Contributing effect of organochlorine exposure on Atlantic bottlenose dolphins during a morbillivirus outbreak

Jennifer Cossaboon,[1] Birgit Puschner,[2] Gina Ylitalo,[3] Deborah Fauquier,[4] and Tracey Goldstein[1]

[1]One Health Institute, [2]Molecular Biosciences, School of Veterinary Medicine, University of California, Davis, CA; [3]Northwest Fisheries Science Center, National Marine Fisheries Service (NMFS), Seattle, WA; [4]Office of Protected Resources, NOAA/NMFS, Silver Spring, MD

Research Question High lifetime organochlorine contaminant exposures → Decreased immune function → Increased viral infection and mortality?

Photo: Marine Mammal Stranding Center, NOAA

Study Objectives

1. Compare ΣPCBs and ΣDDTs in blubber from bottlenose dolphins that died during a dolphin morbillivirus outbreak to assess if exposure correlated with increased susceptibility and death.
2. Compare paired brain and blubber samples to assess if PCB and DDT concentrations in brain tissue are higher, and may be important due to pathological lesions in brain as a result of infection.

Background

Dolphin morbillivirus (DMV; family *Paramyxoviridae*) causes high morbidity and mortality.[1]
- Likely spread via inhalation of respiratory particles.[1]
- Post-mortem lesions include severe pneumonia, encephalitis, and extensive lymphoid tissue depletion.[1]

Over 1,600 Atlantic common bottlenose dolphins died in the 2013-2015 Unusual Mortality Event (UME) along the U.S. East Coast.[2]

Exposure to chemical pollution suspected to increase susceptibility to DMV infection in cetaceans.
- Elevated PCB and DDT concentrations correlated with decreased T lymphocyte responses in an *in vitro* study of blood samples from bottlenose dolphins in Florida.[3]
- Significantly higher mean blubber ΣPCBs in Mediterranean striped dolphins that died during a DMV epizootic (median=778 μg/g lipid) than in blubber biopsies from live-captured individuals (282 μg/g lipid).[4]

Hypothesis

Bottlenose dolphins that died from morbillivirus infection had significantly higher mean ΣPCB and mean ΣDDT blubber concentrations than live bottlenose dolphins that inhabit the same areas and were sampled before the DMV outbreak.

Methods

Stranding networks collected blubber and brain (cerebrum or spinal cord) samples from 15 bottlenose dolphins that died with DMV infection.

To date, tissues from 7 dolphins have been analyzed at the NOAA Northwest Fisheries Science Center (NWFSC) by GC-MS for:

- 51 PCB congeners
- 6 DDT-related compounds
- 15 PBDEs
- 8 chlordanes

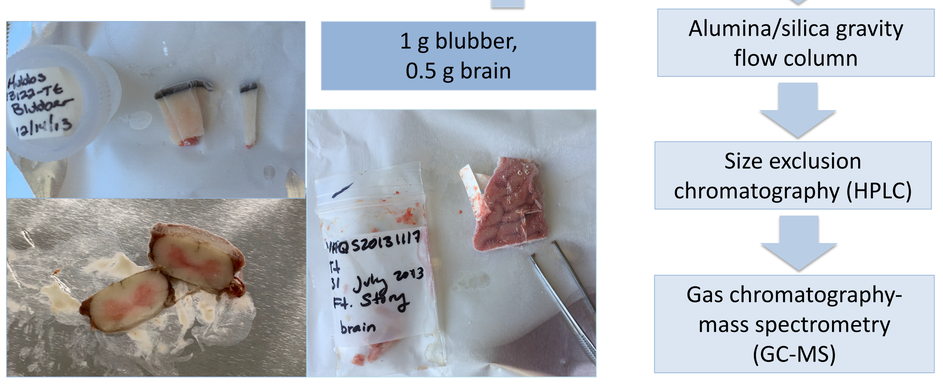

Fig. 1. Flow chart illustrating sample preparation for tissues analysis performed at NWFSC.

Preliminary Results

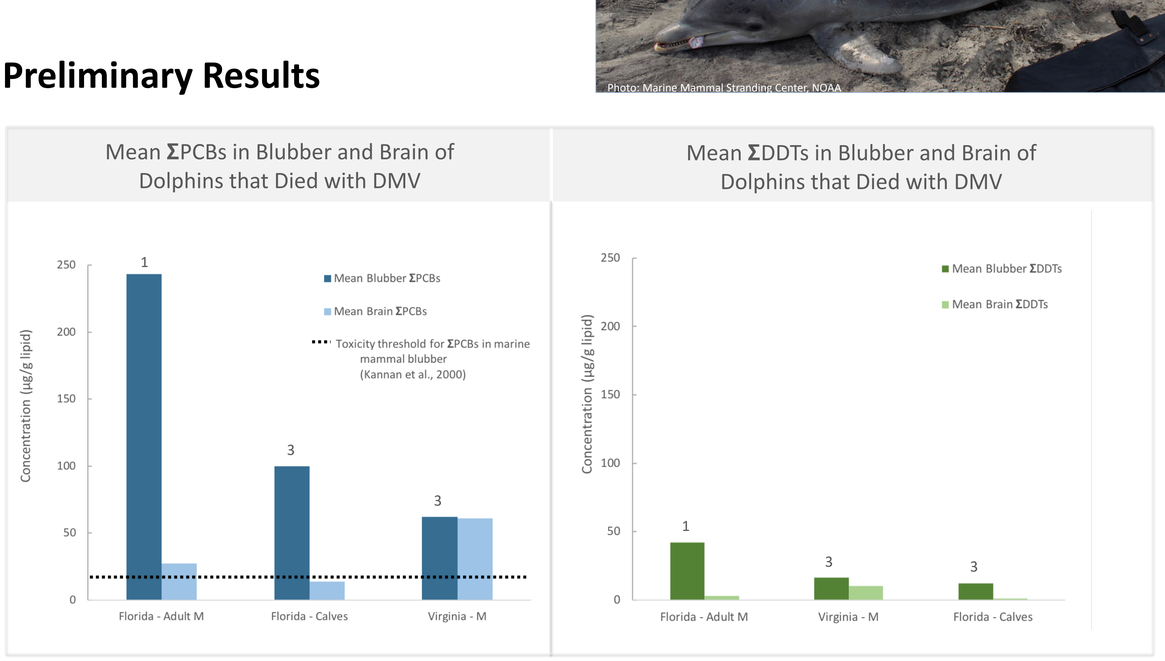

Fig. 2. Mean ΣPCBs and ΣDDTs in blubber and brain of dolphins confirmed to be infected with DMV that stranded during the UME (n=7). Numbers above bars indicate sample size for each group. M = males.

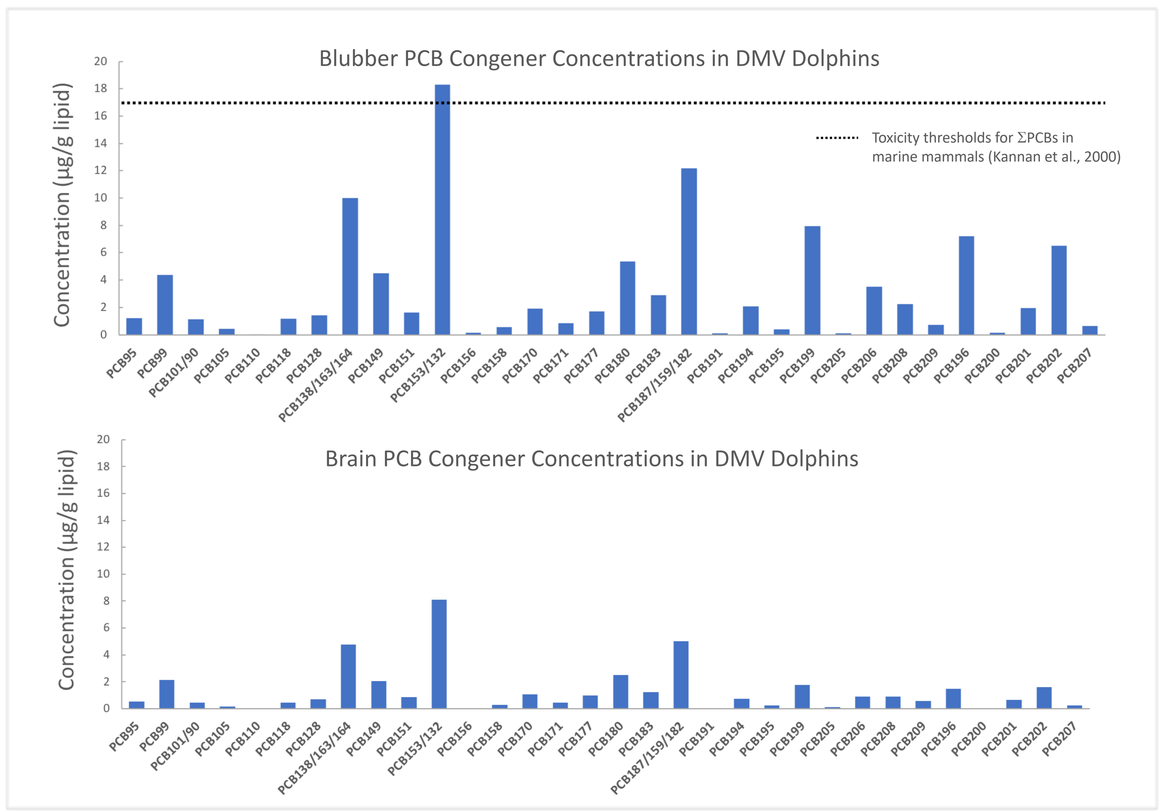

Fig. 3. Mean PCB congener concentrations in blubber (n=7) and brain (n=15) from dolphins infected with DMV that stranded during the UME. PCB 153/132, PCB 187/159/182, and PCB 138/163/164 were detected at the highest concentrations for both tissue types. PCBs below IUPAC no. 95 were excluded due to low concentrations.

ΣPCBs in blubber (n=7) and brain (n=15) of dolphins that died with DMV infection were higher than ΣDDTs across all samples (**Fig. 2**).
- Mean blubber ΣPCBs ranged from 62 – 243 μg/g lipid and ΣDDTs ranged from 12 – 42 μg/g lipid.
- Mean brain ΣPCBs ranged from 14 – 61 μg/g lipid and ΣDDTs ranged from 1 – 10 μg/g lipid.

Mean ΣPCBs and ΣDDTs were higher in blubber than in brain for every dolphin group (**Fig. 2**).

The PCB congener profile was very similar in both blubber and brain samples when adjusted for lipid composition in the two tissue types (**Fig. 3**).
- The most dominant congeners in both blubber and brain samples were:
  1. PCB 153/132
  2. PCB 187/159/182
  3. PCB 138/163/164

Discussion

ΣPCBs in blubber samples (range = 40 – 167 μg/g lipid) were above what are estimated to be toxic threshold values of 17 μg/g lipid (Kannan et al., 2000) for immune impairment in marine mammals.[5]
- The mean blubber PCB 153/132 concentration (18 μg/g) alone exceeds this threshold (**Fig. 3**). PCB 153 is a non-coplanar PCB that has been shown to suppress phagocytosis *in vitro* in bottlenose dolphin immune cells.[6]

Despite the lower concentrations in brain than blubber, the PCB congener profiles were very similar (**Fig. 3**).
- These results indicate that these PCBs are metabolized and cross the blood-brain barrier, with the potential to cause neurotoxicity.[7]

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