Title Effects of the ethylene binding inhibitor, 1-methylcyclopropene, on flue-cured tobacco (*Nicotiana tabacum* L.)

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Abstract

Three experiments were conducted from 2005 to 2008 to determine the effects of 1-methylcyclopropene (1-MCP) on harvest management of flue-cured tobacco. Treatments consisted of 1-MCP at a rate of 0.026 kg ai ha^{-1} and 0.0129 kg ai ha^{-1} applied at: 14 d prior to normal final harvest, 14 and 7 d prior to normal final harvest, 7 d prior to normal final harvest, and 7 and 1 d prior to normal final harvest in all three experiments.

The first experiment was conducted to determine if applications of 1-MCP could increase holding ability and ripening delay in flue-cured tobacco. Holding ability and ripening delay of flue-cured tobacco was not increased by applications of 1-MCP. Value per hectare, grade index, and yield were not affected by applications of 1-MCP, but were reduced when harvest was delayed from the normal.

The second experiment was conducted to determine if applications of 1-MCP could inhibit chemical senescence from applications of 2-chloroethylphosphonic acid in flue-cured tobacco at a rate of 1.68 kg ai ha^{-1} . Chlorophyll meter values were affected when 1-MCP was applied at 14 d alone and 7 d alone in both 2006 and 2008 at either location. Thus in these years and locations, two of the four 1-MCP treatments were effective at inhibiting chemically enhanced senescence from applications of 2-chloroethylphosphonic acid.

The final experiment was conducted to determine the effective concentrations of ethylene and 1-MCP evolving from leaf surfaces after applications of 1-MCP in flue-cured tobacco. Differences in ethylene concentrations suggest that 1-MCP was bound to the receptor site and by 8-hours, 75-97% of the ethylene concentration had evolved from the leaf tissue. Once 2-chloroethylphosphonic acid had been applied for 48 hours, chlorophyll content in all chemical treatments was significantly reduced from that of the non-treated control. These data suggest that 1-MCP potentially occupied ethylene-binding sites and that new binding site were generated to accept ethylene in the form of 2-chloroethylphosphonic acid, initiating enhanced chemical senescence.