APPENDIX B

Antimycin A
Antimycin A is an organic compound that was isolated from the bacterium *Streptomyces girseus* at University of Wisconsin in 1945 (Leben and Keitt 1948, Dunshee, et al. 1949). The chemical formula of antimycin is C_{28}H_{40}N_{2}O_{9} (Rinne and Turner 1991:237), and it inhibits growth of some fungi but does not affect most bacteria. Antimycin was later found to be toxic to fish and was patented as a piscicide in 1964. The formulation proposed for use in the Fossil Creek project is Fintrol-Concentrate (liquid form Antimycin A) and Fintrol 15 (Antimycin A coated sand). Fintrol and Fintrol 15 are registered with the Environmental Protection Agency under registration numbers 39096-2 and 8991-6, respectively. Antimycin A is recognized by the Arizona Department of Environmental Quality as acceptable under the conditions of the Arizona Water Quality Standards for Surface Waters. Antimycin A consists of 10 percent antimycin, a surfactant, and acetone.

Degradation of antimycin is by the following pathway (Hussain 1969):

antimycin A1 → blastmycic acid + antimycin lactone → fatty acids

These degradation compounds have very low toxicity for either fish or mammals (Herr et al. 1967). Detoxification of antimycin is accelerated by pH greater than 7.0 and exposure to sunlight (Lee et al. 1971, Marking and Dawson 1972). When exposed to sunlight, antimycin degrades completely in 1.0 to 1.5 hours (Lee et al. 1971). Degradation of antimycin may also be accelerated by warm water temperature, organic material, and water turbulence (Lee et al. 1971). The above-neutral pH and exposure to sunlight of Fossil Creek in the project area would result in relatively rapid and total degradation of antimycin. For this reason, antimycin application stations need to be established at 100 to 150 meter (about 330 to 490 feet) intervals to maintain desired toxicity levels.

Antimycin acts at a cellular level to interrupt respiration (Schnick 1974:11). Cellular respiration is the process by which oxygen is used to extract energy from organic acids produced by glycolysis, with carbon dioxide being released as the end product (Kirk 1975:301). Cellular respiration occurs in mitochondria, which are organelles in the cytoplasm of cells (DeRobertis and DeRobertis 1980:14). Antimycin interrupts cellular respiration by inhibiting electron transport between cytochrome b and cytochrome c in Complex III of the cellular respiratory chain (Potter and Reif 1952, Rieske et al. 1967a, b).

In addition to rapid natural degradation of antimycin, potassium permanganate (KMnO₄) is used to neutralize antimycin at the downstream end of each treated segment of stream to prevent the piscicide from remaining active outside the treatment area. Potassium permanganate reduces the half-life of antimycin to 7 to 11 minutes in a laboratory setting. The normal half-life of antimycin in laboratory settings can range from 4.6 hours at pH 9.5 to 310 hours at pH 6.5; therefore, KMnO₄ is an excellent neutralizer for antimycin. Potassium permanganate is a strong oxidizing agent and quickly breaks down to naturally occurring compounds that are not toxic (Archer 2001). However, KMnO₄ can be toxic to fish (Tucker and Boyd 1977, Archer 2001). In a laboratory setting, sustained exposure to 2 mg/l KMnO₄ was lethal to rainbow trout, but in antimycin-treated stream water KMnO₄,
is quickly broken down as it reacts to organic material and antimycin. Kemp et al. (1966) found KMnO₄ formed a biologically inert residue when it reacted to organic material. Breakdown components of KMnO₄ (potassium, manganese, and water) are common in nature and have no deleterious environmental effects at concentrations used for neutralization of antimycin (2-4 mg/l; Finlayson et al. 2000). Monitoring stations consisting of caged live fish would be placed at the downstream limit of the project area to verify detoxification of antimycin and KMnO₄.

Literature Cited


