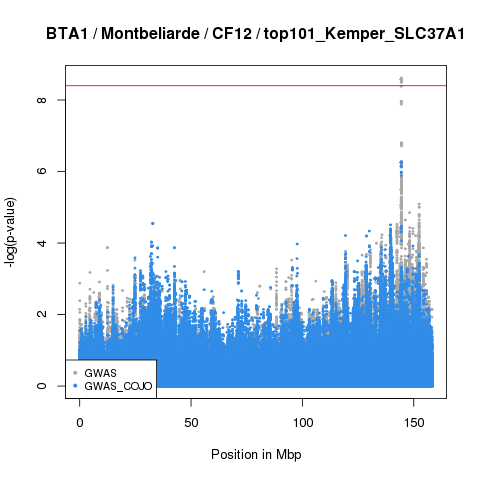
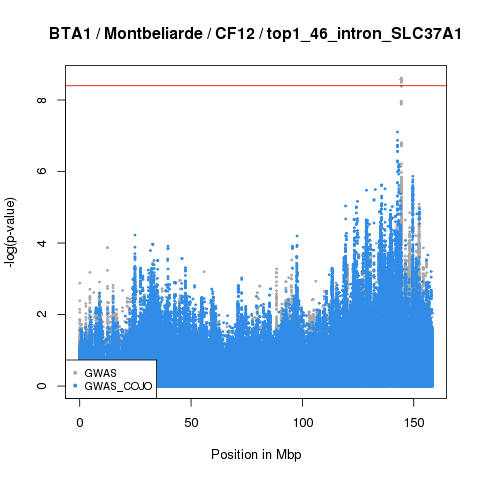
**Figure S1:** -log(*P*-value) plotted against the position of variants detected by GWAS (in grey) and conditional GWAS (GWAS\_COJO; in blue)

**a) On BTA1, in Montbéliarde analysis for the s1-CN phenotype, with the fixed effect of**

a1) the TOP1 variant detected in within-breed Montbéliarde GWAS, at 144,397,274 bp

a2) the TOP1 variant detected in multibreed GWAS, at 144,398,814 bp

a3) the most probable candidate variant detected by Kemper et al (2015), at 144,414,936 bp, ranked 101st in multibreed GWAS

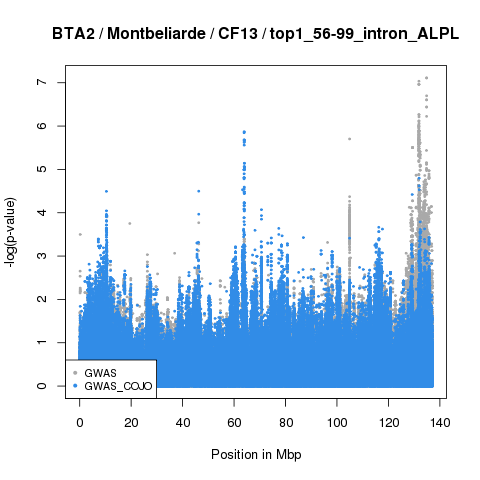
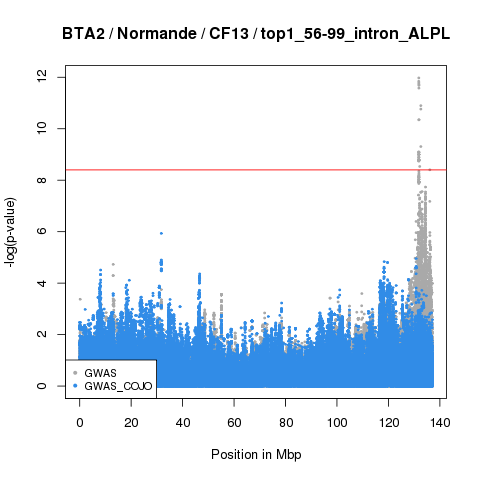
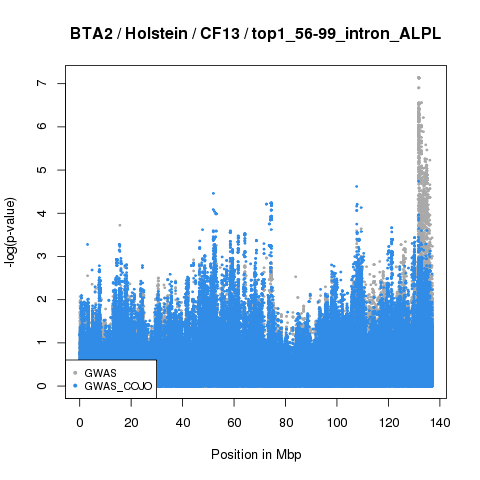


a1)

a3)

a2)

**b) On BTA2, in Montbéliarde, Normande, and Holstein analyses for the s2-CN** **phenotype,** with the fixed effect of the TOP1 intronic variant detected in the within-breed Normande GWAS, at 131,806,882 bp

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Montbéliarde

Holstein

Normande

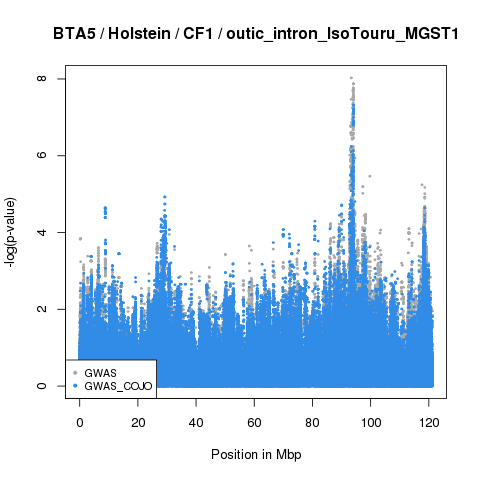
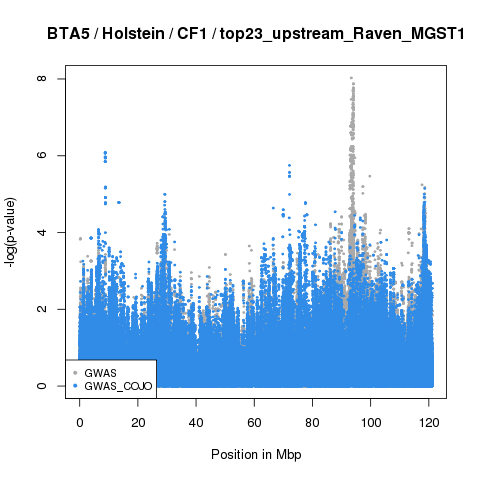
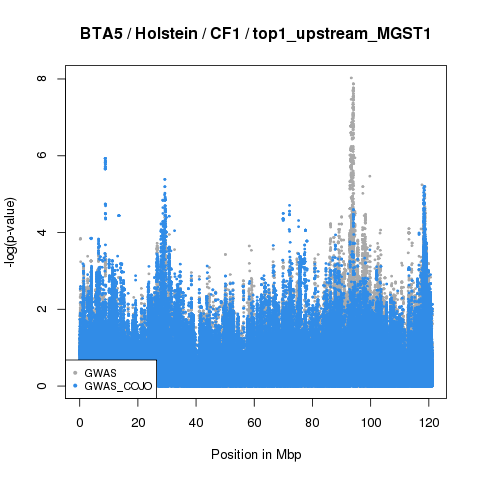
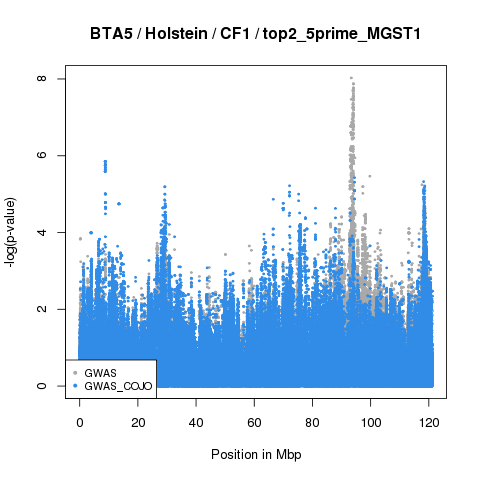
**c) On BTA5, in Holstein analysis for the PC phenotype, with the fixed effect of**

c1) the TOP1 upstream variant detected in the multi-breed GWAS, at 93,950,211 bp

c2) the TOP2 5’-UTR or TOP3 upstream variants detected in the multi-breed GWAS, at 93,950,116 and 93,950,288 bp

c3) the upstream variant detected by Raven et al. (2015) at 93,951,731 bp and ranked 23rd in the multi-breed GWAS

c4) the intronic variant detected by Iso-Touru et al. (2016) at 93,945,694 bp, outside of the confidence interval of the QTL detected in the multi-breed GWAS



c2)

c1)

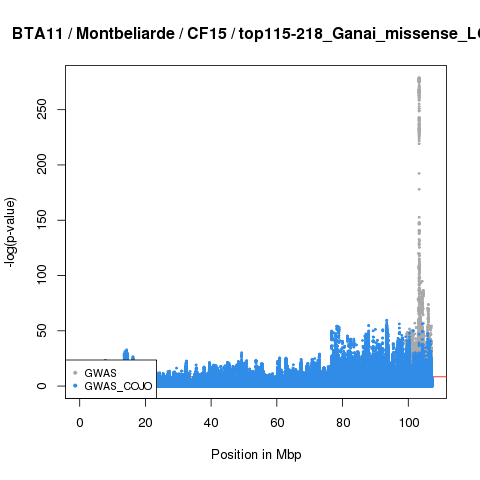
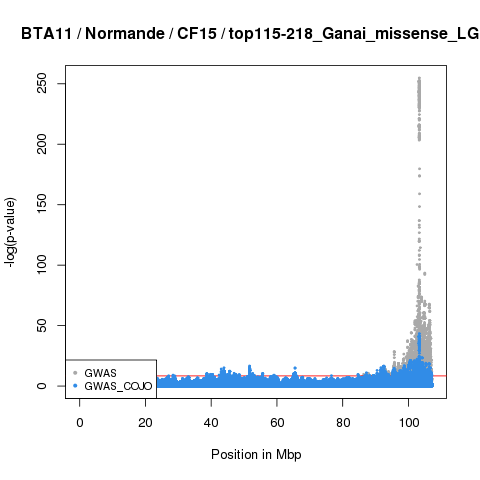
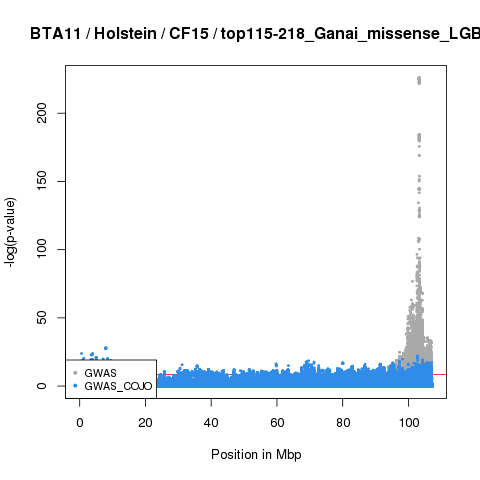
c4)

c3)

**d) On BTA11, in Montbéliarde, Normande, and Holstein analyses for the -LG phenotype, with the fixed effect of**

d1) the missense PAEP mutations that cause the A and B protein variants, at 103,303,475 and 103,304,757 bp (Ganai et al., 2009)

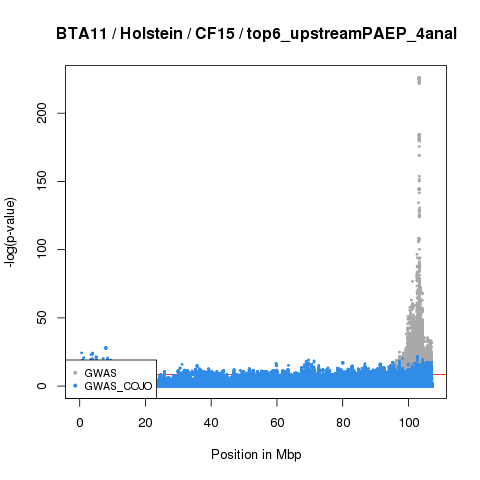
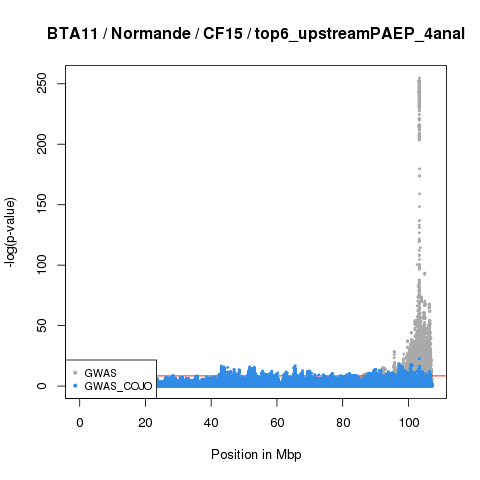
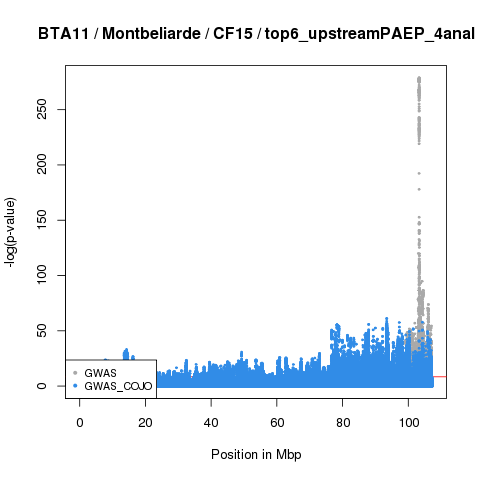
d2) the upstream PAEP variant (103,298,431 bp) ranked in the TOP6 in within- and multi-breed analyses

d1)

Holstein

Normande

Montbéliarde

d2)

Montbéliarde

Holstein

Normande

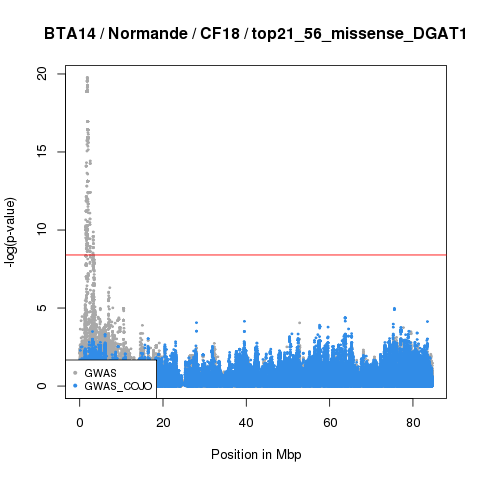
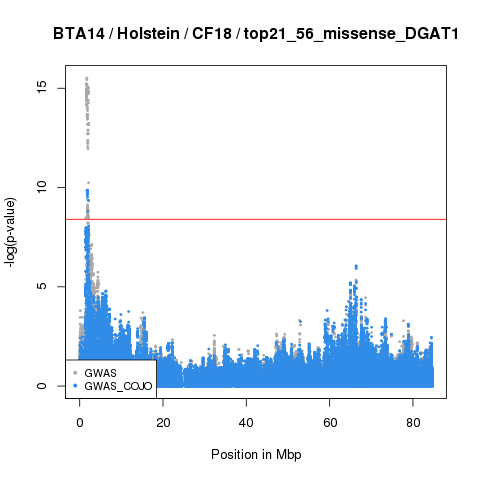
**e) On BTA14, in Normande and Holstein analyses, with the fixed effect of**

e1) the causative K232A *DGAT1* variant for the -CN phenotype, at 1,802,266 bp (Grisart et al., 2001)

e2) the missense *BOP1* variant (1,842,678 bp), ranked 1st in the NOR analysis for the -CN phenotype

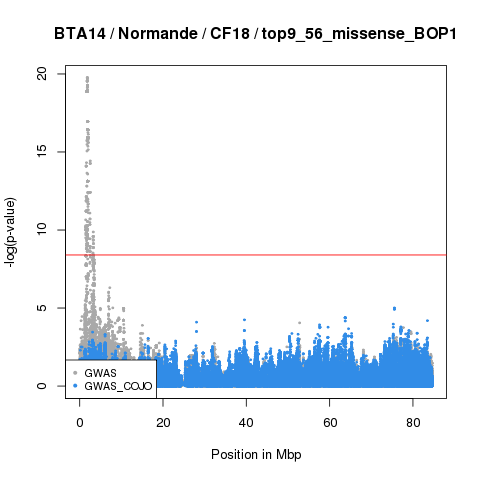
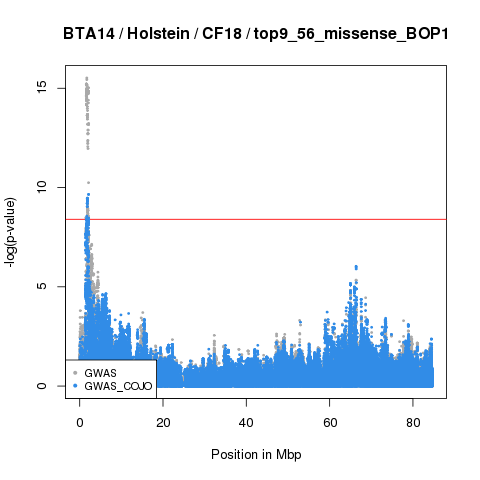
e3) one of the three missense *RECQL4* variants in complete LD (1,617,841, 1,618,978, and 1,619,555 bp), ranked 3rd in the HOL analysis for the -CN phenotype

e4) the missense *MROH1* variant (1,878,165 bp), ranked 14th in the HOL analysis for the s2-CN phenotype

e1)

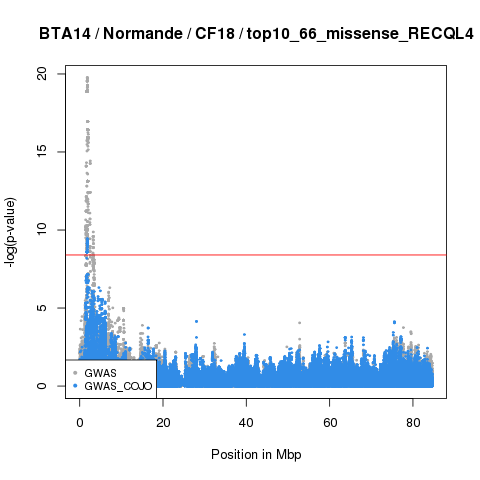
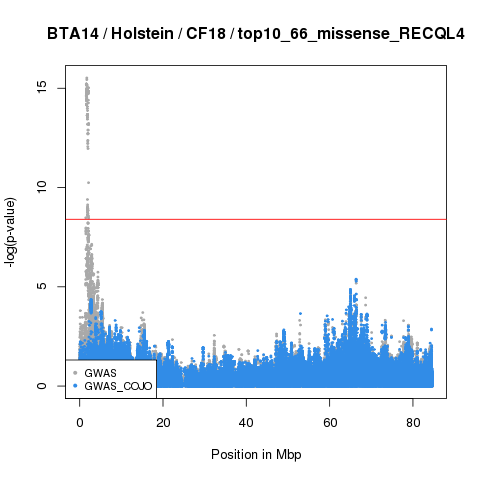
Holstein

Normande

e2)

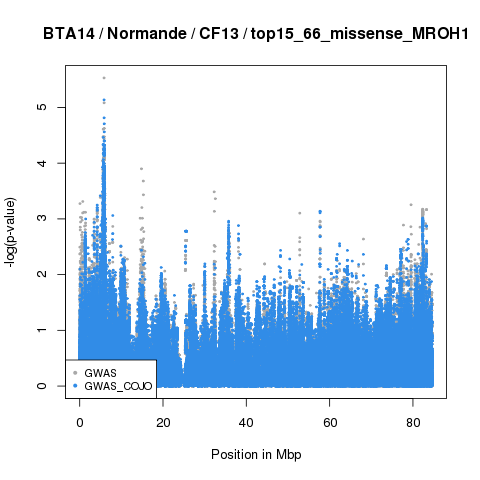
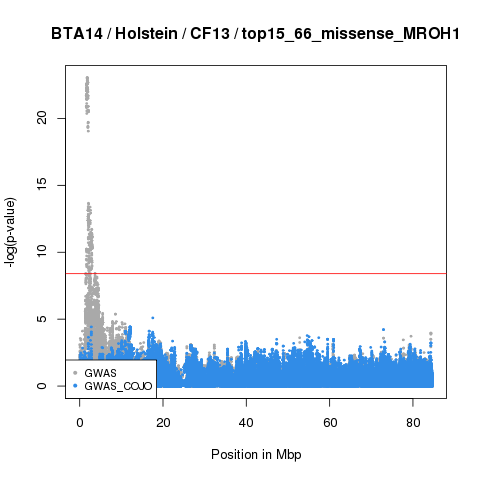
Normande

Holstein

e3)

Normande

Holstein

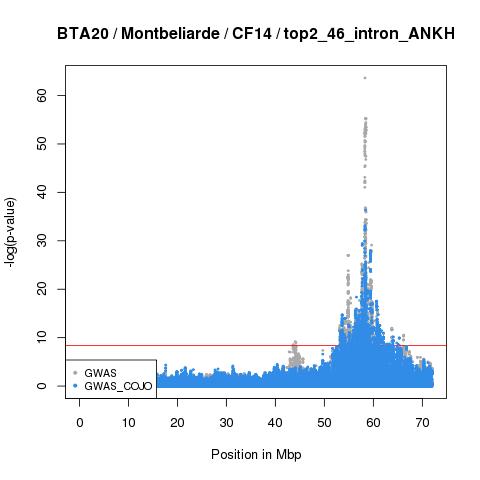
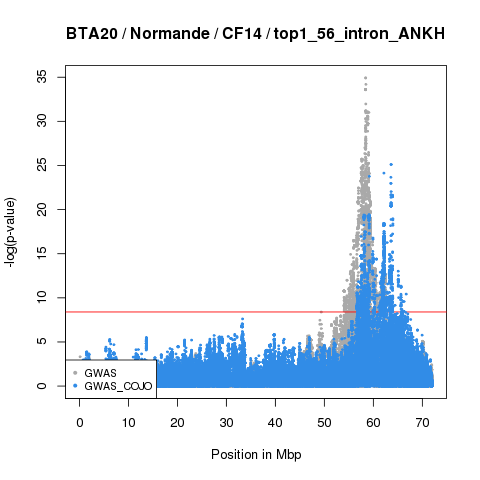
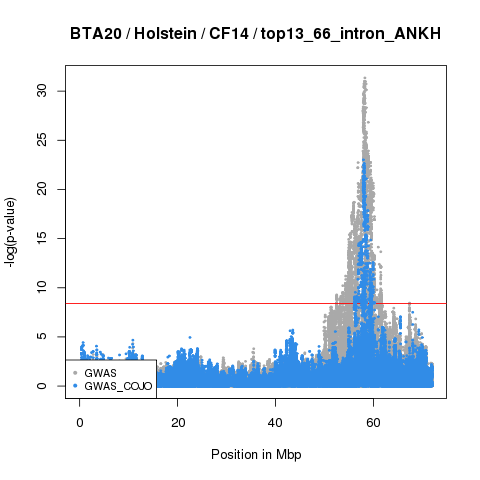


e4)

Normande

Holstein

**f) On BTA20, for the -LA phenotype, with the fixed effect of the most significant variant, located in the *ANKH* gene in Montbéliarde (58,446,550 bp), Normande (58,422,697 bp), and Holstein (58,491,204 bp) analyses**

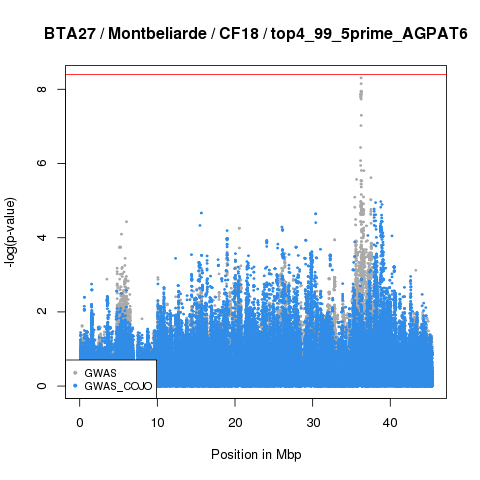
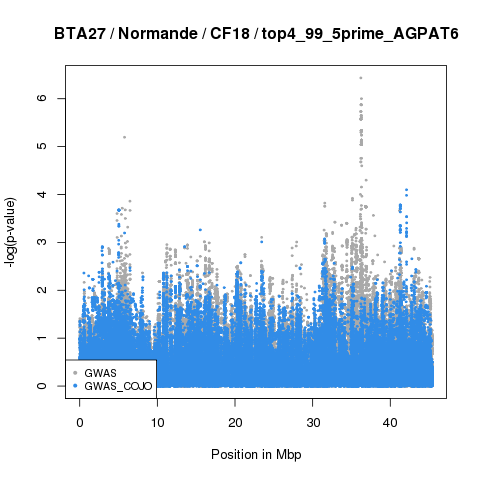
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Holstein

Normande

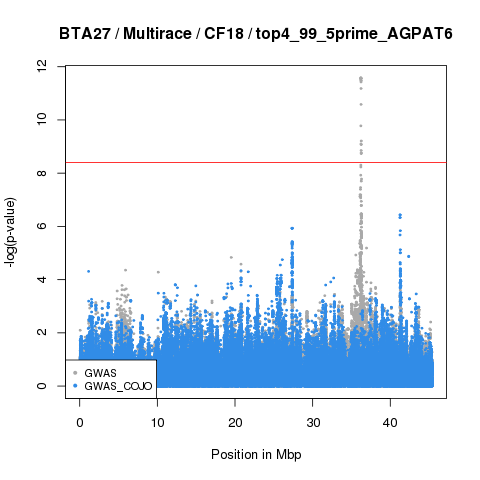
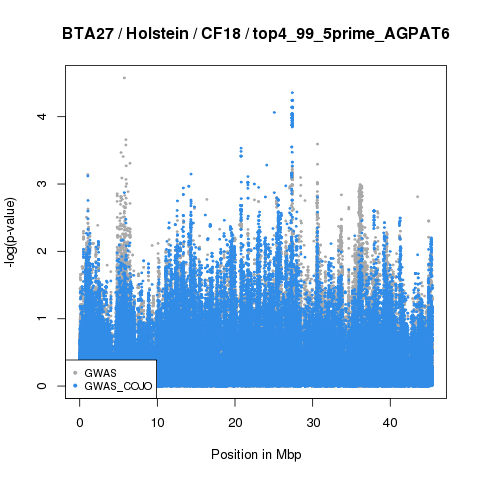
Montbéliarde

**g) On BTA27, for the -CN phenotype, with the fixed effect of one of the five most significant variants located in the *AGPAT6* gene (36,209,319, 36,211,252, 36,211,258, 36,211,708, and 36,212,352 bp) in Montbéliarde, Normande, Holstein, and multi-breed analyses**

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Normande

Montbéliarde

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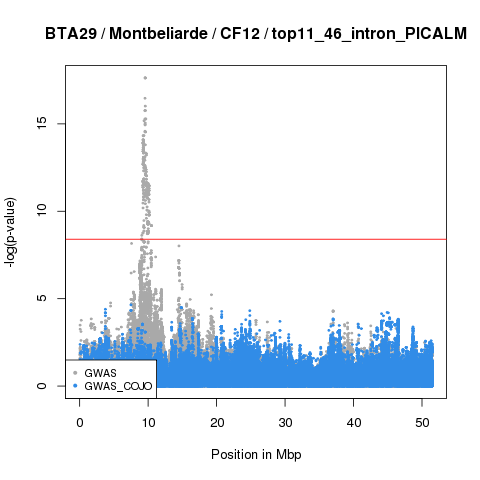
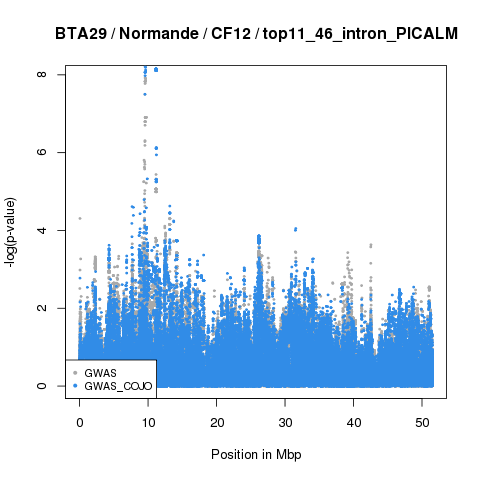
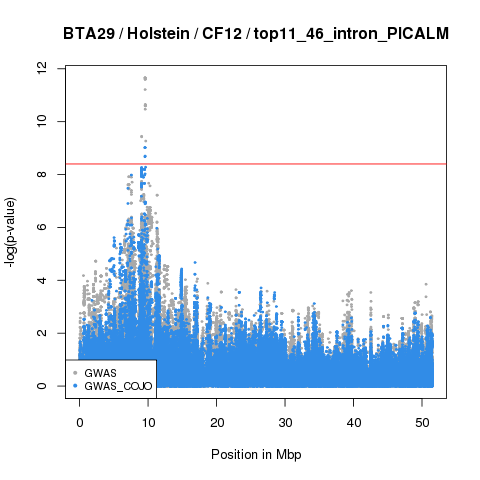
Holstein

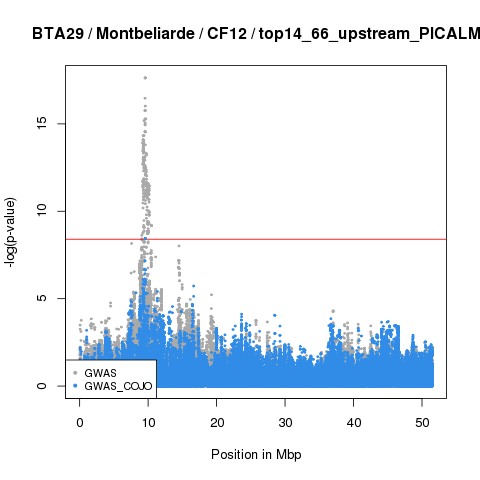
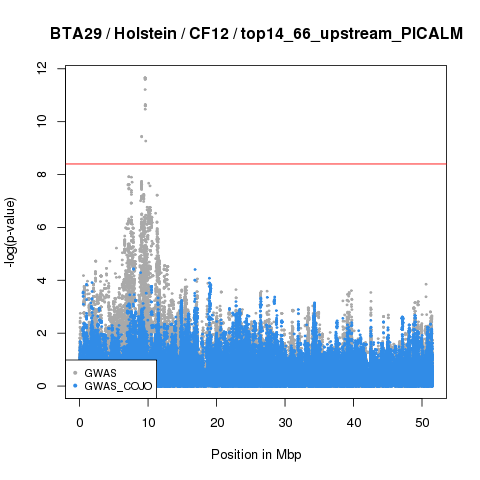
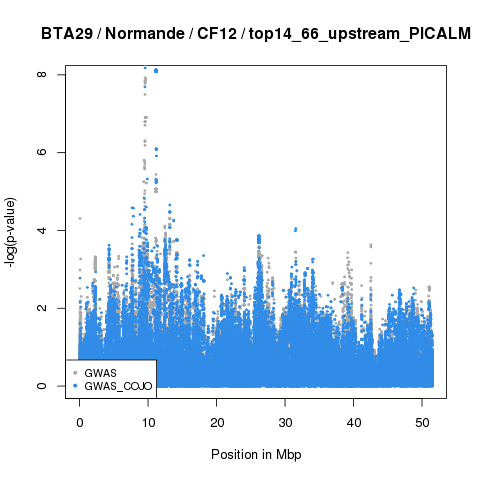
Multi-breed

**h) On BTA29, in Montbéliarde, Normande, and Holstein analyses for the s1-CN phenotype, with the fixed effect of**

h1) the intronic PICALM variants, at 9,651,065 and 9,656,439 bp, ranked 11th in the MON analysis

h2) the upstream PICALM variant, at 9,611,304 bp, ranked 10th in the HOL analysis

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