

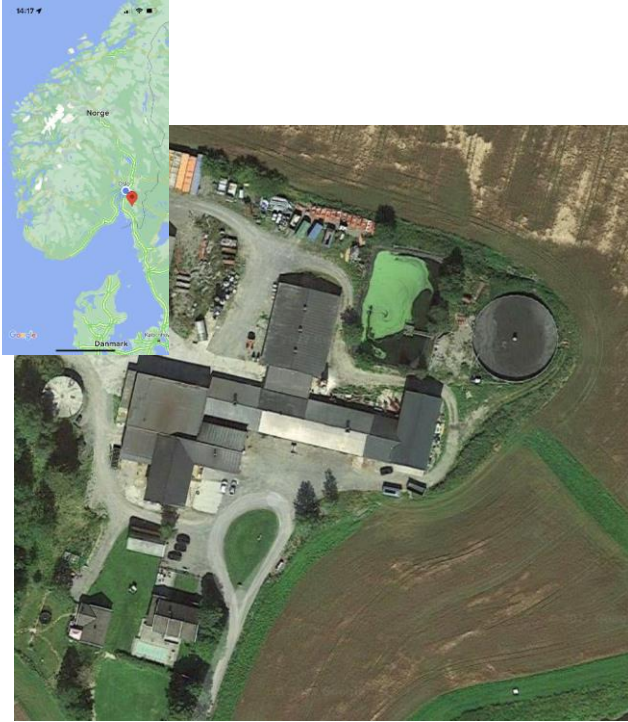


Selenium toxicosis in a Norwegian pig herd

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Precongress ECPHM Resident Workshop 9.-11.5.2022

Herd description



- Finisher herd situated in the south eastern parts of Norway
- Yearly production of approximately 2800 finishers
- Four supplier herds
- Batchwise production, all in-all out by compartment
- Eight units, approximately 170 pigs/unit
- Genetic combination: LZ sows x Duroc (semen)

Case history



October 2021

- 7 - 8 growers showed reluctance to move and stiff gait
- 15 - 20 additional cases the following day
- 2 died
- Growers >40 kg BW
- In two units, most pens affected in various degrees.

Clinical signs

- Reluctance to move
- Stiff gait, posterior incoordination and hind limb paresis
- Body temperature and appetite normal
- No joint swellings, no signs of infectious disease

Differential diagnoses

- stiff gait, posterior incoordination, hind limb paresis, acute death

Insult	Main clinical signs	Consideration
Infectious		
Teschovirus A	Polioencephalomyelitis with high mortality, fever, anorexia, ataxia of the rear legs, nystagmus, convulsions, opisthotonus and coma.	Not likely, never detected in Norway
Encephalomyocarditis virus	Acute disease with sudden death in young pigs due to myocardial failure, anorexia, trembling, staggering gait, paralysis, pyrexia	Not very likely, no fever, older pigs
Arthritis – <i>Streptococcus spp.</i> , <i>Staphylococcus spp.</i> , <i>E.rhusiopathiae</i> , <i>Gl.parasuis</i> , <i>T.pyogenes</i> , <i>M.hyosynoviae</i> , <i>M.hyorhinis</i>	Lameness, fever, inability to rise, joint swelling, pain	Not very likely (- <i>M.hyosynoviae</i>), no signs of infectious disease.
<i>Clostridium botulinum</i>	Progressive weakness (first in front legs), incoordination, staggering gait, paralysis, recumbency, anorexia, blindness, salivation	Rare in swine, but should be considered in afebrile pigs with progressive weakness
Non-infectious		
Nutritional myopathy (Selenium and/or vit E deficiency)	Myocardial hemorrhages, acute death in rapidly growing pigs, pale streaks in muscle, usually 3-8 weeks of age	Likely
Micronutrient deficiencies (Ca, P or Vit D)	Altered gait, lameness, ataxia, tremors, paresis	Likely
Toxicosis – sodium chloride, vit D, vit A	Tremor and motor excitement	Not very likely, deviating symptoms, water access adequate
Se toxicosis	Decreased growth rate, hair loss, paralysis, acute death	Rare, but possible in this case
Osteochondrosis	Progressive lameness affecting one or more limbs due to localized failure of endochondral ossification and necrosis of cartilage	Possible, but many pigs affected
Porcine stress syndrome	Muscle rigidity, pyrexia, rapid rigor, swelling of the back muscles, respiratory distress, discoloured skin, acute death	Not likely, rare, deviating symptoms

Necropsy results

- Three pigs (42.9-64.5 kg) with typical symptoms were submitted to necropsy.

Gross lesions

- Small, diffusely pale liver with reduced texture in two of the pigs (1.2 and 1.3% of BW)
- Osteophyte production, fibrosis, thickened synovial membrane in elbow and hip joint in the heaviest pig
- No claw lesions

Histopathology

- Liver cell degeneration
- No lesions in *N.ischiadicus*, brain or spinal cord.
- No degenerative lesions in heart or skeletal muscle in any of the pigs



Photo: Mette Valheim, Norwegian Veterinary Institute

Herd investigation

- Wet feed mixed on supplier farm, including home-grown cereals, soy flour from a commercial feed mill and a mineral supplement
- Type and amount of mineral supplement (premix) added varied with age
- Changes in the premix delivered 20th September
- First symptoms observed approximately 1-2 weeks later
- Analyses of the premix initiated, and change of diet 18th October
- A total of 318 growers received the same diet
- 68 (21.4%) of the pigs died (n=7) or were euthanized (n=61)





Feed analyses

- Source of selenium: Selenite, NaSe
- The concentration of selenium in the mineral supplement was 1360 mg/kg (values based on 88% dry matter).
- The declared Se concentration was 13 mg/kg.
- The mineral supplement was used in the whole diet at a proportion of 3.086 %



Se concentration (mg/kg) in complete feed	
Upper limit according to EU-legislation	0.5
Maximum tolerable level	4.0
Declared level in case herd	0.4
Calculated level in case herd (88% dry matter)	41.97

Toxicological analyses

Sample*	Animals (n)	Measured Se concentration, mg/kg		Reference values in mg/kg*
		Average	Range	
Liver	3	8.4	5.9 - 12	0.25-1.5
Muscle	3	0.54	0.47 – 0.58	0.08-0.5



Pigs necropsied
17th October

Sample	Animals (n)	Measured Se concentration, mg/kg		Reference values in mg/kg*
		Average	Range	
Serum	10	1.7	0.97 - 2.9	0.08 – 1.0
Muscle	10	0.1	0.098 – 0.11	0.08 – 0.5
Liver	5	2.6	1.8 - 3.9	0.25-1.5
Kidney	5	2.2	1.9 – 2.3	0.9 – 3.0



Pigs slaughtered
9th November

*Puls et al, 1994

Results and follow-up

- The clinical signs improved dramatically within a few days after withdrawal of the selenium-contaminated feed
- Liver Se concentration reduced by approximately 70% (from 8.4 to 2.6 mg/kg) during the first 23 days after feed replacement.
- Liver and kidney from slaughtered pigs were condemned
- No new cases since October 2021
- The producer of the mineral feed conceded a cross-contamination due to technical failure

Selenium

- To avoid Se deficiency, supplementation has become standard practice, especially in areas where soil selenium levels are low (Heile et al., 1998).
- Both organic (selenized yeast, selenomethionine) and inorganic (selenite, sodium selenate) selenium are added to commercial pig feed (Fairweather-Tait et al. 2010; Falk et al., 2022)

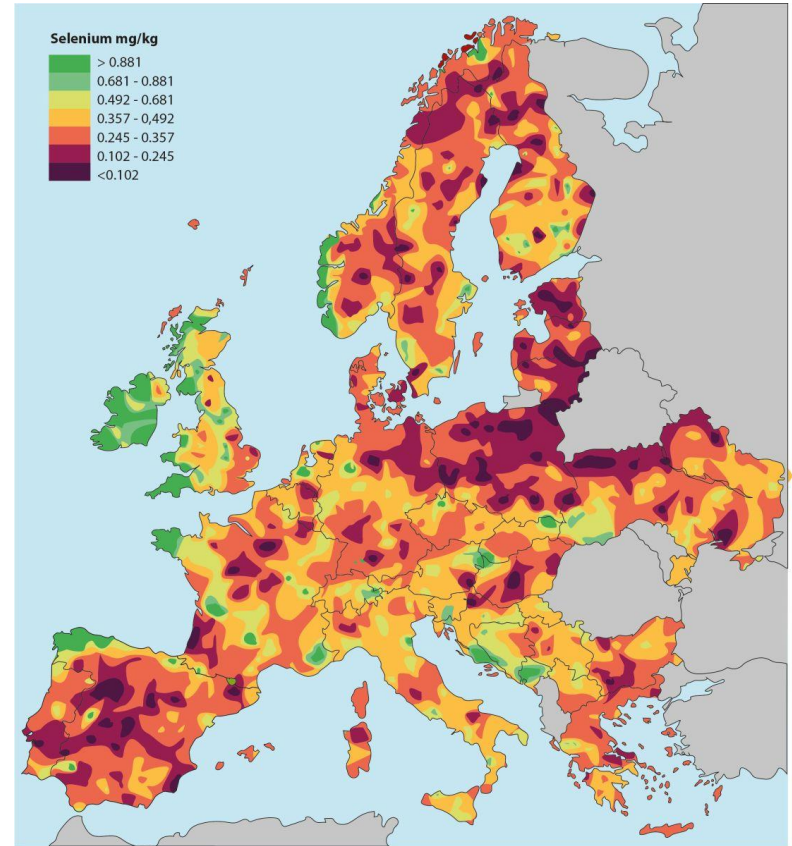


Figure: Haug, 2007

Selenium toxicosis

The European Union (EU) has legislated the concentration of dietary selenium to 0.5 mg/kg, and the maximum tolerable level in diets for swine is 4 ppm (Regulation (EC) No 1831/2003).

Signs of selenium toxicosis in swine include

- anorexia, alopecia, separation of hoof and skin at the coronary band (Panter et al., 1996; Kim and Mahan, 2001)
- degenerative changes in the liver and kidney (Ensley and Radke, 2019)
- paralysis resulting from poliomyelomalacia (Penrith and Robinson, 1996; Davidson-York et al., 1999; Nathues et al., 2010)
- acute death (Nathues et al., 2010)

Concentration, duration of exposure, bioavailability

Liver and kidney Se concentrations greater than 3 ppm are expected with toxicosis (Ensley and Radke, 2019).

Discussion



- Clinical signs, pathological features and analyses of Se concentration in organ and feed samples confirmed the diagnosis of Se toxicosis in the affected farm.
- In contrast to other studies (Wilson et al., 1983; Davidson-York et al., 1999; Casteigneau et al., 2006; Nathues et al., 2010), **bilateral spinal gray matter lesions and claw lesions were not found**
 - Lower Se concentration and/or shorter exposure period?
- Additional blood sampling and diagnostics could have been performed to further explore possible differential diagnoses
- Still deviations in the Se concentration in premix delivered

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**Thank you for
your kind attention**