Is the *cad-n1* Allele Associated with Increased Wood Density or Growth in Full-Sib Families of Loblolly Pine?

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A rare mutant allele (*cad-n1*) of the *cad* gene in loblolly pine (*Pinus taeda* L.) causes a deficiency in the production of cinnamyl alcohol dehydrogenase (CAD). Effects associated with this null allele were examined by comparing wood density and growth of 15-year-old *cad-n1* heterozygotes with their wild-type full-siblings, established in three test series (1, 2 and 3) in two states (South Carolina and Georgia). In each series, *cad-n1* heterozygous selections (A, B and/or C) were crossed with five unrelated wild-type parents, to produce five full-sib families. Series 1 included five crosses each for selections A and B, and series 2 had five crosses with selection C. Five additional crosses for selection A were established in series 3. Each series was established in replicated trials at two different field-test sites, and all tests included a common, unimproved commercial checklot. Test progenies from each cross and at each site were genotyped at the *cad* locus, and assessed for growth and wood density traits. In all, 839 trees were sampled.

We found evidence of large effects on growth and wood density, associated with the *cad-n1* allele. When all three test series were considered in a combined analysis, *cad-n1* heterozygotes were found to have 5% (p = 0.11) greater volume than their wild-type full-sibs. However, the phenotypic effects on wood density or growth are not likely due to the *cad-n1* allele alone, and other loci are probably involved. In series 3, selection A *cad-n1* heterozygotes averaged 17% (p = 0.07) greater volume than their wild type full-sibs, whereas in series 1, they were only 3% greater in volume. This may be due to either different genetic backgrounds between series 1 and 3, or different growing environments. The 17% volume and 3.4% (p = 0.04) wood density increases in series 3 for *cad-n1* heterozygotes were mainly due to selection A being crossed with two particular second parents. It appears that there is an epistatic effect, in that these two second parents contributed certain genetic components that specifically interacted with *cad-n1* to produce the large positive effect. While substantial gains are possible through deployment of relatives carrying the *cad-n1* allele, these gains are family-specific and must be verified for each cross through field testing.

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