

Wilson, J.F. and W.K. Bates. Effects of cycloheximide at low concentrations.

We have observed the following effects of cycloheximide (Actidione) on the Oak Ridge and Rockefeller-Lindegren wild type strains of Neurospora crassa: 1. Detectable inhibition of growth occurs at a concentration of 0.1 $\mu\text{g/ml}$., and a concentration of 0.5 $\mu\text{g/ml}$ causes more than 75% inhibition of growth of standing cultures.

2. Hyphal tip of microcultures (Wilson and Garnjobst 1966 Genetics 53:621) develop abnormal morphology after exposure to concentrations of 0.6, 0.7, or 0.8 $\mu\text{g/ml}$. This change is accompanied by pronounced cytoplasmic flow into the tips, with consequent swelling and dichotomous branching even in the presence of a hypertonic (14%) sucrose solution.

3. Regeneration into punctured cells, typically 100% within 45 minutes at 30°C, is totally inhibited in the presence of 1.0 $\mu\text{g/ml}$. Of a group of cells individually injected with cycloheximide at a concentration of 10.0 mg/ml ., two-thirds survived and one-half of all injected cells retained the ability to regenerate. Production, by absorption, of an intracellular level of antibiotic comparable to that obtained by microinjection would require 100% uptake of the total cycloheximide content of a microchamber filled with a concentration of 1 $\mu\text{g/ml}$.

A complete description of this study requires photographs of regeneration and data too extensive for a brief summary. The complete description will therefore be published elsewhere. We feel that, even on the basis of this brief description, caution must be exercised in the interpretation of data based upon the use of cycloheximide concentrations above 1.0 $\mu\text{g/ml}$. In addition, even though the observations relating to regeneration are consistent with mechanisms based upon inhibition of protein synthesis, the tolerance of cells to the high injected concentration suggests that the toxicity of external cycloheximide may result directly from effects upon the cell membrane, and only secondarily from inhibition of protein synthesis. ■ ■ ■ Department of Biology, University of North Carolina at Greensboro, Greensboro, North Carolina 27412.