0.001–0.009 ppm (concentrations as lipid weight)) were detected in southern bent-wing insectivorous bats (*Miniopterus schreibersii bassanii*) and considered a possible cause of declining populations (Allinson et al., 2006).

The predominance of DDE in the DDT compounds is frequently reported in organism samples (Kelce et al., 1995) and is considered to indicate DDT conversion in tissues and also preferential DDE storage (Aguilar, 1984; Kelce et al., 1995; MacGregor, 1973). The DDE/∑DDT ratio can be used as an index to understand DDT inputs to the environment. This ratio in Beluga whale (*Delphinapterus leucas*) tissues (Borrell and Aguilar, 1987), for example, ranged from 0.3 to 0.8 in the St. Lawrence estuary, Quebec, Canada. The authors suggested that higher values of this index in recent years can be reflective of no new inputs of this pesticide to the environment. This ratio is calculated where possible in Table 1 for the studies on bats reviewed using mean wet weight values listed. Differences in the DDE/∑DDT ratio over different time periods were assessed using *t*-test. The mean ratio was 0.76 over 1970–1990, while it increased to 0.96 over 1991–2006. This is a significant increase ($t = -3.302$, $df = 18.236$, $P < 0.004$) supporting the significant decline in new DDT inputs to the environment over these time periods.

The mean residue concentrations in Table 1 for OC pesticides US (where most of the studies on free bats have occurred) tissue samples (wet weight only) were examined over three specific time periods (1970–1980, 1981–1999 and 2000–2013) to evaluate any time trends. This has had only limited attention to date (Clark et al., 1998) due to the inherent difficulties with bat samples. The problems associated with this analysis in terms of data consistency are discussed previously but the results serve as a first preliminary assessment of the order of concentrations and change in these reported over time for bat tissues. Spatial assessment to rest of the world samples was not viable due to low sample numbers and lack of consistency with data presented. Analysis of variance was used to evaluate the effect of time period and pesticide compounds on residue concentrations reported in bats. Tests for normality and variance homogeneity were carried out and a $\log_{10}$ transformation was applied to the concentration data to stabilize the variances. Significantly different means were separated using 95% confidence intervals (standard error × 1.96), i.e. overlapping errors are not significant (Day and Quinn, 1989).

Results are illustrated in Fig. 1. Concentrations of the seven OC pesticides assessed were significantly different ($F = 13.5$, $df = 6$, $P < 0.001$) at each time period with DDE showing the highest values in each time period. Time period was also significant ($F = 57.1$, $df = 2$, $P < 0.001$), as was the interaction of the compound and time period ($F = 9.8$, $df = 9$, $P < 0.001$). DDD, DDE, DDT and dieldrin show a significant decline from 1970–1980 to 1981–1999 then no significant change to 2000–2013. Of the other compounds assessed HEPX, Oxy and trans-nonachlor (T-nonachlor) showed no significant change between time periods 1970–1980 to 1981–1999, but HEPX and Oxy decreased significantly between 1981–1999 and 2000–2013 (no 2000 data available for T-nonachlor). The data indicate a slow but significant decline in OC pesticide residue concentrations reported in bats, explained in light of the consecutive bans in the US for DDTs in 1972, dieldrin in 1987 and for the heptachlor and chloride parent compounds in 1989 (ATSDR, 1995, 2007). Despite the evidence for reduced exposure, the data in Table 1 and Fig. 1 is clear evidence, however, that exposure does still occur to these long half-life compounds does still occur in countries where they have been banned for many years.

The PCBs have also been reported in many of the studies described above in both US and rest of the world samples (Table 1). For example, Clark et al. (1998) reported PCBs in many samples collected in the US during the 1970s and 80s (e.g. 11.7 ppm in fat tissue), as discussed by Clark and Shore (2001), but, more recently, Kannan et al. (2010) report that PCBs were detected in males and females of *M. lucifugus* in New York (up to 5.8 ppm (lipid weight) in fat tissues). Total PCBs detected in bats in Austria in 2005 reached a maximum concentration of 21.58 ppm in (*Myotis mystacinus*) with a mean of 1.56 ppm (Luftl et al., 2005). In recent Australian samples, a maximum PCB concentration of 0.16 ppm (lipid weight) in carcasses was detected in *M. s. bassanii* (Allinson et al., 2006) and in South India, Senthilkumar et al. (2010)

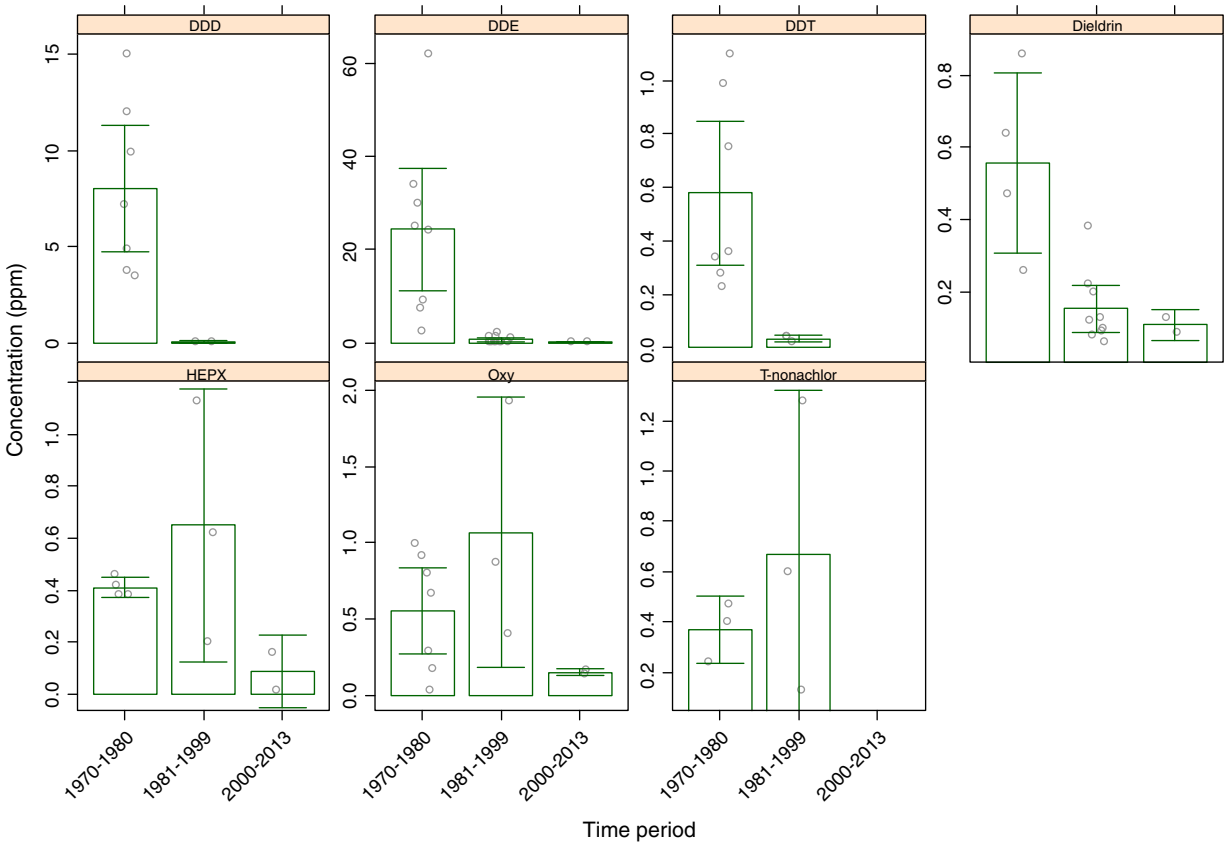

Fig. 1. Mean concentration of seven compounds during three periods of time (1970 to 1980, 1981 to 1999 and 2000 to 2013) in the US. Raw data is shown (○) with means and 95% confidence intervals. No data was available for DDD, DDT and T-nonachlor during 2000 to 2013.

reported the mean total concentration of six PCB congeners in *P. pipistrellus* in the range of 0.19–0.33 ppm. Table 2 summarizes mean PCB concentrations reported in different bat organs including (dry weight, wet weight and lipid weight) over 1970–1980, 1981–1999 and 2000–2013 to illustrate the order of concentrations detected. Statistical analysis of time trends was not possible even in the US due to low sample numbers and data inconsistency. Nevertheless, considering mean wet weights (regardless of country and body organs) a considerable decline in PCBs concentrations seems evident between 1970–1980 and 1981–1999, and between 1981–1999 and 2000–2013. These data again demonstrate the continuing exposure to these persistent compounds worldwide.

2.2. Other pesticides

A number of other studies show evidence that bats are exposed also to a range of other pesticides. McFarland (1998) reported pyrethroid insecticides (used in agricultural pesticides and timber remedial treatments), esfenvalerate and permethrin, in carcasses of *M. lucifugus* from Missouri, US, with permethrin occurring at the greatest concentration (2.5 mean ppm). Sandel (1999) detected cis-permethrin (0.02–0.1 ppm) and trans-permethrin (0.02–0.08 ppm) in guano of *T. brasiliensis* from Missouri, US. Several studies have demonstrated the sub-lethal impacts of pyrethroids to mammals (Baser et al., 2003; Chen et al., 1991; Sutton et al., 2007; Tisch et al., 2002; Vadhana et al., 2010) and Clark and Shore (2001) raised concern about effects in bats.

Eidels and Whitaker (2007) testing nine insectivorous bats in the US (5 *M. sodalis* and 4 *M. septentrionalis*) detected organophosphate pesticides (OPs), chloropyrifos (mean 0.002 ppm), diazinon (mean 0.03 ppm) and methyl parathion (mean 0.02 ppm) in *M. sodalis* carcasses and also chloropyrifos and dichlorovos (mean 0.004, 23.4 ppm, respectively) in guano. Chloropyrifos was also reported (mean 0.18 ppm) in *M. septentrionalis* carcasses. OPs are cholinesterase (ChE) inhibitors (Shore and Douben, 1994) and their adverse impacts on non-target organisms include inhibition of enzyme activity (Bain et al., 2004; Dell'Omo and Shore, 1996), decreasing food consumption (Grue et al., 1997) and reducing reproduction (Sheffield and Lochmiller, 2001).

Allinson et al. (2006), in Australia, reported concentrations of the pesticides tris(4-chlorophenyl) methanol (TCPMOH) (0.009–0.02 ppm) and tris-(4-chlorophenyl) methane (TCPMe) (0.0005–0.001 ppm) (lipid weight) in *M. s. bassanii*. These compounds are used in the production of synthetic high polymers, lightfast dyes for acrylic fibers as well as in agrochemicals (NTP, 2009). According to recent studies their bioaccumulation potential in the marine food chain is comparable to DDT (Falandysz et al., 1999) and they have a similar structure (Minh et al., 2000). The endocrine-disrupting impacts of these compounds have also been well studied (Falandysz et al., 1999; Watanabe et al., 1999) with exposure considered to cause effects in humans and wildlife similar to those observed for DDT (Minh et al., 2000).

As evidenced by the few studies discussed above on pesticides other than the persistent organochlorines, little current data is available elucidating bat exposure and indeed effects for these compounds. In view of the current and widespread use of these pesticides and evident bat exposure and accumulation, additional studies would seem important.

2.3. Polybrominated and polyfluoroalkyl compounds

One recent paper reports on polybrominated biphenyls (PBBs), polybrominated diphenyl ethers (PBDEs) and polyfluoroalkyl compound (PFCs) residues in bats. Kannan et al. (2010) in the US reported PBBs (mean 5.97 ppm) and PBDEs (mean 0.68 ppm) in fat tissues (lipid weight) of diseased male *M. lucifugus* (Table 1) and also PFCs detected in bat livers with perfluorooctanyl sulfonate (PFOS) the predominant

Table 2
The mean concentration of organic compounds (excluding OC pesticides) detected in bats during the years 1970–1980, 1981–1999 and 2000–2013 [in ppm].

Compound	Mean value (1970 to 1980)	Mean value (1981 to 1999)	Mean value (2000 to 2013)	Body part
PCBs	5.15	–	–	Milk, brain, carcasses (wet weight)
	2.00	–	–	Guano (dry weight)
	–	0.24	–	Carcasses and liver (wet weight)
	–	–	0.10	Liver (wet weight)
	–	–	3.25	Carcasses and fat tissues (lipid weight)
Permethrin	–	0.84	–	Carcasses (wet weight)
Cis-permethrin	–	0.06	–	Guano (dry weight)
Trans-permethrin	–	0.05	–	Guano (dry weight)
Esfenvalerate	–	0.19	–	Carcasses (wet weight)
TCPMe	–	–	0.0007	Carcasses (lipid weight)
TCPMOH	–	–	0.01	Carcasses (lipid weight)
Diazinon	–	–	0.003	Carcasses (wet weight)
Methyl parathion	–	–	0.02	Carcasses (wet weight)
Chloropyrifos	–	–	0.004	Guano (wet weight)
	–	–	0.18	Carcasses (wet weight)
Dichlorovos	–	–	0.02	Guano (wet weight)
PBB	–	–	1.51	Fat tissues (lipid weight)
PBDE	–	–	2.90	Fat tissues (lipid weight)
PFOS	–	–	0.09	Liver (wet weight)
PFDS	–	–	0.008	Liver (wet weight)
PFCs	–	–	0.25	Liver (wet weight)
PFNA	–	–	0.056	Liver (wet weight)
DBT	–	–	6.39	Fur (dry weight)

perflourinated compound with a mean concentration of 0.142 ppm wet weight in non-diseased female bats.

Although largely phased out or restricted polybrominated compounds have been incorporated into potentially flammable materials such as plastics, rubbers, textiles and electronics as fire retardants and numerous sources to the environment continue. They are lipophilic, resistant to microbial degradation and are known to bioaccumulate (Hassanin et al., 2004). The PBDEs are thyroid hormone-disrupting compounds, and several laboratories studies on rodents have reported disturbance in thyroid hormone levels with changes in metabolic activity and behavioral anomalies with exposure (Eriksson et al., 2006; Fernie et al., 2008; He et al., 2009). The exposure and continuing accumulation of PBDEs in wildlife manifest in concerns regarding the toxicological impacts and fate of these compounds in the terrestrial ecosystems (Crosse et al., 2012). PFCs are a class of manmade, fluorinated organic compounds that have been used in a variety of consumer and industrial applications for more than 50 years and are known to be persistent and bioaccumulative with numerous current sources to the environment. Toxicity studies in animals report significant reductions in serum cholesterol and/or triglycerides (Seacat et al., 2002; Thibodeaux et al., 2003) and endocrine disruption (Luebker et al., 2005b; Seacat et al., 2002; Thibodeaux et al., 2003) and developmental and reproductive effects with exposure (Lau et al., 2003; Luebker et al., 2005a; Thibodeaux et al., 2003).

In light of the knowledge of toxic effects in mammals, accumulation of these compounds in bat tissues must be recognized as an emerging issue, especially with so little knowledge on residue concentrations and effects.

2.4. Organotin compounds

In the only study reported for bats, Lilley et al. (2013) detected the OTC, tributyltin (TBT) and its metabolite dibutyltin (DBT) in fur of 166 Daubenton's bat (*Myotis daubentonii*) in Finland with 6.39 ppm mean concentration. They reported a highly significant correlation between site sediment TBT concentration and bat fur DBT concentration. Organotin compounds (OTCs) have been used as a component in antifouling paints in marine vessels but have been banned in Industrial Maritime Organization member states since 2008 (Lilley et al., 2013). They are extremely toxic to marine organisms, even at low concentrations (Adema-Hannes and Shenker, 2008; Bao et al., 2011; Chen et al., 2008; McAllister and Kime, 2003), and accumulate in the food chain (Sun et al., 2001). Adverse impacts include ATP production failure, hormone imbalance, impaired hearing and growth and reproductive disorders (Alzieu et al., 1986; Cooke et al., 2004; Gibbs and Bryan, 1986; Nesci et al., 2011; Song et al., 2005; Stenalt et al., 1998).

Table 2 summarizes the mean concentration reported for compounds other than OC pesticides detected in bat tissues between 1981–1999 and 2000–2013. In general studies on compounds other than OC pesticides and PCBs are scarce with most a number of years old. In light of recognized adverse effects for these compounds in mammals, further study of concentrations and effects in bats would seem warranted to fully understand exposure and whether this presents a risk to bat populations now.

3. Issues impacting exposure and accumulation

3.1. Diet, metabolism and foraging habit

Literature reviewed in Table 1 report also on a range of issues that help to understand concentrations reported, exposure and potential effects. Differences in accumulation between different bat species are evident with most understanding gained from OC pesticide studies. Reidinger (1976) reported OC pesticide residues in six south-eastern bat species including long-nosed bats (*Leptonycteris sanborni*), leaf-nosed bats (*Macrotus waterhousii*), pallid bat (*Antrozous pallidus*), *E. fuscus*, *T. brasiliensis*, and western pipistrelle bats (*Pipistrellus hesperus*) in the US. *E. fuscus* collected from the cities had the highest *p,p′*-DDE (120 ppm), *p,p′*-DDD (0.5 ppm) and *p,p′*-DDT (1.0 ppm) median concentrations followed by *T. brasiliensis*. Of the six species sampled, the long-nosed bats *L. sanborni*, which are pollen and nectar feeders, showed lower residue concentrations than the insectivores. Bennett and Thies (2007) also noted that accumulation of persistent compounds was important for the insectivorous bat species, *T. brasiliensis*.

Other studies support this finding. For example, in India, *P. pipistrellus* which feeds on insects, and also fish (Senthilkumar et al., 2001), and is widely found in agricultural lands and domestic areas, showed higher OC pesticide concentrations than the short-nosed fruit bat (*C. sphinx*) and a flying fox (*P. marianus*) (Senthilkumar et al., 2001). Similarly, in Australia, insectivorous forest bats (*Eptesicus/Vespadelus fuscus*) showed higher concentrations of DDT and DDE in the whole body than was detected in fat of fruit bats (*Pteropus. a. gouldii*) (Best, 1973). Insectivorous, higher trophic level, small bat species will be particularly susceptible to

uptake due to high food intake, wide foraging ranges, long life-spans and high metabolic rates (Alleva et al., 2006).

Other factors, however, must also be considered when understanding accumulation. Hernandez et al. (1993) reported higher OC pesticide and PCB residues in *M. schreibersii* than *R. ferrumequinum*, both insectivorous bat species. Lower dietary intake and/or more efficient excretion of OC pesticides in *M. schreibersii* were considered the main reason for the higher concentrations. Higher concentrations of *p,p′*-DDE have been found in brain and carcasses of *T. brasiliensis* compared with cave myotis (*Myotis velifer*), both species having similar diet, activity patterns and roosting behavior (Thies and Thies, 1997). *T. brasiliensis*, however, is a long-range migratory bat with potential for higher exposure. Recent work by Clare et al. (2011) reporting the use of DNA bar-coding to identify bat prey may help to elucidate further dietary factors that influence contaminant exposure of insectivorous species.

Luftl et al. (2005) found significantly (around 5-fold) higher concentrations of lindane (mean 0.1 ppm, maximum 0.72 ppm) in *Myotis emarginatus* liver compared with seven bat other species sampled including *Eptesicus serotinus*, *M. mystacinus*, *Nyctalus noctula*, *Pipistrellus kuhlii*, *P. pipistrellus*, *Phinolophus hipposideros*, *Vespertilio murinus*. They concluded that these bats roosted close to a timber treatment area explaining their higher exposure.

Overall, the literature reports that insectivorous bats show higher contaminant concentrations, but that other factors such as roosting location, foraging habit and bat metabolism significantly influence accumulation. Similar observations have been reported for metal concentrations in bats (Hickey et al., 2001).

3.2. Distribution in body organs and individuals

Work on OC compounds has helped to elucidate distribution of persistent compounds in different bat tissues and relative uptake between different individuals in a population. This has important consequences for the effects of the residue concentrations accumulated. The compounds can appear in a range of bat organs and tissues but concentrations depend on fat content. For example, Shore et al. (1991) exposed pipistrelle bats to PCP and permethrin at 13.11 ± 2.52 μg g^{-1}, both of which are used for timber preservation and treatment. PCP was detected in fat depots, liver, kidneys and in the remainder of the bat carcass. The highest concentration was detected in white fat (median 23.9 ppm), which had the highest lipid content. PCP concentrations in brown fat (median 9.6 ppm) and liver (median 7.3 ppm) were similar, despite the higher lipid content of brown fat. PCP was not detected in bat kidney tissue. On the other hand, permethrin was not detected in the body fur, fat tissues, liver, kidney and carcasses of any bat analyzed. All bats exposed to PCP died within 24 h. Thies et al. (1996) reported for *T. brasiliensis* that the liver contained 2 to 5% of the total *p,p′*-DDE concentrations and the mean concentrations in different organs varied as: liver > kidney > spleen > brain correlating with tissue fat content (Thies et al., 1996).

In *M. s. bassanii*, found in south-eastern South Australia and western Victoria, DDT, DDD and DDE were examined in liver, pectoral muscle, brain and back-depot fat tissues (Mispagel et al., 2004). DDT was detected in only two livers and one back-depot fat tissue (highest concentration 0.126 ppm wet weight in fat tissues). DDD was detected only in brain tissue (highest concentration 0.115 ppm wet weight), but DDE was detected in most tissues (liver, muscle, back-depot fat, brain), with the highest mean concentrations in both male (1.446 ppm) and female (0.978 ppm) (wet weight), in back-depot fat (Mispagel et al., 2004). The mean concentrations of DDE in different organs varied as: back-depot fat tissues > muscle > liver > brain, supporting the earlier findings that persistent compounds usually accumulate in tissues/organs with higher lipid content.

Investigating uptake between different individuals at a site contaminated by past military and industrial uses in Colorado, US, O'Shea et al. (2001), measured DDT, DDE and dieldrin in guano, carcasses, and brains of males (n = 23), females (n = 16), and juvenile (n = 12) of *E. fuscus*. Pesticides were present at the lowest concentrations in female carcasses. Dieldrin levels were highest in juvenile carcasses, but DDE was highest in male carcasses. In the brain samples, the highest concentrations of DDE, DDT, and dieldrin were found in adult males, followed by juveniles and females. The authors concluded that the likely explanation for the consistently lower concentrations in females was that females passed the contaminants to their young through milk during lactation. The concentration in juveniles, however, depended on the juvenile maturity. This is in agreement with the earlier findings of Thies et al. (1996) who found significantly higher concentrations of *p,p′*-DDE in males (n = 12) compared to females (n = 12).

Differences in individual accumulation are not always apparent. For example, Allinson et al. (2006), however, reported no significant differences in pesticide concentrations between males or females of *M. s. bassanii* in Australia except for HCB and Oxy, which were again higher in males. Luftl et al. (2005) reporting OC pesticides and 7 PCB congeners in liver tissues of 16 European bat species found dead or moribund in Austria found no significant difference in concentrations between sexes with the exception of the higher-chlorinated PCBs and lindane, which were higher in juvenile bats. Kannan et al. (2010) examining the concentrations of PCBs, PBDEs, PBBs and OC pesticides in fat tissues of *M. lucifugus* reported no significant difference in the organic contaminant concentration between males (n = 15) and females (n = 13).

Identifying patterns of accumulation obviously suffers from variability associated with the low sample numbers typically available in bat studies. However, males, in some studies, can show higher contaminant concentrations because, it is considered, females pass residues through their milk to the young during nursing. Similarly, higher concentrations reported in juveniles in some studies may result from transfer in milk.

4. Effects of organic contaminants on bats

Exposure to pesticides has been implicated as one of the main factors contributing to bat death and declining populations (Clark, 1981, 1988; Clark et al., 1978a; Kunz et al., 1977; Mitchell-Jones et al., 1989; Ransome, 1989; Stebbings and Griffiths, 1986). Establishing that exposure to organic pollutants is indeed the cause of mortality and population decline is often difficult, however (Clark and Shore, 2001). Exposure not only can lead to lethal and acute effects, but also chronic sub-lethal effects. Acute effects, often leading to early mortality, occur following direct exposure to high concentrations or through secondary poisoning by ingestion of contaminated prey (Berny et al., 1997; Elliot et al., 1996; Mendelssohn and Paz, 1977; Wobeser et al., 2004). Chronic effects occur with smaller doses that do not cause immediate mortality, but can lead to long-term effects such as carcinogenesis, immunotoxicity, endocrine disruption, reproductive failure and altered behavior that may reduce survival (Berny, 2007).

Many authors, as reviewed by Clark and Shore (2001), have reported that OC pesticides can be immediately lethal to bats. Toxicity endpoints reported, however, vary significantly for compound and bat species, and are confounded by the annual cycles of fat deposition. For example, Clark (1988) reported that lethal *p,p′*-DDE concentrations in the brain for *T. brasiliensis* and *M. lucifugus* would be 519 and 603 (ppm wet weight), respectively. Whereas Jefferies (1976) reported mean lethal $\sum$DDT concentration in the brain of *P. pipistrellus* at 95 ppm. Shore et al. (1991) investigating PCP and permethrin toxicity to pipistrelle bats used in remedial timber treatment in England reported that all bats exposed to PCP and PCP/permethrin treated boxes died within 24 and 120 h, respectively. Bats exposed to permethrin treated boxes survived. The authors concluded that PCP was highly toxic to pipistrelle bats at concentrations (13.11 ± 2.52 ppm in carcasses) similar to those used in remedial timber application. On the other hand, the authors acknowledged that the rapid weight loss occurring in first 24 h may have enhanced the toxic effects of PCP. Permethrin had no

significant effect on the survival or the behavior of pipistrelles, although specific physiological effects were not studied. Toxicity endpoints for some of the emerging contaminants reported recently in bat tissues (Table 1), such as the brominated flame retardants have not yet been reported.

Lipid levels have an important influence on the action of highly lipophilic chemicals and must be considered in any valuable assessment of toxicity endpoints (Luckens, 1973). Contaminant accumulation in the fat deposits can prevent lethal concentrations reaching the brain, the most important site of toxic action (Clark and Krynitsky, 1983). With the annual cycles of fat deposition and withdrawal (Krulin and Sealander, 1972) associated with migration and sustained hibernation in many species, however, contaminant release as the fat deposits are metabolized can manifest in lethal outcomes at a stage significantly after exposure. For example, Geluso et al. (1976) reported that exposure to OC residues at sub-lethal concentrations led to lethal concentrations (260–330 ppm) in the brain and significant mortality of juvenile *T. brasiliensis* in New Mexico after the release of DDT from fat deposits during migratory flight. Exposure of nursing pups through the early milk (Laben et al., 1965; Ottoboni and Ferguson, 1969) can also prove fatal during the weaning period and the first migratory flight (McCracken, 1986) which also suggests that exposure is less likely to manifest in effects in late summer and early autumn as fat stores are deposited.

A number of studies implicate bat exposure to OC pesticides and PCBs in reproductive effects (Clark and Shore, 2001). Placental membranes seem to provide only marginal protection for developing embryos against exposure with different compounds showing different propensity for systemic movement (Clark and Lamont, 1976; Thies and McBee, 1994). Again, however, no work is published on this area for other compounds to which bats are obviously being exposed.

Today, when bat exposure is more common at lower, sub-lethal concentrations (based on the data in Table 1), understanding any neurological and physiological effects which may decrease bat survival resilience may be more important for sustaining bat populations in the long-term. For example, Kannan et al. (2010) suspected exposure to sub-lethal concentrations of PCBs, PBDEs, PBBs, OC pesticides and PFCs as a possible reason for *M. lucifugus* suffering from white-nose syndrome in New York State although no significant concentration differences existed between diseased and non-diseased bats. Many of the studies investigating sub-lethal effects again concern the OC pesticides (Clark and Shore, 2001) and report on a relatively limited range of sub-lethal effects and biomarker responses. For example, DDE genotoxicity in *T. brasiliensis* was examined by Thies et al. (1996) using the standard bone marrow chromosomal aberration assay and flow cytometry. No significant differences in chromosal aberrancy or nuclear DNA content variation were found among sexes and sites, but a significant negative relationship was observed between brain DDE concentration and coefficient of variation in DNA content of spleen in males. Esher et al. (1980) observed that both DDT and DDE in the brain of *Pipistrellus subflavus* and *Nycticeius humeralis* inhibited brain ATPase activity in both species. Sub-lethal exposure to lindane on *P. pipistrellus* examined by Swanepoel et al. (1999) showed that metabolic rate significantly increased and was most pronounced in lean individuals potentially leading to energy stress and body mass reduction.

Sub-lethal responses have also been reported in the limited work on other compounds. Amaral et al. (2012) showed that the short-term impacts of exposure on *Artibeus lituratus* to the insecticide spinosad, derived from fermentation of *Sacharopolyspora spinosa*, included decline in hind limb muscle lipid concentration and a significant increase in liver cell diameter. Lilley et al. (2013) investigating DBT effects on general body condition and enzyme activity in *M. daubentonii* reported no significant impact on physiological indicators except lower complement activity (the ability of antibodies and phagocytic cells to clear pathogens) and weaker body immune system. For the organophosphate insecticide (e.g. fenthion, methyl parathion and carbamate) ChE inhibitors, Guillien et al. (1991) observed that the mean brain ChE was decreased significantly 21 days after exposure to fenitrothion in *P. pipistrellus* (residual concentration 0.84 ppm). The authors suggested that risk of trauma in bats as a result of the navigational and foraging ability impairment could be increased and their high energy needs may not be met as a result of the ChE inhibition. O'Shea and Clark (2002) also suggested that inhibition of ChE could result in lower coordination and subsequent disablement in bats.

Effects on many other critical functions are not yet studied. Torpor in bats, for example, is characterized by substantial reductions in body temperature and metabolic rate and appears to be crucial for the long-term survival of many species (Geiser and Brigham, 2000; Turbill and Geiser, 2008). Contaminant effects on torpor are not yet explored. Further, linking any physiological and neurological changes to bat survival and population decline is difficult and also remains unexplored for many contaminants. To conclude, the data available to understand fully sub-lethal contaminant effects on bats is limited to a small range of indicators and contaminants with impacts on bat survival not quantified. Antagonistic and synergic effects of multiple contaminants remain unexplored.

5. Conclusions

This review shows that the OC pesticides and PCBs are detected in bat tissues, frequently, years after compound use was restricted. Time trend analysis indicates that the OC pesticides at least have declined significantly over the 30 years since these compounds were restricted. In recent years, however, it is evident that bats are also exposed to a range of emerging contaminants including the polybrominated flame retardants and perfluorinated compounds. Toxicity endpoints are rarely available, especially for the emerging compounds including PBBs, PBDEs and PFCs. The data available, although limited by small size and high variability, suggests that bats today are exposed generally to lower contaminant concentrations but that these can manifest in a range of sub-lethal neurological and physiological changes that may impact bat survival. Defining concentration endpoints for sub-lethal impacts, especially for the emerging contaminants, and linking these to effects on bat function, behavior or survival, and long term impacts on populations is limited.

Key research challenges include collating a more comprehensive and standardized data base of accumulation concentrations and correlating exposure with effects, both lethal and sub-lethal, including the synergetic and antagonistic effects. These are necessary prerequisites for protection of these critical ecosystem members and the health functioning of the ecosystem they support.

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