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Recommended Citation
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Genetic variation is central to populations, since it produces phenotypic variation that is crucial for adaptation to environmental change. Recent evidence has shown that exposure to environmental toxicants alter allozyme variation in natural populations, which may be due to bottleneck effects or selection for resistance (Gillespie and Guttman, 1999). A toxicity experiment was conducted to determine if exposure of fathead minnow larvae (Pimephales promelas) to copper causes a bottleneck effect in allozyme variation. Fathead minnows were subjected to a 10-day exposure bioassay using copper sulfate (LC50) as the toxicant. One group of larval minnows (86) was exposed to 2.67ppm of copper, while another (86) was maintained in clean water to serve as a reference. Larvae were not fed, nor was the water changed during the bioassay. Thirty-five percent of the fathead minnows died during the copper sulfate exposure, whereas 8% of the reference minnows died. The majority of minnows (86%) exposed to copper died within a 48-hour period of initial exposure. In order to measure allozyme variation, four loci (PGM, GPI-1, GPI-2, and LDH) were analyzed using cellulose acetate electrophoresis. Allele and genotype frequencies of individual loci did not show conclusive evidence for a bottleneck effect. Individual larvae were assigned a multiple locus genotype (MLG) based upon the combination of individual allozyme locus genotypes. Differences in relative frequencies of multiple locus genotypes between the initial population and survivors of exposure may have indicated a bottleneck effect. The number of MLGs (survivor group and mortality group) in the initial population was 17. The number of MLGs in survivors, however, was 11. If the “loss” of MLGs represents a loss of genetic variation in the surviving population, then exposure to copper may have caused a genetic bottleneck effect.