11-2017

The Effects of Heart Medication on the Heart Rates of Drosophila Melanogaster

Felicia A. Baer
Olivet Nazarene University, baerfe@gmail.com

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Felicia Baer[1],
Dr. Dwight Ginn[2]

[1]Felicia Baer, Dept. Biological Sciences, Olivet Nazarene University, Bourbonnais, IL
[2]Dr. Dwight Ginn, Dept. Biological Sciences, Olivet Nazarene University, Bourbonnais, IL

Introduction

Every 40 seconds one American dies of cardiac disease.[1] Current models of human cardiac disease may be similar in anatomy and physiology, but are often expensive and tedious to work with. The current need is for a model organism that is more efficient to work with in lab while still an accurate model of human cardiac disease.

Drosophila melanogaster (*D. mel*) is a more efficient organism to work with in lab due to its short life span, low cost, and the feasibility of culturing.[2,3,4] Anatomically the *D. mel* heart differs from the human heart in that it has only one chamber, one layer of cardiomyocytes, and lacks coronary arteries. It is similar, however, in protein and genetic makeup allowing the *D. mel* heart to develop structural defects, arrhythmias, and cardiomyopathies similarly to the human heart.[5,6]

Atropine increases heart rate by preventing acetylcholine from affecting sinoatrial and atrioventricular nodes by blocking muscarinic acetylcholine receptors (mAchRs). In *D. mel,* one of the two types of mAchRs are similar to humans. [7,8]

Propranolol hydrochloride is a β-blocker that reduces heart rate by blocking beta1-adrenergic receptors (βARs). *D. mel* possesses a family of G-protein receptors that are structurally and functionally related to βARs. [9,10,11]

Hypothesis

I hypothesized that atropine and propranolol hydrochloride in the growth media of third instar larvae would cause an increase and decrease respectively in the heart rates of *D. mel*.

Methods

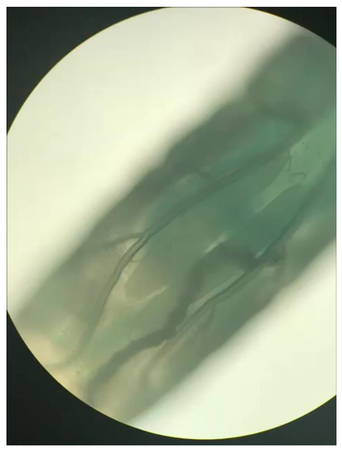
Day 1:
- Adult flies moved to fresh vials

Days 2-5:
- Adult flies mated
- Larvae hatched and grew

Day 6:
- 2st instar larvae moved to fresh media
- Control – no medication
- Experimental groups – 1mM atropine or 1mM propranolol hydrochloride

Day 7:
- 3rd instar larvae heart rates recorded

Recording Heart Rate:
- Three heart rates were recorded in fifteen second intervals for each larva.
- This was done by using a tap counter and a fifteen second timer with a ten second interval of rest in between.
- The three heart rates for each of the fifty larvae in each group were averaged.

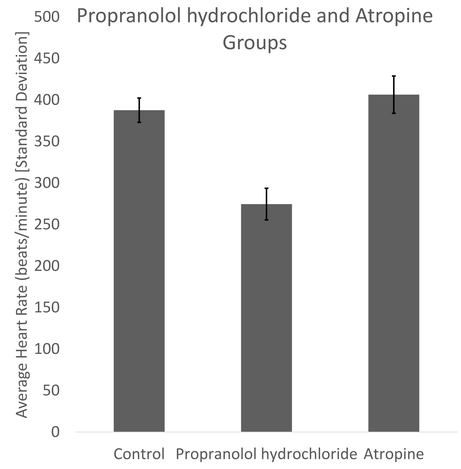


Results

	Average Heart Rate in Beats/min	Standard Deviation
Control	387.653	14.623
Propranolol hydrochloride	274.533	19.084
Atropine	406.373	22.477

Table 1 Average Heart Rate and Standard Deviation of Control, Propranolol hydrochloride, and Atropine Groups

Figure 1 Average Heart Rate of Control, Propranolol hydrochloride and Atropine Groups



T-Test	Significance
Control and Propranolol hydrochloride	3.368E-55
Control and Atropine	3.265E-06

Table 2 T-Test Results and Significance

Discussion

The hypothesis was supported - propranolol hydrochloride decreased heart rate, and atropine increased heart rate in the *D. mel* larvae. This was confirmed by a t-test in which the means for both experimental groups were shown to be significant when compared to the control group.

This research suggests that *D. mel* has potential to be an accurate model of human cardiac disease. Further, since *D. mel* can respond similarly to commonly prescribed heart medication, it could be used in preliminary pharmaceutical testing for new medication.

Acknowledgments

This research was made possible by funding from the ONU Honors Program and supplies and resources from the ONU Department of Biological Sciences.

Contact Information

Felicia Baer: fbaer@olivet.edu
Dr. Dwight Ginn: dginn@olivet.edu

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