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The Effect of Caffeine and Nicotine on Heart Rate

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The use of caffeine and nicotine raises the heart rate of individuals who use the chemicals. Many statistics have shown that the use of the chemicals on college campuses is at a high percentage, we want to test how which one of these chemicals raises the heart rate the most. We tested these chemicals on a daphnia heart because it is very similar to the human heart in structure and the daphnia is transparent making it easy to see the heart through the microscope. We conducted six trials for each of the chemicals, comparing 1 mM of nicotine and 1% of caffeine. We came to the conclusion that nicotine affects the heart rate the most. We expect these findings to be in the interest of those college students who use nicotine on a daily basis, to show how dangerous it is.

Introduction

The use of nicotine and caffeine among students on a college campus have increasingly raised over the past decade (Aria et al., 2010). Students use caffeinated drinks like red bull and coffee to study for tests, and the use of nicotine is very prevalent in the party atmosphere, "social smoking" and the use of electronic cigarettes on college campuses. The relevance of caffeine and nicotine on college campuses is alarmingly high, because we all live on college campuses we would like to test which one of these chemicals causes the heart rate to increase more rapidly. Although we are concerned about the effect on college students, we will be testing the effect of these chemicals on daphnia. The specific compounds that we are using in our experiment are 1 mM Nic for our nicotine

trials and 1.0% Caff for the caffeine trails. The reason we are using the daphnia is because their hearts are very similar to the human heart, and since they are transparent it is easy to study the effect of the nicotine and caffeine on the heart (Stein et al., 1966). To be more specific on the mechanics behind how nicotine and caffeine really work we can see that in nicotine, it binds to nicotinic cholinergic receptors, which are located in the brain, autonomic ganglia, the adrenals and neuromuscular junction (Benowitz, 1997). The main effect of nicotine in a cardiovascular effect on the body is sympathetic neural stimulation (Benowitz, 1997). Like caffeine, nicotine increases the release of catecholamine (Benowitz, 1997), and other neurotransmitters. This release of the catecholamine releases from the adrenals and direct release or enhancement of release from vascular nerve endings (Benowitz,

1997), do lead to increased blood pressure. Human hearts have the vagus nerve, which acts to lower heart rate, daphnia have a similar nerve in their heart which is another reason they were chosen to conduct our experiment. The main question we are inquiring about is, when comparing caffeine to nicotine, which one causes the heart rate to raise higher? We hypothesize that if caffeine is added to the daphnia, then it's heart rate will be higher than when nicotine is added because caffeine is a central stimulant that increases the release of catecholamine, which raises blood pressure. In caffeine central stimulants increase energy, improve attention and alertness, but the down side of these drugs is the increase of blood pressure and heart rate (Biel et al., 1958). If our hypothesis is incorrect then the nicotine will make the daphnia have a higher heart rate than with the caffeine.

Methods

In our experiment we tested how caffeine and nicotine affected the heart rate of daphnia. For this experiment we had to first test the heart rate of a daphnia with no added stimulants to the water it was in. Once that data was collected we collected our round of data with the nicotine and the caffeine. First we used a beaker to collect a sample of the daphnia from an aquarium, once collected you select one of the daphnia using the modified dropper and place it on the depression slide. When the daphnia is on the slide there can't be an access of water surrounding the daphnia, so we had to blot off the extra with a small piece of paper towel, we had to make sure there was a small drop of water surrounding the daphnia so it can survive. Once the round of data is completed with the daphnia with no added stimulants, we began our trials with the 1% of caffeine and the 1 mM nicotine, and about four drops of the solution into the daphnia's water. The daphnia had to swim in the added solution for seven to ten minutes, before we could blot away the access just like we did with the water. Once the daphnia had time to swim in the solution, we placed the depression slide under the microscope, all settings on the microscope were set to the lowest setting. Once everything was in focus we connected scope adapter and a phone to record the ten second long videos of each daphnia. We counted beats per minute of the daphnia's heart by counting the number of beats in the ten seconds then multiplying the number six. We ran six trials for caffeine and six trials for the nicotine, so we could get enough data. Once done with the daphnia we rinsed off the slide with aquarium water. Next we calculated the percent change between the beats per minute once the stimulant was added and the beats per minute before the exposure of the stimulant.

Results

We used a box whiskers chart to display the data we collected for our experiment. The percent change in the beats per minute of the daphnia's heart had the greatest change when the 1mM nicotine was added to the solution. The percent change ranged from -10% to 20% change in the heart beats (see figure one). The caffeine had little effect on the daphnia's heart rate, once the 1% of caffeine was added to the solution the percent of change ranged from -6% to roughly 2% (see figure one).

Discussion

The data from our experiment showed that Nicotine changes the daphnia heart rate more than caffeine. This would mean that our data did indeed answer our question of whether or not caffeine or nicotine affects the heart rate more. The data failed to support our hypothesis because we believed that the caffeine would affect the daphnia heart rate more. We thought that the caffeine would increase the heart rate of the daphnia, and although it did increase, the percent change is smaller than that of Nicotine. Nicotine changed the heart rate of the daphnia more than caffeine; thus, failing to support our hypothesis.



Figure one: Displays the percent change in beats per minute of the daphnia's heart after 1mM of nicotine was added or 1% o caffine.

In order for us to reach this conclusion, we tested multiple daphnia in order to create more accuracy in our experiment. The more daphnia tested the higher the accuracy because if one run is flawed it does not skew the results. Post facto, we realize that our expectations for the experiment addressed the wrong outcome. We focused more on which solution, nicotine or caffeine increased the heart rate more. This is incorrect because it does not address change over all. Instead, we should have thought about what if it decreases the heart rate of the daphnia or if the daphnia is affected by the solution at all. A way to amend our hypothesis is to include that the daphnia heart rate will have a larger percent rate of change when exposed to nicotine because the daphnia seem to not have the proper receptors to the caffeine.

We believe that the daphnia do not have large enough or enough of the adenosine receptors for the adenosine to bind to and increase the heart rate. The adenosine binding to adenosine receptors in the brain is what creates the heart rate to increase (Huang et al., 2005). Since adrenaline is released after the adenosine binds, the heart rate increases therefore the daphnia is not releasing the adrenaline needed to create a large rate of change as we expected in our hypothesis.

In the beginning of the experiment, we thought that the data supported our hypothesis. We exposed the daphnia to caffeine first and saw that the heart rate did in fact increase like we expected with our hypothesis. As we finished up with all the daphnia we noticed that the margin of increase was small, creating a small percent change. We knew for sure that our hypothesis was incorrect after we exposed the daphnia to the nicotine. The changes in the heart rate, whether it increased or decreased, were much greater with nicotine. After noticing the change we decided we needed to figure out why the daphnia did not respond to the caffeine as much as the nicotine.

Besides the adenosine receptors for the daphnia we speculate that size and length of the daphnia could explain our results. For humans, different doses of medicine affect them differently depending on their sizes. Since there the pattern in mammals is the smaller the mammal the higher the heart rate in beats per minute due to their metabolism (Westerhof and Elzinga, 1993). It is possible that the same happened with the daphnia, the smaller daphnia could have already had a higher heart rate which would show little change in their heart rate meanwhile the larger daphnia actually did have a significant change in their heart rate.

Excluding errors in data collection, the data could tell us that there could be something involving the daphnia that created these results. The dosage that the daphnia were exposed simply could not have been enough to create an impact on their heart rate. In humans, large amounts of nicotine or caffeine impact heart rate more than small amounts. It is possible that if the daphnia were exposed to a higher dosage of caffeine our hypothesis could have been supported since there is more caffeine present for the daphnia to take in. Biologically the daphnia could just be more susceptible to nicotine, meaning their bodies intake the nicotine easier and in larger amounts creating a larger percent change than caffeine.

The results we gathered in this experiment we believe have an impact on the society we live in today because of how prevalent the use of nicotine is today, especially on college campuses. Our results showed us that the nicotine had a significant effect on the heart rate. With this information we should be able to have better of understanding of the information if we tested this on humans, specifically on college students. If we proved that nicotine does have the same effect on the students, we could possibly lower the percentage of college students using nicotine.

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