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

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The Effects of Heart Medication on the Heart Rates of *Drosophila melanogaster*

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**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 is 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:
- 2nd 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**

![Figure 1 Average Heart Rate of Control, Propranolol hydrochloride and Atropine Groups](image)

<table>
<thead>
<tr>
<th>Average Heart Rate (beats/minute) [Standard Deviation]</th>
<th>Control</th>
<th>Propranolol hydrochloride</th>
<th>Atropine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beats/min</td>
<td>387.653</td>
<td>274.533</td>
<td>406.373</td>
</tr>
</tbody>
</table>

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

**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.

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**References**