Abstract

Blue mold decay is an important disease of stored apples in North America. The disease is caused by species of *Penicillium* that are difficult to identify with conventional mycological methods. Furthermore, *Penicillium expansum*, has developed resistance to benzimidazole fungicides and information is needed on the nature of this resistance. Twenty-three of 150 isolates collected from packinghouses were selected for identification and fungicide resistance studies using conventional and molecular techniques. The isolates were grown for 1 week on Czapek yeast autolysate, malt extract and 25% glycerol nitrate agars at 5, 25 and 35 °C and on potato dextrose agar at 20 °C amended with benomyl, thiabendazole and diphenylamine. The β-tubulin gene sequence (600–700 bp) of the isolates was also examined taking care to include codons 198 and 200 with forward primers Bt-LEV-Up4 and reverse primer Bt-LEV-Lo1. Based on the information provided by the plate studies regarding appearance and growth rate of the isolates it was possible to separate the faster growing *P. expansum* isolates from the slower growing *P. solitum* isolates. The DNA sequence data was very helpful when used in conjunction with plate tests and removed any ambiguity when trying to separate these closely related species. The DNA sequence at codon 198 corresponded to benomyl resistance if the GAG sequence had a substitution in it. However, benomyl resistance also occurred with the normal GAG sequence at codon 198 in five of the isolates. *P. expansum* isolates with a substitution of GAG to GCG or GTG at codon 198 were highly resistant to benomyl and thiabendazole but only the three isolates with the GCG substitution were highly sensitive to diphenylamine. The remaining benomyl resistant isolates were resistant to diphenylamine except one *P. solitum* isolate that was sensitive.