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Effect of Ethanol on Respiratory Rate of Goldfish

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Biology 202

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Introduction

Alcohol is a known central nervous system depressant and thus effects change on various aspects of physiological functions. Ethanol has long been thought to be a respiratory depressant in animals. However, large doses are usually required to induce noticeable effects on respiration. Deaths from acute alcohol intoxication have long been believed to be due to respiratory failure in humans (Johnstone and Witt 1972). Several studies have documented this. Loomis (1952), Klingman and Haag (1958), and Malt and Baue (1971) found that dogs intoxicated with ethyl alcohol will invariably die of respiratory failure. These effects occur at rather high doses and similar effects are expected to occur with humans (Kaye and Haag 1947). Studies on human alcohol consumption indicate that the effects associated with ethanol ingestion depend on many variables. Whether that person is a chronic user contributes to tolerance to ethyl alcohol. However, an overwhelming amount of evidence supports the hypothesis that alcohol does indeed slow bodily functions. With that in mind we set out to determine if alcohol is, indeed, a depressant then it would effect some sort of change in respiratory rates of goldfish. Specifically, we hypothesized that the alcohol would slow the respiratory rates of our test subjects and that the effects would be dose dependent.

In our experiment our sample fish population would be subjected to different levels of alcohol concentration and opercular beats would be measured. The inebriation vehicle chosen is that of direct addition of the ethanol to the subject's aqueous environment with the assumption that ethanol freely diffuses across the gills (Johnston and Bernard 1983). Thus, blood ethanol concentrations of the fish would be approximately equivalent to that of their surroundings.

Methods and Materials

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