Organic pollutant release from adipose to blood in response to lipomobilisation in the ewe

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Persistent organic pollutants (POPs) contamination of animal products: A major concern for livestock production systems

Listed by the Stockholm convention *UNEP, 2001*

Anthropogenic origins: pesticides, industrial and combustion products…

Toxicological effects on human health and ecosystem

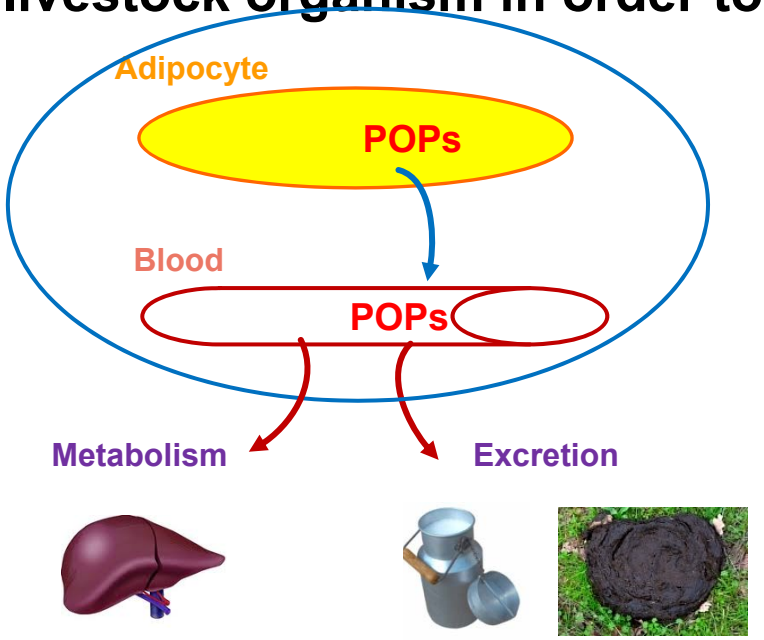
Easily dispersed, poorly metabolized and highly lipophilic:
Persistent in the environment, bioaccumulative in animal fat-rich tissues

Numerous sanitary crises linked to accidental and environmental contamination of livestock

⇒Large economic and social damages

Aims and implications of the study

A need to develop strategies aiming to hasten POPs removal from livestock organism in order to avoid disposal in case of sanitary crisis



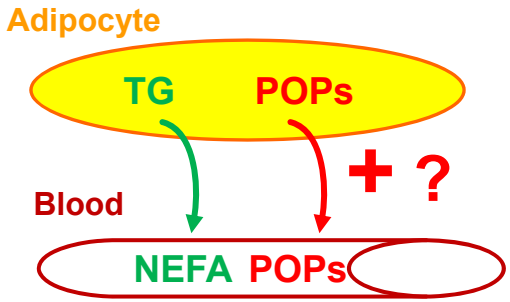
1. Release POPs from storage pool (adipose tissue) to blood

2. Increase POPs hepatic metabolization and/or excretion through feces/milk

POPs: Persistent Organic Pollutants

Aims and implications of the study

Release POPs from their storage pool (adipose tissue) to the blood → **Lipomobilization as inducer of POPs release?**



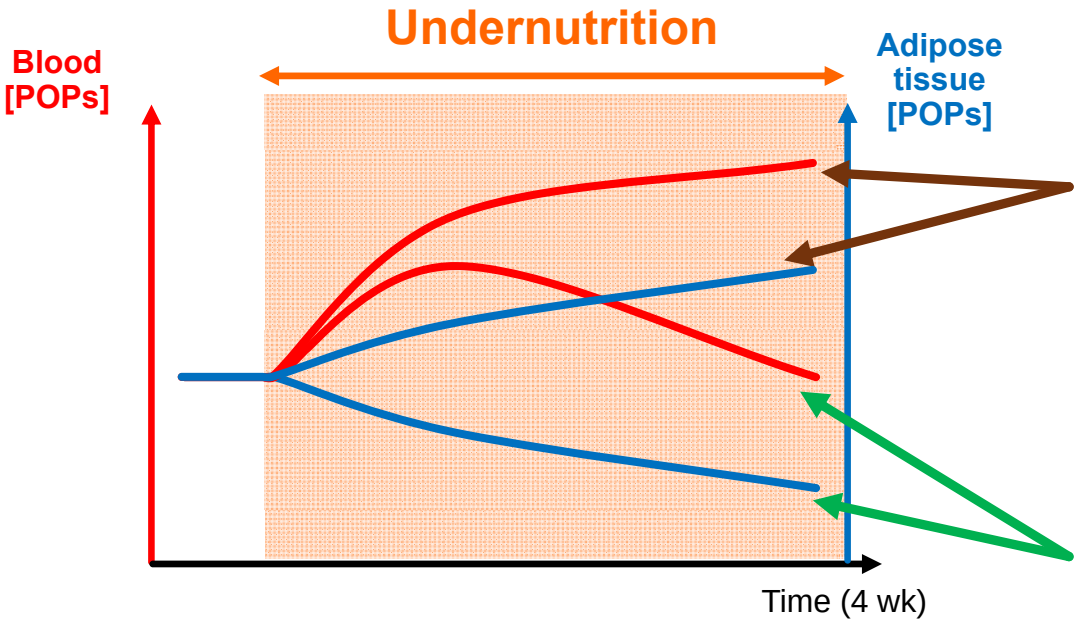
<u>Objective</u>: Test this hypothesis:
Medium-term (1-4 wk) undernutrition

TG: Triglycerides, NEFA: Non-Esterified Fatty Acids, POPs: Persistent Organic Pollutants

Aims and implications of the study

Objective: **Lipomobilization as inducer of POPs release:**

Hypotheses:



POPs are unable to leave the organism (metabolism/excretion)

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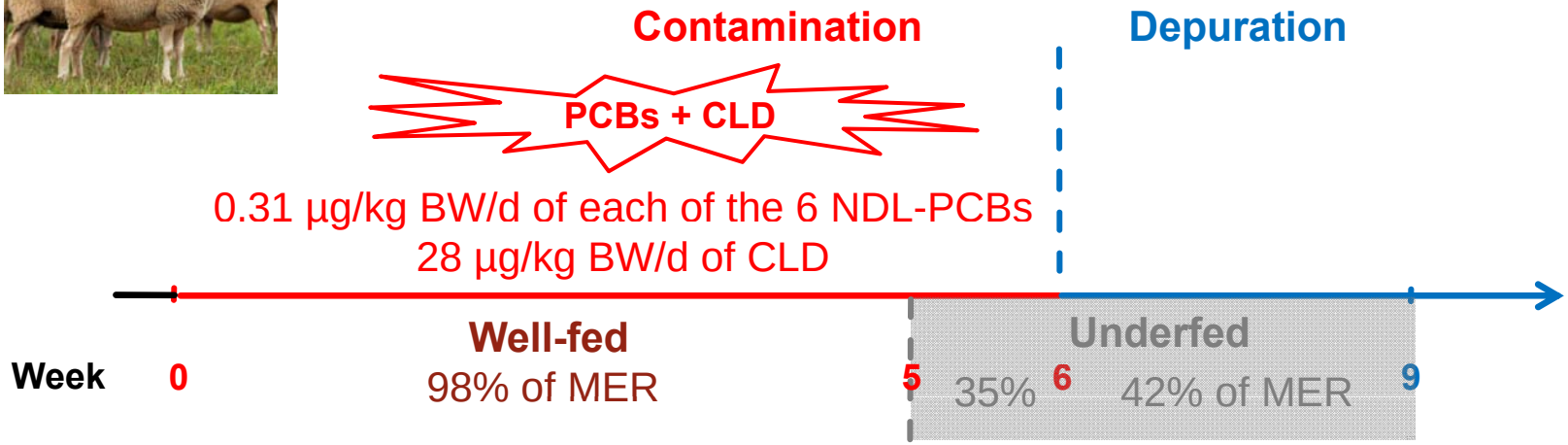
Experimental design

Non-lactating and non-pregnant Romane ewes (*n* = 3)

BW 71 +/-5 kg; BCS 3,4 +/- 0,5

Hay/straw *sub-ad libitum* based diets

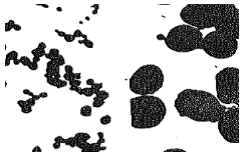


MER: Maintenance Energy Requirements, BW: Body Weight, BCS: Body Condition Score, NDL-PCBs: Non-Dioxin Like Polychlorinated Biphenyls, CLD: Chlordecone

Materials and Methods

Sampling, measurements and analyses

➢ Animal performances and body fatness



•Intakes - daily
•Body weight and nutritional balances - weekly
•Body condition score - every 2 weeks
•Pericaudal subcutaneous adipocyte cellularity – 3-biopsies at the end of each period (i.e., contamination well-fed and underfed and depuration underfed)

➢**Plasma non-esterified fatty acids** (kit)

➢**Serum NDL-PCBs and CLD** (GC-ECD)

7-times longitudinally every 3 to 7 days

➢**Subcutaneous adipose tissue NDL-PCBs** (GC-MS) **and CLD** (LC-MS/MS)

3-biopsies at the end of each period

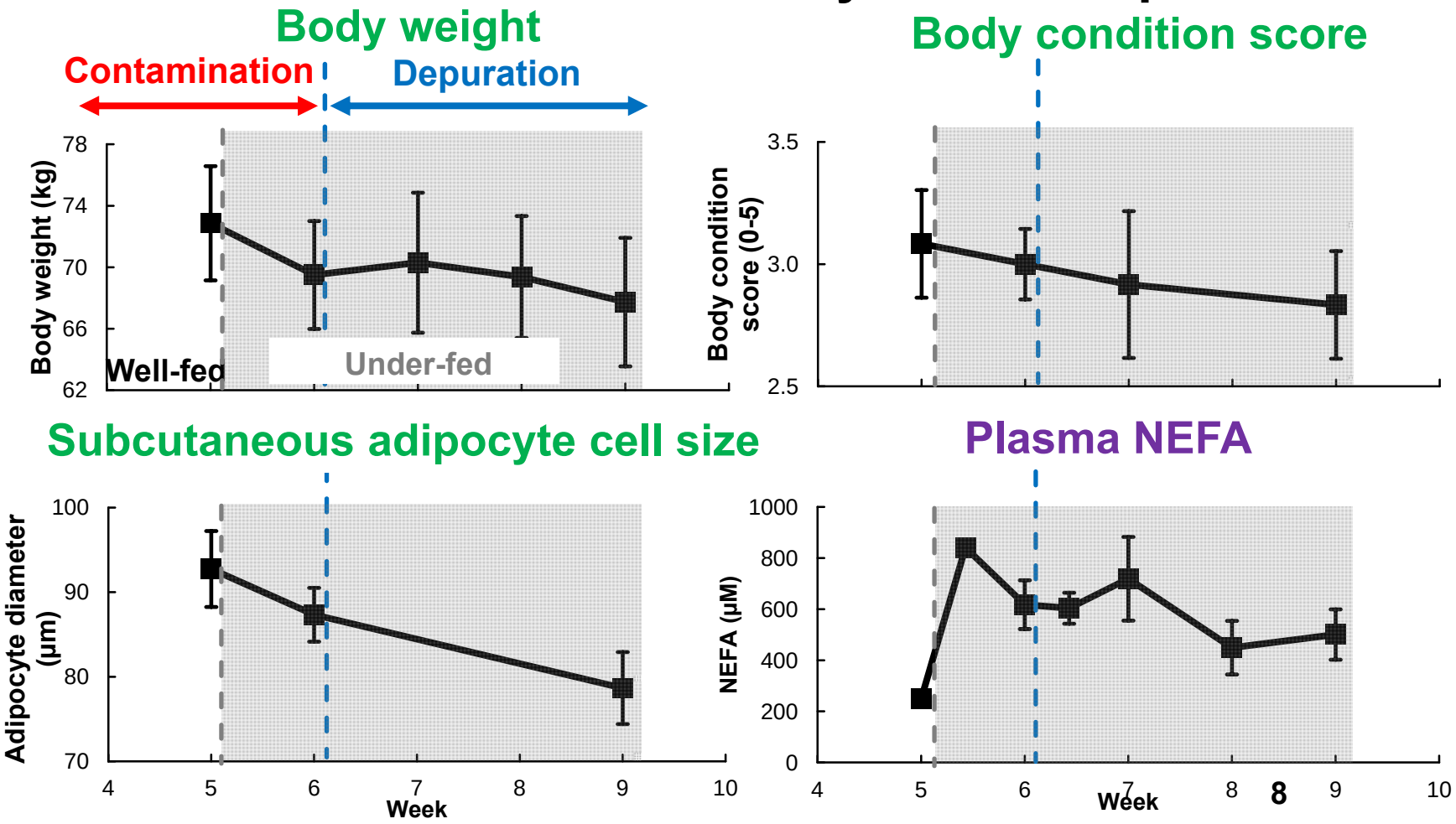
<u>Focus on PCB 180:</u>

Highly-chlorinated and poorly metabolized NDL-PCBs

⇒ Highly bioaccumulative

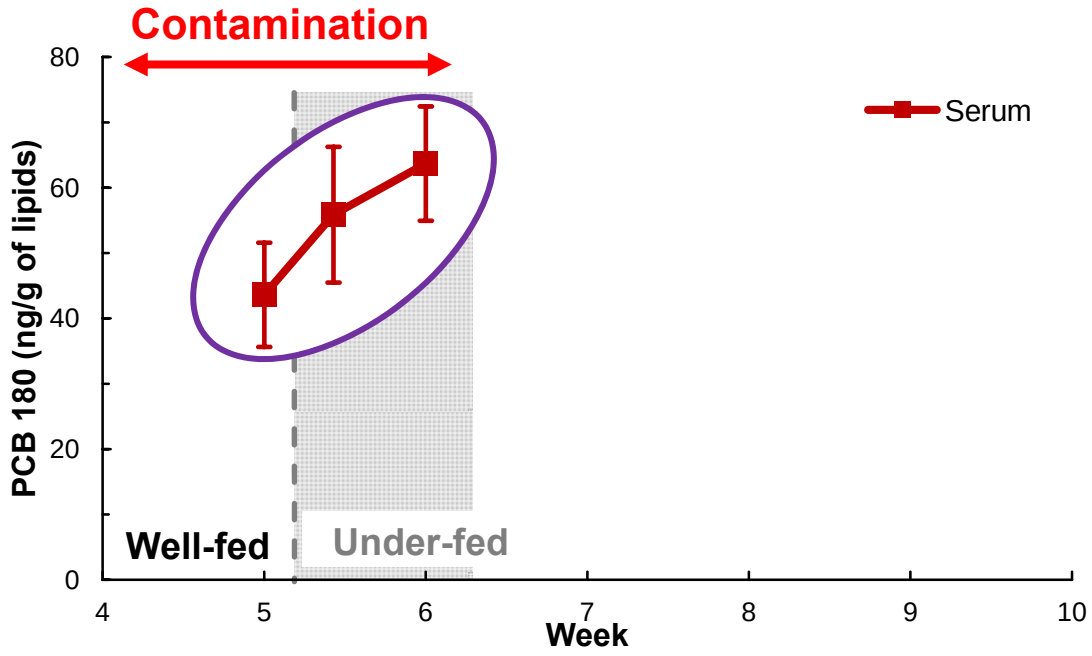
Results

Chronic undernutrition: Body fatness & plasma NEFA



Results

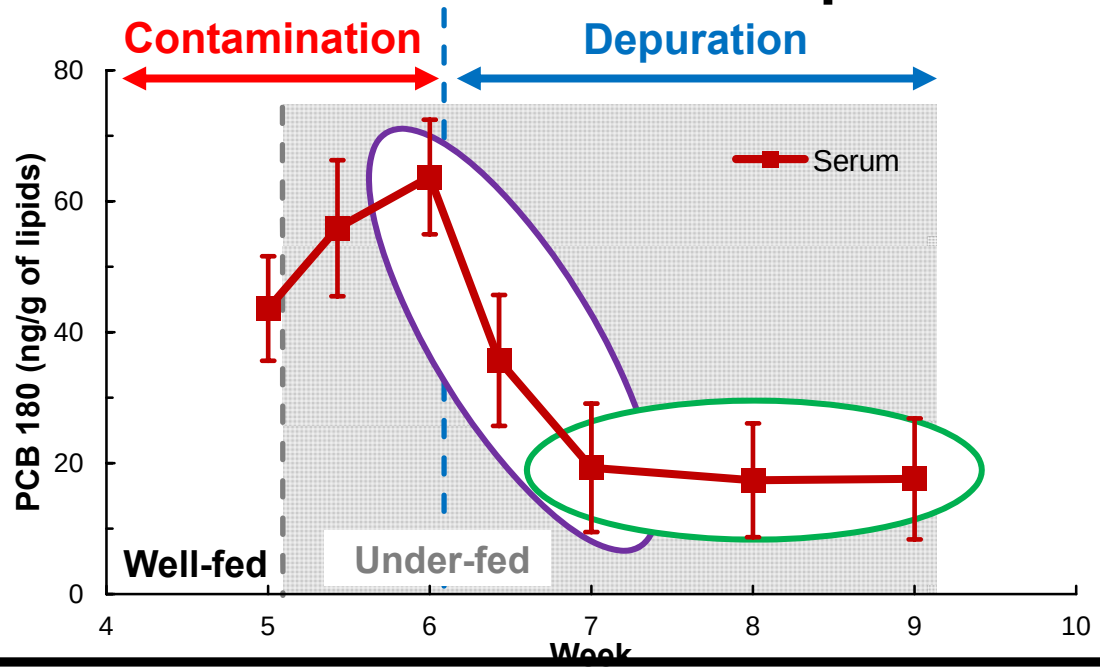
Chronic undernutrition: Serum and adipose tissue PCB 180



Similar observations in growing chicken: 2-4 days of complete starvation => ↑ blood DDT (≈ x 2) ***Donaldson et al., 1968***

Results

Chronic undernutrition: Serum and adipose tissue PCB 180

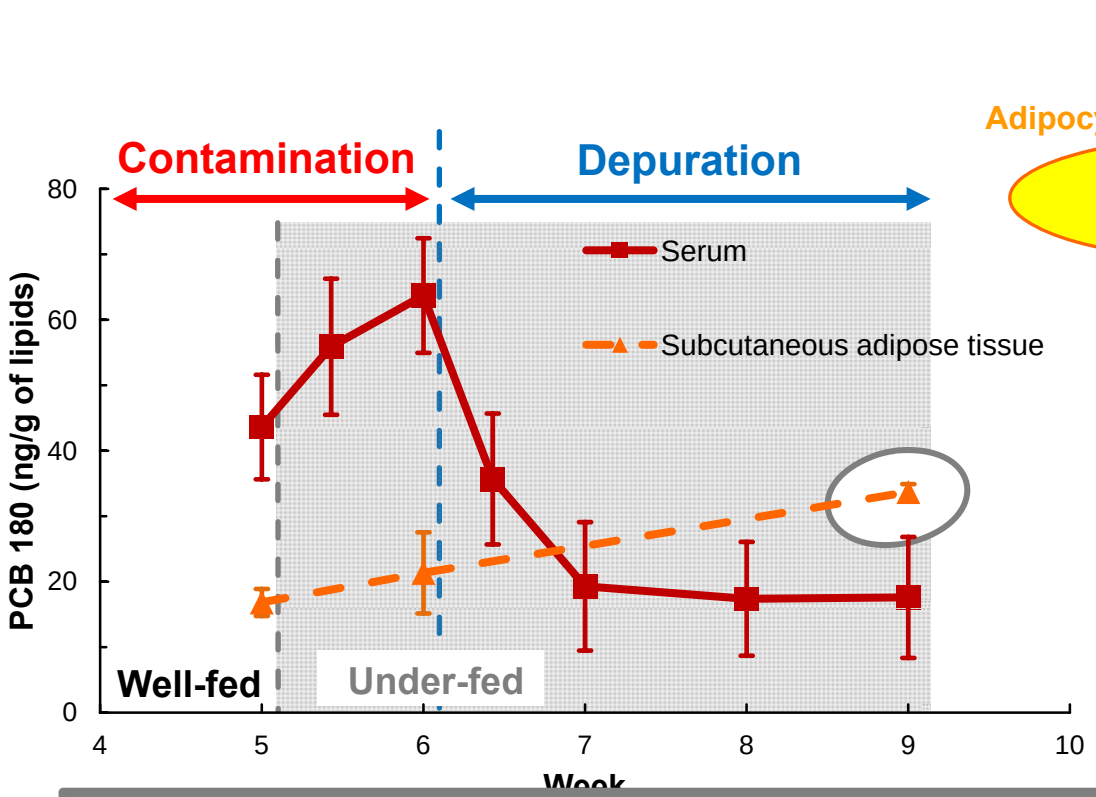


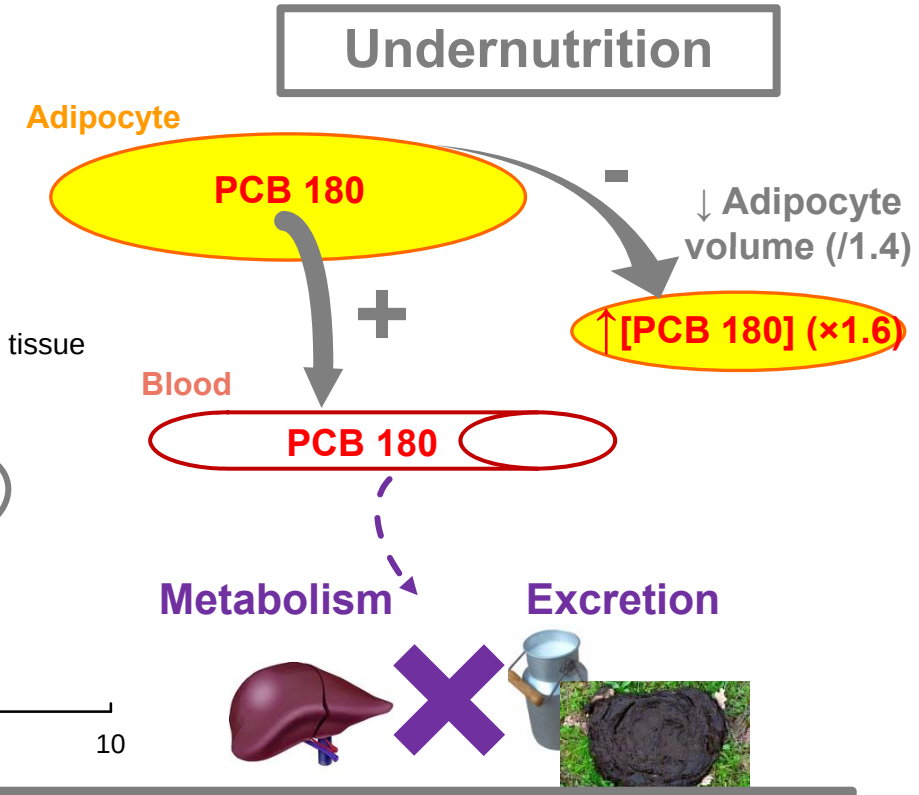
Initial 7-days ↓ in serum PCB 180 after cessation of oral exposure → end of absorption and distribution

Stabilization between 7 and 21 days of depuration

Results

Chronic undernutrition: Serum and adipose tissue PCB 180





Similar observation in growing chicken: 3-wk undernutrition

↑ carcass [PCBs] × 2.4 ↔ ↓ carcass fat mass / 2.4 *Polin et al., 1989*

Conclusions

During contamination, short-term undernutrition (3-7 days) ↑ **serum PCB 180, without significant change in body fatness**

During depuration, 3-wk undernutrition ↑ adipose tissue PCB 180 → linked to ↓ in the size of fat storage pool?

Undernutrition could ↑ poorly-metabolized NDL-PCBs content in blood, but does not seem to be of practical relevance for accelerate depuration of adipose tissue
at least without ↑ the size of excretion pools (milk and fecal lipids outputs)

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