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Genetic variance models for the evaluation of resistance to powdery scab (*Spongospora subterranea* f. sp. *subterranea*) from long-term potato breeding trials

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Abstract Breeding for resistance to soil-borne powdery scab in potato is an important component of the integrated management of this disease. Different genetic variance models within a mixed model framework were applied to data from long-term potato breeding trials, for the genetic evaluation of breeding lines. The multi-environment trial (MET) data came from 12 growing seasons ("years", synonymous with environments) of New Zealand field trials screening for resistance to powdery scab on potato tubers. Pedigree information on a total of 1,031 genotypes was available. Additive components of the genetic effects were important with narrow-sense heritability estimates (and 95 % confidence intervals) from single-year analyses ranging from 0.26 (0.20, 0.44) to 0.57 (0.53, 0.85). Spatial components estimated from the residual plot effects were not important for most years and even when they were significant, estimates were small. In MET analyses, different variance structures for the genetic effects were tested; a homogeneous correlation model (CORH) gave a better fit to the data than a factor analytic FA*k* model of order (*k*), 1 and 2. The year-to-year genetic correlation estimate from CORH was 0.81 and compared with a range of 0.59–0.95 estimated from the FA1 model. There was no strong evidence of non-additive genetic effects with zero or boundary estimates for most years. Models which included the pedigree provided a better fit to the data than models that did not include this relationship information. There was no evidence for genetic improvement in resistance for powdery scab on tubers in the breeding population studied. This suggests that selection pressure for resistance in the past has been weak and greater consideration should be given to selecting parents on empirical breeding values to genetically improve breeding populations for resistance to powdery scab.

Keywords Empirical breeding values · Genetic parameters · Potato breeding programme · Multi-environment trials · MET · Variance components

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Introduction

Powdery scab is a soil-borne disease of potato tubers (*Solanum tuberosum* L.) caused by the Cercozoan pathogen *Spongospora subterranea* f. sp. *subterranea*. This disease is a problem in New Zealand and Australia, with increasing global prominence in many

Fig. 1 **a** Powdery scab lesions on the surface of a potato tuber (photo: Robert Lamberts: **b** *Spongospora* galls on potato roots (photo: Richard Falloon)

other potato-producing regions (Merz 2008; Merz and Falloon 2009). The pathogen causes surface lesions on potato tubers (Fig. 1a). Affected tubers can severely reduce quality, consumer acceptability and productivity for all sectors of the industry; seed tubers will not have the required high health status for sale and both the marketable and factory yields of potatoes (grown for human consumption) will be reduced. Zoospores of the pathogen also infect root epidermis cells, and root galls later develop (Fig. 1b). Root infection adversely affects plant productivity by reducing plant dry weight and tuber yield (Falloon et al. 2005; Shah et al. 2012). Chemical control using pesticides applied to the seed tuber or soil has partial efficacy but is costly, and soil applications of synthetic pesticides are becoming less acceptable because of environmental concerns (Merz and Falloon 2009). Once *S. subterranea* is established in the soil, long cropping rotations are required for potato to avoid the disease because of the longevity of resting spores (Falloon et al. 2011). There are therefore both economic and environmental incentives for the potato industry to develop new cultivars that are resistant to powdery scab, which is considered an important part of an integrated disease management strategy (Genet et al. 2004; Falloon 2008).

The screening of potato breeding lines and potential cultivars for resistance to powdery scab of tubers has been an important component of the New Zealand potato improvement programme since 1991. Identifying resistant lines is important for the selection of new cultivars for industry and also for the selection of new parents to introduce into the programme, with the intention of increasing the level of resistance in the breeding population by recurrent selection. Studies on the genetic basis for resistance to tuber infection have been limited but indicate that genetic control is polygenic. In clonal studies on advanced lines and varieties, individual genotypes could be discriminated in terms of their response to tuber infection on a continuous range of susceptible to resistant clones (Bhattacharya et al. 1985; Falloon et al. 2003). Wastie (1991) found that progeny resistance of tuber infection in seedling families was correlated with the phenotypic resistance of parents, indicating the presence of heritable variation and the potential for early-stage selection for resistance in a breeding programme. The genetic relationship between resistance to tuber powdery scab and root infection is also not well understood, with several studies indicating that genetic control for the two may be independent (van de Graaf et al. 2007; Baldwin et al. 2008; Merz et al. 2012). Harrison et al. (1997) suggested that wild *Solanum* species such as *S. curtilobum* and *S. tuberosum subsp. andigena*, that have shown high levels of resistance to powdery scab, may be useful sources of genetic variation for breeding programmes.

In genetic evaluation, estimates of variance components are required by crop breeders to obtain genetic parameters to plan breeding strategies and to predict breeding values to identify superior parents and breeding lines. Linear mixed models have received increasing attention in recent years for the evaluation of plant breeding multi-environment trials (METs).

These models provide a more realistic representation of the underlying random and error components compared with traditional linear models, by accommodating heterogeneous variances and correlated information from trials and/or relatives through a pedigree (Balzarini 2001; Smith et al. 2005; Piepho et al. 2008a). METs are an important part of plant improvement programmes, to assess the response of genotypes to biotic and abiotic pressures that will invariably fluctuate between different growing seasons and locations. Measurement of genotype × environment (G × E) interaction in crop breeding trials aims to identify high-performing genotypes that are either specific to particular environments, or demonstrate a broad stability across a range of environments. A common approach to modelling G × E effects of genotype performance, following Falconer (1952; cited by Falconer and Mackay 1996), is to consider different environments as different traits. The long-term powdery scab screening trials at The New Zealand Institute for Plant & Food Research Limited (PFR), Lincoln, can be considered as a MET in which every growing season is, in effect, a different environment. At present, the evaluation of breeding lines in the early stages of the PFR potato selection programme assumes independence between genotypes tested within each annual trial as well as independence of genotypes tested across trials and growing seasons. This is common practice in many plant breeding programmes, including potato, but is not a particularly efficient use of data. There are various genetic variance models to describe the structure of the genetic (co)variances within the mixed model framework in MET evaluation (Smith et al. 2001; Crossa et al. 2006; Kelly et al. 2007). One of the simplest, for example, is a homogeneous covariance structure that models different within-trial variances and the same genetic correlation between trials. The most general form (as it attempts to most closely represent the true underlying structure) is an unstructured covariance matrix that contains $t(t+1)/2$ distinct parameters, where t is the number of trials. This approach is not often feasible under a standard REML-based procedure, even when t is not particularly large, as genotype effects are often highly correlated between some trials resulting in singular variance matrices (Kelly et al. 2009; Meyer 2009). A factor analytic (FA) approach has been considered in plant breeding as a parsimonious alternative to the unstructured form of the genetic variance matrix (Piepho 1998; Smith et al. 2001). FA methods aim to simplify the G × E interaction effects into a small number of unobserved latent variables that attempt to explain most of the interaction. Such models have been shown to be computationally efficient and robust, giving good approximations (Thompson et al. 2003; Kelly et al. 2007; Burgueño et al. 2011), and have been applied, for example, to the MET analyses of cane sugar content in sugarcane (Oakey et al. 2007), yield in wheat (Crossa et al. 2006; Burgueño et al. 2007), yield and oil content in canola (Beeck et al. 2010) and fruit weight in mango (Hardner et al. 2012).

This paper compares various forms of the genetic variance matrix following the general approach of Smith (2001) for the quantitative genetic evaluation of resistance to powdery scab in potato. In addition to comparing variance matrices, the usefulness of including pedigree information was also explored, following the approach of Oakey (2007). Comparisons of model fit with variance matrices were made for data collected from early-stage selection trials for the New Zealand PFR potato breeding programme. Trials were carried out across 12 growing seasons and included a total of 1,031 tested genotypes. Including the pedigree information allowed retrospective evaluation of parents based on their empirical breeding values (EBVs), i.e. their ability to transfer powdery scab resistance to progeny, and an assessment of the genetic improvement of resistance to powdery scab in the PFR breeding population. Results showed that a model with simple common correlation which also included pedigree information provided the best fit for these data.

Materials and methods

Data

Data were collected from 12 growing seasons of field trials planted each September from 1998 to 2010 (henceforth designated as "years"). No data were available from 2006. The plant material consisted of 1,031 breeding lines (henceforth used interchangeably with "genotypes") originating from crosses of selected parents (with the absence of any formal mating design). The genotypes had already been through one or two clonal generations of selection for

Table 1 Concurrence of genotypes across 12 years of powdery scab field trials; diagonal entries are the number of genotypes tested in individual years

	1998	1999	2000	2001	2002	2003	2004	2005	2007	2008	2009	2010
1998	160	69	39	4	2	8	5	2	2	2	2	2
1999		140	64	6	2	6	2	2	2	2	2	2
2000			131	39	2	6	2	2	2	2	2	2
2001				140	37	16	8	3	2	2	2	2
2002					122	43	25	14	3	2	2	2
2003						122	45	26	6	2	2	2
2004							113	52	11	2	2	2
2005								113	17	6	4	2
2007									93	32	16	2
2008										136	60	42
2009											136	42
2010												146

tuber yield and quality traits. Many genotypes were related to others planted in the same year or across different years, for example, as full or half siblings. Nine hundred and ninety-nine out of 1,031 tested genotypes had both parents recorded in the pedigree. There were 184 female parents and 80 male parents, 48 of which had acted as both male and female parents. Of the tested genotypes, 901 and 948 of both maternal and paternal grandparents respectively were recorded. Forty-eight tested genotypes had been used as parents and eight were grandparents.

There was reasonable concurrence between adjacent years of genotype entries for most pairs of years (Table 1). Only two genotypes, namely the New Zealand bred cultivars 'Iwa' and 'Gladiator', were represented in every year. Improving connectedness between pairs of years will enhance the reliability of estimates of year to year genetic correlations and the accuracy of EBVs. Genetic links other than those between adjacent years were likely to be improved through pedigree relationships.

The trials were grown at The New Zealand Institute for Plant & Food Research Limited (PFR) farm, Lincoln, Canterbury, New Zealand (latitude 43°38'S, longitude 172°29'E). The soil at the site was a Paparoa silt loam. Trials were planted on the same field site for three consecutive years. After three years, trials were then moved to an adjacent site in the same field. Crop management regimes were consistent for all years. After soil preparation, each trial was planted as a randomized complete block design (RCBD). A single tuber of each genotype was planted in each plot, and each genotype was replicated six times. *Spongospora subterranea* inoculum was made up of macerated potato tubers with severe powdery scab. Each tuber plot was inoculated with approximately 100 ml of inoculum and tubers were then covered with soil. Planting was in mid-September each spring. Moist soil conditions were maintained by regular irrigation through the growing season, particularly during the tuberisation and post-tuberisation periods, to encourage powdery scab development. Plots were harvested in early March after the natural senescence of foliage. All tubers greater than 30 g from each plot were harvested and washed free of soil. Tubers were then assessed for powdery scab severity with a single score assigned to each plot based upon visual assessment of all tubers. Tuber assessment was scored on an ordinal 0–9 scale, where 0 = no visible lesions and 9 = complete surface area covered by powdery scab lesions. This scale was adapted from the scoring scheme described by Falloon et al. (1995). All tuber assessments in the 12 trials were made by the same assessor. The pedigree was built on records from historic PFR field books and a publically-available potato pedigree database (Berloo et al. 2007).

Model selection

To establish an appropriate statistical model, the first stage was to identify the important non-genetic terms following Beeck et al. (2010). Single trials were analysed to establish the importance of terms for each i^{th} trial, estimated with the general form of the linear mixed model:

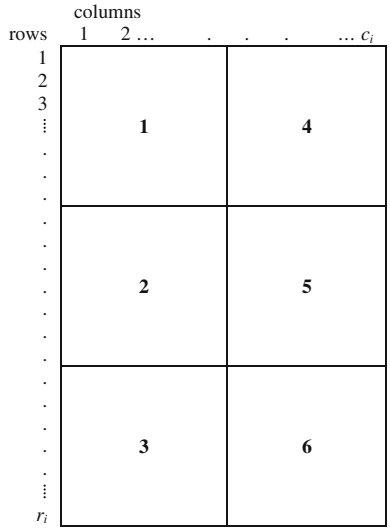

Fig. 2 Illustrative example of the layout of a randomized complete block design powdery scab assessment trial (*i*) with six replicates in r_i rows and c_i columns

$$\mathbf{y} = \mathbf{1}m + \mathbf{Z_1 b} + \mathbf{Z_2 g} + \mathbf{e}$$

where $\mathbf{y}$ is the $n \times 1$ vector of observations for powdery scab severity score, m is the overall trial mean as a fixed effect, $\mathbf{b} \sim N(0, \mathbf{I}\sigma_b^2)$ is the $q\ x\ 1$ vector of random design factors i.e. block or row and/or column effects, $\mathbf{g} \sim N(0, \mathbf{I}\sigma_g^2)$ is the $w\ x\ 1$ vector of random genetic effects, $\mathbf{Z_1}$ ($n\ x\ q$) and $\mathbf{Z_2}$ ($n\ x\ w$) are known incidence matrices that relate the phenotypic observations to their corresponding vectors, $\mathbf{e} \sim N(0, \mathbf{I}\sigma_e^2)$ is the $n\ x\ 1$ vector of random error terms and $\mathbf{I}$ is the appropriate $q\ x\ q$, $w\ x\ w$ or $n\ x\ n$ identity matrix.The error term included an appropriate spatial model to account for local-scale heterogeneity. Various forms of spatial model are possible. However, a separable autoregressive process of order one (AR1) was the only form considered in this study, as this has been shown to provide an adequate variance structure for local spatial trend in crop breeding trials (Smith et al. 2001), and follows the approach described by Gilmour et al. (1997). Each trial (*i*) comprised a rectangular array of r_i rows by c_i columns of n_i plots ($n_i = r_i c_i$), as illustrated in Fig. 2. The best fitting model was selected as the preferred model on the basis of the Akaike information criterion (AIC) goodness-of-fit test: $AIC = -2(\log L - p)$ where $\log L$ is the REML estimate of the log-likelihood and p is a penalty term representing the number of variance parameters fitted.

The model was then developed to incorporate the pedigree, with single trials reanalysed so that the vector of random genetic effects, $\mathbf{g} \sim N(0, \mathbf{I}\sigma_g^2)$, was replaced by the vector of random additive genetic effects, $\mathbf{a} \sim N(0, \mathbf{A}\sigma_a^2)$, where $\mathbf{A}\sigma_\mathbf{a}^\mathbf{2}$ is the variance–covariance matrix of the additive genetic effects (breeding values), with $\mathbf{A}$ as the numerator relationship matrix. Variance components, including the additive variance, and a narrow-sense (h^2) heritability for powdery scab resistance were estimated for each trial. In general, heritability in the narrow-sense was obtained from:

$$h^2 = V_A/V_P = \sigma_a^2/(\sigma_a^2 + \sigma_b^2 + \sigma_e^2);$$

where V_A is the additive genetic variance, V_P is the total (phenotype) variance, σ_a^2 is the variance of the additive variety effects, σ_b^2 represents the variance of the appropriate design factor(s) (e.g. block or row and/or column effects), and σ_e^2 is the random error. Confidence intervals (CI) (95 %) for narrow-sense heritabilities were estimated by jackknifing so that each sample was generated with the n_j genotype removed. The sampled data was then reanalysed to provide the j^{th} partial estimate and the j^{th} pseudovalue of the heritability, and the vector of pseudovalues was used to approximate the CI.

The single trial model was then extended to a multivariate MET analysis by stacking the vectors for the 12 years $(\mathbf{y_1}, \mathbf{y_2}, \ldots, \mathbf{y_{12}})$ of phenotypic observations:

$$\begin{bmatrix} \mathbf{y_1} \\ \mathbf{y_2} \\ \vdots \\ \mathbf{y_{12}} \end{bmatrix} = \begin{bmatrix} \mathbf{1_1}m_1 \\ \mathbf{1_2}m_2 \\ \vdots \\ \mathbf{1_{12}}m_{12} \end{bmatrix} + \begin{bmatrix} \mathbf{Z_{1_1}} & \mathbf{0} & \cdots & \mathbf{0} \\ \mathbf{0} & \mathbf{Z_{1_2}} & \cdots & \mathbf{0} \\ \vdots & \vdots & \ddots & \vdots \\ \mathbf{0} & \mathbf{0} & \cdots & \mathbf{Z_{1_{12}}} \end{bmatrix} \begin{bmatrix} \mathbf{b_1} \\ \mathbf{b_2} \\ \vdots \\ \mathbf{b_{12}} \end{bmatrix} + \begin{bmatrix} \mathbf{Z_{2_1}} & \mathbf{0} & \cdots & \mathbf{0} \\ \mathbf{0} & \mathbf{Z_{2_2}} & \cdots & \mathbf{0} \\ \vdots & \vdots & \ddots & \vdots \\ \mathbf{0} & \mathbf{0} & \cdots & \mathbf{Z_{2_{12}}} \end{bmatrix} \begin{bmatrix} \mathbf{g_1} \\ \mathbf{g_2} \\ \vdots \\ \mathbf{g_{12}} \end{bmatrix} + \begin{bmatrix} \mathbf{e_1} \\ \mathbf{e_2} \\ \vdots \\ \mathbf{e_{12}} \end{bmatrix}$$

and random effects were assumed to follow a multivariate normal distribution with means and variances defined by:

$$\begin{pmatrix} \mathbf{b} \\ \mathbf{g} \\ \mathbf{e} \end{pmatrix} \sim N\left[\begin{pmatrix} \mathbf{0} \\ \mathbf{0} \\ \mathbf{0} \end{pmatrix}, \begin{bmatrix} \mathbf{B_0} \otimes \mathbf{I_b} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{G_0} \otimes \mathbf{I_g} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{R_0} \otimes \mathbf{I_n} \end{bmatrix}\right]$$

where $\mathbf{0}$ are null matrices. $\mathbf{B_0}$, $\mathbf{G_0}$ and $\mathbf{R_0}$ are covariance matrices for design factors (block, row, column), genetic and residual effects, respectively, and $\otimes$ is the direct (Kronecker) product. The matrix $\mathbf{B_0}$ is a diagonal matrix of scaled identity matrices and plot error effects $\mathbf{R_0}$ are assumed to be block diagonal. The same site was used for trials in three consecutive years, but the independence of design factors (block, row, column) and plot errors, so that $\mathbf{B_0} = diag(\mathbf{B_{0_i}})$ and $\mathbf{R} = diag(\mathbf{R_i})$ was assumed. This seemed reasonable as trial alignment was likely to change from year to year so that actual block, row, column and plot positions were relocated. Local spatial trend where necessary, as identified in the single trial analyses outlined previously, was specified through $\mathbf{R}$ as a separable autoregressive (AR1) process (Gilmour et al. 1997) and following Smith (2001), with rows within columns:

$$\mathbf{R_i} = \sigma_i^2 \Sigma \mathbf{c_i} \otimes \Sigma \mathbf{r_i}$$

where σ_i^2 is a scale parameter and $\Sigma \mathbf{c_i}$ and $\Sigma \mathbf{r_i}$ are the $c_i \times c_i$ and $r_i \times r_i$ correlation matrices, respectively, for the column and row dimensions of the trial, i.

The independent genetic component, $\mathbf{G_g} = \mathbf{G_{0_g}} \otimes \mathbf{I}$, was then partitioned into additive and non-additive components to test the importance of additive and non-additive genetic effects, as outlined by Oakey et al. (2007) and Kelly et al. (2009). The assumption was that the variance matrix for the additive genotype effects was a two-way table of genotype by environment effects which had the separable form $\mathbf{G_a} = \mathbf{G_{0_a}} \otimes \mathbf{A}$; where $\mathbf{G_{0_a}}$ is the symmetric and positive definite matrix of additive variances and covariances between environments and $\mathbf{A}$, the numerator relationship matrix, is the symmetric and positive definite (co)variance matrix between genotypes. Similarly, non-additive effects were considered as a two-way table of genotype by environment effects with the variance structure assumed to have the separable form $\mathbf{G_n} = \mathbf{G_{0_n}} \otimes \mathbf{I}$ with independence between non-additive genetic components. This provided the most general form of the models fitted, where the genetic component of the model was partitioned into:

$$\begin{pmatrix} \mathbf{a} \\ \mathbf{n} \end{pmatrix} \sim N\left[\begin{pmatrix} \mathbf{0} \\ \mathbf{0} \end{pmatrix}, \begin{bmatrix} \mathbf{G_{0_a}} \otimes \mathbf{A} & \mathbf{0} \\ \mathbf{0} & \mathbf{G_{0_n}} \otimes \mathbf{I} \end{bmatrix}\right]$$

Using the important non-genetic terms identified within each trial, various forms of the genetic variance matrix were then tested against each other. $\mathbf{G_0}$ is the genetic variance matrix with the diagonal elements representing genetic variances for each trial and the off-diagonal elements representing genetic covariances between pairs of trials (synonymous with environments or years). Definitions of the forms of $\mathbf{G_0}$ were as follows:

SIMPLE: all variances within trials were assumed to be equal and all pairwise covariances between trials were assumed to be independent and therefore zero; **DIAG**: variances within trials were assumed to be different and all pairwise covariances between trials were assumed to be zero; **CORH**: variances within trials were assumed to be different and a constant non-zero correlation was assumed between all pairwise combinations of trials; **FA*k***: factor analytic, (Piepho 1997; Smith et al. 2001), with common factors identified (as the leading principal components) and residuals, or specific (lack-of-fit) variances, so that the genetic (co)variance matrix is, $\mathbf{G_0} = \Lambda\Lambda' + \Psi$, with Λ a $(t \times k)$ matrix of common factors (or environmental loadings) and Ψ a $(t \times t)$ diagonal matrix of specific variances; **RR*k***: (fully) reduced rank analogous to the FA*k* model but all specific variances were assumed absent and fixed at zero (Meyer 2009); **US**: unstructured $\mathbf{G_0}$ with different variances within trials and different covariances between all pairwise combinations of trials.

All models were again tested with and without the pedigree. The analyses of the data were undertaken using ASReml (Gilmour et al. 2006) and R (R Development Core Team 2012), with the mixed models fitted using ASReml-R (Butler et al. 2009). AIC was used as the test criterion for selection of the best model of the various forms of $\mathbf{G_0}$. These were also compared by simulating the response to selection (Piepho and Möhring 2007), based on 1,000 simulation runs. Breeding values estimated from the data were assumed to be the true breeding values. For each simulation run, residuals were resampled (with

replacement) and added to the fitted values. These new data were then reanalysed to give the best linear unbiased predictors (BLUPs) of genotype effects. At selection fraction *s*, the top *s* 100 % genotypes, based on the BLUPs, were identified. A response to selection was then computed as the difference between the mean of the true breeding values of these top genotypes and the mean of the breeding population.

Prediction of breeding values

For each model, empirical breeding values (EBVs) were obtained for all genotypes in the pedigree from the best linear unbiased predictors (BLUPs) of breeding line effects, as, for example, outlined by Smith et al. (2005, 457–459), with all years having equal weighting. For the evaluation of the early-stage powdery scab trials, the aim is to rank genotypes for selection. It was therefore appropriate for breeding lines to be considered as random effects. This contrasts with the comparison of genotypes in late-stage trials, when differences between pairs of varieties are of greater interest. In this case, it is considered that variety effects may be regarded as fixed and genotype values obtained from best linear unbiased estimators (BLUEs) of variety effects, because the BLUP of a specific difference is biased (Smith et al. 2005). Variance parameters are unknown and replaced in the mixed model equations with those estimated from the data, giving empirical BLUEs and empirical BLUPs (respectively, E-BLUEs and E-BLUPs). To detect a genetic trend in powdery scab resistance across years, a mean EBV of tested breeding lines was obtained for each year from the best-fitting variance model, where the cohort for each year was made up of breeding lines in their first year of test. Parametric bootstrapping was used to obtain estimates of the 95 % CI of EBVs for each year.

Results

Data

The phenotypic means of powdery scab severity scores were low for all years (Table 2). There was a poor expression of phenotype in 2002, with severity scores ranging from 0 to 5 resulting in a reduced variance. Non-normal distribution of phenotypic observations for powdery scab severity was found in some years, namely 2002, 2009 and 2010 (Fig. 3). All distributions tended to be positively skewed, particularly in 2010 when the mean was 1.71 and the median was 0.

Single trial analyses

From the visual interpretation of quantile–quantile plots from single trial analyses (plots not shown), assumptions for the normal distribution of residuals were maintained, despite zero score inflation in some years. It was therefore considered appropriate to analyse these data without recourse to an alternative generalized linear mixed modelling (GLMM) approach. Block terms were important in ten years of the trials with the addition of rows and columns only improving model fit in three of these years (Table 3). Spatial terms were positive but small, and only required in four of the twelve years with a maximum correlation of 0.13 for rows in 1998 and 0.09 for columns in 2000. Narrow-sense heritabilities were considered to be moderate, ranging from (95 % CI) 0.26 (0.20, 0.44) in 2002 to 0.57 (0.53, 0.85) in 2007. Additive variance was only 0.33 in 2002 (compared with a range of 0.82–2.50 for other years). Including the pedigree improved the fit in all years with the exception of 2002.

MET analyses

The preferred non-genetic models for each trial were then applied to a MET analysis by combining all of the data. Using the DIAG form for the genetic variance matrix, $\mathbf{G_0}$, non-additive genetic effects were not significant, with their estimates often zero or constrained at the boundary and therefore dropped from further analyses. Results from the testing of various forms of the $\mathbf{G_0}$ are shown in Table 4, with and without the additive genetic relationship matrix **A**. Results could not be obtained from a US genetic variance matrix because of convergence problems. With initial starting values provided, the maximum number of MET trials that could be analysed together with a US matrix was three before running into difficulties.

Based on AIC, CORH with pedigree (Model 3) was the best fitting model (AIC = 17,050) and was very similar to FA1 with pedigree (Model 4) (AIC = 17,056) respectively. RR2 performed better

Table 2 Summary of powdery scab severity scores from field trials, 1998–2010

Year	Observations	Mean	Median	Standard deviation	Range	Dimension (row × column)
1998	972	2.13	2.0	1.65	0.8	108 × 9
1999	864	3.24	3.0	1.80	0.8	96 × 9
2000	810	2.71	2.0	1.77	0.7	90 × 9
2001	864	2.62	2.0	1.59	0.8	96 × 9
2002	756	1.45	1.0	1.10	0.5	84 × 9
2003	756	2.93	3.0	1.48	0.7	84 × 9
2004	702	3.03	3.0	2.08	0.8	78 × 9
2005	702	3.97	4.0	2.19	0.9	39 × 18
2006	–	–	–	–	–	–
2007	630	2.90	2.0	2.02	0.8	126 × 5
2008	840	2.39	2.0	2.03	0.8	168 × 5
2009	840	2.16	2.0	2.07	0.9	168 × 5
2010	900	1.71	0.0	2.29	0.9	90 × 10

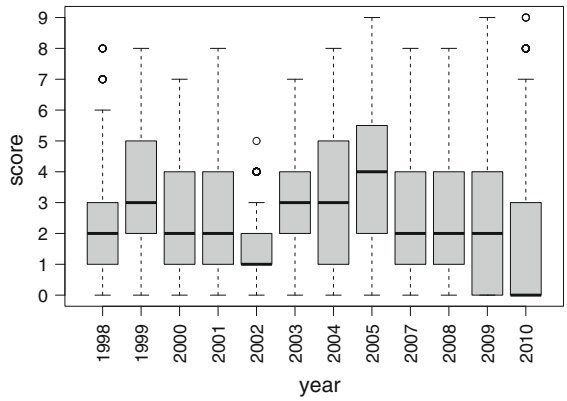

Fig. 3 Box plot distributions of observed powdery scab severity scores from field trials, 1998–2010

than RR1, but could not compete with the CORH or FA*k* models. The year-to-year genetic correlation estimate for CORH was 0.81 while the year-to-year genetic correlations for FA1 ranged from 0.59 to 0.95 and are illustrated in Fig. 4. These were compared with correlations estimated from a bivariate model for each pair of years. There was good agreement between many pairs, but the bivariate results showed a greater range (0.41–0.98) with less extreme values found in the FA analysis. This suggested that the retention of a small number of multiplicative (interaction) terms in this model resulted in a shrinkage of back-transformed correlation estimates.

There was no particular year-to-year trend in genetic correlation estimates from the FA1 model (Fig. 4), although the correlations between 2002 and 2003, and all other years, were mostly below 0.75. This may have been a consequence of the poorer expression of phenotype during these 2 years, particularly in 2002. All other year-to-year correlations (ignoring 2002 and 2003) were greater than 0.75.

The comparison of BLUPs in Fig. 5 shows the shrinkage of empirical breeding values estimated from a DIAG genetic variance structure compared with those estimated from CORH. The product-moment coefficient of correlation between CORH and DIAG was 0.73 and between CORH and FA1 was 0.99. From simulation, the response to selection (at different levels of *s*, the proportion of the population selected) from CORH was slightly greater than for FA1 (Table 5), although the estimates for FA1 were all within the limits of the 95 % CI for CORH.

Table 6 is a (non-exhaustive) list of breeding lines, advanced clones and cultivars of interest that have been used in the past 12 years as parents in the PFR breeding programme. Many of the named cultivars, such as 'Desiree', 'Atlantic', 'Kennebec' and 'Shepody' are popular production cultivars in a number of potato-producing regions of the world. 'Gladiator', a New Zealand-bred cultivar, is highly resistant to powdery scab on tubers, as demonstrated both in New Zealand (Falloon et al. 2003) and Europe (Falloon et al. 2003; Merz et al. 2012). The top ranking genotype is VTN62-33-3, a pollen parent of 'Gladiator', which has been used extensively as a parent in the PFR population in recent years. 'Agria' and 'Kennebec' are popular cultivars widely reported to

Table 3 Variance and spatial components, and heritability estimates from single trial analyses (including pedigrees) for powdery scab field trials, 1998–2010

Trial	Variance components			Column	Residual	Spatial components		h^2[95 % CI][f]
	Additive	Block	Row			AR1 (row)	AR1 (column)	
1998[a]	1.40	0.07			1.35	0.13		0.50 [0.43, 0.56]
1999[a]	1.44	0.06	0.01	0.10	1.62			0.45 [0.36, 0.55]
2000[b]	1.37	0.05			1.55		0.09	0.46 [0.43, 0.68]
2001[b]	1.51	0.05			1.20	0.10	0.05	0.55 [0.49, 0.64]
2002[b]	0.33	0.01			0.95			0.26 [0.20, 0.44]
2003[c]	0.82	0.17			1.41	0.11		0.34 [0.26, 0.45]
2004[c]	2.33	0.14			1.86			0.54 [0.50, 0.74]
2005[c]	1.69			0.28	2.89			0.35 [0.28, 0.52]
2007[d]	2.50	0.01			1.90			0.57 [0.53, 0.85]
2008[d]	2.47	0.09	0.17	0.08	1.84			0.53 [0.44, 0.65]
2009[e]	1.67	0.06	0.06	0.15	2.60			0.37 [0.28, 0.52]
2010[e]	2.40		0.21	0.08	2.93			0.43 [0.38, 0.57]

[a,b,c,d,e] Indicates that the same trial area of land was used for disease screening in the years that share the same letter

[f] 95 % Jackknife CI

Table 4 Summary of variance models, number of variance parameters and goodness of fit for powdery scab severity scores from field trials, 1998–2010

Model	G_0 structure	No. variance parameters	Total	[a]G_g		[b]G_a	
		G_0		[c]AIC	$-2\log L$	[c]AIC	$-2\log L$
1	SIMPLE	1	40	598	17,523	409	17,335
2	DIAG	12	51	506	17,410	335	17,239
3	CORH	13	52	162	17,064	0	16,902
4	FA1	24	63	162	17,042	6	16,887
5	FA2	35	74	165	17,023	10	16,868
6	RR1	12	51	257	17,161	114	17,018
7	RR2	23	62	281	17,163	37	16,919

[a] Genetic variance matrix for independent effects

[b] Genetic variance matrix for additive effects

[c] Expressed as a difference from the best fitting model (Model 3, with pedigree fitted)

be susceptible to tuber infection (Harrison et al. 1997). The current study has shown that these cultivars are more likely to transmit disease susceptibility to progeny given their high New Zealand EBVs.

There was no evidence of a genetic improvement for resistance to powdery scab across years in the population of breeding lines tested between 1998 and 2010, as illustrated in Fig. 6. There was a trend towards greater susceptibility in the population from 2003, but the mean EBVs of the population recovered to 1998 levels in 2009 and 2010.

Discussion

Many plant breeding programmes have access to historical METs and pedigree data, particularly with the application of versatile plant breeding databases. The genetic evaluation of such data requires robust model selection for accurate estimations of both genetic parameters of traits and breeding values of genotypes. This study confirms the usefulness of models that account for heterogeneity amongst different but related environments in that they are better

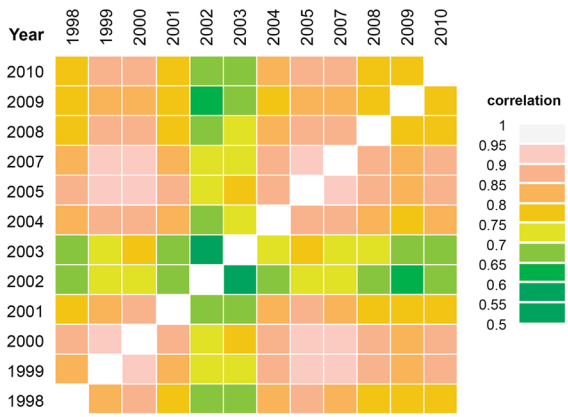

Fig. 4 Graphical illustration of genetic correlations from FA1 model (Model 4, with pedigree) for powdery scab severity trials, 1998–2010

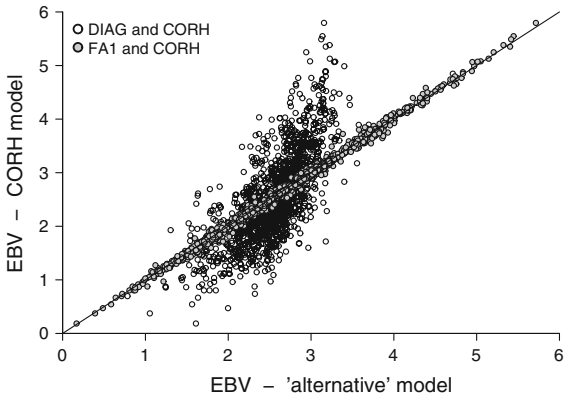

Fig. 5 Scatterplot of estimated breeding values (EBV), with the 'alternative' model representing DIAG (Model 2) or FA1 (Model 4) against the best fitting model CORH (Model 3), so that: *open circles*; DIAG and CORH, and *filled (grey) circles*; FA1 and CORH

than simpler versions where this heterogeneity is assumed to be absent.

Factor analytic (FA) models that incorporate pedigree information have been recommended for use in early stage plant breeding METs as they give accurate predictions of treatment effects (Kelly et al. 2007) and are parsimonious alternatives to the unstructured (US) genetic effects model that are computationally tractable (Thompson et al. 2003). The adoption of these approaches by plant breeders within the linear mixed modelling framework however, has been reported to be slow (Smith et al. 2005; Piepho et al. 2008a; Beeck et al. 2010). A number of plant breeding studies have illustrated the modelling

Table 5 Responses to selection for DIAG, CORH and FA1 variance models computed by simulation at selection fraction *s*

s	Response to selection (95 % CI)		
	DIAG	CORH	FA1
0.10	0.623 (0.614 0.629)	1.647 (1.601 1.681)	1.628 (1.592 1.661)
0.15	0.558 (0.552 0.563)	1.444 (1.412 1.471)	1.430 (1.400 1.455)
0.25	0.467 (0.462 0.470)	1.151 (1.128 1.173)	1.142 (1.120 1.164)
0.35	0.394 (0.391 0.397)	0.918 (0.900 0.934)	0.912 (0.894 0.927)
0.45	0.336 (0.334 0.338)	0.730 (0.715 0.743)	0.725 (0.711 0.737)
0.55	0.283 (0.281 0.285)	0.571 (0.559 0.581)	0.567 (0.555 0.577)
0.65	0.230 (0.229 0.231)	0.432 (0.422 0.441)	0.429 (0.420 0.438)
0.75	0.174 (0.172 0.175)	0.307 (0.300 0.314)	0.305 (0.298 0.312)

of G × E for the analyses of MET data and the benefits of including pedigree information for early stage selection. For instance, studies on canola (Beeck et al. 2010) and barley (Kelly et al. 2009) have demonstrated the superiority of accommodating the pedigree and of FA models over various homogeneous-heterogeneous (co)variance and fully reduced rank (RR) models. The present study also found the FA models were preferred over the RR models (although not as easy to fit), but the application of FA models may not always be necessary for trial data if the heterogeneity of correlated genetic effects between trials is considered to be low. For the long-term trial data analysed in the present study, a more straightforward and parsimonious homogeneous genetic covariance structure (with heterogeneous genetic variances) was found to be adequate when applied to the genetic evaluation of resistance to powdery scab. This was the best fitting model, with a year-to-year genetic correlation estimate of 0.81, which compared with estimates ranging from 0.59 to 0.95 for FA1. From empirical studies under cross-validation, Burgueño et al. (2011) reported that for crop data without G × E crossover interaction, i.e. no re-ranking of genotypes, FA models neither improved nor lost predictive ability (correlation between observed and predicted performance of genotypes)

Table 6 Estimated breeding values (EBVs) for powdery scab score; breeding lines, advanced clones and popular named cultivars used as parents in the New Zealand Plant & Food Research potato breeding programme comparing CORH (Model 3) and DIAG (Model 2), ranked on EBVs from Model 3 (resistant to susceptible)

Individual	Parent		CORH (Model 3) pedigree)		DIAG (Model 2)		
	Maternal	Paternal	EBV	SE_{EBV}	EBV	SE_{EBV}	Rank
VTN62-33-3	(V24/20 × U.KNIGHT) × PROFIJT	VRN I-3 × PROFIJT	0.37	0.37	1.05	0.27	2
GLADIATOR	B5281-1	VTN62-33-3	0.70	0.10	0.78	0.09	1
FIANNA	KONST62-660	AM64-2	1.02	0.33	1.73	0.25	4
761/1	Unknown	X61	1.14	0.45	2.34	0.36	14
981/4	CREBELLA	V394	1.17	0.30	1.62	0.27	3
V394	D47/11	D42/8	1.54	0.18	1.74	0.16	5
MOONLIGHT	1463.1	V394	1.70	0.35	1.98	0.24	7
NADINE	(DESIREExM.PIPER) × vrn-seedling	PENTLAND DELL × vrn-seedling	1.72	0.60	2.36	0.32	15
VAN GOGH	ZPC69-C-239	GLORIA	1.79	0.56	2.49	0.35	21
LONE RANGER	RANGER RUSSET	V394	1.80	0.62	2.14	0.28	8
2765.5	1463.1	STAGE2BLUE	1.81	0.33	2.40	0.29	17
VALOR	CARA	93/2/10	1.81	0.43	2.38	0.30	16
3011.6	FIANNA	ND860-2	2.03	0.30	2.29	0.28	11
GLENNA	10223-7	10300-13	2.09	0.63	2.44	0.34	20
813/28	354/7	FIANNA	2.12	0.37	2.60	0.27	25
DESIREE	URGENTA	DEPESCHE	2.17	0.55	2.31	0.32	12
1194/7	282/9	VTN62-33-3	2.24	0.40	1.78	0.30	6
RED RASCAL	TEKAU	DESIREE	2.30	0.48	2.42	0.29	19
TUTEKURI	Unknown	Unknown	2.30	1.04	2.59	0.38	23
759/3	178/4	B113-6	2.31	0.44	2.42	0.33	18
TEKAU	1584C-10	302.01	2.32	0.67	2.64	0.33	27
940/5	RUA	L115-1	2.35	0.24	2.81	0.21	37
RANGER RUSSET	BUTTE	A6595-3	2.39	0.34	2.66	0.26	29
LAURA	ROSELLA	L6140/2	2.40	0.78	2.62	0.36	26
SUMMER DELIGHT	1858.21	V394	2.43	0.32	2.27	0.26	9
MARKIES	FIANNA	AGRIA	2.45	0.64	2.28	0.31	10
DRAGA	SVP50-2017	MPI19268	2.58	0.71	2.55	0.36	22
MARIS PIPER	Y22/6	ARRAN CAIRN × HERALD	2.62	0.82	2.34	0.34	13
PENTLAND IVORY	PENTLAND CROWN	PENTLAND DELL	2.62	0.97	2.75	0.37	33
MARIS BARD	Y15/139	ULSTER PRINCE	2.72	0.93	2.68	0.36	30
ATLANTIC	WAUSEON	LENAPE	2.73	0.49	2.83	0.34	38
3097.5	WHITE DELIGHT	V99	2.85	0.42	2.65	0.32	28
FRASER	676.34	WHITU	2.89	0.52	2.78	0.33	35
PURPLE HEART	1463.1	STAGE2BLUE	2.97	0.79	2.59	0.33	24
RUA	KATAHDIN	HARFORD	2.99	0.47	2.97	0.27	43
1025/2	KARAKA	L115-1	3.03	0.37	3.17	0.26	51
KAIMAI	RUA	V394	3.06	0.32	2.72	0.23	32
ALLURA	KAIMAI	L115-1	3.11	0.79	3.10	0.29	50
AGRIA	QUARTA	SEMLO	3.18	0.38	2.78	0.28	34

Table 6 continued

Individual	Parent		CORH (Model 3) pedigree)		DIAG (Model 2)		
	Maternal	Paternal	EBV	SE_{EBV}	EBV	SE_{EBV}	Rank
L118-2	H614-1	Unknown	3.21	0.38	3.06	0.31	48
GOLDEN MIRACLE	AGRIA	FRASER	3.24	0.70	2.81	0.33	36
1021/1	FIANNA	L115-1	3.30	0.21	3.02	0.21	45
KARAKA	002/9	V394	3.38	0.29	2.87	0.21	40
2886.3	AGRIA	2221.12	3.43	0.30	3.03	0.26	47
KENNEBEC	USDAB127	USDA96-56	3.45	0.71	3.07	0.33	49
KATAHDIN	USDA40568	USDA24642	3.52	0.75	3.03	0.31	46
KIWITEA	002/9	D42/8	3.53	0.53	2.71	0.31	31
L115-1	H612-3	Unknown	3.55	0.20	3.52	0.17	54
SHEPODY	BAKE-KING	F58050	3.71	0.62	2.93	0.35	41
2955.19	PACIFIC	FRASER	3.87	0.37	2.93	0.32	42
2958.10	285/1	2116.2	3.97	0.35	2.87	0.31	39
2850.6	AGRIA	RUA	4.23	0.48	2.99	0.30	44
COLIBAN	KENNEBEC	V28-12	4.39	0.51	3.25	0.31	53
1155/3	KENNEBEC	KARAKA	5.13	0.40	3.23	0.29	52

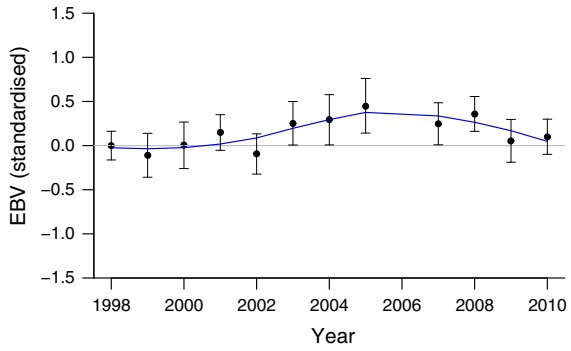

Fig. 6 Mean (standardised) estimated breeding values (EBV) across years for powdery scab assessment trials, 1998–2010. Decreasing values indicate increasing resistance to powdery scab; CORH model with fitted non-parametric smoothing curve and 95 % CI (from parametric bootstrapping)—1998 is the base year (mean EBV = 0)

when compared with a simple linear mixed model (homogenous variances, independent covariances between pairs of sites). For other data, when there was crossover interaction, FA models increased the predictive ability by up to 6 % compared with the simple model form. FA methods were therefore recommended to model G × E regardless of the type of interaction. Using different variance structures for cross-validation and simulation analyses of maize hybrid MET trials, So and Edwards (2011) found no substantial improvement when including heterogenous genetic (co)variance structures over simpler models. This was attributed to poor genetic links between years. Future work in potato will aim to further augment MET trial and pedigree data with molecular information for the evaluation of traits such as resistance to powdery scab. In wheat MET trials, Burgueño et al. (2012) have shown that G × E models that incorporate both pedigree and marker data improved the predictive accuracy over those that included pedigree information alone.

The magnitudes of narrow-sense heritabilities estimated in the present study were, in general, moderate (i.e. between 0.25 and 0.60) and showed the importance of an additive genetic component in the breeding population. Previous research on the inheritance of powdery scab resistance is limited and does not provide any genetic parameter estimates for comparison. From progeny tests, Wastie (1991) concluded that evaluation of parental phenotype for tuber resistance offered an indication of mean progeny performance, which suggested that, in the population studied, the additive genetic component was important and the parental phenotype provided a reasonable representation of its breeding value. The narrow-sense heritability estimates published here are based on

standard formulae found in quantitative genetics texts for individual trials. Cullis et al. (2006), Oakey et al. (2006) and Piepho and Möhring (2007) have proposed alternative methods to calculate heritability in a MET setting, arguing that the standard method overestimates the prediction of genetic gain if variance components have been estimated from more complex covariance models, rather than a simple *genotype + residual* model with balanced data. The method of Piepho and Möhring (2007), which directly simulates the response to selection, was used to compare the different variance models tested in the MET analyses. This showed that there was minimal difference between the two models in terms of the response to selection. This supports the result that the CORH model provided only a slight improvement over the FA1 model with regard to AIC as the selection criterion.

The additive relationship matrix **A** used in the individual model in all publically-available software assumes disomic inheritance. This may not be appropriate when dealing with autopolyploid crops, including potato. This is because of double reduction at meiosis whereby sister chromatids can end up in the same gamete. Under simplistic assumptions, such as no past selection, double reduction or inbreeding, the expected additive genetic covariances both of diploid and tetraploid relatives is equivalent (Lynch and Walsh 1998). Despite the acknowledged approximation of the **A** matrix, the inclusion of pedigree information improved model fit for these potato data. This is in agreement with other crops, such as canola (Beeck et al. 2010) and sugarcane (Oakey et al. 2007), which do not meet the general assumptions required of disomic inheritance and obligate (i.e. non-selfing) outcrossing species. Recent work has generalised the relationship matrix and its inverse to better accommodate autopolyploid crop species (Kerr et al. 2012) and should be considered in future work on the genetic evaluation of tetraploid potato.

There was no evidence of any important non-additive effects in the present study indicating that a genetic model with only additive effects is adequate for the evaluation of resistance to powdery scab. Non-additive effects were tested, following Kelly et al. (2009) and Beeck et al. (2010), by simply assuming independence between non-additive genetic components. Oakey et al. (2007) partitioned the non-additive genetic effects into dominance, via the dominance relationship matrix **D**, and residual genetic components. Fitting a non-additive component may remove confounding that could be present, for example, common environment of siblings, and possibly reduce bias in breeding value estimation. Further, estimates of non-additive effects for individuals could be used to select parental combinations to exploit the non-additive genetic variance (Mrode 1996, p.114; Oakey et al. 2007). Kelly et al. (2009) noted that the estimation of non-additive effects in complex models are likely to often represent only a small proportion of the total genetic variance (e.g. Hill et al. 2008) and are likely to be characterised by large sampling errors.

It is widely reported that there is much variation in the year-to-year incidence and severity of powdery scab and the attributing factors are largely considered to be variations in rainfall and temperature that influence soil conditions and the rate of crop development (reviewed by Harrison et al. 1997; Merz 2008; Merz and Falloon 2009). Screening procedures involving artificial inoculation with pathogens such as *S. subterranea* are expected to inflate the within-trial heritability compared with trials that rely on natural infection. Further, greater stability, or homogeneity, in the estimates of the between-year genetic correlations is likely, particularly if there is some degree of control over the environmental conditions that predispose disease development, such as soil water availability. This may also be the case for many other quantitatively-inherited diseases in various crop species in which progenies are assayed in glasshouse or in field assessments.

As well as the problems associated with tuber infection, such as poor tuber quality and product rejection, *Spongospora* root galling has been shown to reduce root function and plant growth by impeding water and nutrient uptake (Lister et al. 2004; Falloon et al. 2005; Shah et al. 2012). Further research should therefore aim to determine the genetic relationship between tuber infection and root infection. This information will assist in determining an appropriate selection strategy, as simultaneous improvement of both traits is required in the breeding objective. Several studies on clones have found no evidence of correlation between tuber lesion severity and root galling (van de Graaf et al. 2007; Baldwin et al. 2008; Merz et al. 2012), although a positive correlation between the levels of root infection by *S. subterranea* zoosporangium and tuber scab formation has been

reported (Falloon et al. 2003). The common potato processing cultivar 'Russett Burbank' is known to display few tuber symptoms but often shows symptoms of severe root galling. Root symptoms may go unnoticed but development of *S. subterranea* sporosori (containing resting spores) in root galls will provide reservoirs of inoculum that have long-term consequences for potato production because of the long-term survival of resting spores (Merz et al. 2012). Information on root infection is more difficult (and probably more costly) to routinely collect in early stage testing in a breeding programme, suggesting that powdery scab on tubers would be the preferred trait of the two in a selection criterion. Because of the resources required for screening roots and tubers, both may be good candidates for molecular-based selection approaches.

Merz et al. (2012) found no evidence of cultivar × location effect for powdery scab resistance in clones over 4 years in a European study. They concluded that multiple-location testing in a cultivar development programme should not be necessary. Evidence indicated limited *S.subterranea* pathotype variation and suggested that the genetic mechanisms for the pathogen to overcome host resistance were probably less dynamic than those associated with airborne pathogens such as *Phytophthora infestans* (which causes potato late blight). It is unclear if there is a sexual phase in the life-cycle of *S.subterranea* but from genomic analysis of diverse field collections, only two genetically distinct groups had been previously identified (Merz 2008). In a more recent study, a total of 19 haplotypes were identified by Gau et al. (2013). Two of these variants were found to be widely distributed globally, while the other 17 were only found in South America. These results suggest that EBV information from trials in, say, New Zealand may be used to aid selection in other locations with a reasonable level of confidence.

The assumption of homogeneity within block and/or row and column factors in the trials appeared to hold as the analysis of individual trials found no evidence of any important localised spatial effects in most years, using an autoregressive procedure of order one. Even where there was an apparent effect for rows (1998, 2001, 2003) and for columns (2000, 2001) the estimated parameters were small and ranged from 0.10 to 0.13 for rows and 0.05 to 0.09 for columns. Their inclusion (or omission) in future evaluations is likely to make very little difference to final selection decisions. However, the effort required to check spatial effects is minimal compared to the effort and resources invested in a trial, and thus it would be advisable to do so. This study considered the separable autoregressive spatial method only, and chose not to investigate other approaches for spatial adjustment (e.g. Gleeson 1997; Piepho et al. 2008b) as it was not the main purpose of this study. For powdery scab resistance, studies have indicated that the amount of artificially added inoculum in the soil had no effect on the incidence and severity of visual symptoms of lesions on potato tubers (van de Graaf et al. 2007; Shah et al. 2012). Severe infection has been shown to occur when inoculum levels in soil were considered to be low, with no consistent relationship between soil inoculum level and severity of infection (Shah et al. 2012). This may partly explain the apparent lack of important spatial components. Trial sites for the PFR programme are used for three consecutive years and re-innoculated each year. With no reported dose effects, it is unlikely that there will be changes in the importance of spatial components as inoculum distribution across the sites possibly become more uniform over time (less spatial heterogeneity). In contrast, there may be an expectation of more localised spatial heterogeneity (or 'patchiness') as soil inoculum levels build during this period. No such patterns or trends were evident from these 12 years of data. Soil water content affects powdery scab development on tubers, particularly if water content is high over the tuberization period (Merz and Falloon 2009). The PFR powdery scab trials were regularly irrigated over the growing season, to maintain uniform conditions that are conducive for disease development on tubers.

There was no evidence of genetic improvement for powdery scab resistance—that is, no incremental and consistent decrease of mean EBV in the breeding population over the 12 years of trials. This suggests that selection pressure for the disease has been weak, possibly partly due to a slow and reluctant replacement of older and susceptible parents and long generation intervals in the breeding programme. Further, we have little or no understanding of the genetic relationships between powdery scab resistance and other traits that may feature more prominently in a breeding programme. Powdery scab resistance is recognised by breeders to be an important component of an economic breeding objective, but other traits, such as yield and

tuber quality, are likely to exercise much more weight in an (implicit) selection index. Resistant cultivars have been developed, but this has relied on selection of individual clones that display an acceptable level of phenotypic resistance at an advanced stage of selection. It has not been a direct consequence of a population improvement approach. The cultivar 'Gladiator', for example, was developed in the PFR potato breeding programme and has demonstrated a high level of powdery scab resistance in New Zealand and Europe (Merz et al. 2012). Selection of resistant parents based on estimates of their EBVs should help confer resistance to progeny, and therefore be part of a recurrent selection strategy for a population-based improvement of this economically-important characteristic.

Conclusions

This study has shown that a homogeneous genetic correlation model (with heterogeneous variances) was simpler and more parsimonious than a factor analytic model determined from METs for the genetic evaluation of resistance to powdery scab in potato tubers. Simpler models should not be overlooked in the evaluation of plant breeding data if they can compete with more complex forms. Unnecessarily increasing the complexity of models should be avoided if plant breeders are to routinely adopt the genetic evaluation of MET data in a linear mixed model framework for early stage selection. The additive component of variation was important and narrow-sense heritabilities were moderate. The year-to-year genetic correlations were generally high. There was no evidence of non-additive genetic effects, and local-scale spatial heterogeneity was not apparent. Further work is required to determine the genetic relationship between tuber powdery scab and *Spongospora* root infection to help devise a comprehensive breeding strategy for resistance to this pathogen. Exploiting correlated information for the estimation of breeding values in disease screening trials should assist breeders to improve the quantitative resistance to powdery scab of potato tubers in breeding populations. The success of this in a multi-trait selection strategy will, of course, ultimately depend on a number of other factors such as its hitherto unknown genetic relationships with other target traits and the relative importance it is afforded in the selection criterion.

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