Genetics and population analysis

Bayesian robust analysis for genetic architecture of quantitative traits

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ABSTRACT

Motivation: In most quantitative trait locus (QTL) mapping studies, phenotypes are assumed to follow normal distributions. Deviations from this assumption may affect the accuracy of QTL detection and lead to detection of spurious QTLs. To improve the robustness of QTL mapping methods, we replaced the normal distribution for residuals in multiple interacting QTL models with the normal/ independent distributions that are a class of symmetric and long-tailed distributions and are able to accommodate residual outliers. Subsequently, we developed a Bayesian robust analysis strategy for dissecting genetic architecture of quantitative traits and for mapping genome-wide interacting QTLs in line crosses.

Results: Through computer simulations, we showed that our strategy had a similar power for QTL detection compared with traditional methods assuming normal-distributed traits, but had a substantially increased power for non-normal phenotypes. When this strategy was applied to a group of traits associated with outliers, substantially increased power and decreased false positives were observed. Subsequently, we developed a Bayesian robust analysis strategy for dissecting genetic architecture of quantitative traits and for mapping genome-wide interacting QTLs in line crosses.

Supplementary information: Supplementary data are available at Bioinformatics online.

1 INTRODUCTION

In experimental line crosses, most parametric methods for mapping quantitative trait locus (QTL) fall into one of three types of approaches, least-squares, maximum likelihood or Bayesian approach. A common characteristic of these methods is that they all assume normally distributed phenotypes. However, many traits do not follow normal distributions, such as survival time, and others may be the result of human measurement error. This deviation from the normality assumption by phenotypes can render many QTL mapping approaches inappropriate, in senses of less accuracy and effectiveness in QTL detection (Coppieters et al., 1998), and unstable results due to outliers (Pinheiro et al., 2001).

To improve the robustness, various approaches have been developed to deal with non-normal phenotypes in QTL mapping. A simple approach is to adopt parametric methods known for their robustness. However, their robustness for non-normal phenotypes is difficult to establish (e.g. Coppieters et al., 1998; Hackett, 1997; Jansen, 1992; Rebaï, 1997). A second approach is to convert non-normal traits into approximately normal variables through mathematical transformation (Sokal and Rohlf, 1995; Yang et al., 2006). Distribution-free non-parametric methods were also developed for mapping non-normal traits for various population structures (Coppieters et al., 1998; Elsen et al., 1999; Kruglyak and Lander, 1995; Zou et al., 2003). Yet another approach is to replace the normal assumption about the data with other distributions to better fit the trait data (Diao et al., 2004; Feenstra and Skovgaard, 2004; Jansen, 1992; Symons et al., 2002).

When the data is non-normal, assuming that the distributions of random effects and of residuals of Gaussian distributions makes inferences vulnerable to the presence of outliers (Pinheiro et al., 2001). To accommodate these outliers, some symmetric and long-tailed distributions, such as the Student’s t distribution (Dempster et al., 1980; Lange et al., 1989; Rogers and Tukey, 1972), have been suggested for robust estimation. The normal/independent distributions (Andrews and Mallows, 1974; Lange and Sinshheimer, 1993) are a class of symmetric and long-tailed distributions and are used in linear regression models, within a Bayesian framework (Liu, 1996). Fernandez and Steel (1998) applied the method of inverse scaling of the probability density function on the left and on the right side of a non-normal distribution to a symmetric heavy-tailed distribution and have simultaneously captured heavy tails and skewness. Rohr and Hoeschele (2002) have incorporated the Fernandez and Steel’s approach into a Bayesian QTL mapping, developing a robust Bayesian QTL mapping method, which allows for non-normal, continuous distributions of phenotypes within QTL genotypes in single QTL models.

The genetic architecture of quantitative traits includes the number and locations of QTL and their main and epistatic effects. In particular, the unknown number of QTL and possible huge epistatic effects make the dissection for genetic architecture of quantitative traits extremely complex. Fortunately, with a computationally efficient Markov Chain Monte Carlo (MCMC) algorithm, Bayesian model selection frameworks have been developed for identifying epistatic QTL for complex traits (Yi et al., 2005, 2007). However, normal distributions were assumed for these approaches.

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The effects of deviation from this assumption have not been fully addressed.

In this article, we developed a Bayesian robust analysis strategy for studying the genetic architecture of quantitative trait, by combining the flexibility of Bayesian approach in modeling multiple QTL and their interactions and the better phenotypic fitting of symmetric and long-tailed distributions in characterizing non-normal traits. We investigated the robustness of the proposed method by a series of simulations, and applied it to a real dataset in rice. Our method showed an improved power in mapping QTLs with non-normal phenotypes.

2 METHOD

2.1 Genetic model

For simplicity, we consider a mapping population with only two segregating genotypes, e.g., a backcross, double haploid lines (DHLs) or recombinant inbred lines (RILs). However, the method can be applied to other experimental designs, such as F2 design. The phenotypes and molecular marker data were collected on n individuals. Assuming that there are q QTLs responsible for a trait of interest, the phenotypic value yij of individual i can be then described by the following multiple interacting QTL model:

\[ y_i = \mu + \sum_{j=1}^q \alpha_j x_{ij} \beta_j + \epsilon_i \]  

(1)

where \( \mu \) is the population mean, \( \alpha_j \) for \( j = 1, 2, \ldots, q \) is the additive effect of the j-th QTL, \( \beta_j \) is the epistatic effect between j-th QTL and k-th QTL for \( j \neq k \). \( x_{ij} \) is a genotype indicator variable for individual i at locus j and is defined as 1 for one genotype and 0 for the other genotype, and \( \epsilon_i \) is a random environmental error.

To cover outlayers from non-normal distributed phenotypes, we introduce the normal/independent distribution to describe random environmental errors, denoted by \( \epsilon_i \sim N(0, \sigma^2) \), where \( \epsilon_i \) is independent across different QTL and \( \sigma^2 \) is a variance parameter. The type of normal/independent distributions depends on the distribution of \( \epsilon_i \). For instance, if \( \epsilon_i \) is taken to be Gamma(\( \alpha, \beta \)) (\( \alpha, \beta > 0 \)), the normal/independent distribution becomes a u-distribution, \( \epsilon_i \sim \Gamma(\alpha, \beta) \), which is a characteristic distribution for u-shaped and long-tailed distributions.

2.2 Likelihood function

The probability distribution of the phenotype data conditional on all parameters is called the likelihood. Based on model (1), the conditional density of all phenotypes, given the parameters, is

\[ p(y|\mu, \alpha, \beta, \sigma^2) = \prod_{i=1}^n \left( \frac{1}{\sqrt{2\pi\sigma^2}} \right)^{n_i} \exp \left( -\frac{1}{2\sigma^2} \sum_{i=1}^n y_i - \mu \right) \]

where \( y = \{y_i, 1 \leq i \leq n\}, \alpha = \{\alpha_j, 1 \leq j \leq q\}, \beta = \{\beta_j, 1 \leq j \leq q\} \) and \( w = \{w_i, 1 \leq i \leq n\} \), for \( i = 1, 2, \ldots, n \), \( j = 1, 2, \ldots, q \) and \( k = 1, 2, \ldots, q \).

2.3 Prior distribution

As described by Yi et al. (2005), we take L, the maximal number of QTLs as \( L_0 + 3 \sqrt{\frac{n}{L_0}} \), where \( L_0 \) is the prior expected number of all QTLs including main-effect and epistatic QTLs that is determined based on traditional methods. The binary indicator \( c_j \) has an independent prior \( p(c_j) = 1/2 \) for \( j = 1 \) and \( p(c_j) = \frac{1}{2} \) for \( j \neq 1 \), where \( p(c_j) \) is the prior inclusion probability for a certain QTL effect and equals to a predetermined hyper-parameter \( p_{c_j} \) for main effect or \( p_{c_{jk}} \) for epistatic effect.

The population mean \( \mu \) is assumed to have a prior \( p(\mu) \) constant. A hierarchical mixture model is proposed as the prior distribution for each genetic effect, denoted by \( \alpha_j \sim N(0, (\sum_{n=1}^N w_n^2)^{-1}\sigma^2) \) for additive effects and \( \beta_{jk} \sim N(0, (\sum_{m=1}^M w_m^2)^{-1}\sigma^2) \) for the epistatic effects, where \( \sigma^2 \) takes a value such that the prior variance of each QTL effect stays approximately the same as \( \sigma^2 \) increases. Here, we let \( \sigma^2 = n \).

A scaled inverse-\( \chi^2 \) distribution with hyper-parameters \( a \) and \( b \) will be adopted as prior for \( \sigma^2 \), i.e.

\[ \sigma^2 \sim \text{inv-Chi}^2(a, b) \]

The prior for scalar parameter \( df \) is specified based on the form of normal/independent distributions for residual error. The detailed specification of the prior is given in Appendix A.

The prior for position of the j-th QTL is \( p(\gamma_j) = 1/df \), where \( df \) is the length of the marker or adjoining QTLs interval where the j-th QTL resides.

2.4 Posterior distribution and MCMC sampling

The joint posterior density of all unknown parameters is then:

\[ p(\mu, \alpha, \beta, \sigma^2, \gamma, \lambda, \delta, \nu, \xi | y) \propto p(\lambda) p(\delta) \prod_{j=1}^q p(\alpha_j) p(\beta_j) \prod_{j<k} p(\gamma_{jk}) \]

(2)

Joint posterior density (2) by fixing other parameters. For convenience, we adopt as prior for \( \gamma_j, \lambda_j, \delta_j, \nu_j, \xi_j \), \( j = 1 \). The joint posterior density of all unknown parameters need to be derived from the above joint posterior distribution (2) by fixing other parameters. For convenience, we first let

\[ G_i = \mu + \sum_{j=1}^q \alpha_j x_{ij} \beta_j + \sum_{k=1}^q \sum_{j<k} \lambda_{jk} \gamma_{jk} x_{ij} x_{ik} \]

The fully conditional posterior density of the population mean \( \mu \), given all other parameters, can be shown to be a normal distribution with mean \( \hat{\mu} = \sum_j w_j y_j - G_i \) and variance \( \Sigma_j = \sum_j w_j \sigma^2 \).

Conditionally on all other parameters, the QTL effects are mutually independent. In particular, the density of the fully posterior distribution of \( \alpha_j \) is normal with mean \( \hat{\alpha}_j = \sum_k w_k y_{jk} - G_{jk} + G_i \) and variance \( \Sigma_{jk} = \sum_k w_k \Sigma_j \sigma^2 \). Likewise, the conditional posterior distribution of \( \beta_j \) corresponds to the normal with mean \( \hat{\beta}_j = \sum_{k<j} w_k y_{jk} - G_{jk} + G_i \) and variance \( \Sigma_{jk} = \sum_k w_k \Sigma_j \sigma^2 \).

For the residual variance \( \sigma^2 \), the corresponding fully conditional distribution is a scaled inverse \( \gamma^2 \) with parameters \( \nu_0 + n + \nu_1 \) and \( \nu_0 + n + \nu_1 + \sum_k w_k (\nu_1 - G_{jk}) \).

So far, we note that \( \nu_1 \) can be interpreted as a 'weight'. The specific forms of the posterior for \( \nu_1 \) depend on the normal/independent distribution adopted, and the posterior for degree of freedom \( \sigma^2 \) depend on the form of corresponding prior distribution (detailed in Appendix B).
The marginal posterior distribution of $y_k$ is Bernoulli with a probability
\[
p(y_k = 1 | \pi, R) = \frac{\pi R}{1 - \pi R}
\]
where $\pi = \pi_k$ and $R = \left[ \frac{1}{N} \exp \left( -\frac{1}{2} \sum_{i,j} y_{ij} x_{ij}^2 \right) \right]_{i,j = 1, 2, \ldots, q}$ for the additive effect; $\pi = \pi_k$ and $R = \left[ \frac{1}{N} \exp \left( -\frac{1}{2} \sum_{i,j} \delta_{ij} x_{ij}^2 \right) \right]_{i,j = 1, 2, \ldots, q}$ for the epistatic effect. If $y_k$ is sampled to be zero, corresponding to $\alpha = 0$ or $\delta = 0$. Otherwise, $\alpha = 0$ is drawn from its conditional posterior.

3 SIMULATION STUDIES

For convenience of programming, we simulated 61 equally spaced co-dominant markers on a single large chromosome of a length 500 cM for a backcross population with sample sizes of 150 and 300. We simulated the four QTLs, two pairs of which are assumed to mutually interact. The total genetic variance contributed by all main-effect and epistatic QTLs was 45.06%, where the proportion of phenotypic variance contributed by an individual QTL ranged from 0.95% to 11.63%. The population mean and the residual variance were set at $\mu = 5.0$ and $\sigma^2 = 3.0$.

We use non-Bayesian and Bayesian methods to analyze the simulated data. Non-Bayesian mapping is implemented with EM algorithm through two dimensional scan. Detected QTL effects are estimated using multiple QTL imputation. The critical values at significance level of 5% are 3.9 for main effect and 6.7 for epistatic effect, which are obtained with 1000 permutations.

In all Bayesian mapping analysis, we set the prior number of main-effect QTL at three and the prior expected number of epistatic QTL at three, then the upper bound of the number of QTL, main-effect QTL at three and the prior expected number of epistatic QTL was 45.06, where the proportion of phenotypic variance contributed by an individual QTL ranged from 0.95% to 11.63%. The population mean and the residual variance were set at $\mu = 5.0$ and $\sigma^2 = 3.0$.

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is subject to heavy-tailed distribution. The estimates for positions and effects of QTL detected by all methods are fairly close to true parameter values. As expected, the model is more robust with increased heritability and sample size (Tables 2 and 3). Statistical power of QTL detection increases as sample size and genetic contribution proportion increase. The type I error rates of all methods are <6%. On the whole, as statistical power rises, error rate falls.

Table 1. Statistical power of QTL detection (%) and type I error rate (% in the last column) obtained by various mapping methods

<table>
<thead>
<tr>
<th>Sample size</th>
<th>Distribution</th>
<th>QTL no.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>150</td>
<td>Slash</td>
<td>70</td>
</tr>
<tr>
<td>300</td>
<td>Normal</td>
<td>62</td>
</tr>
</tbody>
</table>

Table 2. Mean estimates and SDs (in parentheses) of QTL positions detected by various mapping methods

<table>
<thead>
<tr>
<th>Sample size</th>
<th>Distribution</th>
<th>QTL no.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>150</td>
<td>True position</td>
<td>56</td>
</tr>
<tr>
<td>300</td>
<td>Contaminated</td>
<td>55.7 (3.1)</td>
</tr>
</tbody>
</table>

Table 3. Mean estimates and SDs (in parentheses) of QTL effects detected by various mapping methods

<table>
<thead>
<tr>
<th>Sample size</th>
<th>Distribution</th>
<th>QTL no.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>150</td>
<td>True Effect</td>
<td>0.50 (0.09)</td>
</tr>
<tr>
<td>300</td>
<td>Contaminated</td>
<td>0.45 (0.09)</td>
</tr>
</tbody>
</table>

4 REAL DATA ANALYSIS

A 162 F10 RILs derived from the hybrids of Dasanbyeo (a Korean tongil type rice) × TR22183 (a Chinese japonica variety) had been designed for mapping QTL for traits associated with physical/chemical characteristics and quality of rice. On the basis of the population, we have constructed the framework linkage map of 1437.5 cM long using 208 SSR and STS markers. This map consists of the 16 linkage groups (LGs) for each parental map. We analyzed the data with the Bayesian robust mapping with different sampling residuals from normal distribution and analyzed them with both the Bayesian robust mapping and traditional Bayesian mapping. Results (provided in Section 1 of Supplementary Material) indicated that applying the Bayesian Robust analysis for data being normally distributed had similar powers as using traditional Bayesian mapping methods.
Bayesian robust mapping analysis

type of distributions and traditional Bayesian mapping procedure with normal residuals, respectively.

In all Bayesian analyses, based on results from the interval non-
epistatic mapping (Lander and Botstein, 1989) and two-dimensional
genome scan, the prior number of main-effect QTL was set at \( m_o = 3 \) and the prior expected number of all QTL (\( m_0 \)) was taken to be \( n_0 + 5 \). The upper bound of the number of QTL, \( L \), was then 16.

The initial value of each unknown parameter took the same one as in simulation study. The MCMC was run for 200,000 cycles after the burn-in of 6000 cycles. It was found that the mapping results from 13 of 21 traits of interest support the Bayesian robust mapping procedure. Herein, we take the peak viscosity (PKV) as an example trait to compare the mapping results based on different residual distributions.

The estimates for positions and genetics effects of QTL detected with the Bayesian robust mapping and the traditional bayesian mapping method are listed in Tables 4 and 5, respectively. Apparently, the results from different distributions are comparable: three main-effect QTLs and seven pairs of epistatic QTLs, covering all QTL detected by other methods, are identified with Bayesian robust mapping with a \( t \)-distribution, and followed by one main-effect QTL and four pairs of epistatic QTLs with slash distribution for residuals, one main-effect QTL and three with contaminated normal distribution for residuals and one main-effect QTL and two pairs of epistatic QTL with normal distribution for residuals, whereas only one main effect QTL on seventh LG with non-Bayesian method. This implies that Bayesian robust analysis has higher power than traditional Bayesian model selection and non-Bayesian method. Most of the main-effect and epistatic QTLs increase the PKV in rice, except for a third main-effect QTL and ninth pair of QTLs. All three different cases of two QTLs that involve the epistatic effects are found: (1) both QTLs are the main, as fourth and eighth pairs of QTL; (2) both QTLs are not the main, as seventh pair of QTL and the rest are that only one QTL is the main. Figures 1 and 2 (in Section 2 of supplementary data) plot the one-dimensional profiles of BFs for main effects and two-dimensional profiles of BFs for epistatic effects obtained from Bayesian robust mapping with a \( t \)-distribution for residuals, respectively. They intuitively illustrate the results from Bayesian robust analysis for genetic architecture of quantitative traits.

5 DISCUSSION

Within the framework of Bayesian model selection for mapping genome-wide interacting QTLs, we develop a Bayesian robust mapping strategy for analyzing continuous non-normal quantitative

<table>
<thead>
<tr>
<th>QTL no.</th>
<th>Distribution</th>
</tr>
</thead>
<tbody>
<tr>
<td>( t )</td>
<td>Slash</td>
</tr>
<tr>
<td>1</td>
<td>1.438.7</td>
</tr>
<tr>
<td>2</td>
<td>7.327.6</td>
</tr>
<tr>
<td>3</td>
<td>16.164.5</td>
</tr>
<tr>
<td>4</td>
<td>(1.435.9 \times 16.162.8)</td>
</tr>
<tr>
<td>5</td>
<td>(1.309.3 \times 12.11.5)</td>
</tr>
<tr>
<td>6</td>
<td>(1.445.2 \times 16.23.8)</td>
</tr>
<tr>
<td>7</td>
<td>(1.65.6 \times 12.253.2)</td>
</tr>
<tr>
<td>8</td>
<td>(7.327.6 \times 16.164.5)</td>
</tr>
<tr>
<td>9</td>
<td>(4.24.8 \times 16.160.8)</td>
</tr>
<tr>
<td>10</td>
<td>(9.27.3 \times 16.168.7)</td>
</tr>
</tbody>
</table>

Table 4. Estimated QTL positions (LG-position) obtained from Bayesian robust mapping with different distribution for residual on PKV in rice

<table>
<thead>
<tr>
<th>QTL no.</th>
<th>QTL type</th>
<th>Distribution</th>
</tr>
</thead>
<tbody>
<tr>
<td>( t )</td>
<td>Slash</td>
<td>Contaminated</td>
</tr>
<tr>
<td>1</td>
<td>Main Effect</td>
<td>0.46(1.96)</td>
</tr>
<tr>
<td>2</td>
<td>Main Effect</td>
<td>10.05(6.65)</td>
</tr>
<tr>
<td>3</td>
<td>Main Effect</td>
<td>–4.77(2.77)</td>
</tr>
<tr>
<td>4</td>
<td>Epistatic</td>
<td>13.469.03</td>
</tr>
<tr>
<td>5</td>
<td>Epistatic</td>
<td>9.0(5.13)</td>
</tr>
<tr>
<td>6</td>
<td>Epistatic</td>
<td>7.07(4.29)</td>
</tr>
<tr>
<td>7</td>
<td>Epistatic</td>
<td>8.06(1.77)</td>
</tr>
<tr>
<td>8</td>
<td>Epistatic</td>
<td>273(3.45)</td>
</tr>
<tr>
<td>9</td>
<td>Epistatic</td>
<td>–5.46(3.18)</td>
</tr>
<tr>
<td>10</td>
<td>Epistatic</td>
<td>3.04(2.55)</td>
</tr>
</tbody>
</table>

Table 5. Estimated QTL effects obtained from Bayesian robust mapping with different distribution for residual on PKV in rice

The numbers in parentheses are the 2logBF values.

1037
traits, by replacing the normal distribution for residuals in multiple QTL model with the normal/independent distributions. Compared with Bayesian mapping for normal data, the Bayesian robust mapping strategy additionally sample ‘weight’ $W$ and the robustness parameter $d$ with the Gibbs sampler or Metropolis/Hastings algorithm in the MCMC process. Although computations for the robust models may be more than for their normal counterparts, the flexibility of the Bayesian robust mapping for either non-normal or normal data is enough to compensate for the cost. Of course, if the robustness parameter is assumed to be known, e.g. simply fixed at a small value (Gelman et al., 1995), the implementation of the Bayesian robust mapping will be even easier. In practice, however, unless there is a strong reason to believe in the adequacy of the normality assumption for residuals, it may be safer to use a robust model (Rosa et al., 2003, 2004).

Except for the three common normal/independent distributions discussed in this study, other distributions can also be considered, such as the Laplace and the double exponential distributions. Which distribution is optimal for fitting residuals depends on peculiarities of the dataset, such as the proportion of outliers and how far these are from the ‘center’ of the distribution. The $t$-distribution is the most commonly used thick-tailed distribution for robust inference, and is often a good alternative to a normal distribution. The contaminated normal distribution is the most flexible among the three robust distributions, but at the expense of an additional parameter. The slash distribution, although not often encountered in the literature, is the easiest one to implement in hierarchical modeling, because all conditional posterior distributions have closed forms.

Rohr and Hoeschele (2000) first implemented a robust Bayesian method for mapping QTL. Their study was different from ours in that: (1) their mapping analysis aimed at outbred population whereas ours at inbred; (2) their proposed method was based on a single QTL model whereas ours was based on a multiple QTL model; and (3) they used skewed Student’s $t$-distributions to describe phenotypic residuals in the analysis whereas we adopted a student’s $t$-distribution. In the single QTL model, it may be reasonable to assume that residuals follow skewed Student’s $t$-distributions, because the ‘skewness’ may absorb the effects of other QTLs on phenotypes. However, no ‘skewness’ is necessary for the multiple QTL model. A complete Bayesian mapping requires the sampling of genotypes for QTL and missing markers and aggravates the computational cost of Bayesian robust analyses. To alleviate this problem, we evenly partition the entire genome into small intervals by a number of points and restrict putative QTL to these fixed points, as proposed by (Yi et al., 2005). This strategy greatly reduces computational time by estimating all expected values of indicator variables for putative QTL by using conditional probability of their genotypes on two flanking markers before the MCMC procedure starts. Other ways to improve the efficiency of analyzing many QTL effects with Bayesian model selection include specifying prior inclusion probability for epistasis and using Metropolis/Hastings algorithm to perform fast sampling for binary indicator (Yi et al., 2007).

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Rohr,P.V. and Hoeschele,I. (2002) Bayesian QTL mapping using skewed Student-t distributions, but at the expense of an additional parameter. The slash distribution, although not often encountered in the literature, is the easiest one to implement in hierarchical modeling, because all conditional posterior distributions have closed forms.

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APPENDIX A
Specification of prior for degree of freedom \( df \) in normal/independent distributions

In the \( t \)-distribution, we adopt a flat prior for \( df \) as \( df^{-1} \), yielding \( p(df) \propto df^{-1} \) (Liu, 1996); A Gamma(\( a, b \)) distribution with small positive values of \( a \) and \( b \) (\( b \ll a \)) can be adopted as a prior for \( df \) in the slash distribution; and the prior for \( df \) of contaminated normal distribution involves two parameters, i.e. \( df = (v \tau) \). Herein, a Uniform (0, 1) distribution is used as a prior for \( \tau \) and an independent Beta \( (a, b) \) is adopted as prior a for \( v \).

APPENDIX B
Forms of posteriors for \( w \) and degree of freedom \( df \) in normal/independent distributions

For a \( t \)-distribution, the fully conditional posterior density for each element of \( w \) is a Gamma distribution with parameters \( \frac{1+df}{2} \) and \( \frac{2}{2} \sum_{i=1}^{n} \left( Yi - \mu - \sum_{j=1}^{g} x_{ij} b_{j} \right)^{2} \), corresponding conditional posterior density of \( df \) is

\[
p(df) \propto 2^{\frac{1}{2}} \Gamma\left(\frac{df}{2}\right) \Gamma\left(-\frac{n}{2}\right) \frac{1}{\Gamma\left(\frac{a+1}{2}\right)} df^{-1} \exp\left[-\frac{df}{2} \sum_{i=1}^{n} (w_{i} - \ln w_{i}) \right]
\]

which does not have an explicit form but a Metropolis/Hastings or rejection sampling step (Ripley, 1987) can be embedded in the MCMC scheme to obtain draws for \( df \).

For slash distribution,

\( w_{i} \sim \text{Truncated} - \Gamma\left(\frac{a+1}{2}, \frac{1}{2} \sigma_{i}^{2} \left( Yi - G_{i} \right)^{T} \left( Yi - G_{i} \right) \right) \) with \( df \sim \text{Gamma}(a+n, b-\sum_{i=1}^{n} \ln w_{i}) \).

For contaminated normal distribution, the fully conditional posterior density for \( w_{i} \) is also non-closed form: \( p(w_{i}) \propto w_{i}^{a/2} \left( \frac{1}{\sqrt{2\pi} \sigma_{i}^{2}} \right) \left( 1 - v \right) \left( 1 + \tau \right) \left( \frac{1}{\sqrt{2\pi} \sigma_{i}^{2}} \right) \exp\left[-\frac{1}{2\sigma_{i}^{2}} \left( Yi - G_{i} \right)^{T} \left( Yi - G_{i} \right) \right] \) with \( v \sim \text{Beta} \left( a + \frac{1}{2}, b + \frac{1}{2} \sum_{i=1}^{m} (w_{i} - \tau) \right) \).

\[
p(df) \propto 2^{\frac{1}{2}} \Gamma\left(\frac{df}{2}\right) \Gamma\left(-\frac{n}{2}\right) \frac{1}{\Gamma\left(\frac{a+1}{2}\right)} df^{-1} \exp\left[-\frac{df}{2} \sum_{i=1}^{n} (w_{i} - \ln w_{i}) \right]
\]