INTRODUCTION
An individual’s propensity to drink can be affected by genetic factors as well as environmental influences (e.g., social and familial influences). To study the genetic factors, we examined the genes encoding the enzymes alcohol dehydrogenase in liver (ADH2), mitochondrial aldehyde dehydrogenase (ALDH2), and a reuptake receptor for the brain neurotransmitter serotonin (SLC6A4). We extracted DNA from cheek samples, PCR-amplified the ADH2, ALDH2, and SLC6A4 genes, and analyzed alleles by agarose gel electrophoresis. To study the environmental influences, we compiled selected data from a 94-question survey that was completed by 131 participants. The survey focused on questions regarding family background, social affiliations, and alcohol consumption.

PURPOSE AND HYPOTHESIS
The goal of this experiment was to determine how environmental (especially maternal/paternal influences) and genetic factors influence alcoholic consumption of Chico State students. Due to the Caucasian homogeneity of Chico State students, variation in acetaldehyde alleles should be low. Serotonin receptors, however, vary more in Caucasian populations, and this variation hypothetically predisposes increased alcohol consumption (1).

MATERIALS AND METHODS
1) DNA was extracted from cheek samples. We PCR-amplified the ADH2, ALDH2, and SLC6A4 genes, and analyzed alleles by agarose gel electrophoresis.
2) Student surveys on drinking, drug use, and family history were collected from a 94-question WebCT survey.

RESULTS
The majority of students responding to our survey (n=132) consider themselves social drinkers (Figure 5). Variation in the alcohol dehydrogenase gene was low, while the gene for the serotonin reuptake transporter protein showed some variation. Individuals using antidepressants had interestingly more homozygous LL serotonin transport haplotypes, as observed in previous studies, but this was not a significant result. No strong correlation was observed between maternal drinking behavior and the drinking behavior of the individual, i.e., alcoholic mothers did not typically rear alcoholic children. On average, individuals with mother’s that were moderate drinkers consumed one more drink per sitting, but this finding was also not significant (Figure 6). The survey showed on average that mothers drink less than fathers (Figure 7). The contribution of tobacco use to alcohol consumption showed deviating averages, but was not significant (Figure 8).

DISCUSSION
The null hypothesis, namely that the serotonin transport receptor and alcohol/aldehyde dehydrogenase alleles have no effect on drinking patterns, failed rejection. Caucasian majority of the CSUC student body necessitates a different sample population when testing for the effects of alcohol dehydrogenase on alcoholism. Variations in serotonin reuptake genotypes did not correlate with increased alcohol consumption. They did, however, correlate with previous studies of depression, meaning depressions role in predisposition of alcoholism is not substantiated in this study. Increasing sample size and sample diversity would better elucidate the role of these two genes in predisposition of alcoholic behavior.

CONCLUSIONS
Attempts to correlate drinking behavior with genetic and environmental influences failed to show a significant pattern with any one factor, due to many factors influencing alcohol consumption. This is an interesting topic in light of current events related to alcohol use.

REFERENCES CITED

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