

1 **Marker-based estimates reveal significant non-additive effects in clonally**
2 **propagated cassava (*Manihot esculenta*): implications for the prediction of total**
3 **genetic value and the selection of varieties**

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5 Marnin D. Wolfe^{*}, Peter Kulakow[†], Ismail Y. Rabbi[†], Jean-Luc Jannink^{*‡}

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7 * Department of Plant Breeding and Genetics, Cornell University, Ithaca, NY, USA

8 † International Institute for Tropical Agriculture (IITA), Ibadan, Oyo, Nigeria

9 ‡ USDA-ARS, R.W. Holley Center for Agriculture and Health, Ithaca, NY, USA

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12 **Running Title: Marker-based estimation of non-additive effects in cassava**

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15 **Key words: genomic selection, non-additive effects, cassava**

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18 Corresponding Author:

19 Marnin D. Wolfe

20 Cornell University

21 Department of Plant Breeding and Genetics

22 417 Bradfield Hall

23 306 Tower Road

24 Ithaca, NY 14853

25 Email: wolfemd@gmail.com

26 Phone: 239-595-5081

27 **ABSTRACT**

28

29 In clonally propagated crops, non-additive genetic effects can be effectively
30 exploited by the identification of superior genetic individuals as varieties. Cassava
31 (*Manihot esculenta* Crantz) is a clonally propagated staple food crop that feeds hundreds
32 of millions. We quantified the amount and nature of non-additive genetic variation for
33 key traits in a breeding population of cassava from sub-Saharan Africa using additive and
34 non-additive genome-wide marker-based relationship matrices. We then assessed the
35 accuracy of genomic prediction of additive compared to total (additive plus non-additive)
36 genetic value. We confirmed previous findings based on diallel populations, that non-
37 additive genetic variation is significant, especially for yield traits. Further, we show that
38 we total genetic value correlated more strongly to observed phenotypes than did additive
39 value, although this is constrained by low broad-sense heritability and is not beneficial
40 for traits with already high heritability. We address the implication of these results for
41 cassava breeding and put our work in the context of previous results in cassava, and other
42 plant and animal species.

43

44 INTRODUCTION

45

46 Understanding genetic architecture requires the decomposition of genetic effects
47 into additive, dominance, and epistatic components (Fisher 1918; Cockerham 1954;
48 Kempthorne 1954). However, partitioning genetic variance components is notoriously
49 difficult, requiring specialized breeding designs (e.g. diallel crosses) and pedigree
50 information (Lynch and Walsh 1998) often limiting the genetic diversity that can be
51 sampled in any one given study. Genome-wide molecular marker data now enable the
52 accurate measurement of relatedness in the form of genomic realized relationship
53 matrices (GRMs) (VanRaden 2008; Heffner *et al.* 2009; Lorenz *et al.* 2011b). GRMs
54 provide more accurate relatedness information than pedigrees because they directly
55 measure Mendelian sampling (causing variation in relatedness within relatedness classes
56 such as full-siblings) (Heffner *et al.* 2009). Further, GRMs can measure relationships
57 even in diverse, nominally unrelated samples expanding the potential for studying
58 inheritance in natural and breeding populations (Lorenz *et al.* 2011a).

59 Estimation of narrow-sense heritability and prediction of breeding values in
60 genomic selection programs is becoming increasingly common using additive
61 formulations of GRMs (Visscher *et al.* 2008). Several recent studies have described
62 dominance and epistatic GRMs for the partitioning of non-additive genetic variance using
63 genome-wide SNP markers (Su *et al.* 2012; Vitezica *et al.* 2013; Muñoz *et al.* 2014;
64 Wang *et al.* 2014). Models using these new formulations have been shown to provide
65 improved partitioning of genetic variances relative to pedigree-based approaches (Su *et al.*
66 2012; Muñoz *et al.* 2014). These new models can be used not only to estimate genetic

67 variances but also for genomic prediction of additive and total genetic value in genomic
68 selection breeding programs (Su *et al.* 2012; Vitezica *et al.* 2013; Muñoz *et al.* 2014;
69 Wang *et al.* 2014).

70 Cassava is a vegetatively propagated, staple food crop that is high in starch and
71 feeds half a billion people worldwide (<http://faostat.fao.org>). Efforts to improve cassava
72 genetically with cutting edge methodologies including transgenic and genomic selection
73 (GS) approaches are underway thanks to new genomic resources (Prochnik *et al.* 2012;
74 ICGMC) 2014). Prediction of additive genetic value has recently been evaluated
75 (Oliveira *et al.* 2012; Ly *et al.* 2013) and genomic selection using standard models is
76 currently being tested (<http://www.nextgencassava.org>). Vegetatively propagated crop
77 (e.g. cassava) breeding can exploit non-additive genetic effects by identifying superior
78 clones as varieties (Ceballos *et al.* 2015).

79 Diallel studies in cassava indicate that non-additive genetic effects (i.e. specific
80 combining ability) are strong, particularly for root yield traits (Cach *et al.* 2005, 2006;
81 Calle *et al.* 2005; Jaramillo *et al.* 2005; Perez *et al.* 2005; Pérez *et al.* 2005; Zacarias and
82 Labuschagne 2010; Kulembeka *et al.* 2012; Tumuhimbise *et al.* 2014; Ceballos *et al.*
83 2015; Chalwe *et al.* 2015). If the limited number of parents tested thus far represents the
84 broader cassava breeding germplasm, genetic gains, especially for already low-
85 heritability root yield traits will be slow regardless of the breeding scheme employed (e.g.
86 phenotypic vs. pedigree vs. genomic selection). Breeding gains have indeed been slow in
87 cassava (Ceballos *et al.* 2012) and low accuracies have been reported for genomic
88 prediction of yield compared with cassava mosaic disease (CMD) resistance and dry
89 matter (DM) content (Oliveira *et al.* 2012; Ly *et al.* 2013). However, cassava varieties are

90 evaluated and disseminated to farmers by clonal propagation, meaning that accurate
91 prediction of total (additive plus non-additive) genetic value could contribute to variety
92 selection.

93 In this study, we test the hypothesis that certain cassava traits, especially root
94 yield have relatively large non-additive genetic variance that account for low genomic
95 prediction accuracies previously observed. We estimate additive and non-additive
96 variance components using genomic relationship matrices in two populations of cassava
97 from the International Institute of Tropical Agriculture's (IITA) genomic selection
98 breeding program. Further, we assess the accuracy of predicting additive and total genetic
99 value using the additive and non-additive models. We discuss the origin of non-additive
100 genetic variance in cassava, its potential effect on cassava breeding, and its role in
101 genomic selection strategies for cassava improvement in the future.

102

103 **METHODS**

104 **Germplasm and Phenotyping Trials**

105 We examined additive and non-additive effects in two populations of cassava that
106 have been genotyped and phenotyped as part of the Next Generation Cassava Breeding
107 Program at IITA, Nigeria (<http://www.nextgencassava.org>). The IITA's Genetic Gain
108 (GG) population contains 694 historically important clones, most of which are advanced
109 breeding lines although some are classified as superior landraces. These lines have been
110 selected and maintained clonally since 1970 (Okechukwu and Dixon 2008; Ly *et al.*
111 2013). Most of these materials are derived from the cassava gene pool from West Africa
112 as well as parents derived from the breeding program at Amani Station in Tanzania and

113 hybrids of germplasm introduced from Latin America. Available information on the GG
114 accessions included in our analyses is provided in Table S1.

115 IITA's Genetic Gain trials were conducted in seven locations over 14 years (2000 to
116 2014) in Nigeria for a total of 24,373 observations. Each GG trial comprises a
117 randomized, incomplete block design replicated one or two times per location and year.
118 Since materials have been occasionally lost and new, selected materials are continuously
119 added to the GG, the number of clones trialed in a given year changes gradually across
120 years, generally increasing. The sample sizes, number of replicates and number of clones
121 from the GG in each of the trials (location-year combinations) is provided in Table S2.

122 Theory suggests that founding events and truncation selection can both lead to a
123 conversion of non-additive genetic variation into additive variance. This can happen
124 because of the induction of linkage disequilibrium and reduction in allele frequency (or
125 fixation of alleles) at some loci relative to others (Goodnight 1988; Turelli and Barton
126 2006; Hallander and Waldmann 2007). Consequently, our results might depend on the
127 population examined. We therefore analyzed an additional population: a collection of
128 2187 clones that are the direct descendants of truncation selection on the GG. Briefly, in
129 2012 the GG and all available historical phenotype data was used as a reference
130 population to obtain genome estimated breeding values (GEBVs) using the genomic
131 BLUP (GBLUP) model (VanRaden 2008; Heffner *et al.* 2009). Selection was based on
132 an index that included mean cassava mosaic disease severity (MCMDS), mean cassava
133 bacterial blight disease severity (MCBBS), dry matter content (DM), harvest index (HI)
134 and fresh root weight (RTWT). This index of GEBVs was used to select 83 members of

135 the GG to cross and generated a population of 137 full-sib families, which we refer to as
136 the GS Cycle 1 (C1). The pedigree of the C1 is available in Table S3.

137 Cycle 1 progenies were evaluated in a single clonal evaluation trial during the 2013-
138 2014 field season across three-locations (Ibadan, Ikenne, and Mokwa). For the C1 clonal
139 trial, planting material was only available for one plot of five stands per clone, so each
140 clone was only planted in one of the three locations (Table S2). Clones were assigned to
141 each location so as to equally represent each family in every environment.

142 For both populations, we analyzed the five traits described above (MCMDS, MCBBS,
143 DM, HI, RTWT) plus sprouting ability (SPROUT). MCMDS and MCBBS were scored
144 on a scale of 1 (no symptoms) to 5 (severe symptoms). Most GG trials measured dry
145 matter (DM) by the oven drying method although some trials used the specific gravity
146 method. DM is expressed as a percentage of the fresh weight of roots. Fresh root weight
147 (RTWT) is measured in kilograms per plot and harvest index (HI) is the percent of total
148 biomass per plot (roots plus shoots) that is RTWT. Sprouting ability (SPROUT) was
149 expressed as the percent of planted stakes sprouting at one month after planting.

150

151 **Genotype data**

152 We used genotyping-by-sequencing (GBS) to obtain genome-wide SNP marker data
153 (Elshire *et al.* 2011). We used the ApeKI restriction enzyme as recommended by
154 (Hamblin and Rabbi 2014). SNPs were called using the TASSEL GBS pipeline V4
155 (Glaubitz *et al.* 2014) and aligned to the cassava reference genome, version 5, which is
156 available on Phytozome (<http://phytozome.jgi.doe.gov>) and described by the International
157 Cassava Genetic Map Consortium ((ICGMC) 2014). We removed individuals with more

158 than 80% and markers with >60% missing genotype calls. Also, markers with extreme
159 deviation from Hardy-Weinberg equilibrium (Chi-square > 20) were removed. If there
160 were not at least two reads at a given locus for a given clone, the genotype was set to
161 missing and imputed. SNP marker data was converted to the dosage format (0, 1, 2) and
162 missing data were imputed with the glmnet algorithm in R ([http://cran.r-](http://cran.r-project.org/web/packages/glmnet/index.html)
163 [project.org/web/packages/glmnet/index.html](http://cran.r-project.org/web/packages/glmnet/index.html)) as described in (Wong *et al.* 2014). We
164 used 114,922 markers that passed these filters with a minor allele frequency greater than
165 1% to calculate genomic relationship matrices as described below.

166

167 **Genomic Relationship Matrices**

168 We measured the realized additive, dominance and epistatic relationships in our
169 population using functions that have been described previously (Su *et al.* 2012; Muñoz *et*
170 *al.* 2014). We used the additive realized relationship matrix, **A** as described by Van
171 Raden (VanRaden 2008): $\mathbf{A} = \frac{WW'}{2 \sum_i p_i q_i}$. Here **W** is a matrix of dimension n individuals by
172 m SNP markers. The elements of **W** are the marker dosages ($0 - 2p_i$) for aa, $(1 - 2p_i)$ for
173 Aa and $(2 - 2p_i)$ for AA, where p_i is the frequency of the second allele and q_i is the
174 frequency of the first allele, at the i th locus. The a (or 0) allele refers to the reference
175 genome allele. The **A** matrix was calculated using the *A.mat* function in the *rrBLUP*
176 package (Endelman 2011).

177 As described by Su *et al.* (Su *et al.* 2012) the dominance relationship matrix, **D** is

178 $\mathbf{D} = \frac{HH'}{\sum_i 2p_i q_i (1 - 2p_i q_i)}$ (also see (Muñoz *et al.* 2014)). Where **H** has the same dimensions as

179 **W**, heterozygotes are scored $(1 - 2p_i q_i)$ and homozygotes are $(0 - 2p_i q_i)$. We made a

180 custom modification (available upon request) to the *A.mat* function to produce the **D**

181 matrix. Relationship matrices that capture epistasis can be calculated by taking the
182 hadamard product (element-by-element multiplication; denoted #) of two or more
183 matrices (Henderson 1985). For simplicity, we only explored additive-by-additive (**A#A**)
184 and additive-by-dominance (**A#D**) relationships in this study.

185

186 **Variance component and heritability models**

187 *Single-step, Multi-environment*

188 We used several approaches to estimate the relative importance of additive and non-
189 additive effects in the Genetic Gain and Cycle 1 populations. In the first analysis, we
190 analyzed the multi-year, multi-location GG data with a single random effects model.
191 Since the entire historical phenotype dataset is large (24,373 observations) and was
192 relatively unbalanced in sample size across years and locations, we only analyzed data
193 from trials with >400 individuals. This filter resulted in a dataset of 7745 observations
194 from three locations (Ibadan, Ubiaja, Mokwa) and eight years (2006-2014, excluding
195 2012). All 694 genotyped GG clones were represented in this dataset (Table S2).

196 The models we fit were similar to those described in Ly et al. (Ly *et al.* 2013). The
197 full model was specified as follows: $y = X\beta + Z_{loc.year}l + Z_{rep}r + Z_{add}a + Z_{dom}d +$
198 $Z_{epi}i + \varepsilon$. Here, y represents raw phenotypic observations. In our data, the only fixed
199 effect (β) was an intercept for all traits except RTWT, which contained a covariate
200 accounting for variation in the number of plants harvested per plot. The random effects
201 terms for experimental design terms included a unique intercept for each trial (i.e.
202 location-year combination), $l \sim N(0, I\sigma_l^2)$, where I is the identity matrix and σ_l^2 is the

203 associated variance component as well as a replication effect, nested in location-
204 year combination, $r \sim N(0, I\sigma_r^2)$.

205 The genetic variance component terms included $a \sim N(0, A\sigma_a^2)$, where A is the
206 additive relationship matrix and σ_a^2 is the additive genetic variance component.
207 Similarly, $d \sim N(0, D\sigma_d^2)$, is the dominance effect with covariance D equal to the
208 dominance relationship matrix and σ_d^2 equal to the dominance variance. The
209 epistatic term $i \sim N(0, E\sigma_i^2)$ where the covariance matrix E took the form either of
210 the A#A matrix (additive-by-additive) or the A#D matrix (additive-by-dominance)
211 and the epistatic variance σ_i^2 was correspondingly either $\sigma_{A\#A}^2$ or $\sigma_{A\#D}^2$. The final
212 term, ϵ is the residual variance, assumed to be random and distributed $N(0, \sigma_\epsilon^2)$. The
213 terms X, $Z_{loc.year}$, Z_{rep} , Z_{add} , Z_{dom} and Z_{epi} are incidence matrices relating observations to
214 the levels of each factor. We list the different models fit in Table 1, each of which are
215 variations on the full model described above.

216 The formulation described above was used to fit the GG historical data in a single
217 model. For the C1 progenies only a single trial was available and therefore we fit all data
218 together. Since the C1 trial was conducted across three locations but with no replications
219 we fit the same model for C1 as GG excluding the replication term. The models described
220 above were fit using the *regress* package in R (R Core Team 2015). The *regress* function
221 finds REML solutions to mixed models using the Newton-Raphson algorithm.

222 For each trait, in both the C1 and GG we identified a “best fit” model among the
223 models listed in Table 1 based on the lowest Akaike Information Criterion (AIC; $2*k -$
224 $2*\log(\text{likelihood})$, where k = number parameters estimated). We also examined the log-
225 likelihood of each model and the total genetic variance explained (H^2). The precision of

226 variance component estimates and the dependency among estimates was examined using
227 the asymptotic variance-covariance matrix of estimated parameters (\mathbf{V}). Specifically, we
228 report standard errors for each variance component, defined as the square root of the
229 diagonal of \mathbf{V} . We also converted \mathbf{V} into a correlation matrix (\mathbf{F} , as in (Muñoz *et al.*
230 2014)), where \mathbf{F} is defined as $\mathbf{L}^{-1/2}\mathbf{V}\mathbf{L}^{-1/2}$ and \mathbf{L} is a diagonal matrix containing one over
231 the square root of the diagonal of \mathbf{V} . We use \mathbf{F} to assess the dependency of variance
232 components estimates, especially for comparing results among traits and populations.

233

234 *Within-trial analyses*

235 We used only a subset of the GG trials to estimate variance components in a
236 single multi-environment model. In addition, we leveraged the entire historical GG data
237 by analyzing each trial (N=47, unique location-year combinations) separately. This
238 provided us with 47 estimates of additive, dominance and epistatic variance and we
239 examine the distribution of variance components estimates. As in the multi-environment
240 models, within-trial models were fit with *regress* in R.

241

242 *Genomic prediction and cross-validation*

243 We assessed the influence that modeling non-additive genetic variance
244 components have on genomic prediction using a cross-validation strategy. Because
245 single-step multi-environment models are computationally intensive, we used a two-step
246 approach here. In the first step, we combined data from all available GG and C1 trials
247 using the following mixed model: $y = X\beta + Z_{rep}r + Z_{clone}g + \varepsilon$. In this model, β
248 included a fixed effect for the population mean, the location-year combination and for

249 RTWT only, the number of plants harvested per plot. As in the single-step, multi-
250 environment model for GG, we included the random replication effect $r \sim N(0, I\sigma_r^2)$. In
251 contrast to the previous model, we did not at this stage include a genomic relationship
252 matrix, instead we fit a random effect for clone, $g \sim N(0, I\sigma_g^2)$, where the covariance
253 structure was the identity matrix (I). The BLUP (\hat{g}) for the clone effect therefore
254 represents an estimate of the total genetic value for each individual. The mixed model
255 above was solved using the *lmer* function of the *lme4* R package.

256 In our data, the number of observations per clone ranges from one to 131 with
257 median of two and mean of 5.97 excluding the checks TMEB1 and I30572 which had
258 941 and 902 observations respectively. Pooling information from multiple years and
259 locations, especially when there is so much variation in numbers of observations can
260 introduce bias. Much theoretical development, particularly in animal breeding has been
261 done to address this issue, and we followed the approach recommended by Garrick et al.
262 (Garrick *et al.* 2009). Briefly, BLUPs for clone were deregressed according to $\frac{\hat{g}}{r^2}$ where r^2
263 is the reliability ($1 - \frac{PEV}{\sigma_g^2}$) and PEV is the prediction error variances of the BLUP. In the
264 second step of analysis, where deregressed BLUPs are used as response variables,
265 weights are applied to the diagonal of the error variance-covariance matrix **R**. Weights
266 are calculated as $\frac{1-h^2}{0.1 + \frac{1-r^2}{r^2}h^2}$, where h^2 is the proportion of the total variance explained by
267 the clonal variance component, σ_g^2 (Garrick *et al.* 2009).

268 We implemented a 5-fold cross-validation scheme replicated 25 times to test the
269 accuracy of genomic prediction using the genomic relationship matrices and models
270 described above (Table 1). In this scheme, for each replication, we randomly divided the

271 dataset into five equally sized parts (i.e. folds). We used each fold in turn for validation
272 by removing its phenotypes from the training population and then predicting them. We
273 calculated accuracy as the Pearson correlation between the genomic prediction and the
274 BLUP (\hat{g} , not-deregressed) from the first step. For each model, we calculated accuracy
275 both of the prediction from the additive kernel (where present) and the total genetic value
276 prediction, defined as the sum of the predictions from all available kernels (e.g. additive
277 + dominance + epistasis). Genomic predictions were made using the *EMMREML R*
278 package.

279 All raw genotype and phenotype data are available on www.cassavabase.org [an
280 exact link to be provided upon acceptance of manuscript]. Custom code used for analysis
281 is available upon request.

282

283 **RESULTS**

284 *Single-step, Multi-environment Variance Component Models*

285 Our first assessments of non-additive genetic effects in cassava were single-step
286 multi-environment models implemented in both the Genetic Gain (GG) and the Cycle 1
287 (C1) populations. The five models (Table 1) were initially compared using the AIC.
288 Tables 2 and 3 show the model results including AIC and variance components for the
289 GG and C1, respectively. For HI, MCMDS, and SPROUT the best models were A + D, A
290 + D + AD and A + D + AA, respectively. For these three traits, the best model according
291 to AIC was the same between the GG and C1. For DM the additive only model was best
292 in the GG but an A + D model was selected in C1. For RTWT, in the GG an A + D + AA
293 model was selected but in the C1 the dominance only model was selected. Finally, for

294 MCBBS the additive only model was best in the GG but A + D + AD was selected in the
295 C1.

296 For every trait, when comparing the model achieving the highest broad-sense
297 heritability (H^2), we saw that the H^2 increased from GG to C1. This can be seen most
298 easily in Figure 1, which shows how total explainable genetic variance (H^2) is partitioned
299 among variance components in the C1 and GG (also see Tables 2 & 3). In GG, the
300 additive only model had the highest H^2 for all traits, but in C1 additive + non-additive
301 models always had at least slightly higher H^2 .

302 For DM, the additive component explained more variance across all models in the
303 C1 compared to the GG, e.g. 0.33 (GG, Additive) and 0.51 (C1, Additive). For DM, the
304 A + D + AA model actually had the highest H^2 in the C1 but the variance component for
305 A#A epistasis was not distinguishable from zero (2.05 ± 2.59).

306 For HI, the additive only model was very similar between populations ($h^2 = 0.34$
307 in GG and 0.32 in C1). But for the best model, A + D, more of the total genetic variance
308 is partitioned to C1 (0.28) than in the GG (0.07). Like for DM, the three component
309 models (A + D + AA and A + D + AD) explained the most variance for the C1 ($H^2 =$
310 0.49), but the epistatic components had large standard errors (26.4 ± 18.7 and 24.4 ± 17.6
311 respectively).

312 RTWT was strongly non-additive in both populations. In the GG the A + D + AA
313 model, genetics explained 25% of the phenotypic variance with non-additive variances
314 (D + AA) explained over half of that amount (16% collectively) and the epistatic term
315 was significantly different from zero (0.033 ± 0.014). In the C1, 31% of the phenotypic
316 variance was explainable by dominance alone.

317 MCBBS had a low heritability overall ($H^2 = 0.04$ in the GG for the additive only
318 model, and 0.25 in the C1 for the A + D + AD model). While the limited apparent genetic
319 variance in the GG was best explained additively, in the C1 92% of total genetic variance
320 was non-additive.

321 For MCMDS, the additive model had the highest H^2 (0.79) in the GG but the AIC
322 best model, A + D + AD explained most (0.89) in the C1. The AIC best model for both
323 C1 and GG was the same. In both cases, significant AxD epistasis was detected,
324 explaining 44% of the variance in the GG and 18% in the C1. The additive variance
325 increased from GG (0.25) to C1 (0.64).

326 SPROUT was best modeled with A + D + AA in both populations. Broad-sense
327 heritability increased from 0.22 in GG to 0.39 in C1 for this trait. The dominance
328 component was not significant (5.8 ± 8.6) in the GG but was in the C1 (31.7 ± 26.7).

329 We examined the asymptotic correlation matrices of parameter estimates (**F**) to
330 ascertain the dependency of variance component estimation. Correlation matrices for
331 every trait + model combination are provided for GG in Tables S3-S6 and for the C1 in
332 Tables S7-S9. The correlation between genetic variance components was always negative
333 and was, in general, higher in the GG compared to the C1. Correlations between additive
334 and dominance components were highest in the A+D models (range -0.81 to -0.83 in the
335 GG and -0.5 to -0.61 in the C1). Correlations between A and D components dropped in
336 models with epistasis (range -0.42 to -0.63, GG and -0.26 to -0.58, C1). Correlations
337 between additive and AxA epistatic variances (range -0.09 to -0.29) and AxD epistasis
338 (range -0.07 to -0.22) were low. Correlations between dominance components and

339 epistasis were higher ranging from -0.28 to -0.64 with AxA epistasis and -0.36 to -0.69
340 with AxD epistasis.

341

342 *Within-trial analyses*

343 We also examined variance partitioning within each of 47 GG trials for the 5
344 models described in Table 1. The mean and variability of model parameters (variance
345 components, heritability, etc.) across these trials are summarized in Table S10. Figure 2
346 provides a visual summary of the proportion of phenotypic variability explained by each
347 genetic variance component on average across the trials. We also compared the mean
348 AIC across trials and found them to agree overall with the results of the one-step multi-
349 environment models (Table 2, Table S10). Specifically, the models that fit best in the
350 one-step models were best on average in the within trial analyses for the following traits:
351 DM (additive), HI (A+D), and MCMDS (A+D+AD). However, for RTWT (A+D instead
352 of A+D+AD) and MCBBS (D instead of A) the within trial AIC-best models were
353 different on average from the one-step multi-environment models.

354

355 *Genomic Prediction of Additive and Total Genetic Value*

356 We used cross-validation to assess the accuracy of genomic prediction of additive and
357 total genetic value for the five models (Table 1) in both populations. For additive
358 prediction, the additive only model had higher accuracy for every trait and every
359 population (Figure 3, Table S11-S12). The prediction accuracy for the additive kernel
360 was higher on average in the C1 for DM, MCBBS, MCMDS, and SPROUT compared to
361 the GG but lower for HI and RTWT. Additive accuracies by trait (across models) were

362 highest for DM (range: 0.54-0.60), followed by MCMDS (range: 0.44-0.54) and HI
363 (range: 0.32-0.45). The rest of the traits had similar additive accuracies dependent on the
364 population and model. RTWT had the overall lowest accuracy (range: 0.09-0.36).

365 Across all multi-kernel analyses conducted, prediction of total genetic value was on
366 average 42% more accurate compared to the prediction of the additive kernel in the same
367 model. Compared to the single-kernel additive prediction however, total genetic value
368 predictions were an average of only 6% better (maximum of 26% improvement; Figure 4,
369 Tables S11-S12). By model, improvements in the correlation between total value and
370 phenotype over the additive only model were 5%, 7% and 8% for A+D, A+D+AA and
371 A+D+AD respectively. The additive only model predictions were on average 11% less
372 accuracy in the C1 than in the GG. Additive kernel predictions from models with non-
373 additive components were 7% lower in C1 compared to GG. Total genetic value
374 predictions were also less accurate by 12% in the C1 relative to GG.

375 The models we fit for multi-kernel genomic prediction involved the estimation of
376 weight parameters corresponding to the partitioning of genetic variance among the
377 kernels. Overall, there was a tendency for non-additive kernels (mean of 0.40 for
378 dominance, 0.40 AxA epistasis and 0.46 AxD epistasis) to get more weight than additive
379 (mean of 0.32). Additive prediction accuracies were positively correlated ($r = 0.81$) with
380 weight placed on the additive kernel. The accuracy of total genetic value prediction was
381 negatively correlated ($r = -0.64$) with weight placed on non-additive kernels (Tables S11-
382 S12).

383

384

385 **DISCUSSION**

386 In clonally propagated crops, non-additive genetic effects can be effectively
387 exploited by the identification of superior genetic individuals as varieties. For this reason,
388 we quantified the amount and nature of non-additive genetic variation for key traits in a
389 genomic selection breeding population of cassava from sub-Saharan African. Then we
390 assessed the accuracy of genomic prediction of additive compared to total (additive plus
391 non-additive) genetic value. Using several approaches and datasets based on genome-
392 wide marker data, we confirmed previous findings in cassava based on diallel populations,
393 that non-additive genetic variation is significant, especially for yield traits (Cach *et al.*
394 2005, 2006; Calle *et al.* 2005; Jaramillo *et al.* 2005; Perez *et al.* 2005; Pérez *et al.* 2005;
395 Zacarias and Labuschagne 2010; Kulembeka *et al.* 2012; Tumuhimbise *et al.* 2014;
396 Ceballos *et al.* 2015; Chalwe *et al.* 2015). Further, we found that total genetic value
397 predicted observed phenotype more accurately than additive value, although this is
398 constrained by low broad-sense heritability and is not beneficial for traits with already
399 high heritability (DM, MCMDS). We address the implication of these results for cassava
400 breeding and put our work in the context of previous results in cassava, other plant and
401 animal species below.

402 Our results indicate strong non-additive variance for root yields and mostly
403 additive inheritance of root dry matter content. These findings confirm the conclusions of
404 numerous diallel studies conducted with both Latin American (Cach *et al.* 2005, 2006;
405 Calle *et al.* 2005; Jaramillo *et al.* 2005; Perez *et al.* 2005; Pérez *et al.* 2005) and African
406 cassava (Zacarias and Labuschagne 2010; Kulembeka *et al.* 2012; Tumuhimbise *et al.*
407 2014; Chalwe *et al.* 2015) germplasm (see also (Ceballos *et al.* 2015)). In agreement with

408 the findings of Ly *et al.* (Ly *et al.* 2013), we found cassava mosaic disease severity
409 (MCMDS) to be well predicted with an additive only model. However, we found
410 significant dominance and epistatic components in both populations analyzed. This result
411 is in line with previous diallel studies indicating significant SCA (Tumuhimbise *et al.*
412 2014; Chalwe *et al.* 2015) and genetic mapping studies that identified a single major
413 effect QTL with a dominant CMD resistance effect (Akano *et al.* 2002; Okogbenin *et al.*
414 2012; Rabbi *et al.* 2014). In addition, a recent genome-wide association and prediction
415 study using non-additive genomic relationship matrices (GRMs) found that dominance
416 and especially epistasis explain most of the variance in the region of a large-effect QTL,
417 suggesting multiple interacting loci in the region (**Wolfe *et al.* *in review***). Nearly every
418 study (but see (Zacarias and Labuschagne 2010)) indicates that Harvest Index is mostly
419 explained by additive variance, in agreement with our findings for the GG but in contrast
420 to our results for the C1. Our study is the first to report on the modes of inheritance of
421 CBB and SPROUT.

422 The importance of non-additive genetic variance in evolution by natural and
423 artificial selection is controversial (Hill *et al.* 2008; Crow 2010; Hansen 2013).
424 Nevertheless, numerous studies have found and exploited dominance and epistasis in
425 animal breeding, including dairy (Ahlborn-Breier and Hohenboken 1991; Fuerst and
426 Sölkner 1994; Varona *et al.* 1998; Van Tassell *et al.* 2000; Palucci *et al.* 2007) and beef
427 (Rodriguezalmeida *et al.* 1995) cattle. Diallel studies have indicated significant SCA for
428 maize grain yield (Doerksen *et al.* 2003; Wardyn *et al.* 2007). Aside from cassava,
429 breeding of other non-inbred, clonally propagated species also identify and make use of
430 non-additive effects, including potato (Killick 1977), Eucalyptus (Costa E Silva *et al.*

431 2004) and loblolly pine (Muñoz *et al.* 2014). More recently, marker-based and GRM-
432 based models have identified significant non-additive effects in pigs (Su *et al.* 2012;
433 Nishio and Satoh 2014), mice (Vitezica *et al.* 2013), beef cattle (Bolormaa *et al.* 2015),
434 dairy cows (Morota *et al.* 2014), maize (Dudley and Johnson 2009), soy (Hu *et al.* 2011),
435 loblolly pine (Muñoz *et al.* 2014) and apple (Satish Kumar, Claire Molloy, Patricio
436 Muñoz, Hans Daetwyler, David Chagné 2015). Results from the present study suggest
437 that the combination of genomic selection and hybrid breeding strategies should increase
438 the rate of genetic gain for complex traits such as yield. However, initial investment in
439 identification of complementary heterotic groups with good specific combining ability is
440 required.

441 One of the more interesting aspects of our study relative to previous ones is the
442 comparison between a parental generation (the Genetic Gain) and their offspring (Cycle
443 1), a collection of full- and half-sib families. From GG to C1, the H^2 generally increased.
444 For RTWT, MCBBS and HI this is largely attributable to increased non-additive variance
445 and CMD for which non-additive variance dropped in C1 relative to GG. In contrast to
446 our result, theory suggests that reduction (or fixation) of allele frequencies at some loci
447 relative to others in populations undergoing bottlenecks (Goodnight 1988), inbreeding
448 (Turelli and Barton 2006) or truncation selection (Hallander and Waldmann 2007) should
449 cause a conversion of non-additive (where present) to additive variance. These results
450 have, however, been based on models with finite numbers of loci in linkage equilibrium.
451 Based on the mean diagonal of the kinship matrix, C1 (0.66) does not appear notably
452 more inbred than GG (0.64). We also calculated mean pairwise LD (GG = 0.27, C1 =

453 0.29) and mean LD block size (21.7 kb in GG and 23.1 kb in C1) using PLINK (version
454 1.9, <https://www.cog-genomics.org/plink2>) and found the two generations to be similar.

455 Probably the strongest explanation for the difference in genetic variance
456 components between GG and C1 is the family structure (137 full-sib families from 83
457 outbred parents). In a population of half-sibs $\frac{3}{4}$ of the dominance variance is expressed
458 within families and all of it for full-sib populations (Hallauer *et al.* 2010; Ceballos *et al.*
459 2015). Indeed, increasing the number of full-sib relationships is known to increase the
460 non-additive genetic variance detectable in a population (Varona *et al.* 1998; Van Tassel
461 *et al.* 2010).

462 It is also conceivable that maternal plant effects could increase apparent non-
463 additive effects in C1. The C1 clones in contrast to the GG clones are new, and were
464 derived from stem cuttings of seedling plants germinated in the previous field season
465 (2012-2013). The suggestion is therefore that the quality and vigor of the seedling plant,
466 giving rise to the C1 clones may influence their performance in the 2013-2014 trial. We
467 further caution that comparison of GG and C1 may be biased by the disproportionate
468 amount of data available for the GG.

469 In our study, when additive and non-additive kernels were used together, the
470 ability of the additive kernel to predict the phenotype decreased, suggesting that the
471 additive kernel by itself was absorbing non-additive variance. Estimates of additive
472 genetic variance have previously been shown to capture some non-additive effects (Lu *et*
473 *al.* 1999; Su *et al.* 2012; Zuk *et al.* 2012; Muñoz *et al.* 2014). Predictions models that do
474 not explicitly incorporate non-additive components may therefore achieve gains in the
475 short-term that break down over the long-term (Cockerham and Tachida 1988; Walsh

476 2005; Hansen 2013). Including non-additive GRMs when estimating additive genetic (i.e.
477 breeding) values may therefore provide a less biased, more accurate selection of parents
478 for genomic selection (Costa E Silva *et al.* 2004; Palucci *et al.* 2007). We note that our
479 predictions of total genetic value were focused on parametric models based in
480 quantitative genetic theory. However, many non-parametric and non-linear approaches
481 are available (e.g. RKHS and random forests) that may capture even more non-additive
482 variation than found in our study.

483 Non-additive variation is prevalent in cassava, especially for low heritability traits.
484 This has many important implications for cassava breeding. It explains, in part, why
485 genetic gains have been slow (Ceballos *et al.* 2012). Inbreeding to convert dominance
486 variance to additive and better control epistatic combinations, as in maize, has been
487 suggested as a solution to non-additive genetics (Ceballos *et al.* 2015). Even for low h^2
488 traits and without inbred cassava, using the kinds of models presented in this paper, good
489 parents can be selected based on additive predictions and total genetic value can be
490 simultaneously estimated for the identification of potential commercial varieties, all
491 based on the combination of marker and preliminary field trial data (Heslot and Mark
492 2015). This approach has been previously advocated for plant breeding (Oakey *et al.*
493 2007; Heslot and Mark 2015) and has proven effective in animal breeding, e.g. (Ahlborn-
494 Breier and Hohenboken 1991; Palucci *et al.* 2007; Su *et al.* 2012; Nishio and Satoh 2014).
495 Non-additive models using genomic relationship matrices can thus improve the efficiency
496 and productivity of variety selection pipelines that are the most labor and time intensive
497 part of selecting good cassava clones after crossing.

498
499

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507

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- 688

689 **Figure & Table Legends**

690

691 **Table 1.** Additive plus non-additive effects models tested and their abbreviations.

692

693 **Table 2. Single-step multi-environment model results for the Genetic Gain**
694 **population.** Variance components (\pm standard errors), narrow-sense heritabilities (h^2),
695 proportion of the total phenotypic variance explained by dominance (d^2), additive-by-
696 additive epistasis ($i^2_{A\#A}$), additive-by-dominance epistasis ($i^2_{A\#D}$) and broad-sense
697 heritability (H^2) are provided. Model log-likelihoods and Akaike Information Criterion
698 (AIC) are also given. The best model for each trait (lowest AIC) is highlighted in grey.
699

700 **Table 3. Single-step multi-environment model results for the Cycle 1 population.**
701 Variance components (\pm standard errors), narrow-sense heritabilities (h^2), proportion of
702 the total phenotypic variance explained by dominance (d^2), additive-by-additive epistasis
703 ($i^2_{A\#A}$), additive-by-dominance epistasis ($i^2_{A\#D}$) and broad-sense heritability (H^2) are
704 provided. Model log-likelihoods and Akaike Information Criterion (AIC) are also given.
705 The best model for each trait (lowest AIC) is highlighted in grey.
706

707 **Figure 1. Partitioning of broad-sense heritability among variance components for**
708 **single-step multi-environment models in the Genetic Gain and Cycle 1 populations**
709 **for each trait.** Results from each of five models are shown in each panel broken down by
710 trait (rows) and population (columns). Models include additive only (Additive),
711 dominance only (Dominance), Additive plus Dominance (Add_Dom), Additive plus
712 dominance plus either AxA epistasis (Add_Add_Epistasis) or AxD epistasis
713 (Add_Dom_Epistasis).
714

715 **Figure 2. Partitioning of broad-sense heritability among variance components for**
716 **the within-trial models in the Genetic Gain population.** Three models were fitted for
717 each trait in each of 47 Genetic Gain trials. Each panel contains boxplots showing the
718 distribution proportions of the phenotypic variability explained by a corresponding
719 variance components, including the broad-sense heritability (H^2) across each trial. Red
720 horizontal lines are the median narrow-sense heritability (h^2) from the additive only
721 model. Traits are on columns and three models are on the rows: additive plus dominance
722 (Add_Dom), additive plus dominance plus AxA epistasis (AxA_epi) and additive plus
723 dominance plus AxD epistasis (AxD_epi).
724

725 **Figure 3. Accuracy of additive genetic value prediction in the Genetic Gain and**
726 **Cycle 1 populations.** Boxplots showing the distribution over 25 replicates of 5-fold
727 cross-validation of the prediction accuracy of the additive component from four different
728 models are shown in each panel. The accuracy within the Genetic Gain (red) and Cycle 1
729 (blue) are shown. Traits are in the columns. Accuracy is defined as the correlation
730 between the prediction from the additive kernel in the model and the BLUP from the first
731 stage of analysis where location, year and replicate variability were removed. Models
732 included are: additive only (Add), additive plus dominance (Add_Dom), additive plus
733 dominance plus AxA epistasis (AxA_epi) and additive plus dominance plus AxD
734 epistasis (AxD_epi).

735

736 **Figure 4. Accuracy of total genetic value prediction in the Genetic Gain and Cycle 1**
737 **populations.** Boxplots showing the distribution over 25 replicates of 5-fold cross-
738 validation of the prediction accuracy of the total genetic value from five different models
739 are shown in each panel. The accuracy within the Genetic Gain (red) and Cycle 1 (blue)
740 are shown. Traits are in the columns. Accuracy is defined as the correlation between the
741 sum of predictions from all genetic variance components in the model and the BLUP
742 from the first stage of analysis where location, year and replicate variability were
743 removed. Models included are: additive only (Add), dominance only (Dom), additive
744 plus dominance (Add_Dom), additive plus dominance plus AxA epistasis (AxA_epi) and
745 additive plus dominance plus AxD epistasis (AxD_epi).

746

747 **Supplementary Table 1.** Pedigree and related information for the IITA: Genetic Gain
748 population, Nigeria.

749

750 **Supplementary Table 2.** Sample size (Nobs), replication number (Nreps), clone number
751 (Nclones) and whether a trial (LOC.YEAR) was included in one-step multi-environment
752 models for both populations analyzed.

753

754 **Supplementary Table 3.** Pedigree information for the IITA: GS Cycle 1 population,
755 Nigeria.

756

757 **Supplementary Table 4.** Asymptotic correlation matrices of parameter estimates
758 representing the dependency of variance component estimation for the additive plus
759 dominance model in the Genetic Gain population, by trait.

760

761 **Supplementary Table 5.** Asymptotic correlation matrices of parameter estimates
762 representing the dependency of variance component estimation for the additive plus
763 dominance plus additive-by-additive epistasis model in the Genetic Gain population, by
764 trait.

765

766 **Supplementary Table 6.** Asymptotic correlation matrices of parameter estimates
767 representing the dependency of variance component estimation for the additive plus
768 dominance plus additive-by-dominance epistasis model in the Genetic Gain population,
769 by trait.

770

771 **Supplementary Table 7.** Asymptotic correlation matrices of parameter estimates
772 representing the dependency of variance component estimation for the additive plus
773 dominance model in the Cycle 1 population, by trait.

774

775 **Supplementary Table 8.** Asymptotic correlation matrices of parameter estimates
776 representing the dependency of variance component estimation for the additive plus
777 dominance plus additive-by-additive epistasis model in the Cycle 1 population, by trait.

778

779 **Supplementary Table 9.** Asymptotic correlation matrices of parameter estimates
780 representing the dependency of variance component estimation for the additive plus
781 dominance plus additive-by-dominance epistasis model in the Cycle 1 population, by trait.
782

783 **Supplementary Table 10.** Within-trial model results for the Genetic Gain population.
784 The mean (\pm standard errors) across 47 trials for each trait and model fitted is given for
785 the following model parameters: variance components, narrow-sense heritabilities (h^2),
786 proportion of the total phenotypic variance explained by dominance (d^2), additive-by-
787 additive epistasis ($i^2_{A\#A}$), additive-by-dominance epistasis ($i^2_{A\#D}$) and broad-sense
788 heritability (H^2), trial sample size (N), model log-likelihoods and Akaike Information
789 Criterion (AIC) are also given. The best model for each trait (lowest AIC) is highlighted
790 in grey.
791

792 **Supplementary Table 11.** Results from 25 replicates of 5-fold cross-validation in the
793 Genetic Gain population are given. Mean (\pm standard errors) across the 25 replicates are
794 given for prediction accuracy of each kernel plus total genetic value (sum across all
795 kernels), variance components (V_g and V_e) and kernel weights.
796

797 **Supplementary Table 12.** Results from 25 replicates of 5-fold cross-validation in the
798 Cycle 1 population are given. Mean (\pm standard errors) across the 25 replicates are given
799 for prediction accuracy of each kernel plus total genetic value (sum across all kernels),
800 variance components (V_g and V_e) and kernel weights.
801
802

803 Table 1.

| Model | Relationship Matrices / Variance Components |
|------------|---|
| A | Additive |
| D | Dominance |
| A + D | Additive + Dominance |
| | Additive + Dominance + A#A |
| A + D + AA | Epistasis |
| | Additive + Dominance + A#D |
| A + D + AD | Epistasis |

| Trait | Model | $\sigma^2_{loc,year}$ | σ^2_{rep} | σ^2_{add} | σ^2_{dom} | $\sigma^2_{A\#A}$ | $\sigma^2_{A\#D}$ | σ^2_{error} | h^2 | d^2 | i^2_A #A | i^2_A #D | H^2 | loglik | AIC | |
|-------------|---------------------|-----------------------|------------------|------------------|------------------|-------------------|-------------------|--------------------|------------|-------|---------------|---------------|-------|--------|------|--|
| DM | Additive | 0.0 ± 4.25 | 6.1 ± 5.6 | 10.4 ± 1.0 | | | | 15.0 ± 0.3 | 0.33 | | | | 0.33 | -9457 | 1892 | |
| | Dominance | 0.0 ± 4.17 | 6.1 ± 5.7 | 7.2 ± 0.6 | | | | 15.0 ± 0.3 | | 0.25 | | | 0.25 | -9470 | 1894 | |
| | Add + Dom | 0.0 ± 4.10 | 6.1 ± 5.7 | 8.5 ± 1.6 | | | | 15.0 ± 0.3 | | 0.27 | 0.04 | | 0.31 | -9456 | 1892 | |
| | Add + Dom + AxA Epi | 0.0 ± 4.07 | 6.1 ± 5.7 | 8.2 ± 1.6 | 0.2 ± 1.1 | 1.7 ± 1.5 | | 15.0 ± 0.3 | | 0.26 | 0.01 | 0.05 | 0.32 | -9455 | 1892 | |
| | Add + Dom + AxD Epi | 0.0 ± 4.06 | 6.1 ± 5.8 | 8.1 ± 1.6 | 0.0 ± 1.1 | | | 15.0 ± 0.3 | 1.5 ± 1.2 | 0.26 | 0.00 | 0.05 | 0.31 | -9455 | 1892 | |
| | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | |
| HI | Additive | 48.9 ± 30.5 | 11.8 ± 9.8 | 87.1 ± 7.4 | | | | 10.8 ± 2.0 | 3 | | | | 3 | 10656 | 2130 | |
| | Dominance | 48.7 ± 30.4 | 11.8 ± 9.8 | 58.8 ± 4.7 | | | | 10.8 ± 2.0 | | 3 | | | 3 | 10646 | 2128 | |
| | Add + Dom | 48.9 ± 30.4 | 11.8 ± 9.8 | 57.3 ± 12.0 | 17.6 ± 6.9 | | | 10.8 ± 2.0 | | 2 | 1 | | 3 | 10659 | 2130 | |
| | Add + Dom + AxA Epi | 48.9 ± 30.4 | 11.8 ± 9.8 | 55.6 ± 12.1 | 13.1 ± 8.9 | 8.3 ± 4 | | 10.8 ± 2.0 | | 2 | 1 | 0.0 | 3 | 10659 | 2130 | |
| | Add + Dom + AxD Epi | 48.9 ± 30.4 | 11.8 ± 9.8 | 54.1 ± 11.8 | 7.7 ± 9.3 | | | 10.8 ± 2.0 | 13.2 ± 9.4 | 2 | 0 | 0.1 | 3 | 10660 | 2130 | |
| | | | | | | | | | | | | | | | | |
| logRT WT | Additive | 0.0 ± 0.55 | 0.0 ± 0.14 | 0.0 ± 0.95 | | | | 0.1 ± 0.07 | 28 | | | | 28 | 2362 | 4716 | |
| | Dominance | 0.0 ± 0.56 | 0.0 ± 0.14 | 0.0 ± 0.64 | | | | 0.1 ± 0.07 | | 21 | | | 21 | 2369 | 4731 | |
| | Add + Dom | 0.0 ± 0.56 | 0.0 ± 0.14 | 0.0 ± 0.36 | 0.0 ± 0.13 | | | 0.1 ± 0.07 | | 11 | 12 | | 23 | 2375 | 4740 | |
| | Add + Dom + AxA Epi | 0.0 ± 0.56 | 0.0 ± 0.14 | 0.0 ± 0.29 | 0.0 ± 0.11 | 0.0 ± 0.0 | 0.0 ± 0.0 | 0.1 ± 0.07 | | 9 | 6 | 0.1 | 25 | 2378 | 4744 | |
| | Add + Dom + AxD Epi | 0.0 ± 0.56 | 0.0 ± 0.14 | 0.0 ± 0.31 | 0.0 ± 0.12 | 0.0 ± 0.0 | 0.0 ± 0.0 | 0.1 ± 0.07 | 0.0 ± 0.0 | 10 | 6 | 0.0 | 23 | 2377 | 4743 | |
| | | | | | | | | | | | | | | | | |
| MCB BS | Additive | 0.4 ± 0.73 | 0.0 ± 0.20 | 0.0 ± 0.29 | | | | 0.1 ± 0.09 | 04 | | | | 04 | 2383 | 4759 | |
| | Dominance | 0.4 ± 0.74 | 0.0 ± 0.20 | 0.0 ± 0.22 | | | | 0.1 ± 0.09 | | 03 | | | 03 | 2374 | 4741 | |
| | Add + Dom | 0.4 ± 0.74 | 0.0 ± 0.20 | 0.0 ± 0.23 | 0.0 ± 0.06 | 0.0 ± 0.0 | 0.0 ± 0.0 | 0.1 ± 0.09 | | 03 | 01 | | 04 | 2384 | 4759 | |
| | Add + Dom + AxA Epi | 0.4 ± 0.74 | 0.0 ± 0.20 | 0.0 ± 0.22 | 0.0 ± 0.07 | 0.0 ± 0.0 | 0.0 ± 0.0 | 0.1 ± 0.09 | | 03 | 01 | 0.0 | 04 | 2384 | 4757 | |
| | | | | | | | | | | | | | | | | |

| | | | | | | | | | | | | | | | | | | | |
|------|-----------|-----|------|-----|------|-----|-------|-----|-------|-----|------|-----|-------|----|----|-----|----|-------|------|
| | Add + Dom | 0.4 | 0. | 0.0 | 0. | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.1 | 0.0 | 0. | 0. | 0.0 | 0. | 2384. | 4757 |
| | + AxD Epi | 74 | ± 23 | 20 | ± 01 | 23 | ± 07 | 05 | ± 06 | 00 | ± 06 | 9 | ± 03 | 03 | 01 | 0 | 04 | 78 | .56 |
| MCM | | 0.0 | 0. | 0.0 | 0. | 1.4 | 0.0 | | | | | 0.3 | 0.0 | 0. | | | 0. | | 1255 |
| DS | Additive | 51 | ± 02 | 00 | ± 00 | 4 | ± 9 | | | | | 4 | ± 06 | 79 | | | 79 | -624 | .7 |
| | Dominance | 51 | ± 02 | 00 | ± 00 | | | 0.7 | 0.0 | | | 0.3 | 0.0 | | 0. | | 0. | | 1207 |
| | | | | | | | | 76 | ± 5 | | | 4 | ± 06 | | 66 | | 66 | -600 | .6 |
| | Add + Dom | 51 | ± 02 | 00 | ± 00 | 0 | ± 2 | 0.3 | 0.1 | 0.5 | 0.0 | 0.3 | 0.0 | 0. | 0. | | 0. | | 1202 |
| | Add + Dom | 51 | ± 02 | 00 | ± 00 | 0 | ± 2 | 0 | ± 2 | 67 | ± 8 | 4 | ± 06 | 24 | 45 | | 69 | -596 | .1 |
| | + AxA Epi | 51 | ± 02 | 00 | ± 00 | 8 | ± 1 | 0.2 | 0.1 | 0.1 | 0.0 | 0.3 | 0.0 | 0. | 0. | 0.4 | 0. | | 1180 |
| | Add + Dom | 51 | ± 02 | 00 | ± 00 | 8 | ± 1 | 55 | ± 9 | 0.5 | 0.1 | 4 | ± 06 | 20 | 11 | 2 | 72 | -584 | .7 |
| | + AxD Epi | 51 | ± 02 | 00 | ± 00 | 2 | ± 0 | 0.3 | 0.1 | 0.0 | 0.0 | 0.3 | 0.0 | 0. | 0. | 0.4 | 0. | | 1173 |
| | | 51 | ± 02 | 00 | ± 00 | 2 | ± 0 | 00 | ± 8 | 0.5 | 0.0 | 4 | ± 06 | 25 | 00 | 4 | 69 | -581 | .0 |
| SPRO | | 28. | 13 | | 0. | 70. | | | | | | 22 | | 0. | | | 0. | 10384 | 2076 |
| UT | Additive | 2 | ± .6 | 0.3 | ± 5 | 4 | ± 7.4 | | | | | 8.2 | ± 3.8 | 2 | | | 2 | .9 | 1.9 |
| | Dominance | 2 | ± .6 | 0.3 | ± 5 | | | 48. | | | | 22 | | 0. | | | 0. | 10397 | 2078 |
| | | | | | | | | 1 | ± 4.8 | | | 6.5 | ± 3.8 | 2 | | | 2 | .4 | 6.8 |
| | Add + Dom | 2 | ± .6 | 0.3 | ± 5 | 19. | 10. | 34. | | | | 22 | | 0. | 0. | | 0. | 10399 | 2078 |
| | | | | | | 3 | ± 8 | 2 | ± 8.2 | | | 6.5 | ± 3.8 | 1 | 1 | | 2 | .5 | 9.1 |
| | Add + Dom | 2 | ± .6 | 0.3 | ± 5 | 10. | | | | | | 22 | | 0. | 0. | | 0. | 10410 | 2080 |
| | + AxA Epi | 1 | ± .5 | 0.3 | ± 5 | 9 | ± 9.1 | 5.8 | ± 8.6 | 55. | 12. | 5.4 | ± 3.8 | 0 | 0 | 0.2 | 2 | .4 | 8.7 |
| | | | | | | | | 9 | ± 6 | | | 0 | 0 | | | | | | |
| | Add + Dom | 28. | 13 | | 0. | 12. | | | | | | 22 | | 0. | 0. | | 0. | 10406 | 2080 |
| | + AxD Epi | 1 | ± .5 | 0.3 | ± 5 | 2 | ± 9.4 | 8.4 | ± 9.8 | 36. | 10. | 5.6 | ± 3.8 | 0 | 0 | 0.1 | 2 | .0 | 0.0 |
| | | | | | | | | | | 5 | ± 4 | | | | | | | | |

805

806

807 Table 3

| Trait | Model | $\sigma^2_{loc\ year}$ | | σ^2_{add} | | σ^2_{dom} | | σ^2_{AFA} | | σ^2_{AFD} | | σ^2_{error} | | h^2 | d^2 | \hat{I}^2_{Aa} | \hat{I}^2_{Ad} | H^2 | loglik | AIC |
|---------------------|-----------|------------------------|---------|------------------|---------|------------------|---------|------------------|---------|------------------|----------|--------------------|-----|-------|-------|------------------|------------------|--------|--------|--------|
| DM | Additive | 8.3 | | 20. | | | | | | | | 11. | | 0.5 | | | | 0.5 | 3544.1 | 7094.3 |
| | | 2 ± 8.3 | | 9 ± 2.3 | | | | | | | | 5 ± 0.6 | | 1 | | | | 1 | 58 | 16 |
| | Dominance | 8.6 | | | | 16. | | | | | | 10. | | | 0.4 | | | 0.4 | 3585.3 | 7176.6 |
| | | 8 ± 8.7 | | | | 0 ± 1.6 | | | | | | 6 ± 0.7 | | 5 | | | | 5 | 17 | 34 |
| | Add + Dom | 8.3 | | 17. | | 3.4 | | 1.5 | | | | 10. | | 0.4 | 0.0 | | | 0.5 | 3537.9 | 7083.9 |
| | 8 ± 8.4 | | 3 ± 2.5 | | ± 1.5 | | | | | | 7 ± 0.7 | | 3 | 8 | | | 2 | 53 | 07 | |
| Add + Dom + AxA Epi | 8.4 | | 16. | | 2.8 | | 1.6 | 2.0 | 2.5 | | 10. | | 0.4 | 0.0 | 0.0 | | 0.5 | 3537.5 | 7084.9 | |
| | 2 ± 8.4 | | 6 ± 2.5 | | ± 1.6 | | | 50 ± 91 | | | 4 ± 0.8 | | 1 | 7 | 5 | | 3 | 00 | 99 | |
| Add + Dom + AxD Epi | 8.3 | | 17. | | 3.4 | | 1.7 | | | 0.0 | 2.2 | 10. | | 0.4 | 0.0 | | 0.0 | 0.5 | 3537.9 | 7085.9 |
| | 8 ± 8.4 | | 3 ± 2.6 | | ± 1.7 | | | | | 01 ± 40 | | 7 ± 0.9 | | 3 | 8 | | 0 | 2 | 54 | 08 |
| HI | Additive | 19. | 19. | 69. | 10. | | | | | | | 124 | | 0.3 | | | | 0.3 | 3653.6 | 7301.2 |
| | | 5 ± 7 | | 2 ± 6 | | | | | | | | .7 ± 4.7 | | 2 | | | | 2 | 34 | 67 |
| | Dominance | 19. | 19. | | | 86. | 10. | | | | | 103 | | | 0.4 | | | 0.4 | 3666.1 | 7326.2 |
| | | 1 ± 3 | | | | 6 ± 2 | | | | | | .1 ± 5.2 | | 1 | | | | 1 | 24 | 48 |
| | Add + Dom | 19. | 19. | 32. | 12. | 61. | 12. | | | | | 103 | | 0.1 | 0.2 | | | 0.4 | 3672.2 | 7336.5 |
| | 2 ± 4 | | 3 ± 3 | | 8 ± 0 | | | | | | .8 ± 5.2 | | 5 | 8 | | | 3 | 56 | 12 | |
| Add + Dom + AxA Epi | 19. | 19. | 29. | 12. | 57. | 12. | 26. | 18. | | | 97. | | 0.1 | 0.2 | 0.1 | | 0.4 | 3673.1 | 7336.3 | |
| | 2 ± 3 | | 2 ± 4 | | 8 ± 6 | | 4 ± 7 | | | | 4 ± 6.3 | | 3 | 5 | 1 | | 9 | 88 | 75 | |
| Add + Dom + AxD Epi | 19. | 19. | 28. | 12. | 56. | 13. | | | 24. | 17. | 96. | | 0.1 | 0.2 | | 0.1 | 0.4 | 3673.0 | 7336.1 | |
| | 2 ± 4 | | 8 ± 4 | | 4 ± 1 | | | | 4 ± 6 | | 0 ± 6.9 | | 3 | 5 | | 1 | 9 | 92 | 83 | |
| logRT WT | Additive | 0.0 | 0.0 | 0.1 | 0.0 | | | | | | | 0.2 | 0.0 | 0.2 | | | | 0.2 | 160.58 | 315.16 |
| | | 07 ± 08 | | 01 ± 18 | | | | | | | | 8 ± 10 | | 6 | | | | 6 | 0 | 0 |
| | Dominance | 0.0 | 0.0 | | | 0.1 | 0.0 | | | | | 0.2 | 0.0 | | 0.3 | | | 0.3 | 183.50 | 361.01 |
| | | 06 ± 07 | | | | 16 ± 18 | | | | | | 5 ± 11 | | 1 | | | | 1 | 9 | 8 |
| | Add + Dom | 0.0 | 0.0 | 0.0 | 0.0 | 0.1 | 0.0 | | | | | 0.2 | 0.0 | 0.0 | 0.2 | | | 0.3 | 184.19 | 360.38 |
| | 07 ± 07 | | 20 ± 19 | | 03 ± 22 | | | | | | 5 ± 11 | | 5 | 7 | | | 3 | 3 | 5 | |
| Add + Dom + AxA Epi | 0.0 | 0.0 | 0.0 | 0.0 | 0.1 | 0.0 | 0.0 | 0.0 | | | 0.2 | 0.0 | 0.0 | 0.2 | 0.0 | | 0.3 | 184.19 | 358.38 | |
| | 07 ± 07 | | 20 ± 20 | | 03 ± 24 | | 00 ± 34 | | | | 5 ± 14 | | 5 | 7 | 0 | | 3 | 3 | 5 | |
| Add + Dom + AxD Epi | 0.0 | 0.0 | 0.0 | 0.0 | 0.1 | 0.0 | | | 0.0 | 0.0 | 0.2 | 0.0 | 0.0 | 0.2 | | 0.0 | 0.3 | 184.19 | 358.38 | |
| | 07 ± 07 | | 20 ± 20 | | 03 ± 25 | | | | 00 ± 35 | | 5 ± 15 | | 5 | 7 | | 0 | 3 | 3 | 5 | |
| MCBS | Additive | 0.0 | 0.0 | 0.0 | 0.0 | | | | | | | 0.1 | 0.0 | 0.1 | | | | 0.1 | 751.23 | 1496.4 |
| | | 47 ± 5 | | 26 ± 07 | | | | | | | | 9 ± 06 | | 0 | | | | 0 | 8 | 77 |
| | Dominance | 0.0 | 0.0 | | | 0.0 | 0.0 | | | | | 0.1 | 0.0 | | 0.1 | | | 0.1 | 754.60 | 1503.2 |
| | 48 ± 5 | | | | 33 ± 08 | | | | | | 8 ± 07 | | | 3 | | | 3 | 3 | 06 | |
| Add + Dom | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | | | | | 0.1 | 0.0 | 0.0 | 0.1 | | | 0.1 | 754.98 | - | |
| | 48 ± 5 | | 08 ± 09 | | 25 ± 10 | | | | | | 8 ± 07 | | 3 | 0 | | | 3 | 2 | 1501.9 | |







