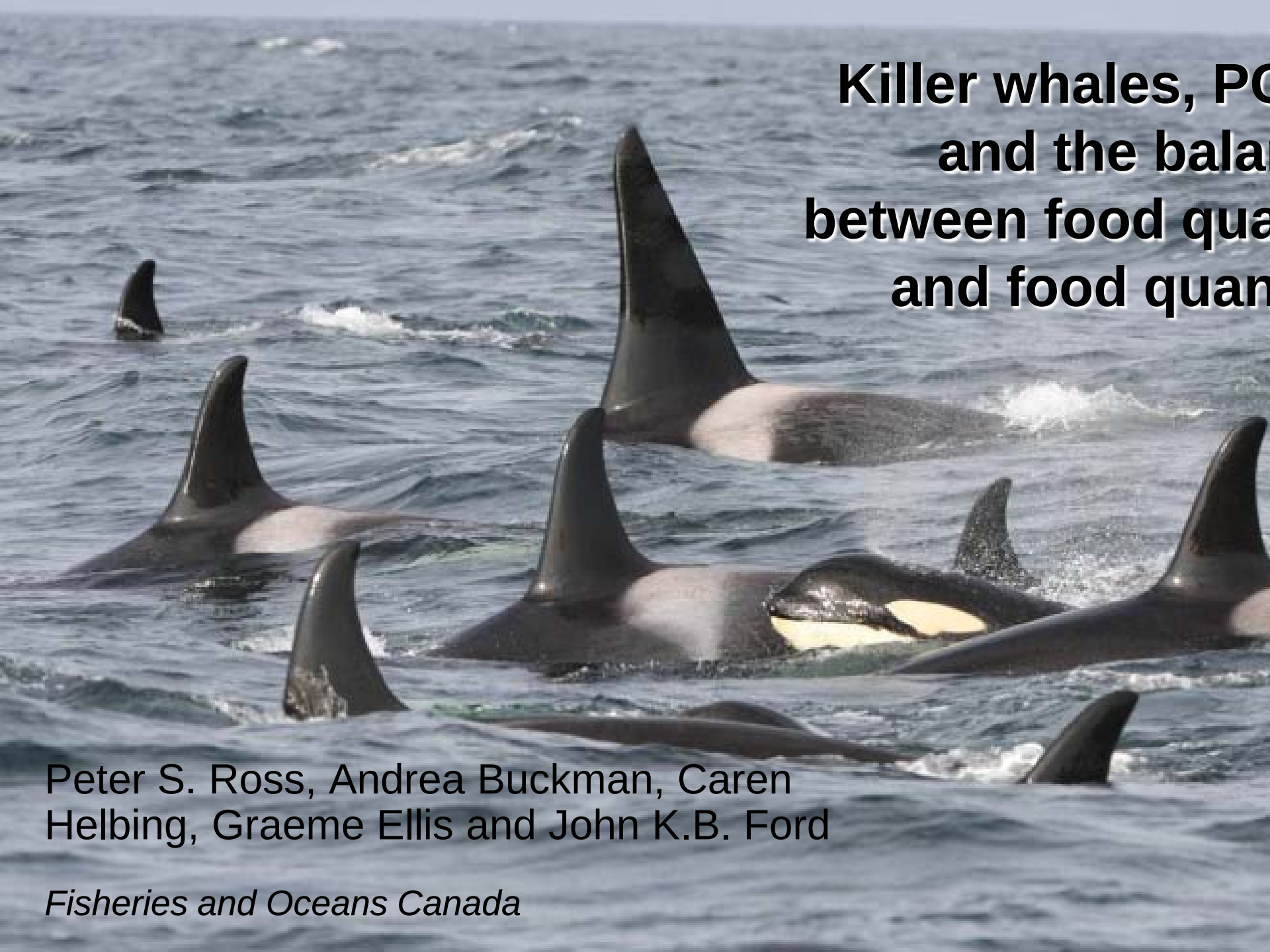
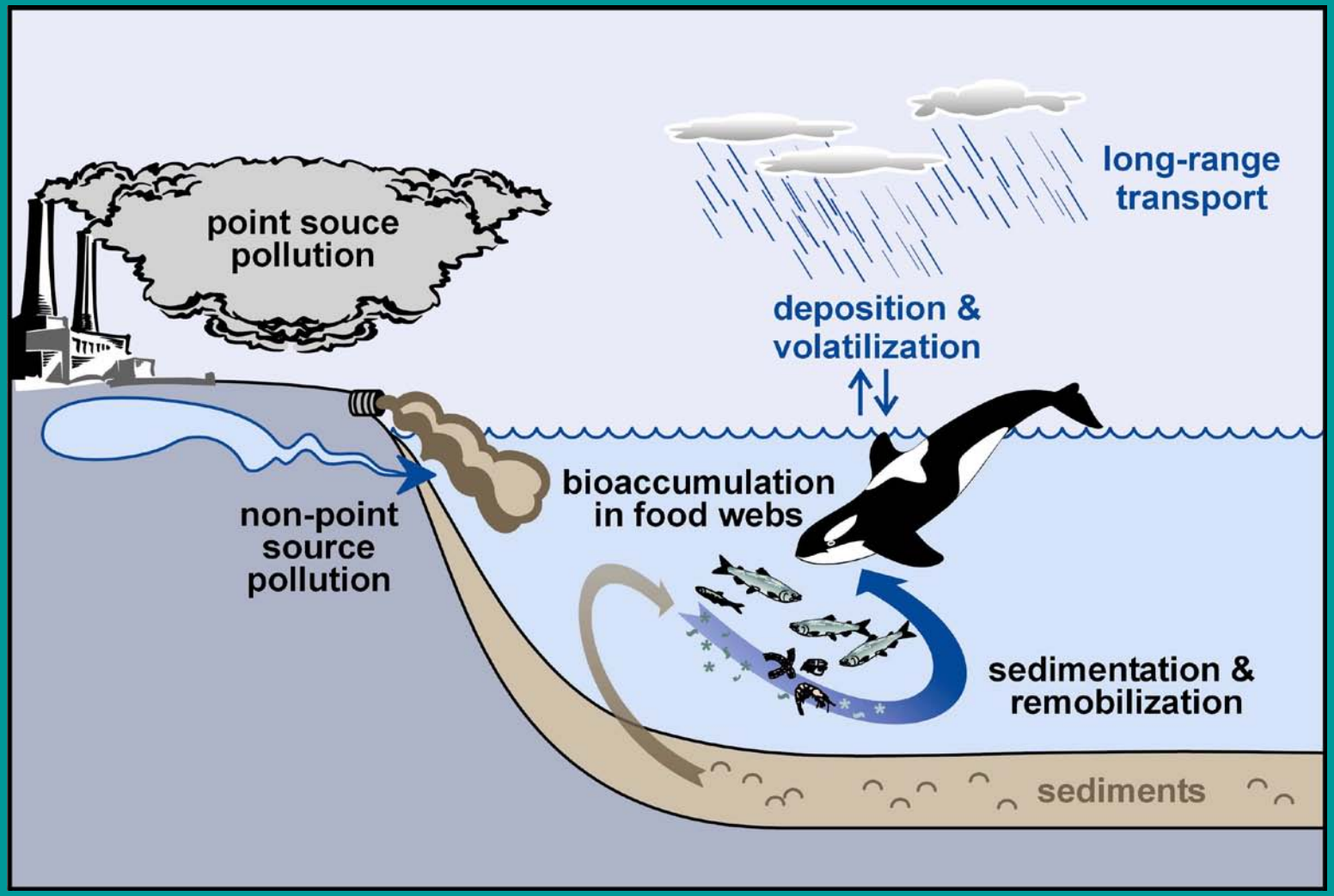


Killer whales, PC
and the bala
between food qua
and food quan

Peter S. Ross, Andrea Buckman, Caren Helbing, Graeme Ellis and John K.B. Ford

Fisheries and Oceans Canada

Food quantity and quality: PCBs and other POPs are fat-soluble, partition into aquatic food webs, and biomagnify



PCBs have been implicated in adverse effects in marine mammals

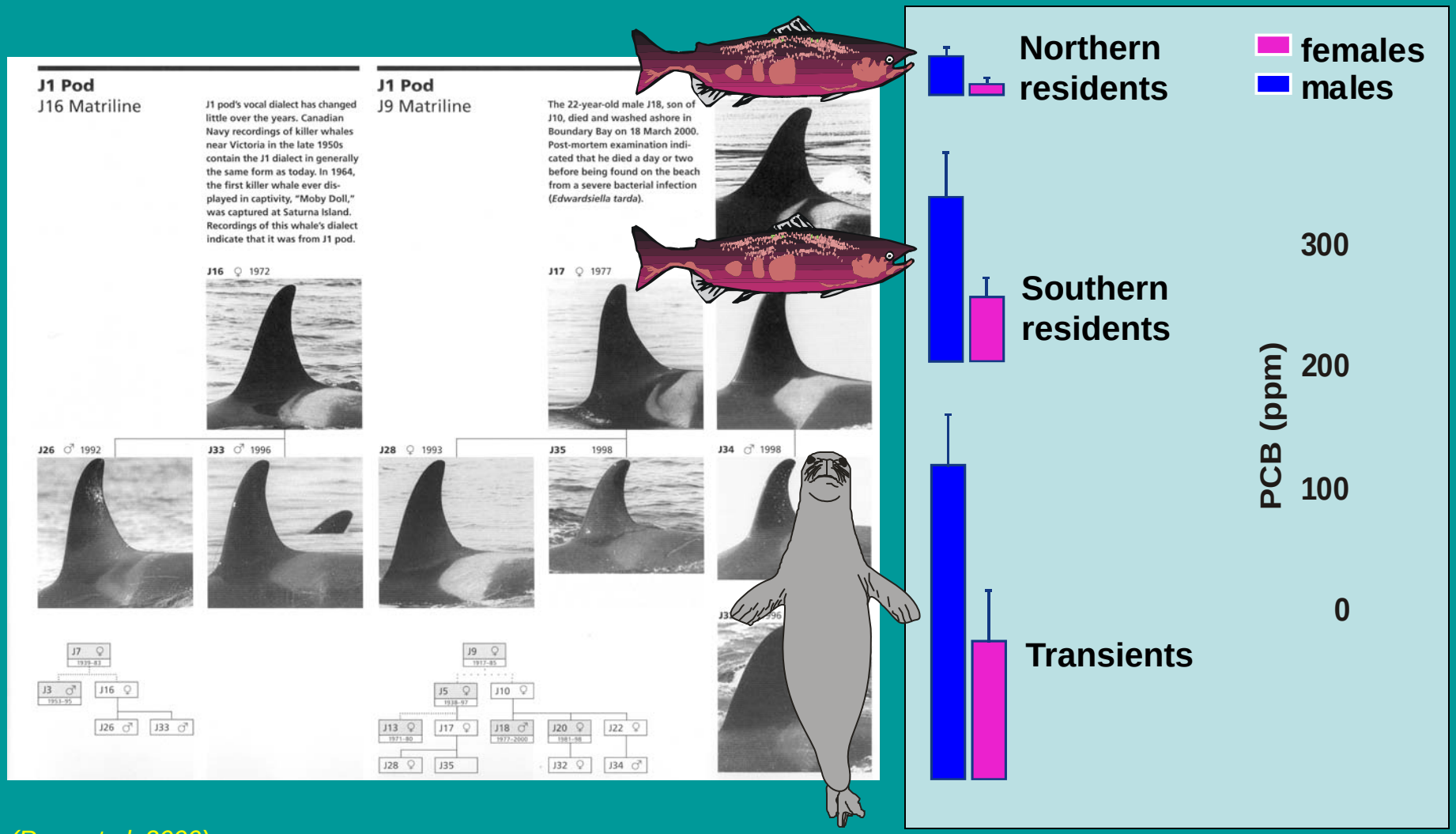
PDV mass mortalities in Europe

- *Seals fed contaminated fish:*
  - decreased reproductive success;
  - immunotoxicity;
  - disruption of vitamin A and thyroid hormone levels.

- *Epidemiological studies:*
  - reduced reproduction associated with PCBs;
  - endocrine disruption associated with PCBs.

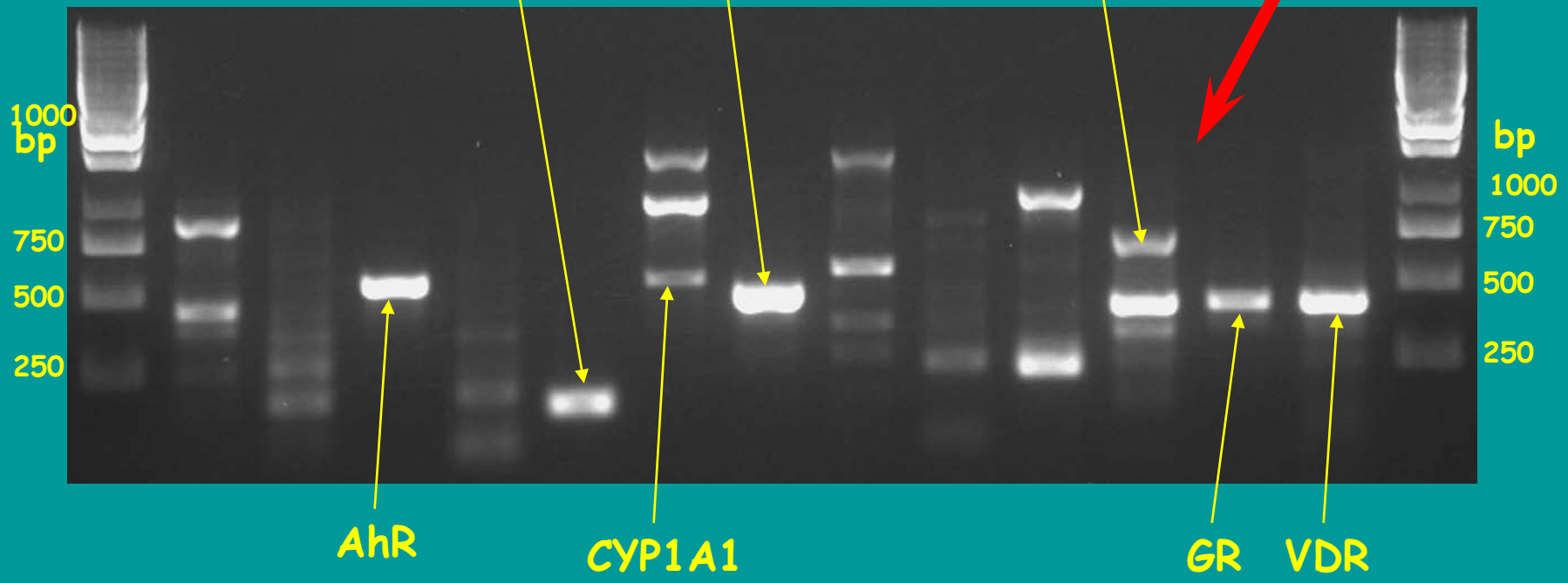
- *Marine mammals inhabiting contaminated regions:*
  - decreased reproductive success;
  - increased incidence of disease.

Killer whales are highly contaminated with legacy PCBs: age, sex and feeding preferences influence accumulation



(Ross et al, 2000)

A new killer whale health toolbox: Real-time QPCR to explore health of killer whales

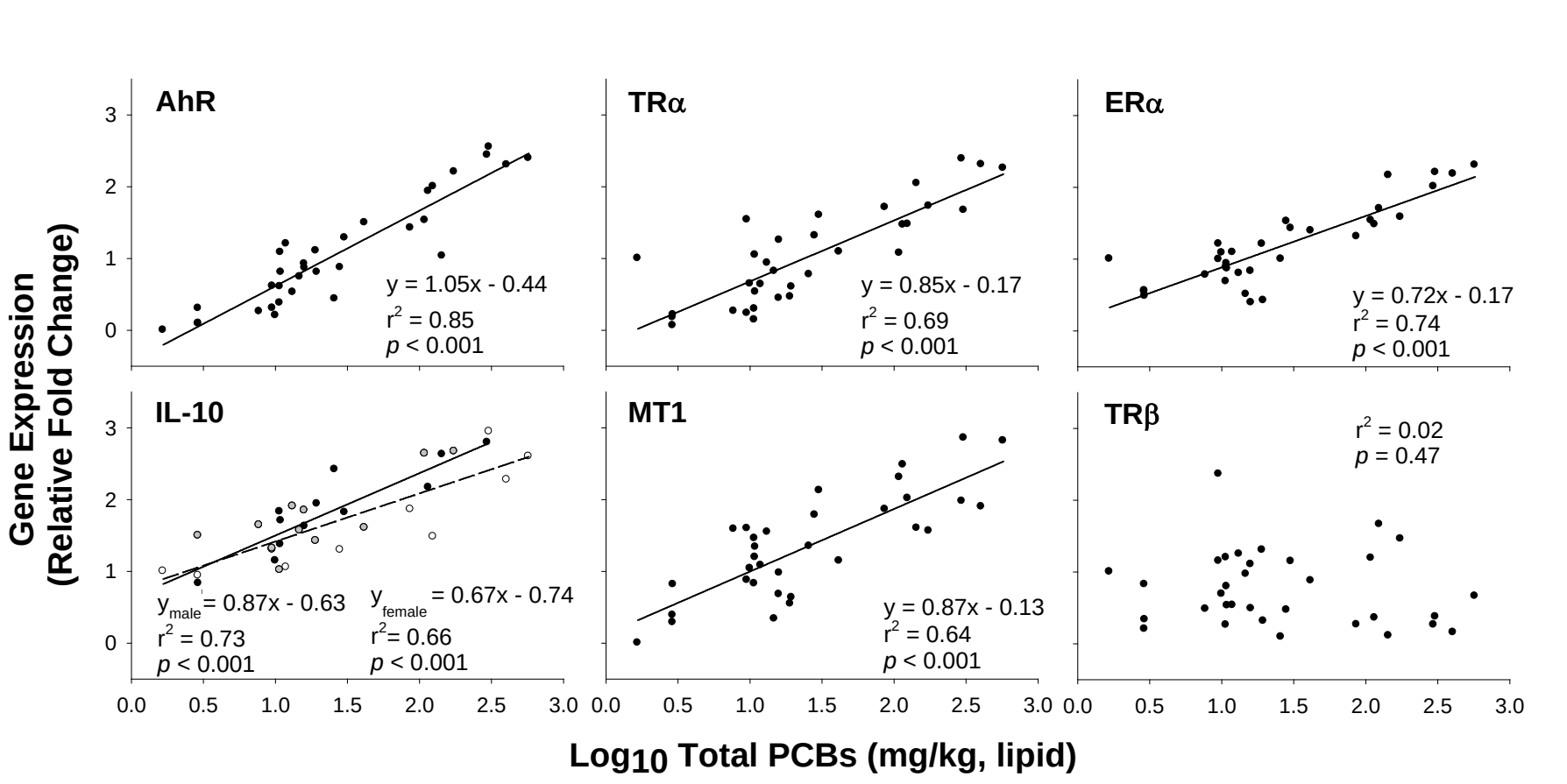


QPCR

- Primer QA
  - Validation of primers using a 3-tiered scheme to evaluate amplification integrity, verify amplicon, and establish suitability for ΔΔCt method
- QPCR on killer whale samples
  - Standardized protocols on blubber and skin samples
- Relative change and normalizer
  - ribosomal protein L8 (RpL8) was normalizer
  - glyceraldehyde-3-phosphate dehydrogenase (GAPDH), and cytoplasmic β actin (CBA) were not suitable since they were not invariant
  - Relative change was calculated by anchoring the data to a single individual

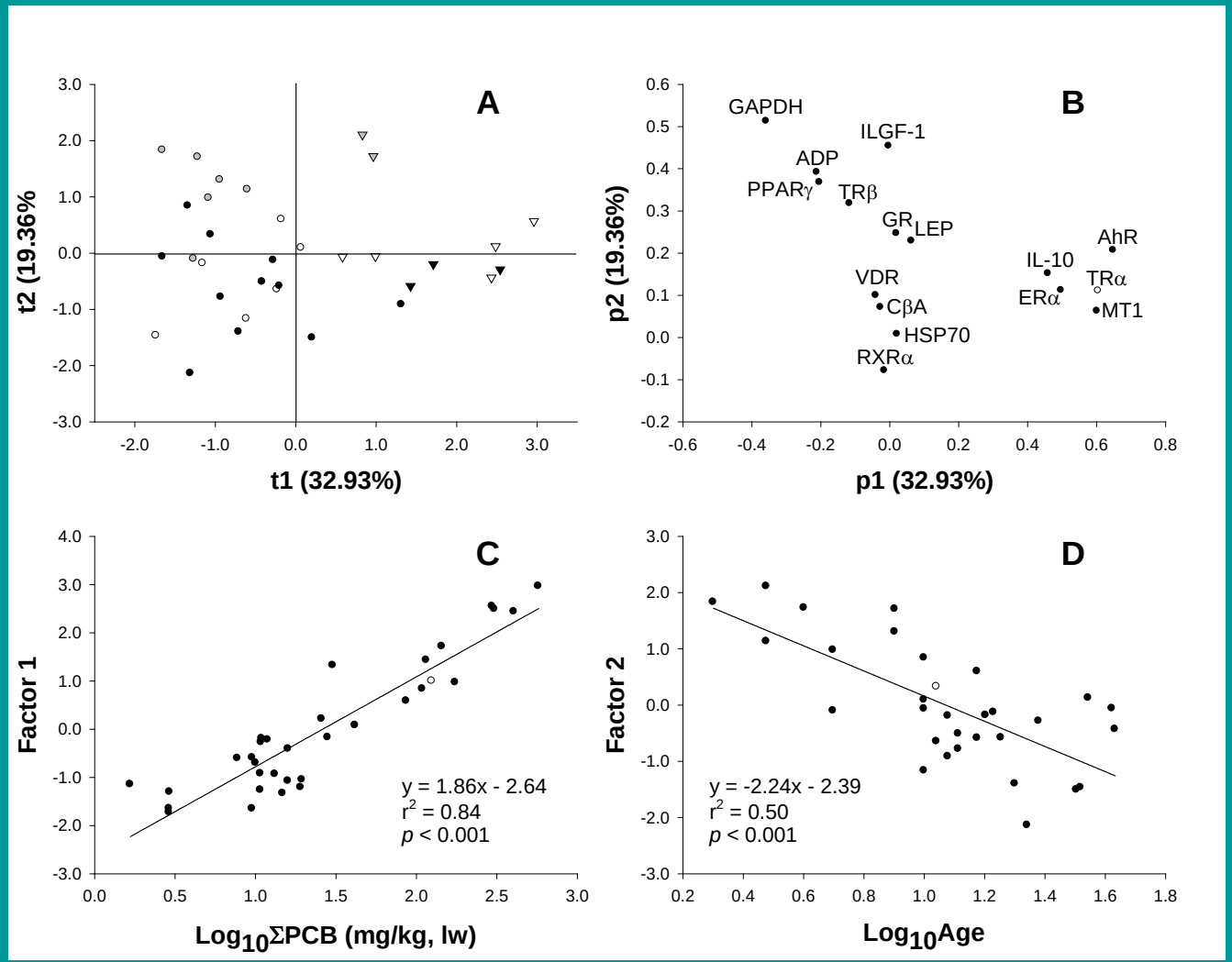
5 out of 11 transcripts were correlated with PCBs:

Aryl hydrocarbon, thyroid, estrogen, intrleukin-10 and metallothionein receptors



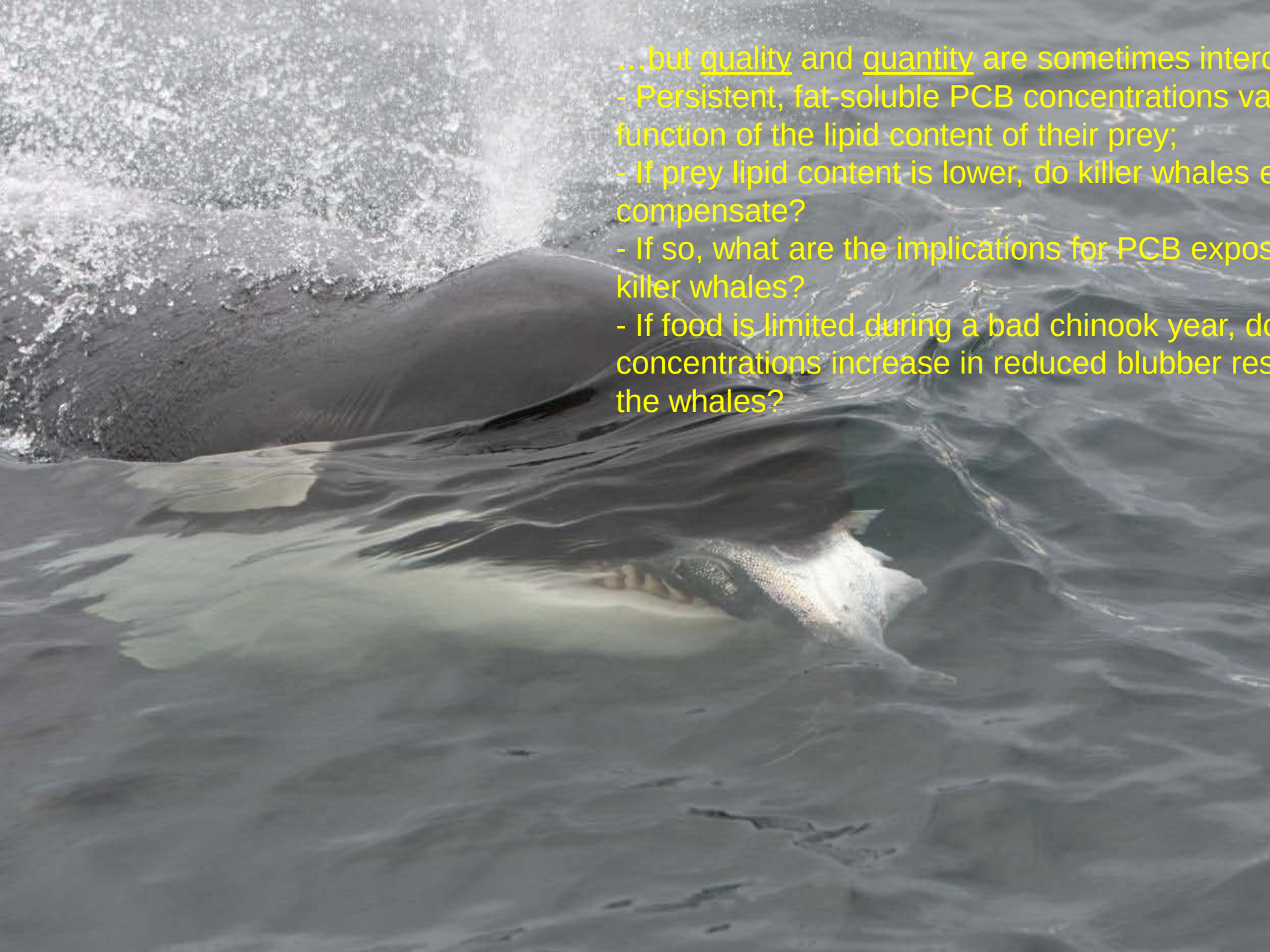
Principal Components Analysis (PCA) implicates PCBs (33%) and age (19%) in gene expression profiles in killer whale biopsies

(Buckman et al, in press)



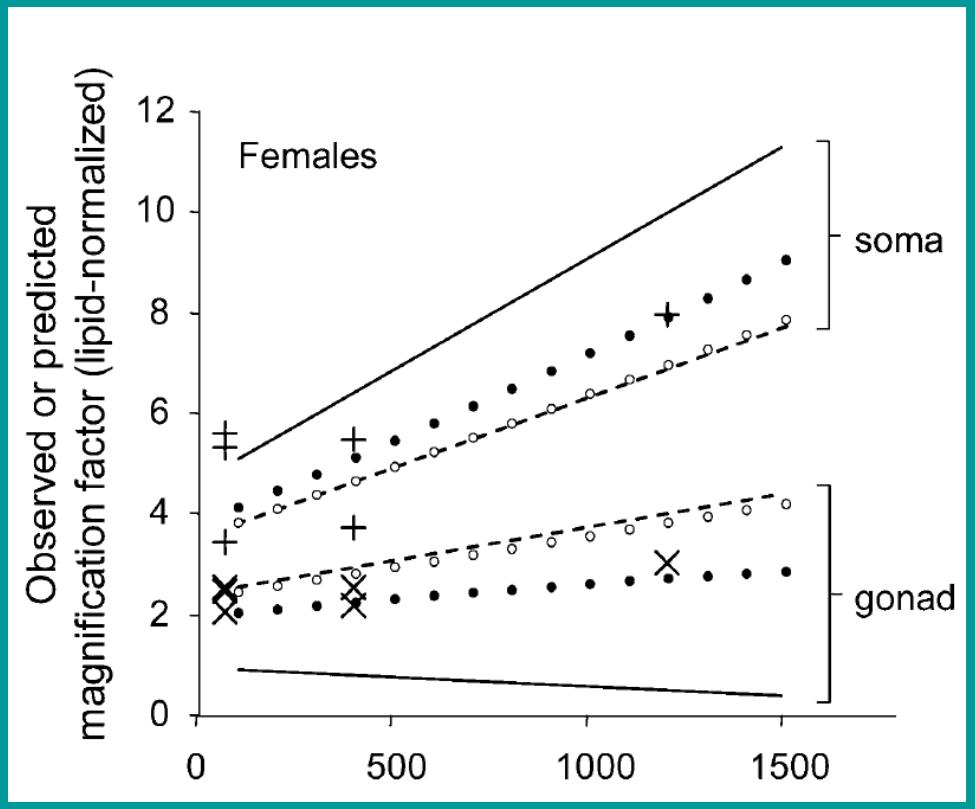
PCBs and killer whale health:

- PCBs are toxic and are disrupting endocrine function at the cell-signalling level;
- The population level consequences are not clear, but a weight of evidence suggests the SRES population to be more ‘vulnerable’ to endocrine-mediated effects (development, reproduction, immune system);
- Since PCBs in killer whales come from their prey, food *quality* is a serious concern…



…but quality and quantity are sometimes interc
- Persistent, fat-soluble PCB concentrations va
function of the lipid content of their prey;
- If prey lipid content is lower, do killer whales e
compensate?
- If so, what are the implications for PCB expos
killer whales?
- If food is limited during a bad chinook year, do
concentrations increase in reduced blubber res
the whales?

As salmon return to natal stream, lipid content dwindles, resulting in a steady increase in PCBs and related compounds in remaining fat



Km migration

(Fraser sockeye PCBs; De Bruyn et al ES&T 2007)

Depending on when (where) these chinook are intercepted, killer whales will consume prey with different levels of lipid.

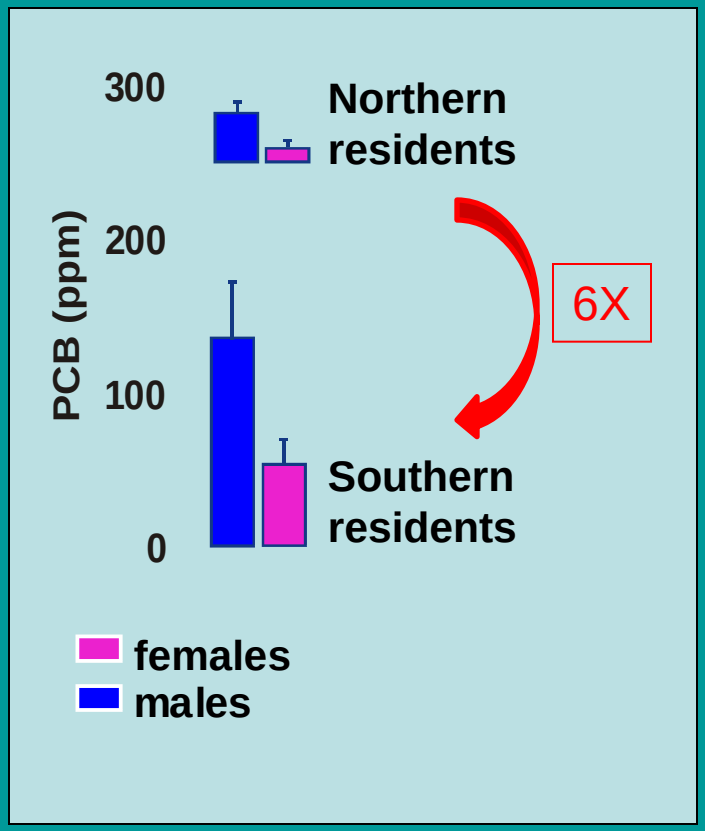
So, if killer whales eat on a lipid basis, they will

1) eat *more* lipid-poor chinook and
2) ingest more PCBs

	Estimated daily intake µg/d (except for ΣPCDD, ΣPCDF, and ΣTEQ ng/d)		
Sum congeners/isomers[a]	Northern residents	Southern residents	Southern residents (lipid-equivalent)
ΣPCB	308.49	1,248.00	2,051.38
ΣPBDE	No data	410.62	674.95
ΣPCDD	19.75	33.67	55.34
ΣPCDF	17.17	47.82	78.60
ΣDDT (DDT, DDD, DDE)	49.72	272.88	448.55
HCH (α-, β-, γ-)	77.76	51.74	85.05
ΣTEQ (PCB, PCDD, PCDF)	9.00	36.85	60.58

(Chinook; Cullon et al., ET&C 2008)

Contaminant data suggest that resident killer whales eat more chinook when lipid content is low; PCB results from biopsies suggest that SRES kw's eat 50% more salmon that their NRES counterparts



Wet weight scenario for daily PCB intakes:

N Residents: 308 ug

S Residents: 1248 ug (4x)

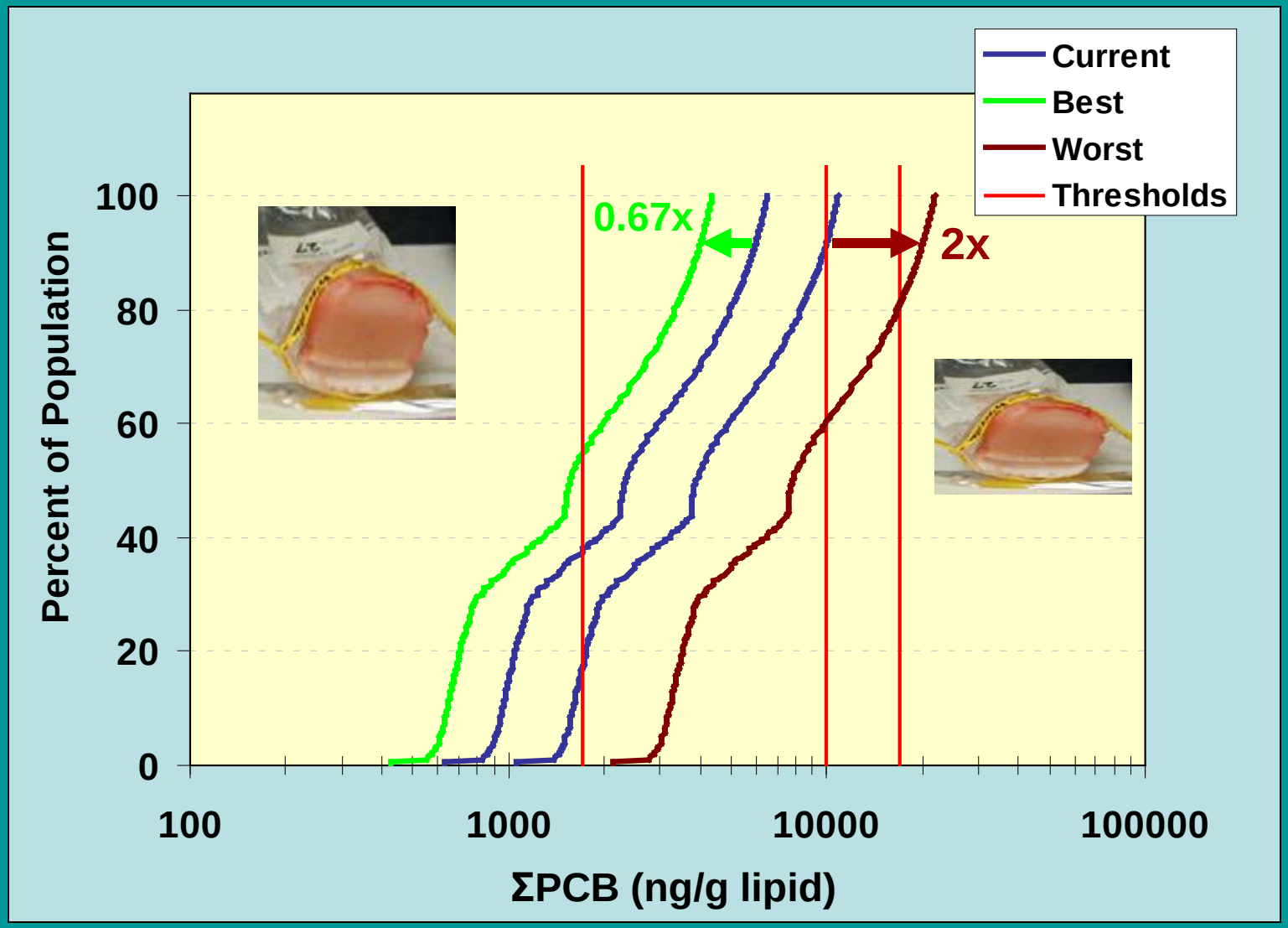
Lipid adjusted scenario for daily PCB intakes:

N Residents: 308 ug

S Residents: 2051 ug (6X)

(Ross et al 2000 MPB; Cullon et al ET&C 2009)

Low chinook years may heighten PCB-related health risks as killer whales lose blubber and mobilize higher concentrations of PCBs (as gauged against three effects thresholds)



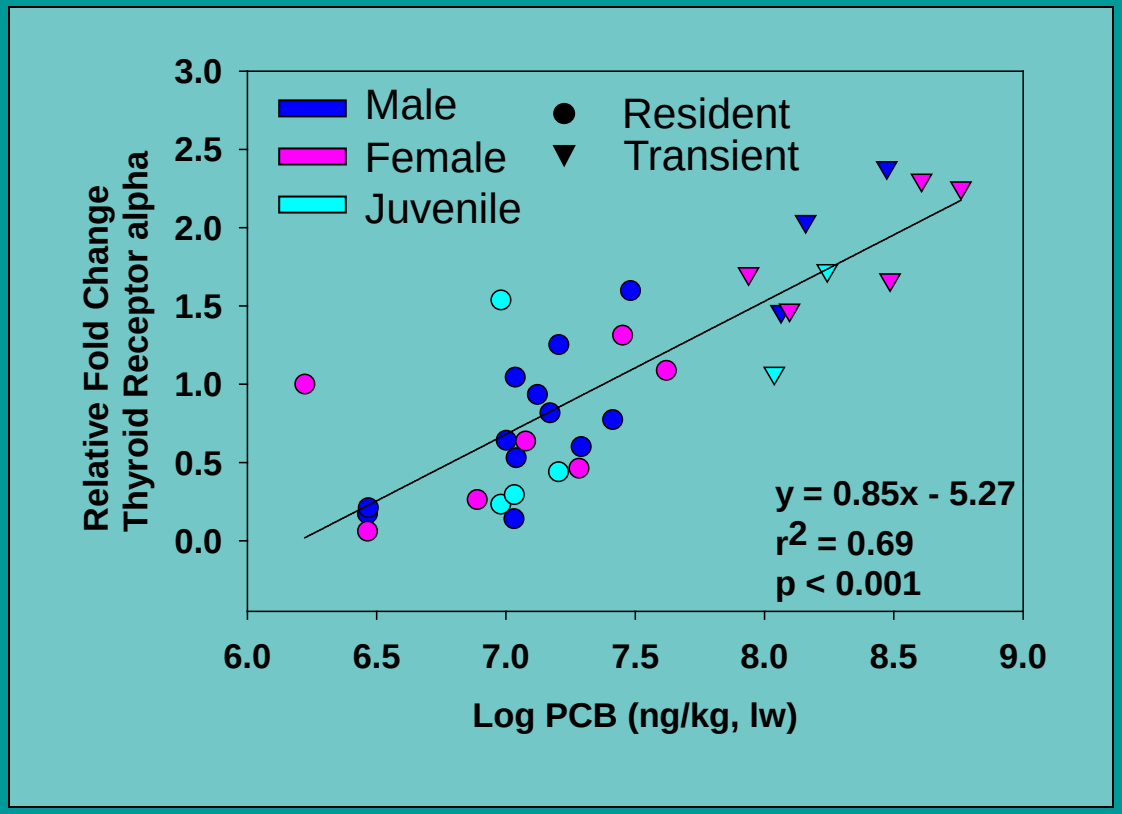
Summary

- Southern resident killer whales are highly PCB-contaminated;
- PCBs come from their prey (Puget Sd chinook have 3-5 x higher PCBs than other west coast chinook; O'Neill et al 2009)
- Age and sex are major determinants of PCB concentrations in killer whales;
- Several important gene expression endpoints are correlated with PCB concentrations in killer whales;
- These PCB-related effects may increase the vulnerability of killer whales to population level consequences (disease, reproduction), especially in the face of additional stressors (disease, climate change, noise);
- Reduced lipid content in both prey and in killer whales will result in higher PCB concentrations in remaining lipid;
- These changes may:
  - increase the delivery of PCBs to killer whales;
  - increase the health risks to the whales themselves.

Thank you

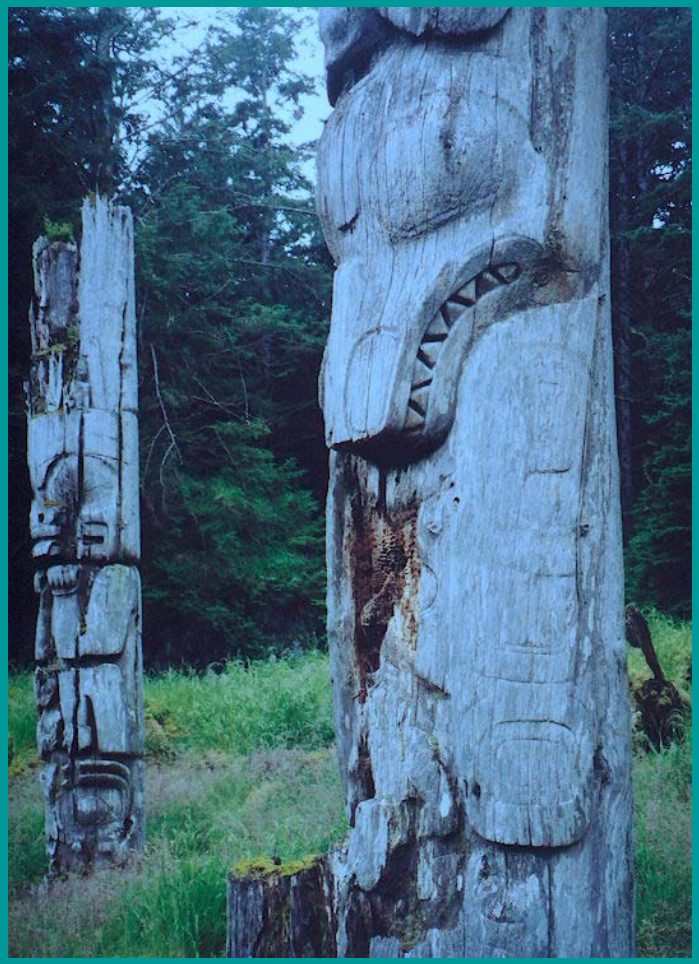
- Species at Risk Act Science Fund (Fisheries and Oceans Canada)
- Graeme Ellis, Neil Dangerfield, Lance Barrett-Lennard, Nik Veldhoen
- Marilyn Joyce, Paul Cottrell, Stephen Raverty
- Photo credits Graeme Ellis, Ingrid Visser, Brian Gisborne, John K.B. Ford, Peter S. Ross

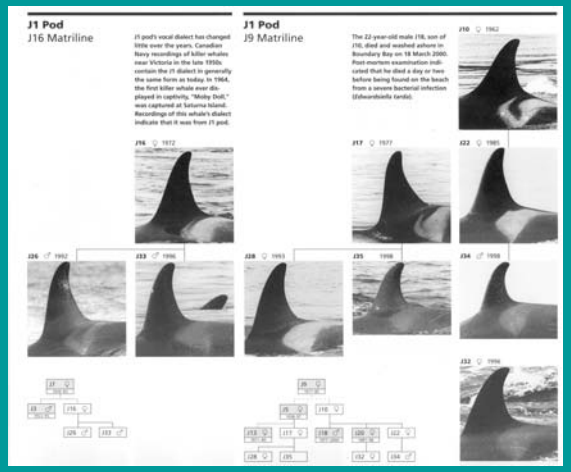
PCBs increase Thyroid Receptor gene expression in killer whales: Results suggest that PCBs increase metabolic turnover



(Buckman et al, 2011 accepted)

Recovery goal: "Ensure the long-term viability of resident killer whale populations by achieving and maintaining demographic conditions that preserve their reproductive potential, genetic variation, and cultural continuity." (Listed 2001)





Know your study animals: age, sex and feeding preferences enabled us to understand and/or rule out confounding factors

Why are killer whales so vulnerable?

- **Long lives (up to 90-100 years);**
- **High trophic levels;**
- **Large habitat needs.**