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Quantitative trait loci for production traits in pigs: a combined analysis of two Meishan × Large White populations

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Summary

Combined analysis of data from two or more resource populations can improve the power and accuracy of QTL mapping and allow some cross-validation of results. In this study, we performed a genome-wide scan using combined data from two F_2 populations derived from a cross between Large White and Chinese Meishan pigs. A total of 739 pigs were included in the analysis. In total 187 markers were genotyped in the two populations, including 115 markers genotyped in both populations, and these markers covered 2282 cM of the pig genome with an average of 13.58 cM between markers. Seven traits (teat number, birth weight, weaning weight, test-end weight, fat depth at shoulder, fat depth at mid back and fat depth at loin) were analysed for both individual populations and the combined population. There were 9 (2, 10), 1 (4, 4) and 14 (5, 18) QTL that achieved 1% genome-wide, 5% genome-wide and suggestive significance levels respectively in population 1 (population 2, combined population). Additive effects of QTL detected in the two populations at all significance levels were largely consistent suggesting that the QTL represent real genetic effects, but this was not the case for dominance or imprinting effects. There were also a number of significant interactions between detected QTL effects and population.

Keywords fat, growth rate, mapping, QTL, swine.

Introduction

Following the first QTL mapping study of pigs in 1994 (Andersson *et al.* 1994), a number of other studies in this species have followed (from Rothschild *et al.* 1995 to Ren *et al.* 2006). At the time of writing, information on 1675 published QTL across 18 pig autosomes and the X and Y chromosomes are recorded in the pig QTL database (PigQTLdb, http://www.animalgenome.org/QTLdb/pig.html). These mapping studies have contributed to the identification of some economically important major genes including *PRKAG3* (Milan *et al.* 2000) and *IGF2* (Van Laere *et al.* 2003).

In most cases, data from different studies have been analysed separately; however, there have been joint analyses of specific chromosomes (Walling *et al.* 2000; Kim *et al.* 2005; Pérez-Enciso *et al.* 2005). Walling *et al.* (2000) collated data from almost 3000 pigs from seven F_2 crosses between Western commercial breeds and either the European wild boar or the Chinese Meishan breed, and scanned chromosome 4 for birth weight (BW), mean backfat depth and growth rate from birth to slaughter or end of test. A QTL influencing BW found in one population was confirmed by the joint analysis. Kim *et al.* (2005) combined the data from a Berkshire × Yorkshire F_2 population and a Berkshire × Duroc F_2 population, and scanned chromosomes 2, 6, 13 and 18 for 26 traits. Based on their results, they suggested that combined analysis using a range of QTL models increased the power of QTL mapping. Pérez-Enciso *et al.* (2005) demonstrated the advantages of a multibreed analysis for analysing the X chromosome with data from five different crosses.

In principle, combined analysis could increase power to detect QTL or confirm the QTL only detected in one population, and improve the accuracy with which QTL parameters are estimated, especially where individual populations were small (Lander & Kruglyak 1995). Combined analysis

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may also permit the testing of more highly parameterized or complicated models with the increased population size, facilitating model testing and estimation of parameters. On the other hand, different populations will have different founder animals, potentially from different breeds. One can gain information from a combined analysis in the form of explicit tests for interaction of QTL parameters with population. Such tests can show that results from different populations are truly different (e.g. Walling *et al.* 2000), a conclusion that it may be difficult or impossible to draw from *post hoc* comparisons of results from populations analysed separately. Nonetheless, if there are interactions between QTL and population, this can negate the potential additional power that can be obtained from the larger population. Furthermore, different populations will often be reared in different environments and with different testing regimes. These environmental effects need to be taken into account in the analysis and may lead to genotype × environment interactions that can decrease the power and accuracy of the analysis of QTL effects in the combined population.

Two populations from Roslin Institute (Edinburgh, UK) were produced by crossing Large White to Chinese Meishan in 1992 and 1995 respectively, and have been partially analysed independently. Walling *et al.* (1998) searched for QTL for growth rate and fat traits on chromosome 4 (SSC4) in the first population (P1). The second population (P2) was used, for example, to perform genome scans for QTL influencing locomotion, osteochondrosis-related traits and boar taint (Lee *et al.* 2003, 2005). In this study, we collate the data from these two F_2 populations, and perform a genome scan for QTL of seven production traits recorded in both populations in the individual datasets and the combined dataset.

Materials and methods

Data collection

Data were collected from two F_2 crosses between Large White and Chinese Meishan at the Roslin Institute. The founders of the two F_2 crosses were taken from different generations of the same purebred populations at Roslin Institute. The first population (P1) was produced in 1992, and 441 F_2 individuals were involved in this study (Walling *et al.* 1998). The second population (P2) was developed in 1995; 292 F_2 individuals were used in this study (Lee *et al.* 2003). The traits common to both populations, as well as the number of observations, mean, standard deviation, minimum and maximum for each trait, are shown in Table 1. To facilitate joint analysis of the data from the two populations, the phenotypes were standardized in each population separately to a mean of zero and variance of one prior to any further analysis. Individuals without any marker genotypes or common trait phenotypes were eliminated from the study. The total number of F_2 animals used in QTL mapping was between 726 [fat depth at loin (LBF)] and 733 [teat number (TN)] in the combined population (P3).

Table 1 The traits measured in the two populations.

Trait	*N*	Mean	SD	Min	Max
P1: TN	441	15.08	1.134	12.00	19.00
BW (kg)	441	1.24	0.248	0.57	2.08
WW (kg)	441	8.23	1.829	2.00	14.50
ETW (kg)	441	81.95	10.621	49.00	118.00
SBF (mm)	440	32.53	7.643	15.00	67.00
LBF (mm)	439	17.50	5.075	5.00	32.00
MBF (mm)	439	18.21	6.058	8.00	43.00
P2: TN	292	14.84	1.348	12.00	18.00
BW (kg)	292	1.23	0.204	0.62	1.72
WW (kg)	292	7.83	2.073	3.50	15.50
ETW (kg)	292	85.15	2.824	76.00	100.00
SBF (mm)	287	29.71	6.177	15.00	48.00
LBF (mm)	287	16.90	3.969	9.00	29.00
MBF (mm)	288	16.09	4.396	9.00	31.00

TN, teat number; BW, body weight at birth; WW, body weight at weaning; ETW, body weight at end test; SBF, fat depth at shoulder; LBF, fat depth at loin; MBF, fat at mid back.

Markers and map construction

There were 171 markers genotyped in P1 and 131 in P2, including 115 common markers. Marker details are available from http://www.thearkdb.org. All 187 markers with at least 280 F_2 animals genotyped were used to produce the linkage maps. Linkage maps of the three populations were re-derived for our data using CRIMAP 2.4 (Green *et al.* 1994). Maps for each chromosome were derived using the BUILD option within CRIMAP assuming the same map distances in males and females. The markers that were not placed on the linkage map by the BUILD option were positioned to their most likely positions using the ALL and FLIPS*n* options. After the initial linkage maps were constructed, the FLIPS*n* option was used to confirm the order of the markers. If the number of markers on a chromosome was ≤8, then *n* was the number of the markers. If the number of markers was >8, then *n* was equal to 6.

QTL analysis

The GLM procedure in SAS was used to determine which factors to include in the QTL mapping analysis (SAS9.0; SAS Institute Inc.). Factors reaching the 5% significance level were kept in the model as a fixed effect or covariate; other factors were dropped from the model. The model for all traits except weaning weight (WW) included sex as a fixed effect, and population was included as a fixed effect in the analysis

of the combined population for the seven traits. Body weight at the end of test was also fitted as a covariate for fat depth at mid back (MBF), fat depth at shoulder (SBF) and LBF. Body weight at birth was fitted as a covariate for WW and test-end weight (ETW), the age of weaning was fitted as a covariate for WW and the age at the end of test was fitted as a covariate for ETW.

The statistical approach adopted for QTL analysis was developed by Haley *et al.* (1994) for a cross between outbred lines. This method is based on an assumption that the QTL are fixed for alternative alleles in the two founder breeds. In this article, we define alleles from Large White and Meishan as *Q* and *q* respectively. The probabilities of each F_2 offspring being each of the four QTL genotypes (*QQ*, *Qq*, *qQ* and *qq*) were estimated at each analysis point in the genome conditionally upon the marker genotypes. A linear model with the additive, dominance and imprinting effects of a QTL at a given position and other fixed effects was fitted by least squares for each trait. The additive and dominance effects of a QTL at a given position were defined as the deviation from the mean of the two homozygotes of animals homozygous for the Large White allele or animals heterozygous for the Large White and Meishan alleles respectively. The imprinting effect was estimated as the difference between the heterozygous animals inheriting a *Q* from their father (*Qq*) and heterozygous animals that received a *Q* from their mother (*qQ*). Negative values for the additive effect means that the allele from Meishan increases the numerical value of the trait. A negative value for the dominance effect indicates that the allele from Meishan is dominant. A negative value for the imprinting effect implies that the QTL exhibits maternal expression.

Significance thresholds were determined by permutation (Churchill & Doerge 1994). Three level threshold values were used: the suggestive, 5% and 1% genome-wide threshold values. The 5% and 1% genome-wide threshold values were obtained directly from the experiment-wise permutation of 1000 iterations (Churchill & Doerge 1994). The 5% chromosome-wide threshold was considered as the suggestive significance level, obtained following de Koning *et al.* (2001) as: $P_{\text{Genome–wide}} = 1 - (1 - P_{\text{Chromosome–wide}})^{1/r}$, where r is the proportion of total genome length attributed to the chromosome.

A forward and backward selection interval mapping approach was used for QTL mapping as follows:

1 The whole genome was scanned for a single QTL. If one or more QTL were detected at the genome-wide suggestive significance level (as determined by permutation) the QTL with biggest *F*-value was considered to be the first QTL. If no QTL were detected, the analysis of this trait in the population would be considered finished.

2 A two-QTL model was used to reanalyse the chromosome where the first QTL was located. In addition, using the first QTL as a genetic background effect, we searched for QTL on the remaining chromosomes.

3 Comparing the significance levels of the tests performed in the second step, the most significant QTL (assuming it at least achieved the suggestive level) was considered the second QTL.

4 Using the second QTL as a genetic background effect, the position and effects of the first QTL were re-estimated. If the position of the first QTL changed, the new parameters of the first QTL were used as a genetic background effect and the position and effect of the second QTL were re-estimated. This iteration was continued until the parameters of the two QTL remained unchanged.

5 Using the two QTL as genetic background effects, the genome was scanned to detect a third QTL.

6 The last four steps were repeated, until no new suggestive or more significant QTL were found when using all previously detected QTL as genetic background effects.

7 Finally, using all of the other QTL as genetic backgrounds, the position and effects of each individual QTL were re-estimated.

The significance level of the estimated additive, dominance and imprinting effects of a QTL was determined by an *F*-distribution. Because the traits of the combined population were standardized, the effects of the QTL in the combined population needed to be back-transformed to the original scale by multiplication by the standard deviation of the particular trait in the relevant population.

To investigate whether the effect of the QTL differed between populations, QTL that reached suggestive or higher significance level in P1, P2 or P3 were reanalysed in P3 including an interaction between the QTL and the population but otherwise using the same model as used in the combined analysis. The QTL for which the interaction was to be tested was fixed at the position where they were found in P1, P2 or P3. An *F*-test was performed to determine the significance level of the interaction between the QTL and the population. The *F*-value was the ratio of the difference between the residual sum of squares (RSS) of the model without population (reduced model) and of the model with it (full model) to the mean square (MS) of the full model, and the numerator and the denominator of the degree of freedom (d.f.) were equal to 3 and the population size minus the number of effects fixed in the full model respectively.

For QTL that reached a 5% genome-wide or higher significance level in the combined population, the interaction between QTL and gender was tested in the combined population using an *F*-test to determine the significance level of the interaction between the QTL and the sex. The steps to calculate the *F*-value and d.f. were analogous to those described for testing interactions between QTL and population. When a QTL reached at least the suggestive significance level the bootstrap approach (Visscher *et al.* 1996) was used to estimate the 95% confidence interval using 2000 bootstrap resamples.

The percentage of trait variance (Var%) explained by each QTL was calculated using following formula:

$$\text{Var}\% = \frac{(\text{MS}_{\text{reduce1}} - \text{MS}_{\text{full}})}{\text{MS}_{\text{reduce}}} \times 100,$$

where MS_{full}, $\text{MS}_{\text{reduce1}}$ and $\text{MS}_{\text{reduce}}$ were the mean squares (MS) of the models containing all detected QTL, containing all detected QTL except the one of interest and omitting all QTL respectively.

Results

Linkage map

Most of the markers had the same order in the three populations, except some very closely linked markers on SSC7 and *TCF1* on SSC14 (Fig. S1). The order of all markers was same as the order of the USDA2.0 average maps (http://www.marc.usda.gov/genome/swine/swine.html) except *S0091* and *SW776* on chromosome 2. These two markers were exchanged in their order, the difference of the LOD score was 9.02 between orders. In order to compare the results in the three populations, only the linkage map derived from the combined data set was used in QTL mapping. The combined linkage map consisted of 187 markers, spanning 2281.9 cM of the whole genome at an average spacing of 13.58 cM between markers.

The marker information content (Knott *et al.* 1998) in population P3 was mostly between 0.5 and 0.9, but dropping below these values in a few troughs between more distantly spaced markers. Information content is shown in Fig. S2.

Thresholds

Separate significance thresholds were estimated for each trait via 1000-iteration experiment-wide permutations. The averages of the suggestive, 5% and 1% genome-wide threshold values and their standard deviations (in parentheses) were 4.08 (0.034), 6.46 (0.128) and 7.78 (0.263) respectively. For a model with fixed interaction between the QTL and the population, the average critical values (and their standard deviations) of the suggestive, 5% and 1% genome-wide significance levels were 2.84 (0.025), 4.14 (0.066) and 4.88 (0.097).

Individual-population analyses

Table 2 gives the results from the individual-population analyses. There were 9 (2), 1 (4) and 14 (5) QTL that achieved 1% genome-wide, 5% genome-wide and suggestive significance level in P1 (P2) respectively. The genome scans revealed some QTL in common in the two populations, especially on SSC1 and SSC7 for fat traits. On the distal end of SSC1, we found a 1% genome-wide QTL for MBF at almost the same position in the two populations. At the same location on SSC1, we also located a QTL for SBF that had the same significance level in the two populations. In the middle of SSC7, we found the same situation. In most cases, allele effects were in the direction expected from the breed difference; however, effects of QTL affecting LBF, MBF and SBF on SSC7 were in the opposite direction, with the Meishan alleles decreasing subcutaneous fat deposition as reported by ourselves and others previously (Rohrer 2000; Yue *et al.* 2003).

There were a number of apparent differences in the QTL detected in the two populations. There were one 5% (MBF, at 77 cM on SSC2) and five 1% genome-wide QTL found in P1, with no equivalent QTL found in P2. Two 5% genome-wide QTL were mapped in P2 where no QTL were found in P1.

Combined analyses

The results from the combined analyses are given in Table 3. There were 10, 4 and 18 QTL that achieved 1% genome-wide, 5% genome-wide and suggestive significance levels respectively. Almost all the QTL at 1% genome-wide significance in P1 and P2 also reached 1% genome-wide significance in P3 (Fig. 1a,b) except the two QTL for WW and ETW in P1, which however achieved 5% genome-wide significance in P3 (Fig. 1c,d). One QTL for MBF at the 5% genome-wide significance level mapped to SSC2 in P1 and was found as a suggestive QTL in P3, whereas another QTL at the same significance level for TN on SSC16 was detected in P2 but was not found in P3. Other 5% genome-wide QTL found in P1 or P2 were detected in P3 at least at the same significance level. Seven suggestive QTL (four from P1 and three from P2) were also identified in the combined population, where two QTL (one from each population) reached the 5% genome-wide significance level and one QTL (from P1) reached the 1% genome-wide significance level. However, most QTL significant only at the suggestive level in the individual populations (10 in P1 and two in P2) were not identified in P3.

Interaction between QTL and population

The significant results from the test for the interactions between QTL and population are given in Table 4. Twelve QTL that reached the suggestive or higher significance level found in P1 and/or P2 had significant interactions with population. In nine instances for a QTL detected in P1 there was no evidence of a similar effect in P2 (BW, SSC7; ETW, SSC2, 4, 6 and 18; MBF, SSC2; TN, SSC3 and 17 and WW, SSC1) and in one instance for a QTL detected in P2 there is no evidence for a similar effect in P1 (SBF, SSC5). Despite the lack of evidence for a QTL in one of the populations and evidence of a significant interaction, in three of these cases analysis of the combined population ignoring the interaction identified QTL significant at the suggestive level (MBF on SSC2) or at the 5% genome-wide significance level (ETW

Table 2 Results of the QTL mapping of the individual populations.

Trait	Population	Chr	Position (cM)	*F*-value[1]	CI	ADD ± SE[2]	DOM ± SE[2]	IMP ± SE[2]	Var (%)
BW	P1	7	125	4.59	66–159	−0.02 ± 0.02	**−0.11 ± 0.03**	0.04 ± 0.02	2.32
BW	P1	16	0	4.69	0–110	***−0.13 ± 0.04***	−0.07 ± 0.11	0.01 ± 0.04	2.48
ETW	P1	2	73	4.20	15–126	**−1.81 ± 0.70**	1.36 ± 1.02	1.48 ± 0.75	1.95
ETW	P1	4	61	***11.01***	52–100	***4.72 ± 0.82***	0.76 ± 1.16	0.04 ± 0.74	6.16
ETW	P1	6	43	6.11	29–193	*3.85 ± 1.20*	**−6.53 ± 2.78**	2.22 ± 1.22	3.12
ETW	P1	18	12	5.63	0–45	*2.16 ± 0.85*	**3.34 ± 1.49**	**1.83 ± 0.89**	2.82
LBF	P1	5	107	5.01	0–125	*−0.89 ± 0.43*	−1.21 ± 0.67	**−1.18 ± 0.41**	2.52
LBF	P1	7	74	***11.94***	49–104.5	***2.25 ± 0.36***	−0.81 ± 0.56	−0.13 ± 0.37	6.87
LBF	P1	9	26	4.16	0–131	*0.97 ± 0.41*	−1.49 ± 0.69	0.57 ± 0.47	1.98
MBF	P1	1	133	***8.98***	46.5–134	***−1.44 ± 0.31***	**−0.91 ± 0.44**	−0.23 ± 0.32	4.67
MBF	P2	1	134	***10.23***	123–134	***−1.34 ± 0.26***	**−0.97 ± 0.41**	0.59 ± 0.31	7.69
MBF	P1	2	77	**6.87**	9–129.5	*−0.98 ± 0.30*	**1.00 ± 0.45**	**0.73 ± 0.32**	3.43
MBF	P2	5	68	**7.55**	18–93	*−1.04 ± 0.32*	−0.16 ± 0.49	***−1.05 ± 0.30***	5.45
MBF	P2	5	135	5.85	7–135	−0.55 ± 0.29	−0.80 ± 0.42	***1.10 ± 0.34***	4.04
MBF	P1	7	72	***12.79***	52–83	***1.81 ± 0.31***	−0.37 ± 0.43	−0.53 ± 0.29	6.90
MBF	P2	7	63	**6.74**	52–97.5	***1.45 ± 0.33***	0.09 ± 0.49	0.19 ± 0.30	4.78
MBF	P2	9	72	4.42	0–89	0.09 ± 0.29	−0.89 ± 0.41	**0.73 ± 0.27**	2.85
MBF	P1	11	50	4.70	18–76	−0.39 ± 0.32	0.44 ± 0.49	*−1.12 ± 0.34*	2.16
MBF	P2	X	66	4.87	0–92	−0.76 ± 0.32	**1.07 ± 0.45**	**0.61 ± 0.29**	3.23
SBF	P1	1	131	***9.15***	109–134	***−2.26 ± 0.48***	−1.66 ± 0.72	−0.20 ± 0.50	5.00
SBF	P2	1	123	***8.06***	0–132	***−1.87 ± 0.54***	*−3.11 ± 0.90*	0.02 ± 0.56	6.27
SBF	P1	2	79	6.18	7.5–112	*−1.42 ± 0.45*	0.86 ± 0.71	*1.34 ± 0.50*	3.18
SBF	P2	5	132	4.74	0–135	*−1.33 ± 0.49*	−1.74 ± 0.79	0.93 ± 0.57	3.32
SBF	P1	7	69	***8.60***	50–159	***2.11 ± 0.46***	−0.91 ± 0.63	−0.80 ± 0.43	4.67
SBF	P2	7	70	**6.69**	0–159	***2.20 ± 0.59***	0.33 ± 0.96	**1.20 ± 0.56**	5.05
SBF	P2	X	64	6.51	0–74	***−1.93 ± 0.54***	1.63 ± 0.79	**1.02 ± 0.50**	4.89
TN	P1	1	114	***9.18***	34–132	***0.53 ± 0.10***	0.05 ± 0.19	−0.02 ± 0.10	4.81
TN	P1	3	108	4.38	0–118	*−0.21 ± 0.07*	0.19 ± 0.11	−0.11 ± 0.08	1.99
TN	P1	4	53	5.42	0–68	***−0.29 ± 0.09***	−0.25 ± 0.14	−0.17 ± 0.08	2.59
TN	P1	6	0	5.27	0–220	***0.33 ± 0.10***	0.14 ± 0.19	0.20 ± 0.11	2.51
TN	P1	12	85	***7.93***	2–97	***−0.59 ± 0.12***	0.12 ± 0.26	−0.01 ± 0.12	4.07
TN	P2	16	6	**6.55**	0–110	*0.53 ± 0.17*	0.35 ± 0.40	**0.67 ± 0.22**	5.45
TN	P1	17	36	5.56	26.5–103	*0.22 ± 0.08*	**0.28 ± 0.11**	0.09 ± 0.08	2.68
WW	P1	1	114	***9.70***	54–129	***−0.63 ± 0.13***	0.36 ± 0.24	0.16 ± 0.13	5.57
WW	P1	17	0	4.42	0–103	−0.19 ± 0.15	0.03 ± 0.30	*−0.42 ± 0.14*	2.18

See Table 1 for expansions.

Var (%), percentage of trait variance explained by the QTL.

[1]Regular = suggestive significant, bold = 5% genome-wide significant, bold and italic = 1% genome-wide significant.

[2]Regular = not significant, bold = $P < 0.05$, italic = $P < 0.01$, bold and italic = $P < 0.001$.

on SSC4 and WW on SSC1). In one case for a QTL detected in P2 there was a QTL with a similar additive effect in P1, but unlike P2, P1 displayed no evidence for an imprinting effect (MBF; SSC5). Just one QTL found in both P1 and P2 had a significant interaction with the population (the QTL of SBF on SSC7). The estimates of the additive and dominance effect were similar in the two populations so the imprinting effect that was only found in P2 may have caused the interaction between QTL and population.

Interaction between QTL and sex

The QTL detected in P3 had no significant interactions with sex except the QTL for SBF on the X chromosome. The interaction on this chromosome was caused by the interaction between the sex and dominance effects and the interaction between the sex and imprinting effects, not by the interaction between the sex and additive effects of the QTL ($P = 0.7521$). This is a technical artefact resulting from the impossibility of estimating these effects in heterogametic males.

Discussion

Earlier studies have combined data from two or more populations to scan selected chromosomes to map QTL (Walling *et al.* 2000; Kim *et al.* 2005), but to our knowledge this study is the first genome-wide scan to map QTL in pigs in this way. From the results presented here, the benefits of the combined analysis are apparent. More QTL were mapped in

Table 3 Results from the QTL mapping of the combined population.

					P1			P2			
Trait	Chr	Position (cM)	*F*-value[1]	CI	ADD ± SE[2]	DOM ± SE[2]	IMP ± SE[2]	ADD ± SE[2]	DOM ± SE[2]	IMP ± SE[2]	Var (%)
BW	1	0	4.66	0–125	***0.05 ± 0.02***	0.03 ± 0.03	−0.01 ± 0.02	***0.05 ± 0.01***	0.02 ± 0.02	−0.01 ± 0.01	1.47
BW	14	52	4.60	4–113	***0.05 ± 0.01***	−0.01 ± 0.02	0.00 ± 0.01	***0.04 ± 0.01***	−0.01 ± 0.02	0.00 ± 0.01	1.44
ETW	3	43	4.38	3–132	***2.24 ± 0.63***	0.41 ± 0.99	−0.50 ± 0.66	***0.60 ± 0.17***	0.11 ± 0.26	−0.13 ± 0.18	1.33
ETW	4	61	**6.56**	29.5–112	***2.84 ± 0.65***	−0.09 ± 0.96	−0.40 ± 0.62	***0.75 ± 0.17***	−0.02 ± 0.26	−0.11 ± 0.17	2.19
ETW	7	67	5.81	26.5–143	*−2.06 ± 0.63*	**2.11 ± 0.91**	0.80 ± 0.59	*−0.55 ± 0.17*	**0.56 ± 0.24**	0.21 ± 0.16	1.89
LBF	1	134	4.56	0–134	***−0.85 ± 0.25***	−0.49 ± 0.35	0.12 ± 0.26	***−0.66 ± 0.20***	−0.38 ± 0.28	0.10 ± 0.21	1.38
LBF	5	108	4.28	0–117	−0.53 ± 0.28	−0.67 ± 0.44	−0.74 ± 0.28	−0.42 ± 0.22	−0.52 ± 0.34	−0.58 ± 0.22	1.27
LBF	7	74	**12.0**	62–113	***1.62 ± 0.28***	−0.34 ± 0.42	0.16 ± 0.27	***1.27 ± 0.22***	−0.26 ± 0.33	0.13 ± 0.21	4.26
LBF	X	65	5.59	0–74.5	***−1.21 ± 0.32***	0.85 ± 0.53	0.33 ± 0.31	***−0.95 ± 0.25***	0.66 ± 0.42	0.25 ± 0.24	1.77
MBF	1	134	**18.5**	125–134	***−1.84 ± 0.28***	***−1.41 ± 0.40***	0.10 ± 0.30	***−1.34 ± 0.20***	***−1.02 ± 0.29***	0.08 ± 0.21	5.91
MBF	2	76	5.01	4–130	*−0.79 ± 0.27*	0.23 ± 0.41	**0.73 ± 0.29**	*−0.57 ± 0.20*	0.17 ± 0.30	**0.53 ± 0.21**	1.36
MBF	4	80	5.78	23–121	−0.46 ± 0.29	0.94 ± 0.42	***0.84 ± 0.29***	−0.33 ± 0.21	0.68 ± 0.30	***0.61 ± 0.21***	1.61
MBF	5	69	**8.46**	51–107	***−1.42 ± 0.30***	0.56 ± 0.46	−0.35 ± 0.29	***−1.03 ± 0.22***	0.40 ± 0.34	−0.26 ± 0.21	2.51
MBF	7	71	**18.8**	59–75	***2.22 ± 0.31***	−0.8 ± 0.44	−0.22 ± 0.29	***1.61 ± 0.22***	−0.58 ± 0.32	−0.16 ± 0.21	6.00
MBF	9	70	4.97	0–117	**0.56 ± 0.28**	−0.84 ± 0.39	**0.61 ± 0.28**	**0.41 ± 0.20**	−0.61 ± 0.28	**0.44 ± 0.20**	1.33
MBF	10	88	4.37	7–115	−0.64 ± 0.34	−0.29 ± 0.57	***1.07 ± 0.33***	−0.47 ± 0.25	−0.21 ± 0.42	***0.77 ± 0.24***	1.13
MBF	12	16	5.98	0–57.5	−0.23 ± 0.33	*1.84 ± 0.53*	−0.79 ± 0.33	−0.17 ± 0.24	*1.33 ± 0.39*	−0.58 ± 0.24	1.68
MBF	X	64	5.06	0–92	−1.04 ± 0.36	0.72 ± 0.60	**0.93 ± 0.35**	−0.76 ± 0.26	0.52 ± 0.44	**0.67 ± 0.25**	1.37
SBF	1	126	**14.4**	117–134	***−2.22 ± 0.42***	***−2.68 ± 0.68***	0.03 ± 0.43	***−1.79 ± 0.34***	***−2.16 ± 0.55***	0.03 ± 0.35	4.85
SBF	2	82	**7.55**	63–111	***−1.46 ± 0.36***	0.35 ± 0.57	**0.93 ± 0.39**	***−1.18 ± 0.29***	0.28 ± 0.46	**0.75 ± 0.31**	2.36
SBF	7	69	**11.0**	62–80	***2.19 ± 0.40***	−0.91 ± 0.57	0.18 ± 0.37	***1.77 ± 0.32***	−0.74 ± 0.46	0.14 ± 0.30	3.61
SBF	10	122	4.16	4–122	−0.58 ± 0.42	0.24 ± 0.70	***1.45 ± 0.44***	−0.47 ± 0.34	0.19 ± 0.57	***1.17 ± 0.36***	1.14
SBF	X	63	**7.37**	0–68	***−1.74 ± 0.48***	*2.09 ± 0.81*	**0.94 ± 0.46**	***−1.41 ± 0.39***	**1.69 ± 0.66**	**0.76 ± 0.37**	2.30
TN	1	110	**13.1**	53.5–128	***0.45 ± 0.07***	0.06 ± 0.12	−0.03 ± 0.08	***0.54 ± 0.09***	0.07 ± 0.14	−0.03 ± 0.09	4.43
TN	4	52	6.11	2–83	*−0.19 ± 0.07*	−0.15 ± 0.11	***−0.21 ± 0.07***	*−0.23 ± 0.08*	−0.18 ± 0.13	***−0.24 ± 0.08***	1.87
TN	7	107	5.44	20–132	***0.26 ± 0.07***	0.05 ± 0.11	−0.07 ± 0.07	***0.31 ± 0.08***	0.07 ± 0.13	−0.08 ± 0.09	1.63
TN	10	91	**8.57**	69–102	***0.31 ± 0.08***	−0.13 ± 0.11	***−0.24 ± 0.08***	***0.36 ± 0.09***	−0.16 ± 0.13	***−0.28 ± 0.10***	2.78
TN	10	51	6.36	30–69	***−0.29 ± 0.08***	−0.13 ± 0.11	0.15 ± 0.08	***−0.34 ± 0.09***	−0.16 ± 0.14	0.18 ± 0.10	1.96
TN	11	0	4.54	0–56	−0.1 ± 0.06	0.03 ± 0.09	***−0.21 ± 0.07***	−0.12 ± 0.07	0.04 ± 0.11	***−0.25 ± 0.08***	1.30
TN	12	84	**8.47**	17–97	***−0.46 ± 0.09***	−0.13 ± 0.20	−0.04 ± 0.09	***−0.55 ± 0.11***	−0.16 ± 0.23	−0.05 ± 0.11	2.74
WW	1	130	**6.68**	16–134	***−0.32 ± 0.08***	−0.02 ± 0.11	0.09 ± 0.08	***−0.37 ± 0.09***	−0.03 ± 0.13	0.10 ± 0.09	2.25
WW	17	0	**7.29**	0–38.5	−0.16 ± 0.12	−0.09 ± 0.26	***−0.48 ± 0.12***	−0.18 ± 0.13	−0.1 ± 0.30	***−0.54 ± 0.13***	2.49

See Table 1 for expansions.

Var (%), percentage of trait variance explained by the QTL.

[1]Regular = suggestive significant, bold = 5% genome-wide significant, bold and italic = 1% genome-wide significant.

[2]Regular = non-significant, bold = $P < 0.05$, italic = $P < 0.01$, bold and italic = $P < 0.001$.

the combined population (Tables 2 & 3), detected QTL were mapped more accurately and the analysis of population interactions provided a diagnostic indication of the robustness of the QTL.

There were eight more QTL (one 1% genome-wide, three 5% genome-wide and four suggestive) in P3 than in P1, and 21 more QTL (eight 1% genome-wide and 13 suggestive) in P3 than in P2. An imprinted QTL for TN on SSC10 (between *SW1041* and *SW951*) that was identified in P3 (at the 1% genome-wide significance level) but not in either P1 or P2 confirms earlier research. Hirooka *et al.* (2001) found a QTL ($P < 0.1\%$ genome-wide) for TN in the same region (between *SW920* and *SW951*), and other researchers have mapped a QTL for TN in the vicinity (Rohrer 2000; Dragos-Wendrich *et al.* 2003; Rodriguez *et al.* 2005). In this case, the larger sample size from our combined analysis has allowed us to identify two linked QTL with opposite effects some 40 cM apart. The effects of these QTL tend to cancel each other out and the smaller sample sizes of the individual populations were not sufficient to tease them apart (Tables 1 & 3).

The single QTL found at the 1% significance level in the P3 population but not detected in either P1 or P2 is discussed above. But note that there were also 13 suggestive QTL found in P3 that were not found in either P1 or P2. There were only four QTL/trait combinations that were significant at any level in both P1 and P2 and these represented the two major QTL affecting fatness (MBF and SBF) on SSC1 and SSC7. There were 20 QTL/trait combinations significant in P1 but not significant in P2 and there were seven QTL/trait combinations significant in P2 but not P1.

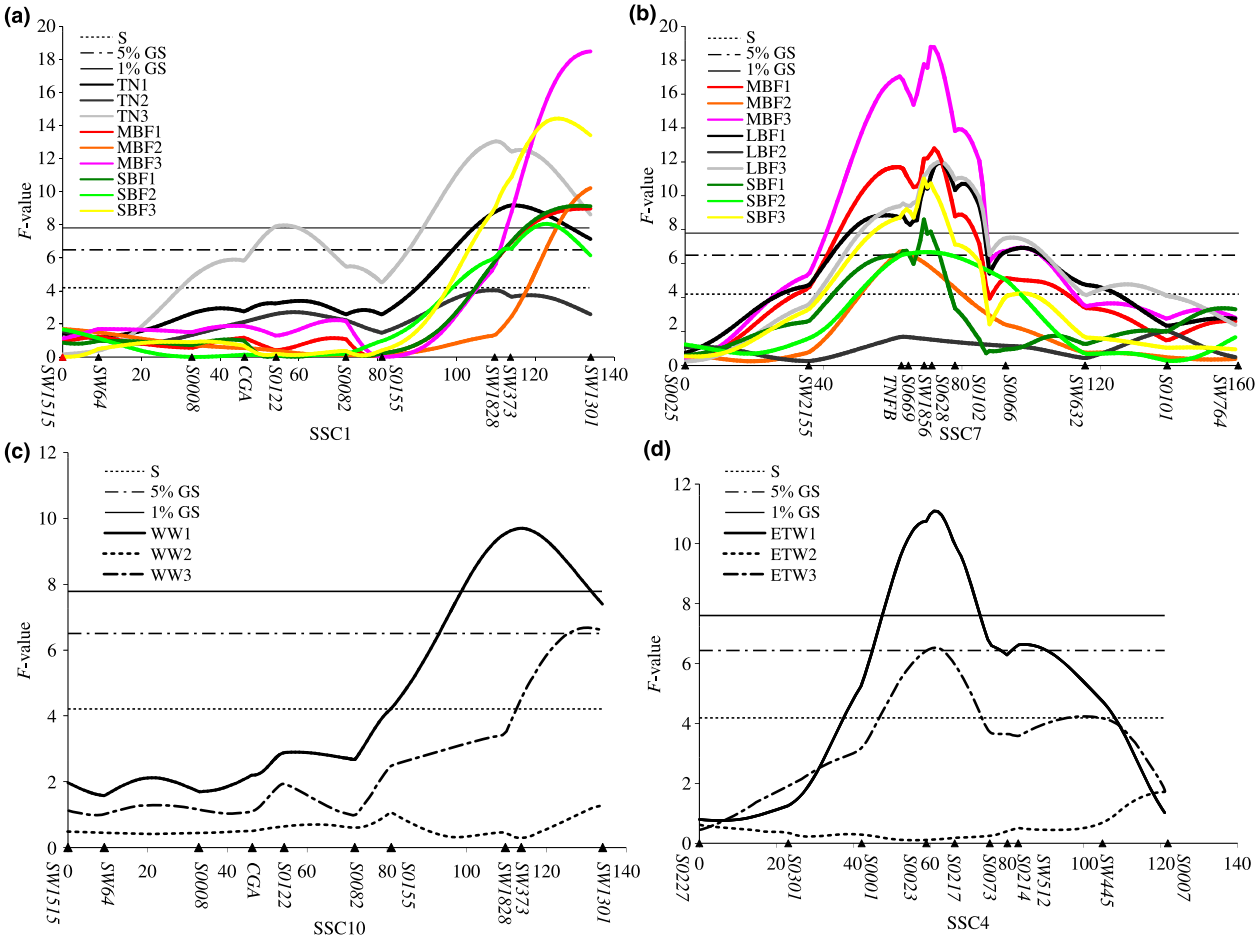

Figure 1 Estimated test statistics across the chromosomes. S, 5% GS and 1% GS mean the critical value of the suggestive significance level, the 5% genome-wide significance level and the 1% genome-wide significance level respectively. (a) Chromosome 1, (b) chromosome 7, (c) chromosome 10, (d) chromosome 4.

Table 4 Significant interactions between the QTL and population.

				P1			P2				
Trait	Chr	Position (cM)	*F*-value[1]	ADD ± SE[2]	DOM ± SE[2]	IMP ± SE[2]	ADD ± SE[2]	DOM ± SE[2]	IMP ± SE[2]	*P*-value[3]	Var (%)
BW	7	125	2.62	−0.02 ± 0.02	−0.11 ± 0.03	0.04 ± 0.02	0.00 ± 0.02	0.04 ± 0.04	0.01 ± 0.02	*0.032*	1.29
ETW	2	73	*3.41*	*−1.66 ± 0.73*	1.28 ± 1.07	*1.52 ± 0.77*	0.29 ± 0.23	**1.00 ± 0.36**	−0.15 ± 0.25	*0.013*	1.88
ETW	4	61	***4.86***	***4.50 ± 0.85***	0.36 ± 1.20	−0.52 ± 0.77	0.15 ± 0.27	−0.14 ± 0.43	−0.09 ± 0.28	*0.026*	3.01
ETW	6	43	2.53	**3.25 ± 1.24**	−4.78 ± 2.91	1.82 ± 1.27	0.15 ± 0.26	0.75 ± 0.40	0.08 ± 0.27	*0.036*	1.19
ETW	18	12	*3.46*	*1.92 ± 0.89*	*3.44 ± 1.53*	1.59 ± 0.92	0.60 ± 0.32	−0.47 ± 0.52	−0.48 ± 0.30	*0.017*	1.92
MBF	2	76	**4.31**	−1.11 ± 0.35	1.00 ± 0.52	**1.14 ± 0.38**	−0.17 ± 0.31	−0.70 ± 0.47	0.11 ± 0.32	*0.014*	2.21
MBF	5	69	***5.71***	−1.20 ± 0.38	1.13 ± 0.59	0.17 ± 0.38	***−1.28 ± 0.35***	−0.31 ± 0.55	−0.87 ± 0.34	*0.034*	3.14
SBF	5	132	*3.42*	0.19 ± 0.49	0.37 ± 0.77	−0.77 ± 0.56	−1.27 ± 0.49	−2.05 ± 0.78	*1.25 ± 0.56*	***<0.001***	1.72
SBF	7	69	***7.07***	***1.86 ± 0.48***	−0.94 ± 0.65	−0.50 ± 0.44	***2.27 ± 0.58***	−0.70 ± 0.93	*1.37 ± 0.54*	*0.029*	4.34
TN	3	108	2.2	*−0.16 ± 0.08*	0.13 ± 0.11	−0.14 ± 0.09	0.01 ± 0.12	−0.15 ± 0.17	0.23 ± 0.13	*0.021*	0.88
TN	17	36	*3.41*	**0.24 ± 0.08**	*0.23 ± 0.11*	0.11 ± 0.08	−0.08 ± 0.14	−0.28 ± 0.23	0.21 ± 0.15	*0.030*	1.74
WW	1	130	***5.08***	***−0.47 ± 0.10***	0.16 ± 0.15	0.11 ± 0.10	−0.10 ± 0.14	−0.36 ± 0.21	0.06 ± 0.14	*0.017*	3.19

See Table 1 for expansions.

Var (%), percentage of trait variance explained by the QTL.

[1]Regular = non-significant, italic = suggestive significant, bold = 5% genome-wide significant, bold and italic = 1% genome-wide significant.

[2]Regular = non-significant, italic = $P < 0.05$, bold = $P < 0.01$, bold and italic = $P < 0.001$.

[3]*P*-value of the interaction test; italic = $P < 0.05$, bold and italic = $P < 0.001$.

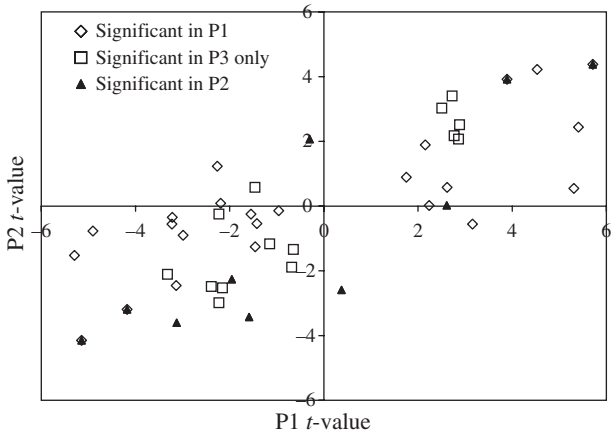

Figure 2 Additive effect *t*-values in populations P1 and P2 for QTL significant in P1 and P2 and for QTL only significant in the combined population (P3).

However, consistency between the two populations was greater than it appears above. For QTL detected as significant at any level in P1, we can compare test statistics for estimated additive effects in P1 (selected as the larger and hence more powerful study) with those in the independent P2 population. Figure 2 plots the estimated *t*-value for the additive effect of the QTL (i.e. the estimated additive effect over its standard error) in P2 against that from P1 taken from the analysis of QTL × population interactions. There was a strong positive relationship between these two *t*-values, with the correlation between the *t*-values being 0.79. This indicates that discrepancies in QTL detected in the two populations was in large part due to the limited power of the individual populations to detect QTL, such that a QTL detected in one population was often not replicated in the second simply because the power of detection was low. From this, we may conclude that most of the QTL detected at the suggestive level or higher in the individual populations are likely to be true effects rather than false positives (i.e. type I errors). The slope of the regression of P2 *t*-values onto P1 *t*-values was 0.46. Thus the additive effects were generally less significant in P2 than in P1. This is presumably partly a reflection of the smaller size of the P2 population, but may also reflect the well-known effect of stringent significance thresholds and low power in inflating estimates of QTL effects in the population in which they were detected.

Figure 2 also shows estimated *t*-values for additive effects in P1 and P2 for QTL that were detected as significant at the suggestive level or higher either in P2 or only in P3 (but in this latter case not in P1 or P2 on their own). Again these showed a very strong positive relationship, with correlations of 0.88 and 0.90 respectively. In this case, the slopes of the regression of P2 *t*-values onto P1 *t*-values were 0.88 and 0.89 respectively. Although this consistency gives some confidence that these effects are also true positives, we should also note that detection of a QTL in P3 when it has been detected in neither P1 nor P2 is only likely when the effects in the two populations are consistent. So a high correlation of estimates between P1 and P2 for QTL only detected in P3 is not unexpected.

To estimate the importance of the additive effect of the detected QTL in causing the phenotypic difference between Large White and Meishan, we summed the additive effects of the QTL of the traits, after back-transformation to their original scales, and compared them where possible with the reported phenotypic difference between Large White and Meishan (Haley *et al.* 1992, 1995). More than 40% of the total phenotypic difference between the breeds can be accounted for by the estimated effects of the detected QTL for the traits BW, LBF, MBF and SBF (Table 5). However, these are likely to be overestimates due to selection of those effects passing a relatively stringent significant threshold. TN additive effects explain little of the breed difference but imprinting effects are surprisingly important (Table 3). The estimated additive QTL effects for WW are in the opposite direction from those expected from the breed contrast. However, most of the difference in WW between Meishan and Large White pigs is controlled by the genotype of the dam and not that of the piglet (Haley *et al.* 1995). In this QTL mapping study, we were examining the effect of individuals' genotypes and hence QTL on their own WW. Haley

Table 5 The total additive effects explained by the detected QTL in P3.

Trait	Var (%)	$P1_{LW\text{-}MS}$[1]	$P2_{LW\text{-}MS}$[1]	$R_{LW\text{-}MS}$[1]	Reference
BW (kg)	2.91	0.214	0.176	0.450	Haley *et al.* (1992)
ETW (kg)	5.41	6.031	1.603	–	–
LBF (mm)	8.68	−1.951	−1.526	−4.365	Haley *et al.* (1992)
MBF (mm)	22.90	−7.285	−5.287	−7.300	Haley *et al.* (1992)
SBF (mm)	14.26	−7.628	−6.165	−12.165	Haley *et al.* (1992)
TN	16.71	−0.038	−0.046	−2.880	Haley *et al.* (1995)
WW (kg)	4.74	−0.972	−1.101	2.008	Haley *et al.* (1992)

See Table 1 for expansions.

Var (%), percentage of trait variance explained by all of the detected QTL in P3.

[1]$P1_{LW\text{-}MS}$, $P2_{LW\text{-}MS}$ and $R_{LW\text{-}MS}$ are the estimated summed additive effects between Large White and Meishan in P1 and P2 and the estimated purebred difference taken from the references respectively.

et al. (1995) estimated that the additive direct effect of the mean piglet weight at weaning was −0.44 (without linear covariates) and −0.28 (with linear covariates); i.e. the genotype of a Meishan piglet acted to increase its WW. In contrast, the larger effect of its dam was in the opposite direction. Our QTL results are thus consistent with these results in showing a positive effect of Meishan alleles in the piglet on WW. The detected QTL explain an estimated 2.9% (for BW) to 22.9% (for MBF) of the total trait variance in the F_2 population. Even for TN, where little of the breed difference is accounted for, 16.7% of the variance in the F_2 is explained (as the balance of positive and negative QTL effects explains little of the breed difference whilst causing substantial genetic variance in the F_2). Note that we cannot easily relate these estimates of variance explained to the total genetic variance in the population as the latter is difficult to predict or estimate accurately with the data available here on a cross between two outbred populations.

Twenty-six of the 32 QTL significant at the suggestive or higher level in the combined analysis have significant additive effects (Table 3) and we have seen above that these seem relatively consistent across populations. Only six of the 32 QTL have evidence of a significant dominance component, whilst a surprising 13 have a significant imprinting effect (more properly named a parent-of-origin effect as we have no evidence for its biological cause). However, the dominance and imprinting effects appear much less consistent across populations than the additive effect. For QTL detected as significant in P1, the correlation with P2 of estimated *t*-values for dominance effects is −0.02 and for imprinting effects is 0.21, neither of these correlations being significantly different from zero. This result provides less confidence in these estimates than in the estimates of the additive effects. This comes about presumably because most of the dominance (81.25%) and imprinting (59.38%) effects are not significantly greater than zero and their estimators are not reliable.

The direct test for interactions between QTL effects and population highlights some real differences between populations P1 and P2. This finding underlines the fact that there is more to the differences between the two populations than just an issue of low power, meaning that a QTL detected in one population may not be detected in a second. These interactions could be due to a number of factors, such as incomplete standardization of the data between populations, or real genetic interactions between QTL and background genotype or environment or perhaps genetic heterogeneity between population founders (e.g. due to segregation of QTL within breeds). Where QTL demonstrate interactions with population we should be more careful in interpreting their results and taking them forward for further study. However, even in the set of QTL with significant interactions, there was a level of consistency between populations. For the 10 QTL detected in P1 that showed a significant interaction, the correlation of the *t*-values of additive effects in populations P1 and P2 was 0.50, with the test for significance from zero being suggestive ($P = 0.05$–0.10).

Combined analysis can also narrow the confidence interval of the estimated location of a QTL. It can be seen that almost all QTL at 5% genome-wide significance in one population were repeated in at least a second population at the suggestive or higher significance levels (with the exception of the QTL for TN on SSC10 in population P3 and the QTL for TN on SSC16 in P2). Thus, we used the results from these 16 QTL to compare the estimated confidence intervals between populations. When compared to P1 the confidence intervals were decreased by 32% in P3 and when compared to P2 the confidence intervals were decreased by 45% in P3, with the population size increasing by 66% or 150% respectively. These reductions in the confidence interval may translate to commensurate reductions in the number of markers required to span the region in follow-up studies aimed at improving resolution or tracking the inheritance of the QTL in other samples and populations.

In conclusion, joint analysis of two data sets has provided greater power to detect QTL and greater confidence in the reality of detected QTL and improved genetic estimates. The challenges of combining data from more diverse sources than those analysed here would be greater, but our earlier studies have shown that these challenges are not insurmountable and the rewards make such analyses worth pursuing.

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Supporting information

Additional supporting information may be found in the online version of this article.

Figure S1 The sex-averaged linkage maps for the three populations. (Pop1 = population 1; Pop2 = population 2; Joint = combined population.)

Figure S2 Information content. Markers genotyped only in population 1 (regular font), in population 2 (italic font) and in both populations (bold font). Map distances were based on the combined data. The average information contents were the mean of additive content, dominance content and imprint content using marker data from population 1 (squares), population 2 (triangles) and the combined data (solid line).

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