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Genetic parameters for early growth and disease resistance in a cloned F₂ hybrid progeny of *Eucalyptus urophylla* × *grandis*

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The VERACEL breeding program includes several advanced generation *urograndis* hybrids (F₂) from crosses of selected F₁. To understand the performance and genetics of these F₂, we established F₂-cloned progeny trials with 1,350 clones from 35 families, each family with 8 to 35 cloned progenies. These families stem from crosses between known F₁ *urograndis* female parents and pollen mixtures of other selected parents. The experimental design comprised 15 trials with 95 clones each, arranged in randomized blocks and linked by 7 common commercial hybrid clones. These experiments were performed in two sites contrasting for physiological disorder (PD) incidence. Due to incomplete pedigree of the families, the model fitted assumed the additive genetic relationship between sibs as half-sibs. The non-additive genetic component was estimated from clone effects within half-sib families. A multisite individual tree genetic model was fitted for diameter, height, PD, *Calonectria*, and rust incidence for trees up to 1 year old: $y_{ijklmn} = \mu + g_i + g_{ij} + s_m + c_n + t_{bkl} + s_{gim} + s_{gijm} + s_{cmn} + e_{ijklmn}$; where random effects are g_{ij} (additive genetic effects), g_i (all non-additive genetic effects of clones), and e_{ijklmn} (residuals); and fixed effects are s_m , which is either site with higher or lower productivity and PD incidence, c_n is F₁ or F₂, and t_{bkl} is trial-block interaction. The same model was used for diseases and PD, but assumed a binomial distribution with a logit link function. PD was analysed only for the higher PD incidence site, thus excluding interactions with the other site. Results indicate that at the high PD incidence site, height and diameter growth was greater (~40%), but it also had higher mortality (40% vs. 22%) and higher PD incidence (51% vs. 23%). Compared with F₁ controls, *Calonectria* incidence was higher in F₂ (~10%) but similar at both sites, as was rust, although with lower incidence (~5%). Multisite analysis revealed low additive genetic variance and moderate total genetic variance (A+NA) for growth, diseases, and PD. For growth, narrow- and broad-sense heritability were $h^2=0.11$ and $H^2=0.40$. The additive genetic correlation between the two sites was close to 1 ($r_A \sim 0.9$), whereas the non-additive genetic correlation was lower ($r_{NA} \sim 0.5$). The narrow- and broad-sense heritability for diseases and PD was low (between 0.10 and 0.15). The low additive genetic variance for growth, diseases, and PD constrains substantial gains from parental selection within F₁. However, clonal selection would still be effective due to considerable non-additive effects. Further studies using a large set of various populations are needed to validate these findings.

