The Effects of Nicotine on Heart Rates in the Atlantic Surf Clam (*Spisula solidissima)*

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Introduction

Many drugs and chemicals work in the body by either mimicking or blocking the effects of the natural chemicals in the body (Cobb, 2002). Additionally they may stimulate or block the release of a chemical from the body. Nicotine has many different effects in the body, but usually one effect dominates giving the overall observed response. In humans, and in all mammals as is known, nicotine increases the heart rate (Cooper, Hausman 2004). This is because nicotine stimulates the release of adrenalin and noradrenalin, a chemical very similar to adrenalin. Collectively these chemicals are termed catecholamines. (Bourzac 2000). The fact that nicotine is working via this pathway can be demonstrated by drugs that block catecholamine receptors, for example b-blockers, which you may have heard about. These drugs block the effect of nicotine increasing heart rate. Nicotine causes a slight increase in the concentrations of adrenalin in the blood (Wikipedia, 2004). However an injection of adrenalin that gives the same increase in blood concentrations as seen with nicotine does not increase the heart rate as much as is observed with nicotine (Wikipedia 2004). Therefore the nicotine must be having more localised effects and it is assumed that it stimulates the local release of catecholamines in the heart from local nerve terminals (Bourzac 2000). Nicotine is a drug that can be found naturally present in the leaves of tobacco plants. It increases the contraction rates and beats per minute of the heart muscle, as well by decreasing blood circulation, in turn narrowing the width of blood vessels through which the blood travels (Bourzac, 2000). Thus, this narrowing of the blood vessels can eventually lead to heart disease, stroke or even death (Cobb, 2002). When an individual is exposed to such a chemical in small doses , nicotinic acetylcholine receptors are initiated or activated (Cobb, 2002). Such activation of the nicotinic acetylcholine receptors lead to an increase in the flow of adrenaline. It should be noted that adrenalin happens to be a stimulatory protein. (Cobb, 2002) Furthermore, an increased flow in adrenaline causes an increase in heart rate as a final effector. (Wikipedia 2004).

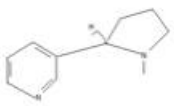

Fig 1. Here a figure of the structure of Nicotine is expressed.
(Structure adapted from www.math.rutgers.edu/~zeiberg/family/nicotine.gif)

It should be noted that heart rate is commonly defined as how many heart muscle heart contractions occur in a single minute. Thus, such processes are referred to in the unitary citation of beats per minute (bpm). The intracellular pathways can be activated or deactivated in a variety of mechanisms each leading to imparting a different effect onto the functioning behaviors of the heart muscle. For example, the human body increases the heart rate when an outside factor stimulates an intravenous, and intracellular response, such as the nicotine illiciting the release of adrenaline into the blood.

In this experiment, the effects of nictotine on the contraction of heart cells of the Atlantic Surf clam was studied. It is hypothesized that since due to the fact that nicotine stimulates an increase in adrenalin in the blood, the presence of nicotine will increase heart rate. This hypothesis is of extereme pertinence because studying the effects of nicotine on heart contraction will help to better understand how an introduction of nicotine into the system of an

organism, such as humans, increases or decreases heart rate, which may be a direct cause of heart disease in the human population (Bourzac, 2000). Similarly, this hypothesis is intriguing because results obtained from the study may shed light on how and why heart cells contract when coming in contact with the nicotine. In this experiment, Atlantic Surf clams were used to serve as the mechanism by which the effects of nicotine would be studied on the heart. Nicotine would be administered in liquid form onto the site of the beating heart, perhaps 3 drops at a time, and then the heart would be observed for any change in beating rates. If none were observed, more nicotine would be administered. Atlantic Surf clams were chosen for this study, because much is known about their intracellular mechanisms, such as chemical signaling of cells from different tissues, and significant study had been administered with them in researching the effects of other chemicals on tissues within the animals (Cobb, 2002). In this experiment, the inner contents of the clam were opened, with as much of the tissue being kept in contact as possible. The heart of the clam was located by observing beat like motions underneath the gills, and when the tissue covering the gills was pulled back, small amounts of nicotine were administered directly to the region near or surrounding the heart, and had been observed for an increase or decrease in the heart activity.

Materials and Methods

Materials:

Atlantic Surf Clams Medium
silver plated scalpel
sea water at room temperature,
small container bucket,
incubator set at 18 degrees C,
Scholar Chemistry Nicotine Solution (1.5X10^-6 mg/m^3)
dissection microscope
Nikon SMZ660 Digital Camera
Spot Insight QE Camera
Macintosh computer (OSX operating system
microscope lights,
Dissection knife,
Petri dishes,
Kimwipes,
pipettes.
Professor Robert L. Morris Urchinology Laboratory
BTV Pro, Microsoft Office Picture Manager
ICUC Center - Wheaton College : Science Center: 26 East Main St. Norton, MA 02766

Methods:

A group of 6 Atlantic Surf C clams were obtained. Next, the clams were carefully opened by cutting into the muscle tissue located near the hinge of the clam. The two shells were then pulled open, exposing the inner contents, anus, heart, gills, foot. The clam being studied was placed in a petri dish with some salt water so that the gills and tissue covering the heart such as the gills would become flexible and was pulled back to expose the beating heart, which is located near the hinge of the clam. A nicotine solution of 1.5X10^-6 concentration was obtained in liquid form. 1 pippete was filled at a time. 3 drops were then added at a time to the heart and its surrounding regions. BTV Pro was opened to begin video recording of the observations of the effect on heart cell beating rates. The clam being studied was gently positioned underneath the microscope lense so that the video camera (BTV Pro) transmitted a clear shot of the clam and its functioning cardiovascular system. Eight video clips were captured as the heart rate changed (either quickening or slowing down), each video clip lasting five minutes in duration. Some of the movie clips fast forwarded to better and quickly observe the movement of the cells. Each movie clip was reviewed one at a time and the time it took for one heart contraction to occur, was measured. Finally, averages of the data were taken. For example, in between heart contractions seconds were counted. Each clam underwent 3 trials, with each trial undergoing the addition of 3 drops of nicotine administered to the heart. If an increase in contraction was noticed, it was noted on paper with how many drops were administered.

Results

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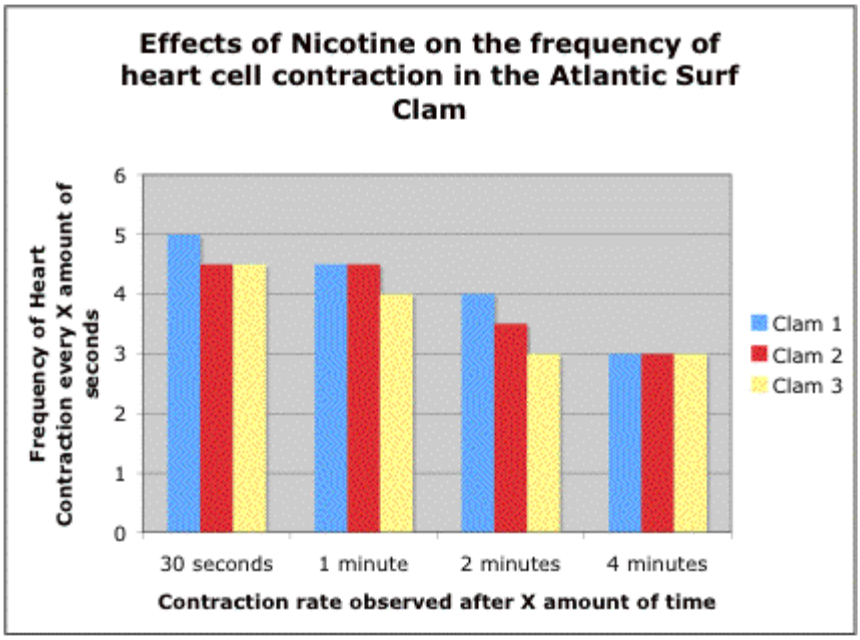

Fig 2. This graph expresses evidence that nicotine affects the clam heart cells after administering the nicotine, 3 drops at a time to each of the three clams.

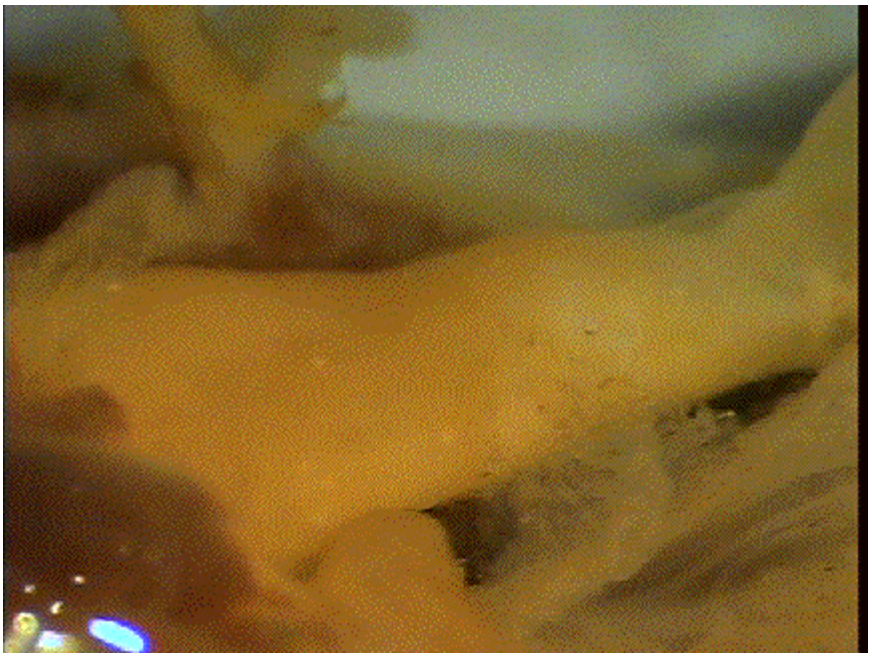

Fig 3. Ths image shows the heart contraction of the Atlantic Surf Clam as it contracts in response to the stimulatory nature of nicotine, which initiates the release and increase of adrenaline levels in the blood, which in turn constricts blood vessels, which cause the heart cell contraction rate to increase.

In Figure 2, it can be observed that as time increased, and more drops of nicotine were administered, the heart rates in all three clams ended up increasing. For instance, in Clam 1, the heart rate increased from 1 heart contraction every 5 seconds, to one heart contraction every 3 seconds. In clam 2, heart rate initially stayed stable at one contraction every 4.5 seconds, up until 1 minute after administering the three drops of nicotine, however increased to 1 contraction every 3.5 seconds after 2 minutes, and even lower, to one contraction every 3 seconds after 4 minutes. In the case of Clam 3, the heart cell contraction increased from 1 contraction every 4.5 seconds to one contraction every 4 seconds after 1 minute, and then decreased from one every 4 seconds to one contraction every 3 seconds and remained stable thereafter.

Cell contraction for the control clam remained stable for the most part, however in clams where nicotine was added, heart cell contraction, and tissue movement greatly increased as more and more amounts of the nicotine were added. For example, after 30 seconds, only three drops of the nicotine solution was added to the heart of Clam I. After 1 minute, 6 drops had been added, (three drops in each addition). This addition accounts for the increase in heart contraction from one contraction every 5 seconds to one contraction every 4.5 seconds. At the end of the trial, the heart cell contraction had increased to an astounding rate of one contraction every 3 seconds. Similar results were found to prove true in both Clam II and Clam III as heart rates readily increased as more nicotine was added.

Discussion

In this preliminary experiment, data suggest that nicotine does in fact greatly increase the heart rate in the Atlantic Surf Clam (*Spisula solidissima).* These results suggest that as more nicotine was applied to the cardiovascular system of the Atlantic Surf Clam, more adrenaline receptors had their ligands bound to them and as a result more adrenaline was released into the bloodstream. The release adrenaline into the blood stream in turn could have interacted with the receptors located on the endothelial cells located on the lining of the veins and arteries leading to the heart. This activation may have led to a narrowing of the arteries and circulatory constriction, thus causing the heart contraction rate to increase to make up for the addition of this added strain (Wikipedia, 2004). Thus, in conclusion the hypothesis that an addition or increase of nicotine to the cardiovascular system of the Atlantic Surf Clam increased heart contraction was upheld via results of this laboratory experiment.

Data for this experiment was quantified by measuring the amount of time it took for one heart contraction to occur after the nicotine had been administered. This was repeated for each clam specimen and a graph was then made that reflected the results that were observed. It should be noted that my collaborator, Phuentsho Wangmo, studied the effect of nicotine on cilia motility in the gills of the clam and used the same source of nicotine and similar methods and procedure.

Sources of error in this laboratory include the fact that the clam specimens, when being handled and opened, may have sustained injuries or perforations to vital organs and tissues in their system. For example, in one clam specimen that was observed, it could be observed that the gills and respiratory tract sustained a rip due to the incision that was made when opening the clam. To avoid encountering this problem in the future it would be suggested to carefully identitfy the adductor muscles and hinge of the clam before making an incisions so as to not perforate important tissues by accident, which could result in the premature death of the organism. An additional source of error that was encountered in this laboratory was the accidental measuring error of nicotine drops being administered. For example, when administering the drops, occasionally one large drop would form at the end of the pipette or two very small drops would be squeezed out. Confusion ensued as to whether to consider such drops as one or two drops. This may have skewed the data irrevocably as even half of one drop of nicotine can cause enormous side effects and change in behavior in a clam whose biological functioning system is much smaller and fragile than that of a human or other large organism. To avoid this problem in the future, it is suggested that smaller amounts of nicotine be taken up into the pippete at any given time, and slow, steady squeezing be administered to the bulb when begining to administer the nicotine. Such careful, cautious pipetting should reduce the appearance of drops that could be considered either one large or two small in size. Another problem with this laboratory is that too few trials were conducted. Conclusive evidence to support the hypothesis formulated in this laboratory cannot be formulated by just three trials. This is due to many factors. For example, the three animals that were studied may have been sick, weak or otherwise different from most other specimens representative of this species. By obtaining more clam specimens and running far more trials, the possibility of incorrect data and results would be greatly reduced.

In order to obtain data that would more strongly support the formulated hypothesis it would be recommended

to study the detailed effects that nicotine and its chemical structure have on activating and releasing adrenaline that is stored in intracellular compartments. Similarly, it would be interesting to study how the b blocking receptors function to increase heart rate in response to nicotine.

In conclusion, preliminary experiment tended to indicate that as time proceeds and nicotine addition remains constant and added in equal doses, heart contraction will increase in biological organisms both in a physiological and cellular manner and via cellular mechanisms. <>

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