

Evaluation of the maximum limit for selenium in feed for salmonids

Ørnsrud R.^{1*}, Amlund H.¹, Olsvik P.A.¹, Rasinger J.D.¹, Sundal T.K.², Hamre K.¹, Hillestad M.³, Buttle L.⁴, Lundebye A-K.¹ & Berntssen M.H.G.¹

1 The National Institute of Nutrition and Seafood Research (NIFES), Bergen Norway
2 Department of Biology, University of Bergen, Norway
3 Biomar AS, Myre, Norway
4 EWOS Innovation, Dirdal, Norway



*Presenter (robin.ornsrud@nifes.no)

INTRODUCTION
AIM AND
EXPERIMENTAL

Introduction

Maximum limits (ML) for essential minerals in animal feeds have been established by the European Union to protect animal welfare, consumer- and user safety and minimize environmental impact. Farmed Atlantic salmon have traditionally been fed diets mainly based on marine ingredients with sufficient levels of Se. As ingredients of plant origin replace marine ingredients, Se levels in commercial fish feeds decrease.

Consequently, the feed may need to be supplemented with Se to compensate for reduced levels. Such supplementation to the natural marine Se levels is currently not permitted due to the EU MLs in feeds for salmon. These MLs are currently 0.5 mg total Se/kg feed, of which 0.2 mg/kg feed can be provided as an organic form of Se.

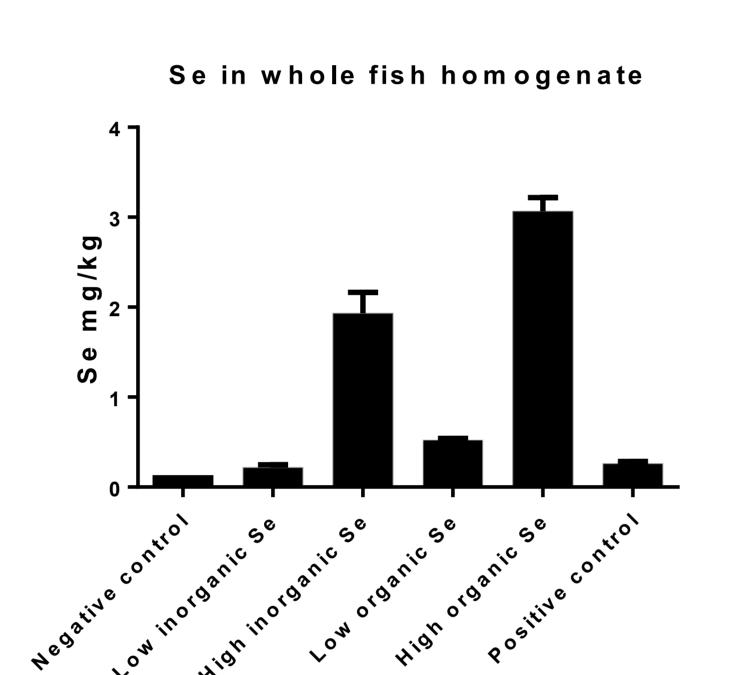
The current European ML for Se in fish feed is generic for all animal categories and not specific for fish. In order to establish a scientifically-based ML for Se in feed for salmonids, a risk assessment on the dietary toxicity of both organic and inorganic forms of Se in Atlantic salmon is required. Prior to a risk assessment, appropriate toxic mode of action and markers of toxic exposures have to be established. The aim of this study was to establish biomarkers of dietary Se toxicity in Atlantic salmon.

Materials and methods

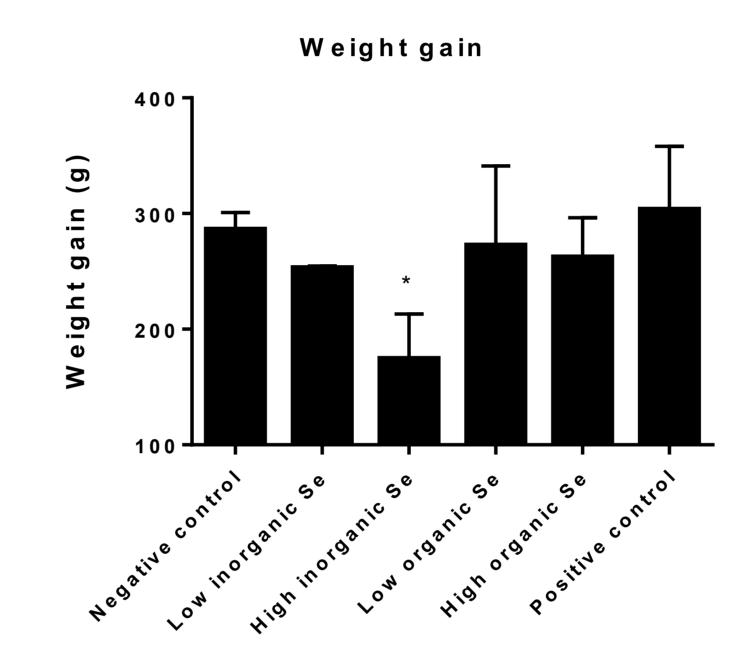
A 3 month feeding experiment with six diets was performed using Atlantic salmon (*Salmo salar* L.) at 500g. A basal diet with a low level of Se (0.35 mg Se/kg), was used as a negative control. Two diets supplemented with selenite (DSM) constituted a Low inorganic (1.1 mg Se/kg) and a High inorganic (15 mg Se/kg) diet. Two diets supplemented with organic Se as L-selenomethionine (AlkoSel®) constituted a Low organic (2.1 mg Se/kg) and a High organic (15 mg Se/kg) diet. Finally, a fish meal based diet served as a Positive control (0.89 mg Se/kg).

Liver biopsies were submitted to non-targeted (metabolomic screening) and targeted analysis (vitamin E, vitamin C, TBARS). Whole body samples were analysed for Se levels and lipid content.

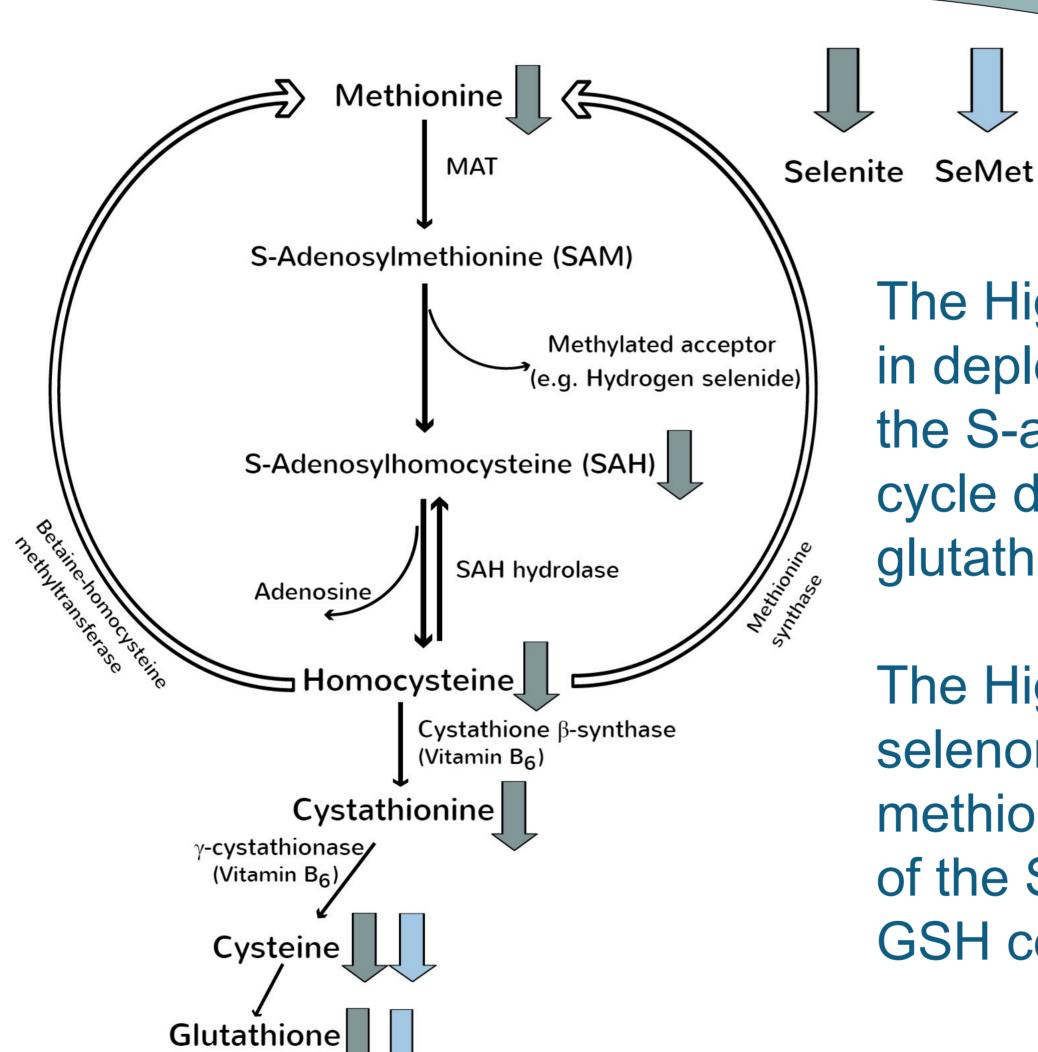
RESULTS AND DISCUSSION



The dietary treatments resulted in different whole body accumulation of Se



The High inorganic diet resulted in lower weight gain



The High inorganic diet resulted in depletion of intermediates in the S-adenosylmethionine (SAM) cycle due to increased glutathione (GSH) consumption

The High organic diet supplied selenomethionine, a source of methionine, preventing depletion of the SAM cycle, but increased GSH consumption

Vitamin C

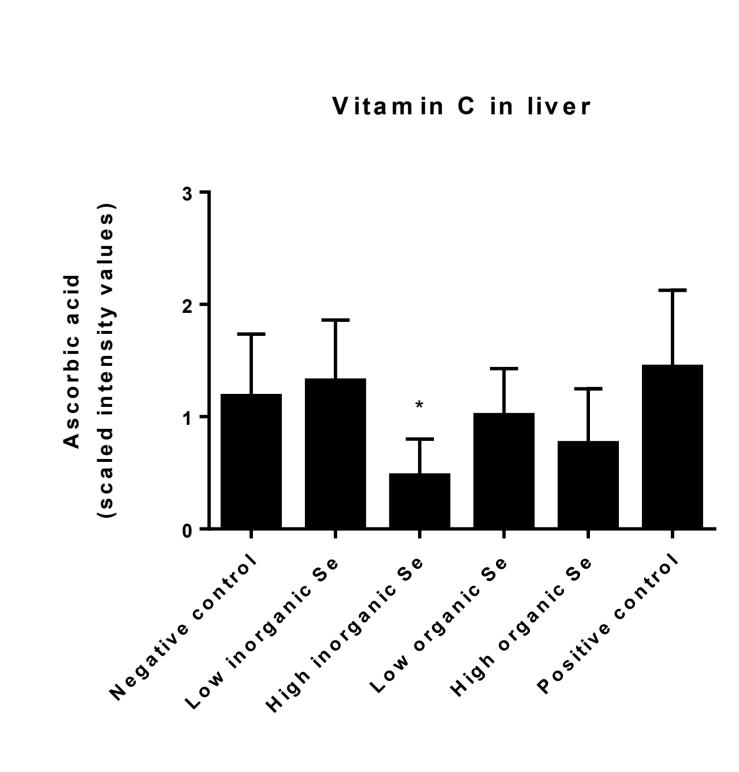
Cycle

Dehydro-

ascorbate

Radical

Vitamin E

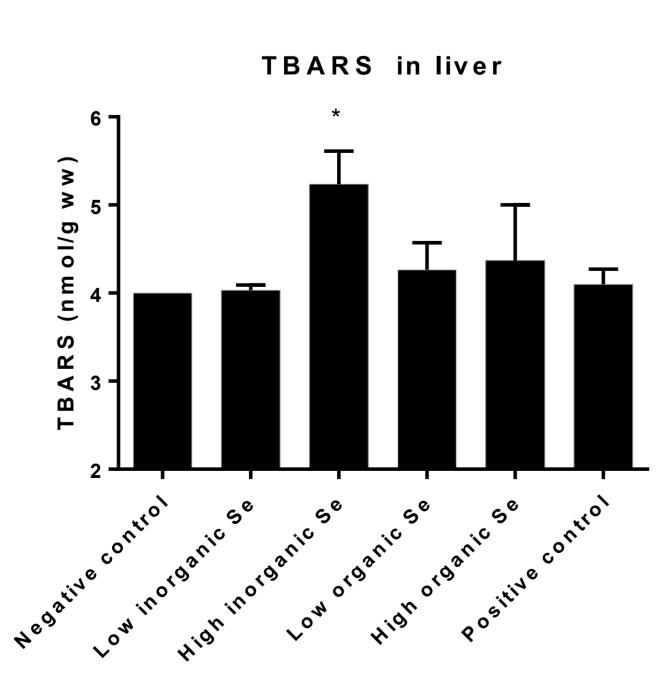


Depletion of GSH led to

vitamin E and vitamin C

increased consumption of

Vitamin E (α -tocopherol) in liver



Oxidative stress led to formation of lipid peroxidation products

GSH, vitamin C and vitamin E form an antioxidant network that prevents oxidation of lipids

Cytosol

Thiol

Cycle

Dihydrolipoic aci

*Van Meeteren et al 2005

NAD(P)H

GSH Reductase

NAD(P)++ H+

SUMMARY

- Fish fed the High organic Se diet showed the highest Se accumulation in whole fish
- > Reduced levels of glutathione (GSH), intermediates in the S-adenosylmethionine (SAM) cycle, vitamin C and vitamin E and elevated levels of TBARS were seen in fish fed the High inorganic Se diet.

ROOH, ROH

R00', R0'

Radicals

- > The toxic mode of action caused by excessive Se, appears to have been oxidative stress, where selenite showed a higher toxic potential than selenomethionine
- Markers of oxidative stress are suitable as markers of Se toxicity



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