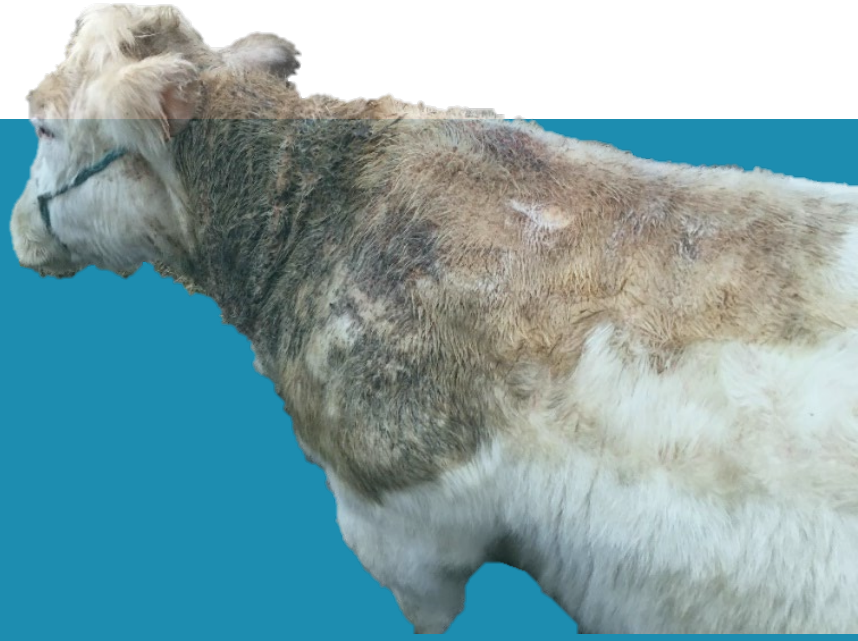


Combatting psoroptic mange susceptibility in Belgian Blue cattle: can breeding provide a solution?

Roel Meyermans, S. Janssens, W. Gorssen, X. Hubin, W. Veulemans, E. Claerebout, C. Charlier and N. Buys

Session 85: " Genetics of novel health and welfare traits "

CABG Center for Animal Breeding and Genetics

KU LEUVEN

Belgian Blue cattle

Pictures from: http://www.awenet.be/

Beef cattle breed

50% of Belgian national cattle herd

Worldwide: sires used in crossbreeding

± 2000 breeders

High degree of muscling (“double muscling”) due to a mutation in the *myostatin* gene

High ADG (1.2 kg/day during fattening, 1.6kg/day between 7 – 13 months old)

Low feed conversion, lean meat and high carcass yield > 82 %

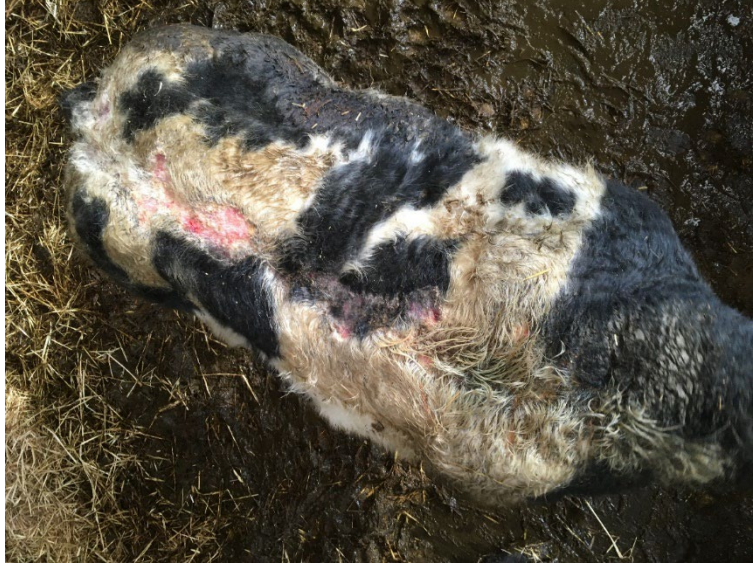
Psoroptic mange

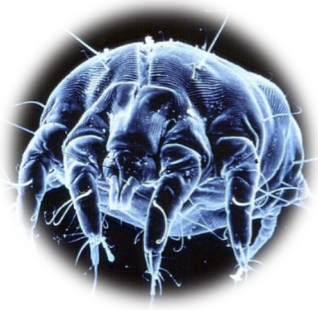
Skin disease

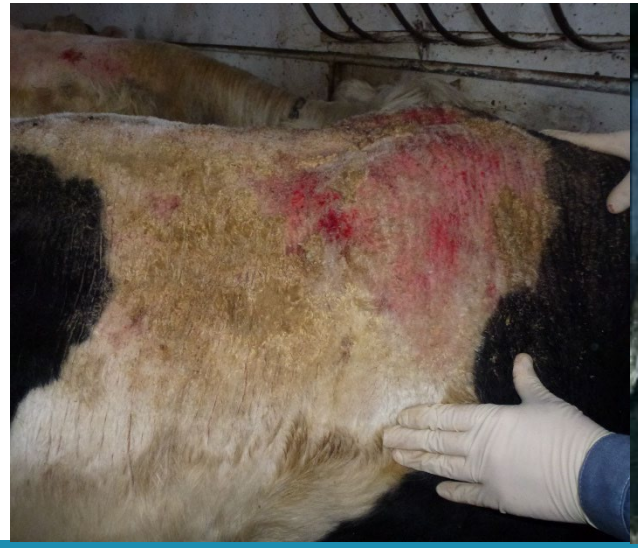
Caused by mites *(Psoroptes ovis)*

Consequences:

- Decrease of animal welfare
- Economic losses:
  - Loss in daily gain
  - Leather quality decreases
  - Treatment cost







Psoroptic mange

74% of all beef farms in Flanders are confronted with psoroptic mange (Sarre et al. 2012), but also in Wallonia it is a severe problem

Currently ‘controlled’ via acaricide drugs, but:

- Often ineffective
- Costly and time consuming
- Resistance of the mites
  (Sarre et al., 2015; Van Mol et al., 2020))

Psoroptic mange sensitivity

Belgian Blue is extremely sensitive to psoroptic mange compared to other breeds

Even within the same environment, some animals are more sensitive than others

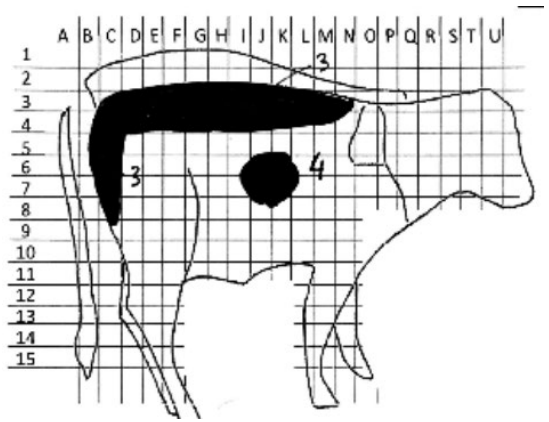
Farmers: *“Some sire lines produce more sensitive offspring than others“*

→ Is selection possible ?

Animal sampling

1,975 cattle sampled from 2011-2020 in winter

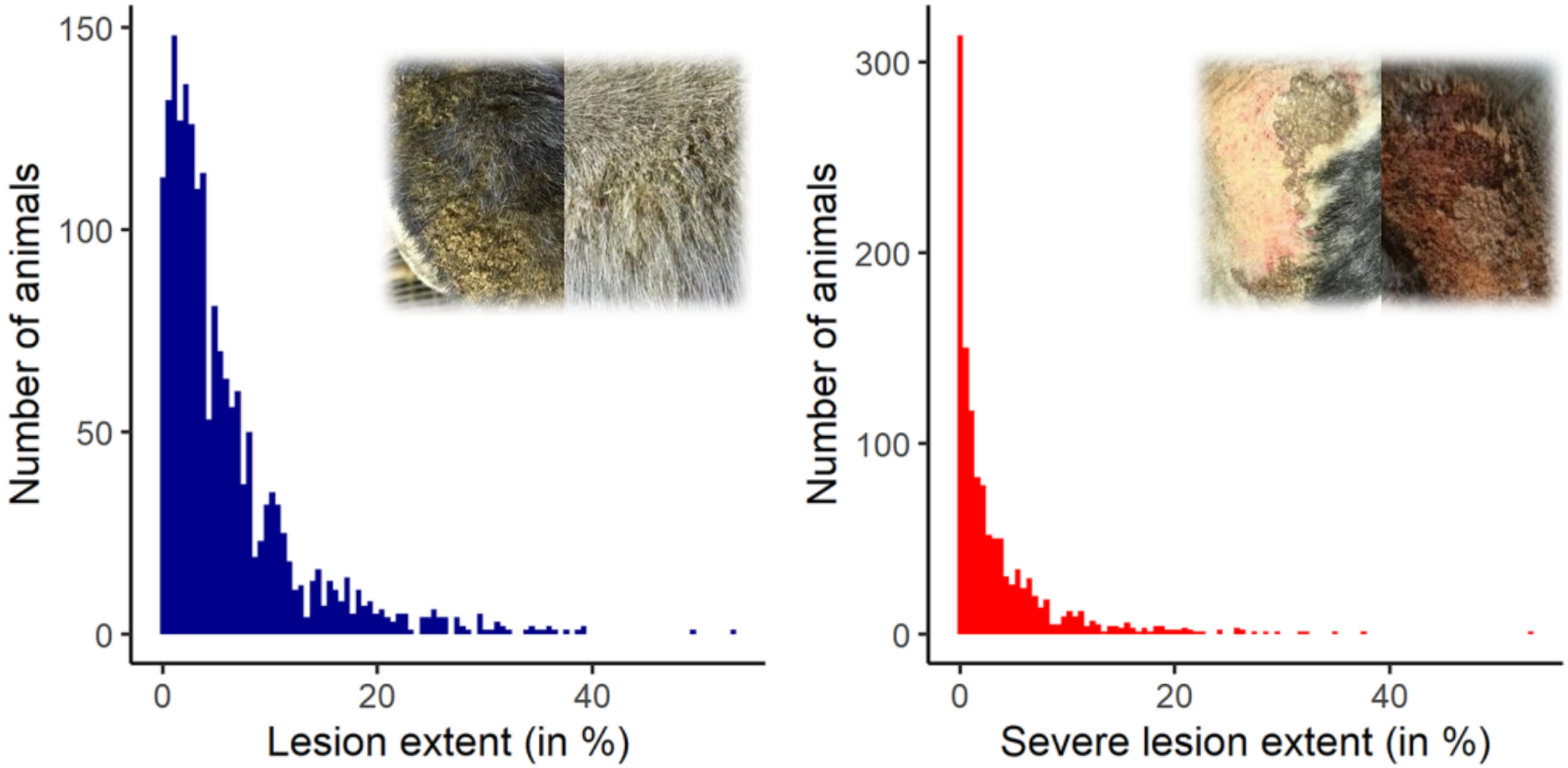
- Registration of lesion size and severity assesment
- Presence of (live) *P. ovis*
- Labour intensive !



DNA samples → genotyped on EuroGenomics MD or Illumina BovineSNP50 array

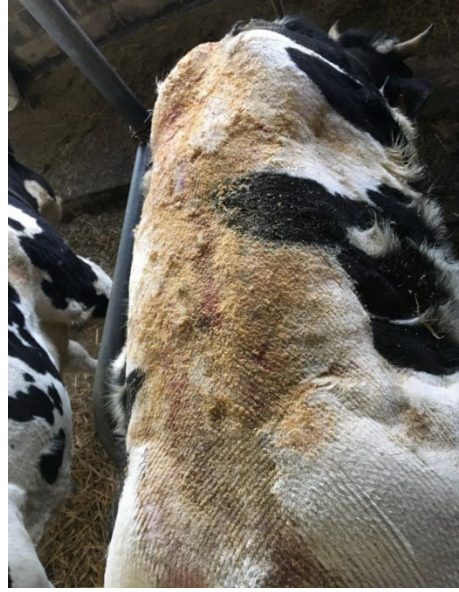
Correlations between successive screenings (2w) were high (±0.74, n=655)

Animal sampling



Histograms of lesion extent (LE) and severe lesion extent (SLE). LE and SLE are expressed as % of body surface of the animal and show a right-skewed distribution of psoroptic lesion extent

Heritability of mange susceptibility



Animal model, using ssGBLUP (remlf90)

Corrected for sex, coat color, birth year, age and project (fixed) and contemporary group (random)

Trait	Additive genetic variance	Contemporary group variance	Residual variance	h^2	c_g^2
Lesion extent	3.06 (1.30)	19.06 (3.04)	23.27 (1.45)	0.067 (0.03)	0.420 (0.04)
Severe lesion extent	3.17 (1.00)	17.12 (3.41)	14.40 (1.16)	0.091 (0.03)	0.494 (0.05)
Mite count	516.20 (180.41)	622.50 (130.38)	3042 (190.25)	0.124 (0.04)	0.149 (0.03)

h^2: heritability
c_g^2: proportion of variance explained by contemporary group effects
SE between brackets

Genomics of mange susceptibility

Case (n=241) – Control (n=192) approach

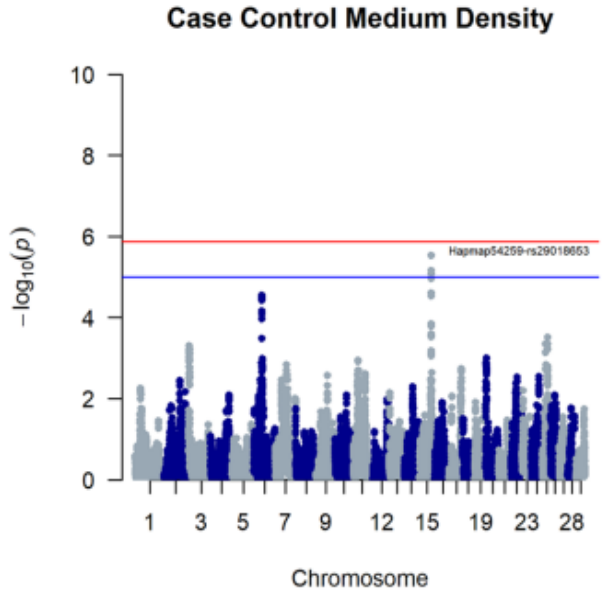
&

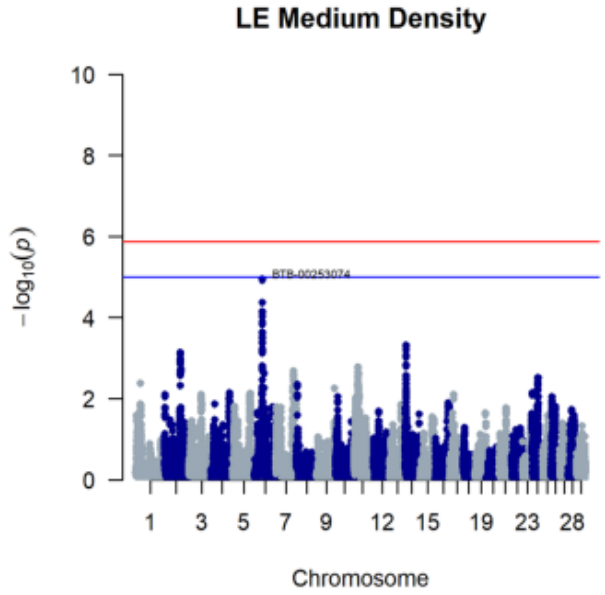
Quantitative approach for lesion extent (n=1,296)

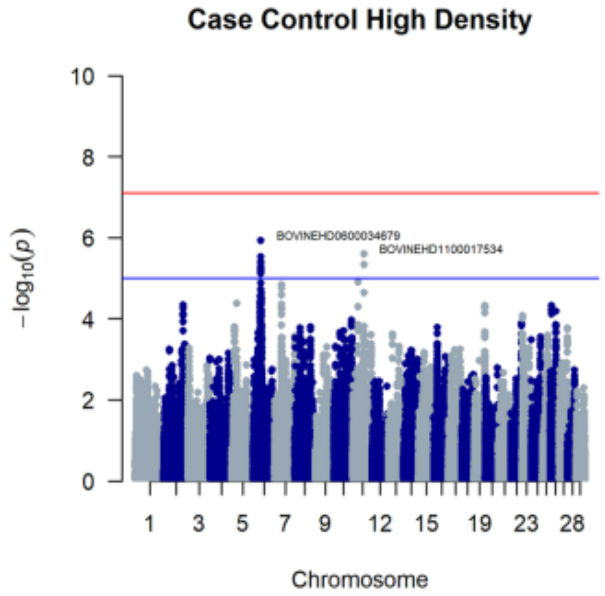
Genotypes phased and imputed to HD using BEAGLE 3.3.2

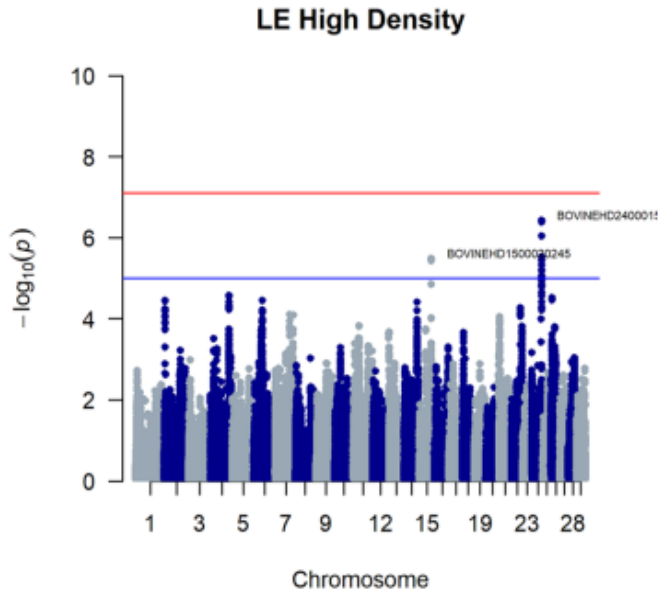
Haplotypes were assigned using HiddenPHASE 1.1

GWAS was performed using GLASCOW (linear-mixed model)


Case Control Medium Density
Hapmap54259-rs29018653
$-\log_{10}(p)$
Chromosome


LE Medium Density
BTB-00253074
$-\log_{10}(p)$
Chromosome


Case Control High Density
BOVINEHD0800034679
BOVINEHD1100017534
$-\log_{10}(p)$
Chromosome


LE High Density
$-\log_{10}(p)$
Chromosome

Genomics of mange susceptibility

Meyermans *et al. Genetics Selection Evolution* (2024) 56:52
https://doi.org/10.1186/s12711-024-00921-7

Genetics Selection Evolution

RESEARCH ARTICLE **Open Access**

Genetic and genomic analysis of Belgian Blue's susceptibility for psoroptic mange

Roel Meyermans[1*], Steven Janssens[1], Annelies Coussé[1], Susanne Tinel[1], Wim Gorssen[1], Fabrice Lepot[2], Xavier Hubin[2], Patrick Mayeres[2], Wim Veulemans[3], Nathalie De Wilde[4], Tom Druet[5], Michel Georges[5], Carole Charlier[5], Edwin Claerebout[4] and Nadine Buys[1]

SCAN ME

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Can we select against mange susceptiblity?

Heritabilities for mange susceptiblity were low (± 7-9%) with a single observation

→ still possible to select for traits with a low h²

$$\Delta G = \frac{i_{sire} + i_{dam}}{2} \times \sigma_p \times h^2$$

[Smith, 1969; Falconer & Mackay, 1996]

Allows a ΔG of 0.85% lesion coverage in one generation
if "full" selection against susceptiblity would be possible …

With a mean lesion coverage of 3.38% in the screened population
→ 26% reduction (!)

Can we select against mange susceptiblity?

Current model: ± 2,000 phenotyped animals, total number of animals in pedigree (± 10,000)

Breeding bulls are raised in “high health” environments

→ phenotype offspring

To obtain reliable EBVs (rel > 0.5): > 47 offspring per bull necessary

In our dataset: 19 bulls with > 10 offspring phenotyped & 443 sires in total

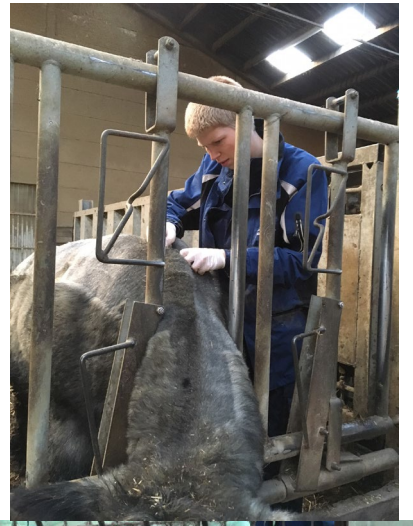
Can we select against mange susceptiblity?

Setup routine data collection

- Camera / image analysis ?

Rule out non - *P. ovis* infestations !

Marker assisted selection ?



Take home message

- Sensitivity of psoroptic mange susceptiblity is heritable
- Estimation of genetic parameters is possible, selection can be feasible
- Genomic candidate regions have been identified
- But: *In the world of genomics, phenotyping is king*

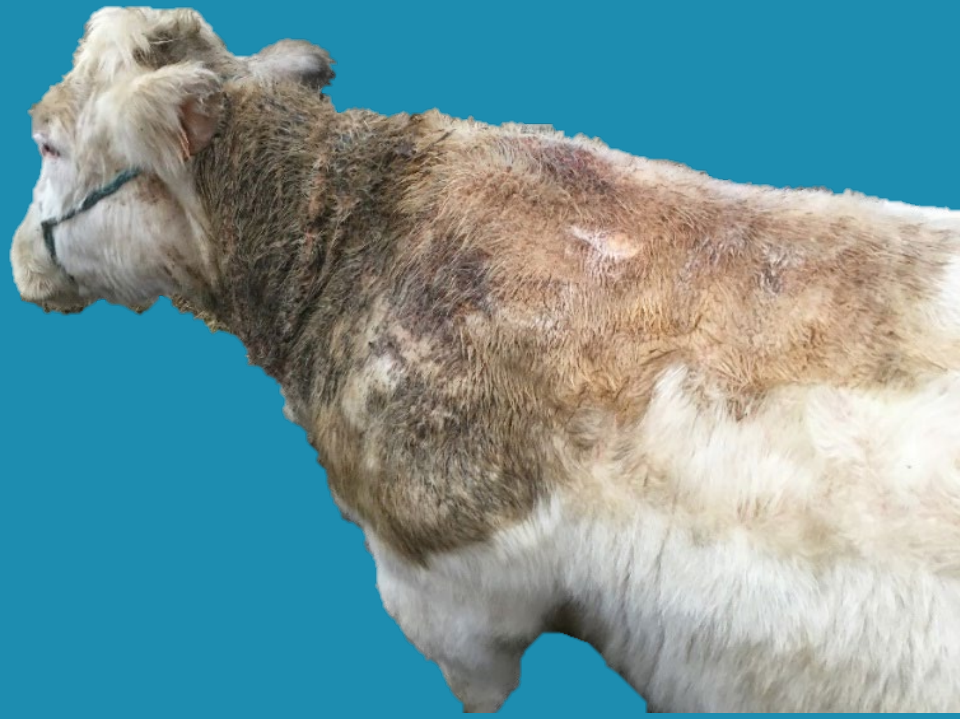
Partners

- Universities
- Herdbooks
- Funding

RT17/1 & RT 11/5

Fellowship: 1S37119N & 1S37121N

PDMT2/23/035



Thank you for your attention

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