Students – additional information that expands on the slide content has been included in the speaker notes section for your convenience. If speaker notes are available you will see a speech bubble icon in the top left corner. Hover over the icon to see the speaker notes for that slide.

UBC SHARC 2016 Workshop

Designing & Presenting a Research Poster

Linda Herbert

Student Research Coordinator, Faculty of Medicine, UBC

Ada Lo

UBC MD 2019

Science Communication

Tips for effectively sharing your research

Part 3: Designing & presenting a research poster

Linda Herbert, MSc

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What is the purpose of a research poster?

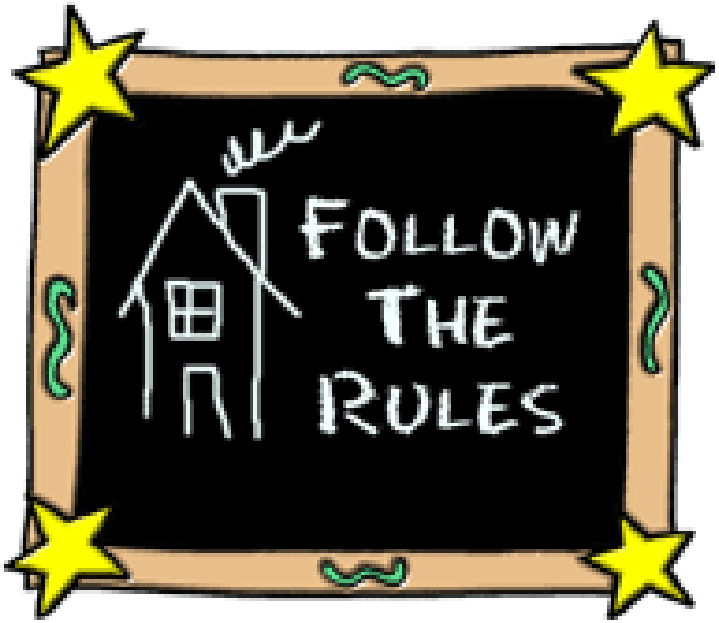
Realities of a Poster Session…

Where to start?

1. Confirm the guidelines/constraints
2. Distil your research – keep it simple!
3. Start designing…

1. Confirm guidelines/constraints

- Orientation
- Size
- Format?
- Content?



SHARC Guidelines

- Any orientation
- Width ≤ 36” (ideally)

2. Distil your research

- Keep it simple!
- Focus on the important stuff
- Eliminate superfluous detail
- Be clear and concise
- Lead the reader/viewer
- Picture/diagram vs words
- 1-2 take home messages
- Think like an abstract!

Photo: Roger Ferrer Ibáñez flickr

EXAMPLE

My awesome poster title

Methods

- This is where I explain the methodology with a huge section of text.
- It is really time consuming to read and the audience either stops listening me while they are reading or, more likely, they just ignore my poster because it looks a little overwhelming and they don't want to spend 15 minutes reading the ridiculous amount of text I've included here.
- Seriously, just looking at this much text in one block will make me skip a poster!
- Are you even still reading at this point?
- Do you have any idea what I have been saying while you have been reading?

Another poster section

- A whole bunch more text is down here.
- Look at all the text crammed on this poster!

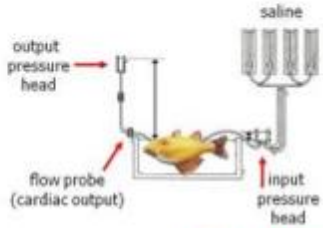
The final poster section

- I just love text and I think everyone else should love it too!
- People should spend 15 minutes just reading my poster, that is realistic right?
- They should also be required to stand really close so that they can read this teeny, tiny text I have used.

EXAMPLE

My awesome poster title

The *in situ* perfused heart



Cardiac power output = flow x pressure generated
(i.e. ATP demand) **($P_{IN} - P_{OUT}$)**

The final poster section

- I just love text and I think everyone else should love it too!
- People should spend 15 minutes just reading my poster, that is realistic right?
- They should also be required to stand really close so that they can read this teeny, tiny text I have used.

Another poster section

- A whole bunch more text is down here.
- Look at all the text crammed on this poster!

3. Designing & presenting your poster

- Know your audience
- Be engaging
- Think visually
- Go the extra mile
- Practice, practice, practice

1. Know your Audience

Who?

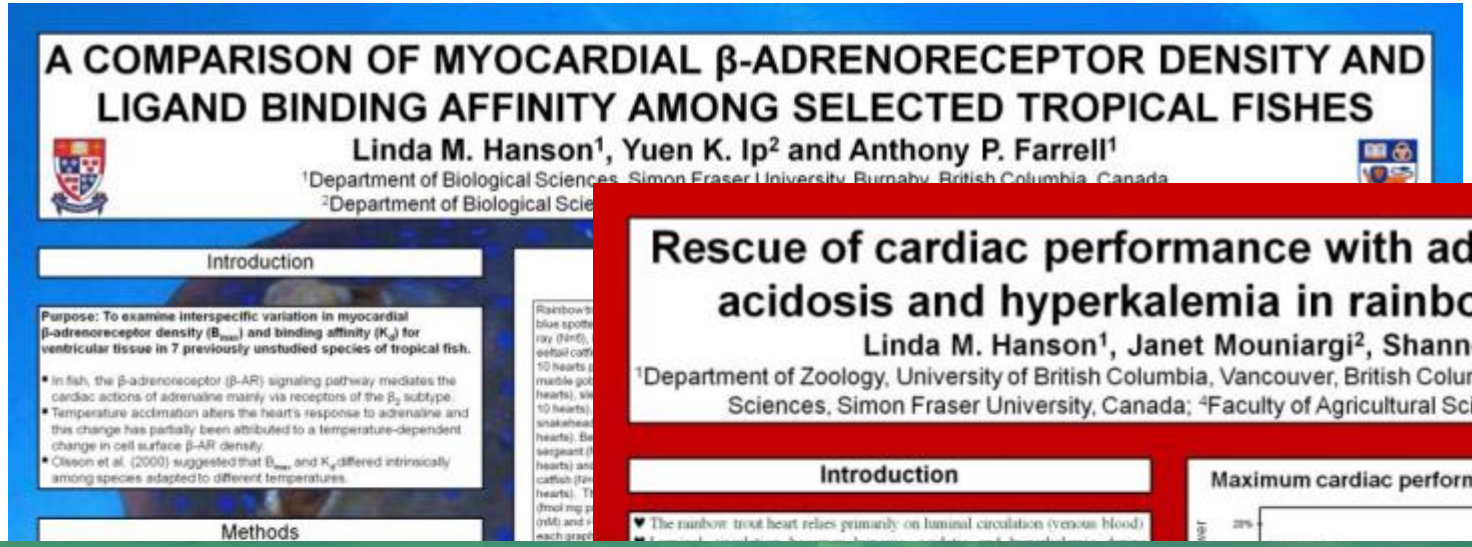
When?

What?

Where?

Examples

Know your audience



2. Be engaging

Manuscript ≠ Poster

Limited time

Engaging title

but^appropriate

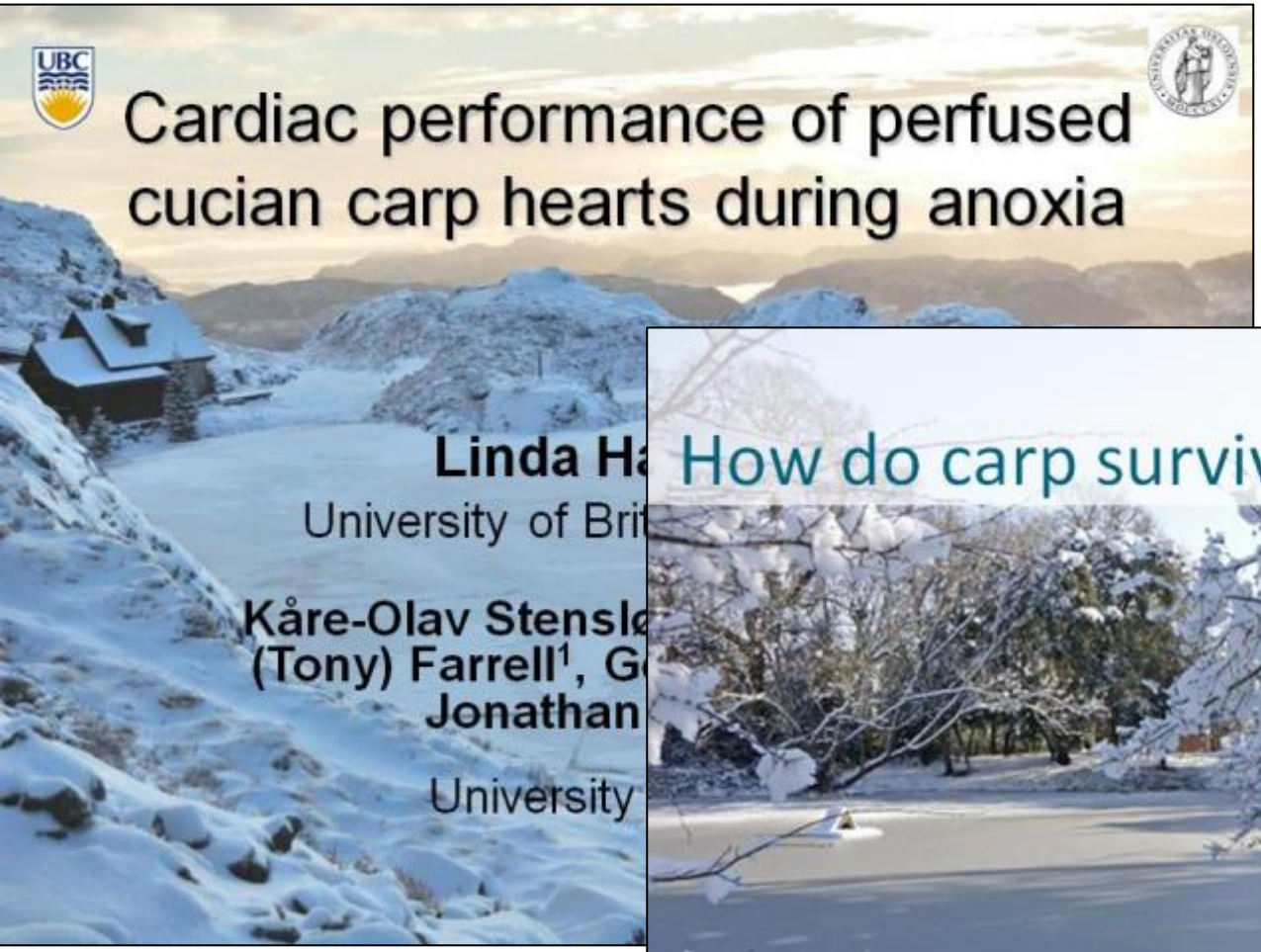
Get excited

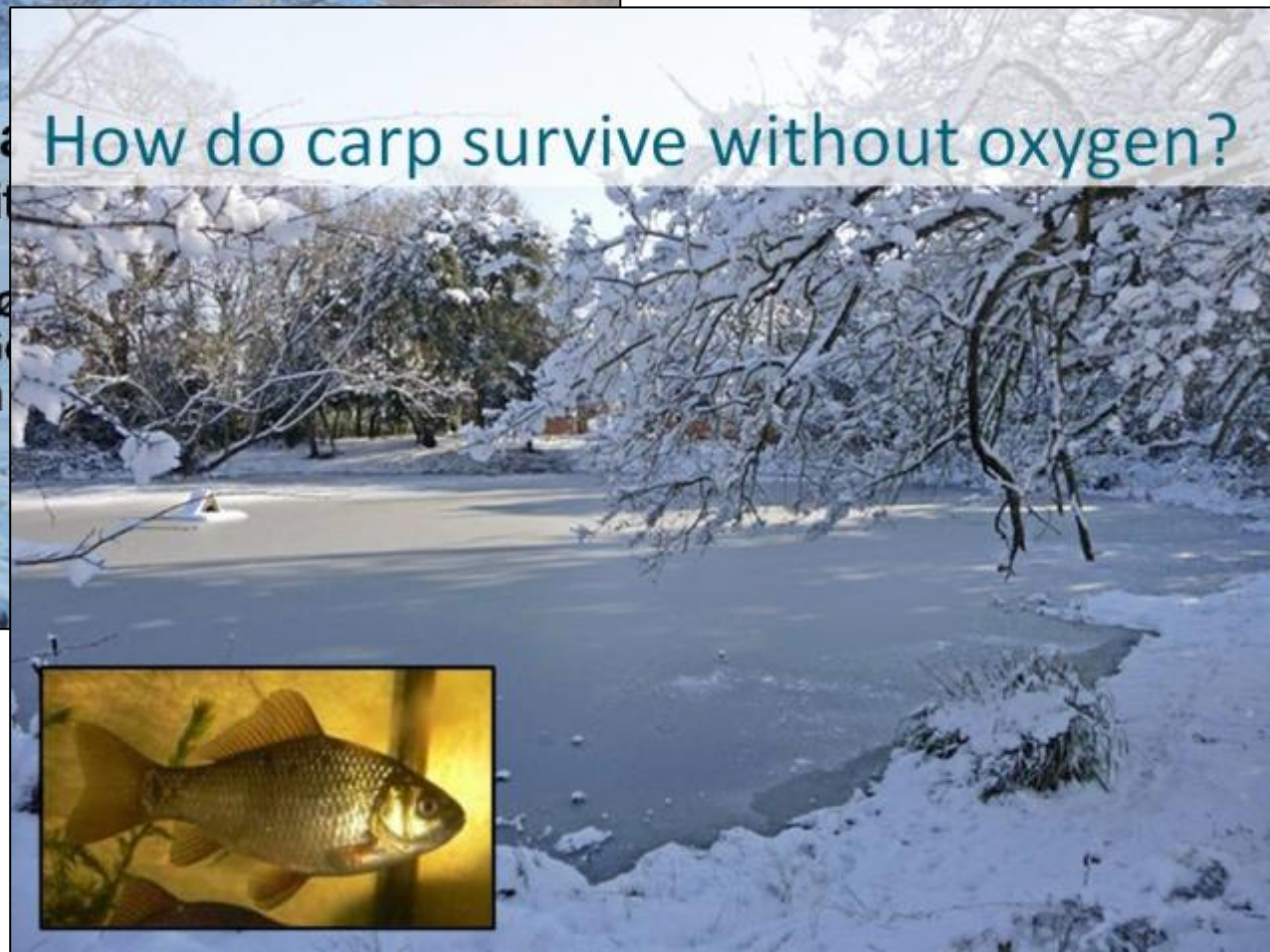
Tell a story

Be yourself

Examples

Engaging title





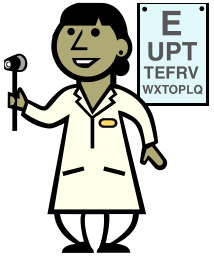
3. Think visually

Do you think that anyone can actually read the this tiny, tiny font…
…or that they want to spend 20 minutes reading one poster?

Font choice

Summarize

Images

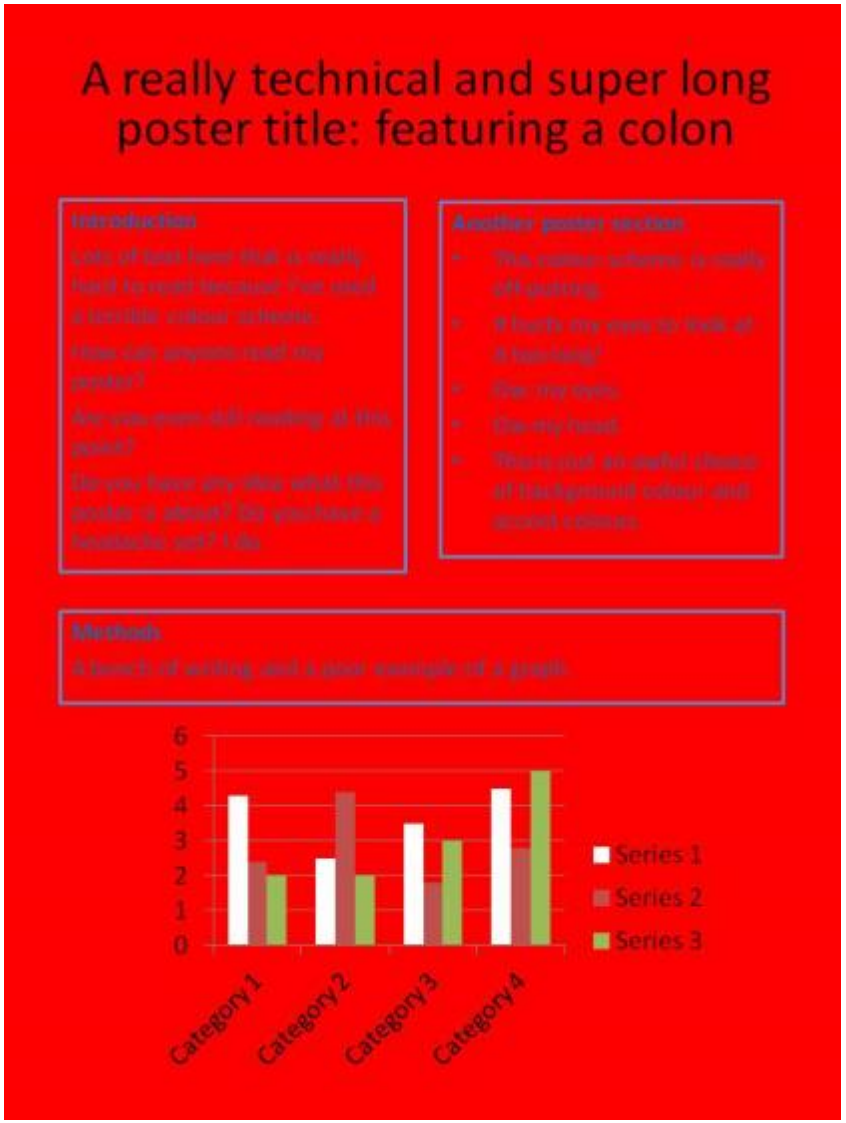


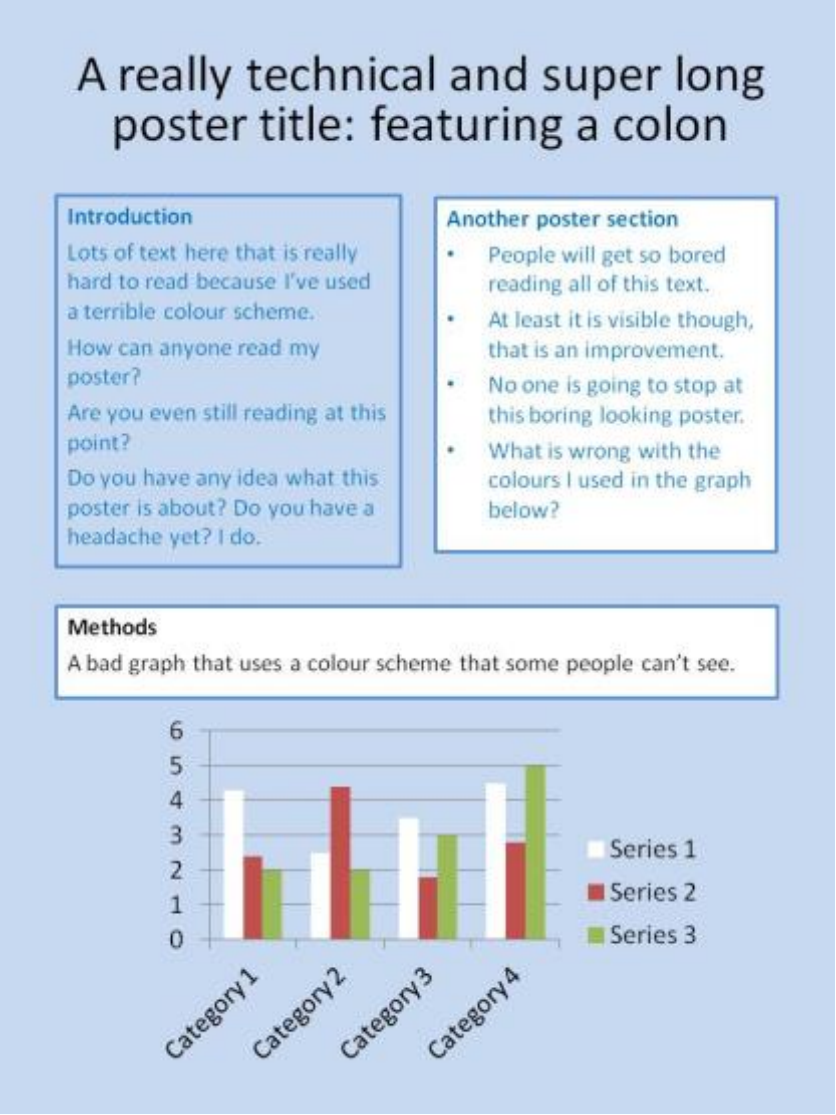
Readability

White space

No distractions

Examples


A really technical and super long poster title: featuring a colon
Introduction
Lots of text here that is really hard to read because I've used a terrible colour scheme.
How can anyone read my poster?
Are you even still reading at this point?
Do you have any idea what this poster is about? Do you have a headache yet? I do.
Another poster section
This colour scheme is really off-putting.
It hurts my eyes to look at it (seriously)
Ew, my eyes.
Ew, my head.
There is not an awful choice of background colour and accent colours.
Methods
A bad graph that uses a colour scheme that some people can't see.
6
5
4
3
2
1
0
Series 1
Series 2
Series 3
Category 1
Category 2
Category 3
Category 4


A really technical and super long poster title: featuring a colon
Introduction
Lots of text here that is really hard to read because I've used a terrible colour scheme.
How can anyone read my poster?
Are you even still reading at this point?
Do you have any idea what this poster is about? Do you have a headache yet? I do.
Another poster section
People will get so bored reading all of this text.
At least it is visible though, that is an improvement.
No one is going to stop at this boring looking poster.
What is wrong with the colours I used in the graph below?
Methods
A bad graph that uses a colour scheme that some people can't see.
6
5
4
3
2
1
0
Series 1
Series 2
Series 3
Category 1
Category 2
Category 3
Category 4

Examples

Here is a bunch of text do you think it is easy to read these words when they are justified?
What about when we use superdyduperdy long words that really mess up the spacing.

Here is a bunch of text that is not justified do you think it is easier to read these words when they are not justified?
What about the superdyduperdy long words, how do they look?

Examples

Rescue of cardiac performance with adrenergic stimulation during hypoxia, acidosis and hyperkalemia in rainbow trout (*Onchorhynchus mykiss*)

Linda M. Hanson[1], Janet Mouniargi[2], Shannon Obradovich[3], and Anthony P. Farrell[4]

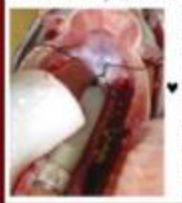
[1]Department of Zoology, University of British Columbia, Vancouver, British Columbia, Canada; [2]Zoophysiology, Gothenburg University, Sweden; [3]Biological Sciences, Simon Fraser University, Canada; [4]Faculty of Agricultural Sciences & Dept. of Zoology, University of British Columbia, Canada.

Introduction

- The rainbow trout heart relies primarily on luminal circulation (venous blood)
- Luminal circulation becomes hypoxia, acidotic and hyperkalemic during strenuous exercise, factors that are highly detrimental to cardiac performance
- Nevertheless, the rainbow trout heart must maintain a high cardiac performance under these conditions
- **We hypothesize that Adrenergic stimulation plays a critical role in maintaining maximum cardiac performance under conditions of strenuous exercise (hypoxia, hyperkalemia and acidosis).**
- In addition, we were interested in determining the hypoxic thresholds for cardiac collapse under hypoxia alone, and under strenuous exercise conditions with tonic and maximal adrenergic stimulation

Technique – The Perfused Heart

- This *in situ* preparation isolates the heart in terms of perfusate delivery and collection while leaving the pericardium intact, allowing for assessment of maximum cardiac performance
- The input cannula is introduced into the sinus venosus via a hepatic vein and the output cannula is inserted into the ventral aorta (Farrell et al., 1986)



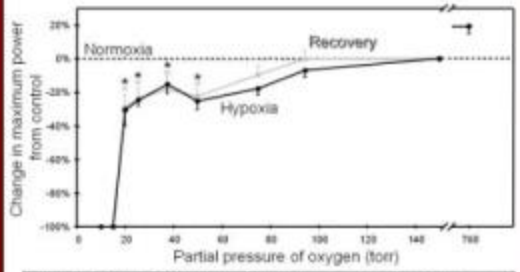
Experimental Procedure

- Maximum cardiac performance of *in situ* perfused rainbow trout hearts was assessed at 10°C under varying levels of hypoxia (94-10 torr), both alone and in conjunction with hyperkalemic (5 mM), acidotic (pH 7.5) exposure
- In addition, the hypoxic, hyperkalemic, acidotic exposure was done with both tonic (5 nM) and maximal adrenergic stimulation (500 nM)
- Sequential 15 min perfusions were done for individual hearts as follows:

1. normoxic (150 torr O_2, pH 7.9, 5 nM adrenaline)
2. hypoxic (pH 7.9, 5 nM adrenaline)*
3. recovery/normoxic (150 torr O_2, pH 7.9, 5 nM adrenaline)
4. strenuous exercise (hypoxic, 5 mM K^+, pH 7.5, 5 nM adrenaline)*
5. strenuous exercise with adrenergic stimulation (hypoxic, 5 mM K^+, pH 7.5, 500 nM adrenaline)*

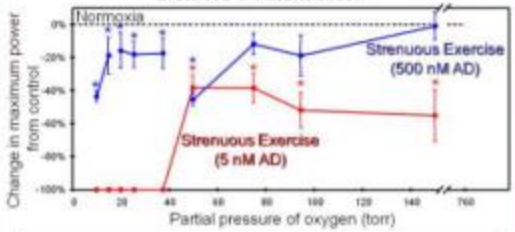
*For experiments conducted below hypoxic thresholds hearts were not exposed to lethal steps

Maximum cardiac performance under hypoxia



Hypoxia & Hypoxic Recovery: Maximum power of perfused rainbow trout hearts was assessed under normoxic conditions (150 torr O_2), specific levels of hypoxia (indicated on the x axis), and then again under normoxic conditions (recovery). Each P_{O_2} value indicates a separate group of hearts (N=4-10). Values plotted are change from control ± SEM. *denotes significant differences from control (repeated measures ANOVA P<0.05).

Maximum cardiac performance under strenuous exercise conditions



Hypoxia, hyperkalemia and acidosis: Following recovery (see above), maximum power of perfused rainbow trout hearts was assessed under specific levels of hypoxia (indicated on the x axis), in conjunction with hyperkalemia (5 mM KCl) and acidosis (pH 7.5) to simulate strenuous exercise conditions, first with tonic adrenergic stimulation (5 nM) and then with maximal stimulation (500 nM). Each P_{O_2} value indicates a separate group of hearts (N=4-10). At P_{O_2} levels ≤ 37 torr hearts were not exposed to the hypoxic, hyperkalemic, acidotic saline with tonic adrenergic stimulation. In addition, at P_{O_2} levels ≤ 15 torr hearts did not receive prior exposure to hypoxia alone. Values are plotted as change from control ± SEM. *denotes significant differences from control (repeated measures ANOVA P<0.05).

Results & Conclusions

Hypoxia Alone

- Exposure to hypoxic perfusate ≤ 50 torr resulted in significant reductions in maximum cardiac performance
- In addition, full recovery upon return to normoxic conditions was not seen
- Exposure to hypoxic perfusate ≤ 20 torr was lethal thus the threshold for cardiac collapse under hypoxia occurred between 15-20 torr

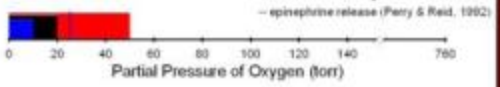
Hypoxia, Hyperkalemia & Acidosis

- With tonic levels of adrenergic stimulation maximum performance during simulated strenuous exercise conditions was significantly decreased
- The threshold for cardiac collapse under the above conditions was between 37-50 torr as exposure to perfusates ≤ 50 torr was lethal

Rescue With Adrenergic Stimulation

- Maximum adrenergic stimulation restored cardiac performance in hearts previously exposed to strenuous exercise conditions when P_{O_2} ≥ 75 torr
- Maximal adrenergic stimulation protected cardiac performance during exposure where P_{O_2} ≤ 37 torr, conditions that would otherwise be lethal
- However, even with adrenergic stimulation maximum performance was significantly decreased from that observed during normoxia

Thresholds for cardiac collapse



The right endpoint indicates the hypoxic level below which irreversible cardiac arrest was observed after ≥ 5 minutes of exposure

Take Home Message

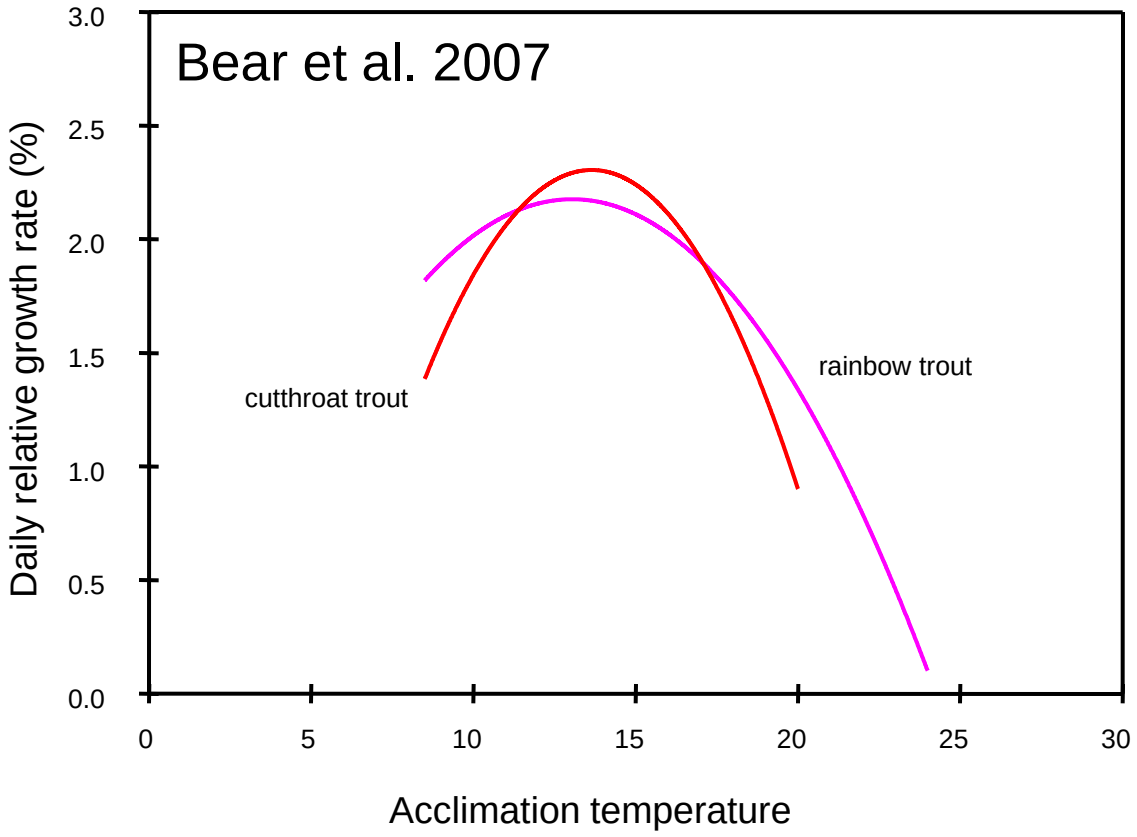
Under conditions simulating strenuous exercise, adrenergic stimulation plays a critical role in maintaining cardiac performance, raising the threshold for cardiac collapse to hypoxic levels similar to those seen in vivo.

References

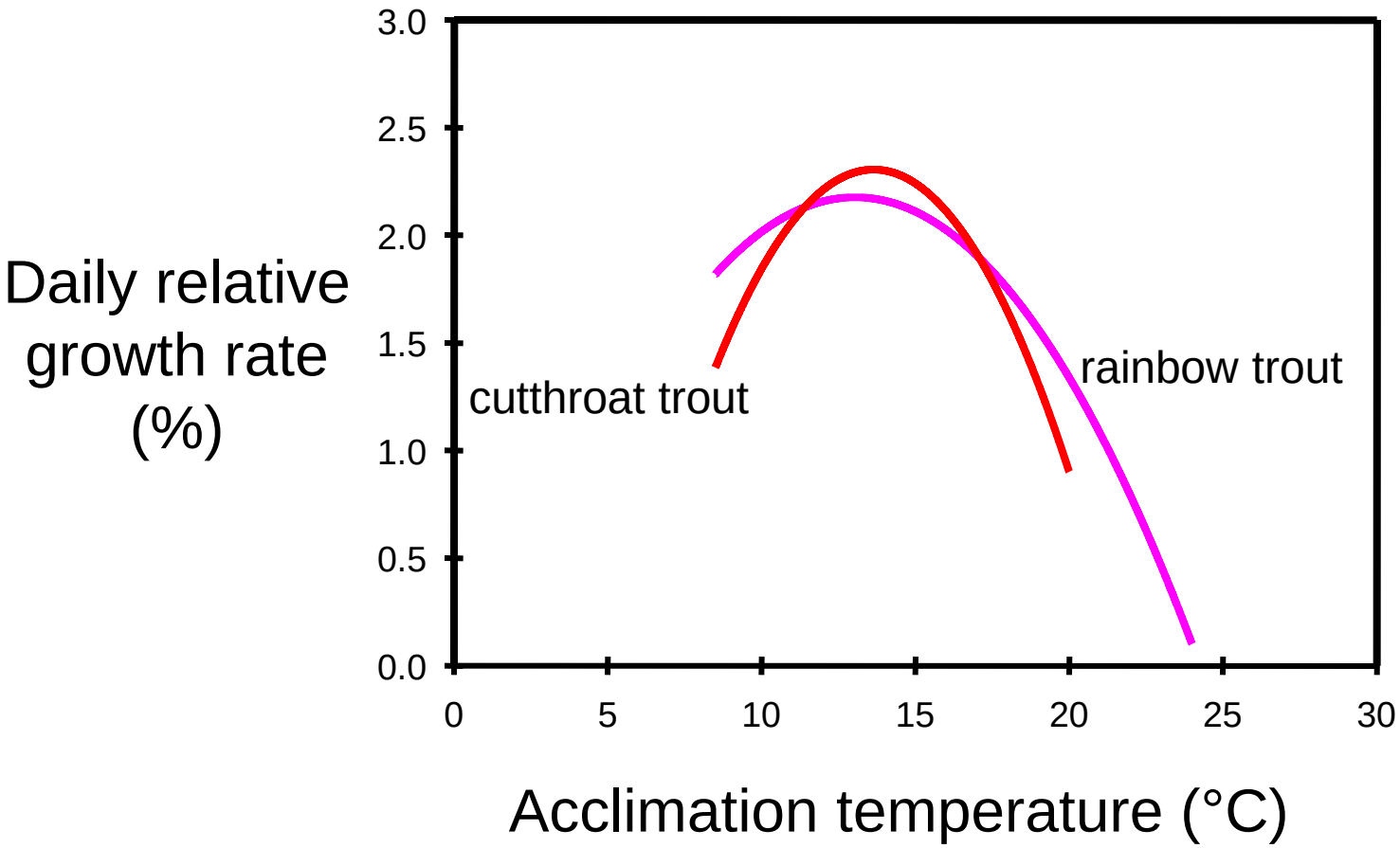
Farrell AP, MacLeod KR, Chauvey B (1986) Intrinsic mechanical properties of the perfused rainbow trout heart and the effects of catecholamines and extracellular calcium under control and acidotic conditions. J Exp Biol 125:319-345

Perry SF & Reid SD (1992) Relationship between blood O2 content and catecholamine levels during hypoxemia in rainbow trout and American eel. Am J Physiol 263: R240-249

Bad graph versus better graph



Bad graph versus better graph



Bear et al. 2007

Examples for discussion 1 / 6

A COMPARISON OF MYOCARDIAL β-ADRENORECEPTOR DENSITY AND LIGAND BINDING AFFINITY AMONG SELECTED TROPICAL FISHES

Linda M. Hanson[1], Yuen K. Ip[2] and Anthony P. Farrell[1]

[1]Department of Biological Sciences, Simon Fraser University, Burnaby, British Columbia, Canada

[2]Department of Biological Sciences, National University of Singapore, Republic of Singapore

Introduction

Purpose: To examine interspecific variation in myocardial β-adrenoreceptor density (B_{max}) and binding affinity (K_d) for ventricular tissue in 7 previously unstudied species of tropical fish.

- In fish, the β-adrenoreceptor (β-AR) signaling pathway mediates the cardiac actions of adrenaline mainly via receptors of the β_2 subtype.
- Temperature acclimation alters the heart's response to adrenaline and this change has partially been attributed to a temperature-dependent change in cell surface β-AR density.
- Olsson et al. (2000) suggested that B_{max} and K_d differed intrinsically among species adapted to different temperatures.

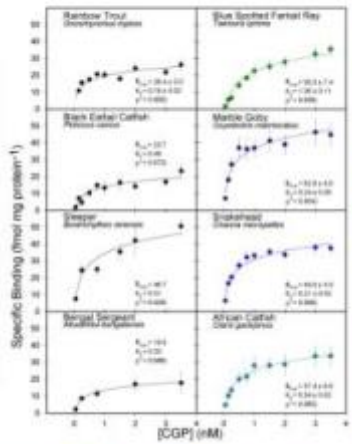
Methods

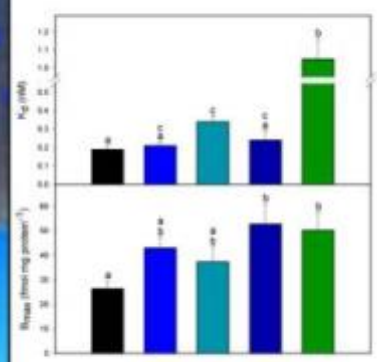
- B_{max} and K_d were determined for ventricular punches using a tritiated ligand technique (Watson-Wright et al., 1989; Gamperl et al., 1994).
- Ventricular tissue punches were incubated with the hydrophilic β_2-adrenoreceptor ligand [^{3}H] CGP-12177.
- The mass of individual ventricles determined the degree of replication for binding assays.
- Binding curves were replicated up to 6 times (N).
- Binding parameters were determined using a Scatchard plot as described by Zivin and Waud (1982).
- The control group (rainbow trout) was compared separately with the tropical elasmobranchs, the tropical freshwater teleosts and the tropical saltwater teleosts.

Results

- Our results for rainbow trout compare favorably with previous studies done at similar temperatures.
- B_{max} values ranged from 19.5 to 52.8 ± 8.0 fmol mg protein^{-1}.
- The highest B_{max} values were observed in marble goby, blue spotted fantail ray, sleeper, and snakehead.
- B_{max} was significantly higher than rainbow trout (P<0.05) in both blue spotted fantail ray and marble goby.
- Ligand binding affinity (K_d) varied from 0.19 ± 0 .02 to 1.05 ± 0.11 nM.
- K_d values for blue spotted fantail ray and African catfish were both significantly higher than rainbow trout (P<0.05).

Rainbow trout (N=6), blue spotted fantail ray (N=6), black eeltail catfish (N=1, 10 hearts pooled), marble goby (N=6, 19 hearts), sleeper (N=1, 10 hearts), snakehead (N=6, 17 hearts), Bengal sergeant (N=1, 10 hearts) and African catfish (N=6, 15 hearts). The B_{max} (fmol mg protein^{-1}), K_d (nM) and r^2 values for each graph are indicated. Values are shown as $\bar{x}$ ± SEM.





[^{3}H]CGP-12177 dissociation constant (K_d) and β-adrenoreceptor density (B_{max}). Values are $\bar{x}$ ± SEM. Dissimilar letters denote significant differences at P<0.05. N=6 for all species.

Conclusions

Tropical Marine Elasmobranchs

- Blue spotted fantail ray is the first elasmobranch in which β-AR density and binding affinity have been characterized.
- B_{max} was significantly higher than rainbow trout (P<0.05).
- K_d (1.05 nM) was double that observed in any other species (0.48 nM).
- The significantly lower binding affinity observed may be due to variation in β-AR subtypes between teleosts and elasmobranchs.

Tropical Freshwater Teleosts

- β-AR density values tended to be higher in tropical freshwater teleosts when compared with temperate rainbow trout.
- The difference was statistically significant for marble goby (P<0.05).
- High variation within species meant differences for other tropical freshwater teleosts did not reach statistical significance.
- K_d of African catfish was significantly different from rainbow trout.

Tropical Marine Teleosts

- Neither B_{max} nor K_d differed significantly from rainbow trout.

Discussion

- The present results suggest that B_{max} is higher in freshwater, but not marine, tropical species.
- However, Olsson et al. (2000) reported high B_{max} values for both marine tropical species (mahimahi = 46.9, skipjack tuna = 41.3) and marine temperate species (sockeye salmon = 47.5).
- No clear phylogenetic or environmental pattern of β–AR values is evident.

Future Research

- A comparison of β–AR density values between both temperate and tropical saltwater and freshwater teleosts within family groups.
- Studies of β–AR density and binding affinity in elasmobranchs.
- Characterization of β-AR subtypes in elasmobranchs.

References

Gamperl AK, Wilkinson M, Boutilier RG. (1994) β-Adrenoreceptors in the trout (*Oncorhynchus mykiss*) heart: characterization, quantification and effects of repeated catecholamine exposure. Gen Comp Endocrinol 95:259-272

Olsson H, Yee N, Shiels HA, Brauner C, Farrell AP. (2000) A comparison of myocardial β-adrenoreceptor density and ligand binding affinity among selected teleost fishes. J Comp Physiol B 170:545-550

Watson-Wright WM, Arnour JA, Johnstone DE, Wilkinson M. (1989) Myocardial slice: a physiological approach to β-adrenergic ([^{3}H]CGP-12177) receptor binding in hamster and guinea heart. J Pharmacol Methods 22:37-47

Zivin JA and Waud DR. (1982) How to analyze binding, enzyme and uptake data: the simplest case, a single phase. Life Sci 30:1491-1422

Hepatic Portal Vein Cannulation Technique in Fish

Erika J. Eliason[1], Anders Kiessling[2], Anders Karlsson[2], Brankica Djordjevic[2], A.P. (Tony) Farrell[1,3]
[1]Department of Zoology, University of British Columbia, Canada, [2]Department of Agricultural and Aquacultural Sciences, Norwegian University of Life Sciences, Ås, Norway, [3]Faculty of Land and Food Systems, University of British Columbia, Canada
Email: eeliason@interchange.ubc.ca

Introduction

Non-stressful sampling of blood from the hepatic portal vein (HPV) in fish has tremendous value for both nutritionists and physiologists. Furthermore, the combination dorsal aorta (DA) and HPV cannulation techniques enables the examination of nutrient uptake and hepatic metabolic transformation, as well as systemic physiological changes associated with gut function (such as acid-base balance and ion and osmotic regulation) in greater detail and with greater precision than previously possible.

Objectives

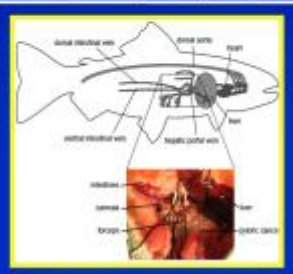
#1 - Evaluate a chronic hepatic portal vein cannulation technique in Atlantic salmon (*Salmo salar*. L)

#2 - Measure the plasma amino acid profiles of blood simultaneously sampled from the hepatic portal vein and dorsal aorta following a meal in rainbow trout (*Oncorhynchus mykiss*)

HPV Cannulation Method

1. Prepare for surgery

- Cannula: PE 50 tubing with a bubble, fitted with a 2-3 cm silastic tubing tip, cut at a 45° angle
- Fish (600-1200 g) are initially anesthetized in buffered 0.1 g l^{-1} MS-222 and maintained on the surgery bubble using gill irrigation with chilled, aerated, buffered 0.05 g l^{-1} MS-222



2. Make an incision into the body cavity

- Location: just posterior to the pectoral fin
- Cut the skin with a scalpel and extend the cut through the muscle of the body wall with Mayo scissors
- Use cotton swabs to carefully retract pyloric caeca to locate a branch of the ventral or dorsal intestinal vein

3. Isolate a branch of the intestinal vein

- Use fine curved forceps to isolate a 0.75 cm section of the desired vessel, about 5 cm from the main HPV
- Slip 2 pieces of 2-0 silk around the vessel, tightening the posterior thread to occlude the vessel
- Cut into the vessel at a 45° angle (V-shaped) between the 2 pieces of silk using microscissors

4. Insert the cannula and close the wound

- Advance the cannula into the vessel, towards into the hepatic portal vein
- Tighten the silk threads around either side of the bubble
- Flush and fill the cannula with heparinized saline (150 IU ml^{-1}) and close the wound with interrupted sutures (2-0 silk) every 4-5 mm

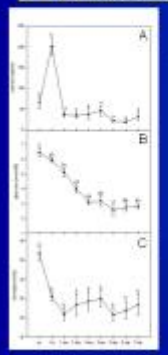
5. Return the fish to the experimental tank

- Total surgery time should be ~45 min, and the fish should fully recover from the anesthesia within 5 min
- Allow fish to freely swim in the experimental tanks fitted with a shelter, 5 cm above the water surface

Study #1: Monitor the fish's recovery from surgery in the experimental tank by measuring blood variables for 7 days in unfed fish
Study #2: Allow rainbow trout to recover for 1 day following the HPV and DA cannulations before force-feeding them a meal of 1% of their body mass. Follow changes in DA and HPV blood samples 0,3,6,12,24 & 48 h postprandial

Results

Fig. 1: Assessing the HPV Technique



Baseline levels for plasma cortisol (19-45 ng ml^{-1}), plasma glucose (2.6-3.2 mmol l^{-1}) and Hct (21-26%) were all within the normal range for healthy, unstressed fish

1A. Plasma Cortisol

- Plasma cortisol returned to a baseline level by 24 h

1B. Plasma Glucose

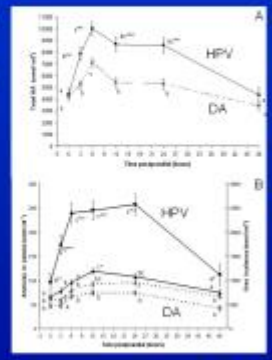
- Plasma glucose steadily declined to a stable baseline by day 3

1C. Hematocrit

- Hematocrit steadily declined to a stable baseline level by 24 h

Suggests that internal hemorrhaging and stress were not serious problems

Fig. 2: Postprandial plasma amino acid profiles



2A. Total Plasma Amino Acids

- Postprandial peaks in plasma amino acids in both the DA (open squares) and HPV (closed squares) lasted from 3 h to beyond 24 h, but had returned to baseline levels by 48 h
- Total plasma amino acids were always significantly higher in the HPV than the DA, suggesting either dilution or hepatic biotransformation occurred during the first pass of blood through the liver
- DA and HPV differences were not always maintained for single amino acids (e.g. valine, cysteine, tryptophan) providing definitive evidence of hepatic biotransformation

2B. Total Plasma Ammonia and Urea

- Postprandial peaks in plasma ammonia lasted from 3 h in the HPV (black squares) and 6 h in the DA (white squares) and returned to baseline by 48 h
- Postprandial plasma urea levels peaked in both the HPV (black triangles) and DA (white triangles) by 6 h and returned to baseline levels by 48 h
- Differences in plasma ammonia levels in the HPV compared to the DA indicate intestinal catabolism of free amino acids

For more information, please see Eliason et al. (2007) PNAS, in press and Karlsson et al. (2006) [illegible]

N values = 12, statistically significant differences (p<0.05) over time are indicated by differing letters. A statistically significant difference (p<0.05) between the hepatic portal vein and dorsal aorta is indicated by an asterisk.

Conclusions

Successful HPV cannulation technique – Key blood variables returned to baseline levels within 3 days post-surgery
Hepatic metabolism – Some amino acids undergo hepatic transformation during their first pass through the liver

Funding from the Natural Sciences and Engineering Research Council of Canada (NSERC), the Norwegian Research Council Center of Excellence, Aquaculture Protein Centre (APC) and the Department of Animal and Aquacultural Sciences, Norwegian University of Life Sciences, Norway

Cardiovascular and respiratory responses to hypoxia in bar-headed geese (*Anser indicus*) and barnacle geese (*Branta leucopsis*)

Sabine L. Lague[1,*], Beverly Chua[1], Anthony P. Farrell[1], Yuxiang Wang[2], and William K. Milsom[1]
[1] Department of Zoology, University of British Columbia, Vancouver, Canada, [2] Department of Biology, Queen's University, Kingston, Canada
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Introduction

Avian Flight: Rest (Resting $\dot{V}O_2$) → Flight (10-20 fold ↑ in $\dot{V}O_2$ (1)) → Flight at high altitude

Challenges:
1. ↓ Air density (↑ O_2 demand; 2)
2. ↓ PO_2 (↓ O_2 drive)

∴ High altitude flight = potential mismatch between O_2 supply and O_2 demand; however, despite these barriers **bar-headed geese** routinely migrate at >6000 m (3), where the inspired PO_2 is <50% of sea level (4).

Enhanced O_2 Transport Cascade

High Flier (e.g. bar-headed goose)

- Ventilation — More effective breathing pattern; Enhanced hypoxic ventilatory response (HVR)
- Pulmonary O_2 diffusion
- Circulatory O_2 delivery — Larger hearts; ↑ Hemoglobin O_2 affinity
- Muscle O_2 diffusion
- Muscle O_2 utilization
- Muscle ATP turnover

Ventilation and Circulation: both are convective steps and can be immediately regulated, thus likely playing an important role in response to hypoxia.

Hypoxic cardiovascular response (HCVR): Cardiovascular changes that accompany hypoxia exposure and regulate tissue O_2 delivery. Characteristic: Unknown.

Hypoxic ventilatory response (HVR): Ventilatory changes that accompany hypoxia exposure and ensure blood-O_2 loading. Characteristic: ↑ total volume in bar-headed geese.

Research Question: How do the HCVR and HVR vary across species and altitude?

Study Groups

Study Group	Altitude (m)	F_IO_2
High altitude bar-headed geese (wild and captive)	4000	0.07
	1000	0.09
	1200	0.12
Sea level bar-headed geese and barnacle geese		0.21

Methods

- **Study Animals:** N=5-7 adult birds from each of study group
- **Variables:** see below: respiratory, metabolic, and cardiovascular
- **Surgery:** cannulation of right brachial artery and vein
- **Protocol:** each bird was exposed at rest to stepwise decreases in F_IO_2 of 25-min each (diagram below: normoxia in purple; hypoxia in blue)
- **Blood Samples:** at 15-min in all exposures except recovery (5, 25-min)

Control | Hypoxia | Recovery

N_2 | Air | Pneumotach | F_IO_2 Analyzer | O_2 Analyzer | Arterial Blood | Venous Blood

Results

Question 1: Do the HVR and HCVR differ between bar-headed and barnacle geese at low altitude? → YES

Question 2: Is there plasticity in the HCVR and HVR between bar-headed geese at high and low altitude? → YES

Q.1 Q.2

A. Blood O_2 saturation: High altitude > Low altitude > Barnacle Geese; bar-headed geese > bar-headed geese

B. HVR - Means of increasing $\dot{V}_{TOT}$: We confirmed that bar-headed geese predominantly ↑ $\dot{V}_{TOT}$ by ↑ V_T (5), whereas barnacle geese ↑ $\dot{V}_{TOT}$ by ↑ f_R like other lowland geese.

C. HCVR - Means of increasing Q: All groups ↑ Q primarily by ↑ V_T, with minimal contribution of f_H.

Q.1

A.1 HCVR Recovery: Despite having similar HCVRs, f_H is elevated in barnacle geese during recovery. Thus, they were less able to meet O_2 demand.

B.1 Blood Acid-Base: During hypoxia both species exhibit respiratory alkalosis. Barnacle geese are likely reliant on anaerobic metabolism during 0.09 and 0.07 F_IO_2, and do not fully recover their pH_a.

C.1 HCVR $\dot{V}O_2$ relationships: $\dot{V}O_2$ is linearly related to f_H and O_2 pulse in barnacle geese, and only to O_2 pulse in bar-headed geese.

Q.2

A.2 HVR sensitivity: $\dot{V}_{TOT}$ in bar-headed geese at high altitude is less sensitive than at low altitude. It increases at a lower P_IO_2.

B.2 HCVR sensitivity: Q in bar-headed geese at high altitude is less sensitive than at low altitude. It increases at a lower P_IO_2.

C.2 f_H sensitivity: High altitude bar-headed geese increase f_H only at very low P_IO_2 values after V_T was already maximized.

Summary

Legend: Hypoxic sensitivity; Ability to meet O_2 supply and demand

Hypoxic ventilatory response (HVR): Barnacle geese > Low altitude bar-headed geese > High altitude bar-headed geese

Hypoxic cardiovascular response (HCVR): Barnacle geese = Low altitude bar-headed geese > High altitude bar-headed geese

Research Significance

Comprehensive study of the HCVR and HVR in bar-headed and barnacle geese

Bar-headed geese are better able to meet O_2 supply and demand during hypoxia than barnacle geese.

Altitude matters! The HCVR and HVR are plastic and less sensitive in high altitude bar-headed geese.

This is significant because most current physiological research is from geese raised at low altitude.

Geese have an incredible capacity to increase stroke volume to facilitate perfusion increases during hypoxia. Waterfowl research (much of it completed during exercise – not hypoxia) has generally concluded that heart rate (rather than stroke volume) is the primary contributor to cardiac output.

References

(1) Butler et al. (1977). J. Exp. Biol. 71: 7-26. (2) Altshuler et al. (2004). PNAS 101: 17731-17736. (3) Hawkes et al. (2011). PNAS 108:9516-9519. (4) Dejours (1975). P. of Comp. Resp. Physiol. Elsevier. (5) Scott (2011). J. Exp. Biol. 214: 2455-2462.

Acknowledgements and Funding

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Photo credit: Samasekharam Sandeep.

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HIV-1 Tat Protein Induces Downregulation of CD127 Transcripts in CD8 T-Cells

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1) Ottawa Health Research Institute, 2) University of Ottawa - Department of Biochemistry, Microbiology and Immunology, 3) The Ottawa Hospital - Department of Infectious Diseases

Summary

• We have recently established that HIV-1 Tat protein (Tat) causes a specific downregulation of Interleukin-7 receptor-alpha (CD127) on CD8 T-cells.

•This downregulation is both time and dose dependent

•Tat has previously been shown to downregulate IL-2 gene expression in Jurkat cells through alterations in the AP1 complex.

• Since Tat is known to effect the transcriptional regulation of other cellular genes, we hypothesize that this down regulation by Tat occurs at the level of transcription initiation within the CD127 gene promoter

• Addition of purified Tat protein to CD8 T-cells induced a significant decrease in the level of CD127 mRNA. The majority of CD8 T-cells cultured in media alone remained $CD127^{hi}$ over 24 hours and contained high levels of CD127 transcripts. In contrast, the bulk of the CD8 T-cells cultured in the presence of Tat shifted to $CD127^{lo}$ and demonstrated a 6-fold decrease in CD127 mRNA ($p < 0.05$).

• To determine if Tat affected CD127 transcript stability, mRNA levels were measured in the presence and absence of Tat in cells transcriptionally arrested with Actinomycin D. In CD8 T-cells treated with Actinomycin D (5mg/ml) or Actinomycin D plus Tat (10 mg/ml) for 12 and 24 hours, equivalent levels of CD127 mRNA were found indicating Tat does not enhance CD127 mRNA degradation.

• Future work will include a mutational analysis of the putative human CD127 promoter examining transcriptional activity in the presence and absence of Tat protein.

Background

• CD8 T-Cells are required for recognition and control of viral replication during infection

• CD8 T-Cell functions are impaired during HIV infection. Although viral specific T-Cells persist in blood, they do not appear to respond to antigen or show cytolytic function.

• Interleukin-7 (IL-7) is essential for CD8 T-Cell proliferation and function.

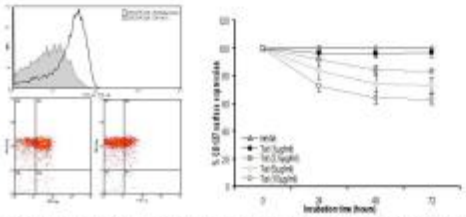
• The IL-7 receptor is composed of two chains, a unique alpha chain (CD127) and a common gamma chain (CD132) that is shared among IL-2, IL-4, IL-7 IL-9, IL-15 and IL-21 receptors

• We have previously shown that during active HIV replication, levels of surface CD127 on CD8 T-cells are significantly lower when compared to healthy controls.

• We have also shown that the HIV-1 Tat (Tat) protein specifically downregulates CD127 surface expression on CD8 T-cells.

•This effect is specific to (CD127) and is both time and dose dependent

•Downregulation of CD127 by Tat leads to impaired CD8 T-cell proliferation and cytolytic capacity.



HIV Tat protein downregulates CD127 expression on CD8 T-cells in a time and Dose dependent fashion.

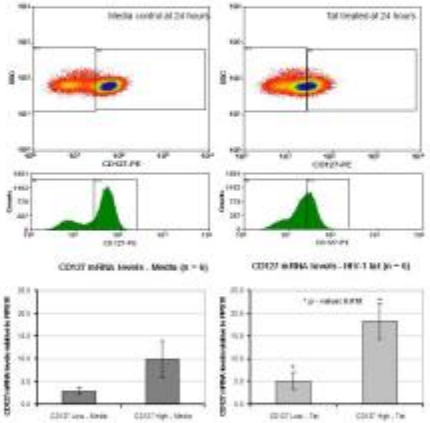
Hypothesis

Downregulation of CD127 expression on CD8 T-cells by Tat occurs at the level of transcription initiation.

Results

Does Tat Decrease the level of CD127 mRNA transcripts in CD8 T-Cells?

• To determine if Tat causes a decrease in CD127 transcripts, cells were treated with or without Tat (10 µg/mL) and after 24 hours were sorted by FACS into $CD127^{hi}$ and $CD127^{lo}$ populations.

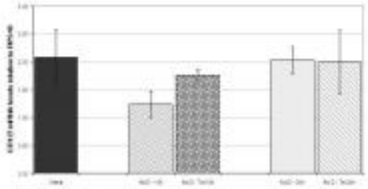


The majority of CD8 T-cells cultured in media alone remained $CD127^{hi}$ over 24 hours and contained high levels of CD127 transcripts.

The bulk of the CD8 T-cell population cultured in the presence of Tat shifted to $CD127^{lo}$ and demonstrated a 6-fold decrease in CD127 mRNA ($p < 0.05$).

Does Tat induce Transcript Degradation?

• CD8 T-Cells were transcriptionally arrested with Actinomycin D (5mg/mL) in the presence and absence of Tat. Following incubation for 12 hours (n=2) and 24 hours (n=4), CD127 transcript levels were analyzed using quantitative PCR (normalized to RPS18).



• Cells were treated with Actinomycin D (5 mg/ml) or Actinomycin D plus Tat (10 µg/ml) for 12 and 24 hours.

•This suggests that Tat does not enhance CD127 mRNA degradation.

Conclusions

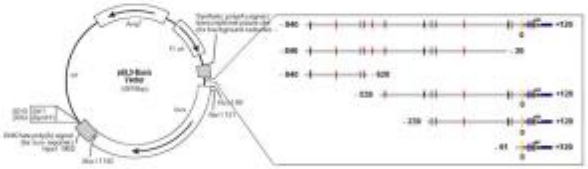
• **Tat induces a decrease in the rate of CD127 gene transcription in CD8 T-cells resulting in a shift from $CD127^{hi}$ surface expression to $CD127^{lo}$.**

• **This decrease is not due to cell death or mRNA degradation.**

Future Work

• Putative Transcriptional Factor Binding sites within the CD127 Promoter and potential enhancer region.

• The CD127 Promoter region (1.1 kb) and five truncation mutants have been cloned upstream of the luciferase reporter gene.



Methods

CD8+ T-Cell Isolation:

CD8 T-cells from healthy HIV seronegative volunteers (n=6) were isolated using the AutoMACS Microbead CD8+ Isolation System. The cells were allowed to recover overnight in RPMI-1640 with 20% FCS.

Transcript Studies:

The cells were incubated either in medium alone or in the presence of purified Tat protein (10 µg/ml) for 12 or 24 hours. The cells were then sorted by FACS into $CD127^{hi}$ and $CD127^{lo}$ populations. Total RNA was harvested

Degradation Studies:

CD8 T-Cells were transcriptionally arrested by pre-treatment for 2h with Actinomycin D (5 mg/mL). Cells were then treated with purified Tat protein (10 µg/mL) for 12 or 24 hours when total RNA was harvested.

CD127 Transcript Quantitation:

CD127 transcripts were measured using real-time PCR and normalized to the expression of the RPS18 reference gene

DIFFERENTIAL EXPRESSION OF SEMAPHORIN-4F IN AXOTOMIZED CNS VERSUS PNS NEURONS

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1. ICORD (International Collaboration on Repair Discoveries), 2. Dept. of Zoology, 3. Dept. of Surgery, 4. Dept. of Anatomy, University of British Columbia, Vancouver, BC, Canada, 5. Sumitomo Pharmaceuticals Co., Osaka, Japan.

Introduction:

While peripheral nervous system (PNS) neurons are able to regenerate their axons after injury, central nervous system (CNS) neurons typically fail to do so. Studies suggest that one contributing factor to the inability of CNS neurons to regenerate is the presence of inhibitory members of the Semaphorin family of guidance molecules. However, the function of many Semaphorins in the nervous system is still unknown.

Here, we compared the expression of transmembrane Semaphorin-4F (Sema-4F) in the non-regenerating neurons of the red nucleus following cervical spinal cord lesion (CNS injury), versus the regenerating neurons of the facial nucleus after transection of the facial nerve (PNS injury). Using *in situ* hybridization, immunohistochemistry and western blotting, we found an increase in Sema-4F expression in rat facial motoneurons several days following axotomy, and this upregulation was maintained for several weeks. In contrast, while rubrospinal neurons show no change in Sema-4F mRNA levels following axotomy, Sema-4F protein levels may be down-regulated. We propose that neuronal expression of Sema-4F following axotomy may in fact be beneficial to regenerating neurons.

Objective:

To compare the level of Semaphorin-4F (Sema-4F) mRNA and protein expression, following either:

(1) PNS Injury: Transection of the Facial Nerve to axotomize the **Facial Motor Neurons- FMNs** (a model of regenerating neurons).

(2) CNS Injury: A lateral hemi-section of the cervical spinal cord to axotomize the **Rubrospinal Neurons – RSNs** (a model of non-regenerating neurons).

Materials and Methods:

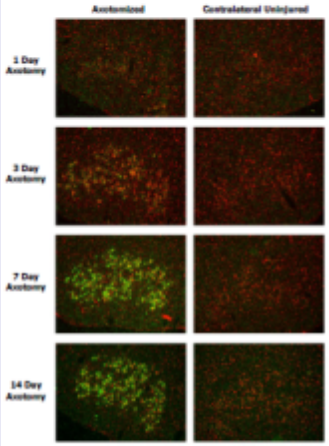
Surgical Procedures:
For all experiments, adult male Sprague-Daly rats (200-250 grams) were used. All animals were anaesthetized i.p. with ketamine (70 mg/kg) and xylazine (10 mg/kg) and killed with chloral hydrate (100 mg/kg i.p.). **Rubrospinal Tract Model of CNS injury:** The spinal cord was exposed at the cervical level (C3/C4) and a lateral hemi-section performed. Animals were killed at 3, 7, and 14 days post-injury. **Facial Nerve Model of PNS injury:** The branches of the left facial nerve was exposed unilaterally close to its point of of exit at the stylomastoid foramen and all branches were axotomized by the re-sectioning a small portion of the nerve to prevent regeneration. Animals were killed at 1, 3, 7, and 14 days post-injury.

Immunohistochemistry:
Sema-4F expressing motoneurons were visualized with Sema-4F antibodies (Sumitomo Pharmaceuticals) coupled to a biotinylated secondary antibody (1:250 Jackson Immuno). Signal was further amplified with a Tyramide Signal Amplification kit (TSA, Perkin-Elmer). Fluorescent staining of Nissl was used as counterstain.

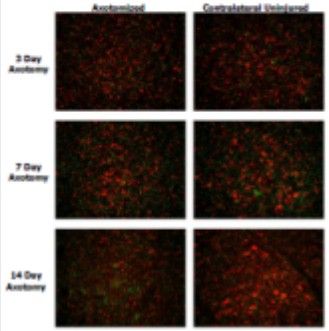
***In Situ* Hybridization:**
Sema-4F mRNA expression was detected using radioactively labeled (^{35}P), 50-mer oligonucleotide probes for Rat Sema-4F (AB002563). Following hybridization, facial sections were left for 4 weeks, and rubrospinal sections for 10 weeks, prior to slide development. Ethidium Bromide (0.01%) was used as a counterstain to visualize neurons.

Western Blot Analysis:
Protein from Facial (n=8) and Red (n=6) nuclei was extracted, separated by SDS-PAGE (7.5%) and transferred to a PVDF membrane (Immobilon-P, Millipore). Membranes were incubated with Sema-4F antibody (1:1000); bound to a peroxidase conjugated secondary antibody (Jackson Laboratories), and protein visualized with ECL substrate (Amersham). Membranes were stripped re-probed with an Actin antibody (1:1000, ICN) as a loading control.

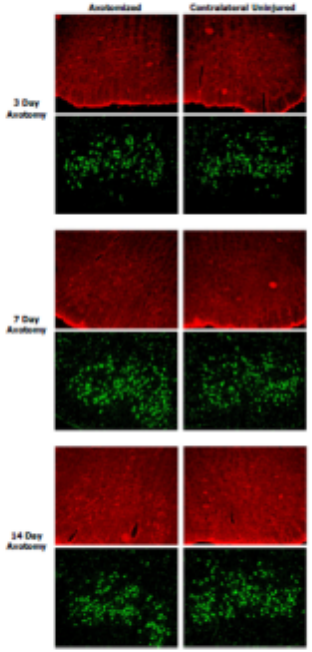
Sema-4F mRNA signal is increased in axotomized FMNs



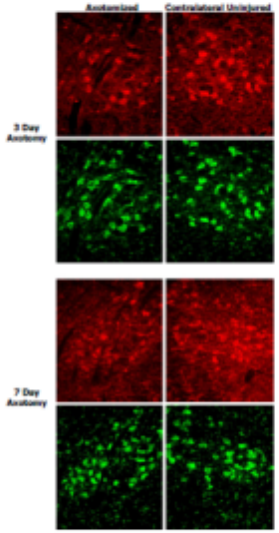
Sema-4F mRNA signal is NOT increased in axotomized RSNs



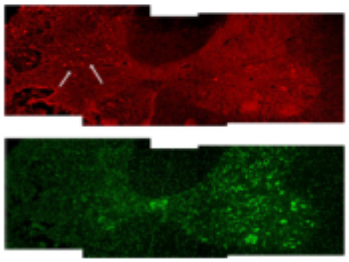
Sema-4F immunoreactivity is increased in axotomized FMNs



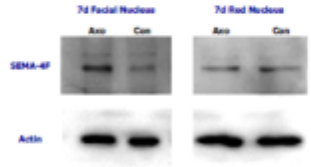
Sema-4F immunoreactivity may decline in axotomized RSNs



Sema-4F immunoreactivity is seen adjacent to the spinal cord injury site 7 days post-injury



Sema-4F Western Blot



Summary and Conclusions:

(1) Following injury, axotomized Facial Motoneurons upregulate both Sema-4F mRNA and protein.

(2) In contrast, while axotomized Rubrospinal Neurons show no change in Sema-4F mRNA levels, Sema-4F protein levels may be down-regulated following injury.

(3) Cells present in the spinal cord gray matter, adjacent to the spinal cord injury site, express Sema-4F.

Conclusion:

The observation that Sema-4F is upregulated after a PNS, but not a CNS, injury, may suggest that Sema-4F plays a beneficial role in regenerating neurons.

Acknowledgements:

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CHRISTOPHER REEVE PARALYSIS FOUNDATION

Fisheries and Oceans Canada | Pêches et Océans Canada

DETECTION OF *Kudoa thrysites* DNA FROM SEAWATER USING GRAVITY-FLOW FILTRATION AND qPCR

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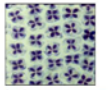
Introduction

The myxosporean, *Kudoa thrysites* is endemic to the marine environment in the Pacific Northwest of North America. Infection of farmed Atlantic salmon muscle is of concern to aquaculturists whereby loses due to this parasite are upwards of $50 million annually[1]. Infected muscle contains plasmodia filled with *Kudoa* myxospores while actinospores are found in seawater. Infection is characterised by host post-mortem myoliquefaction or 'soft flesh syndrome' caused by a protease that digests muscle fibres resulting in compromised quality of the fish product.

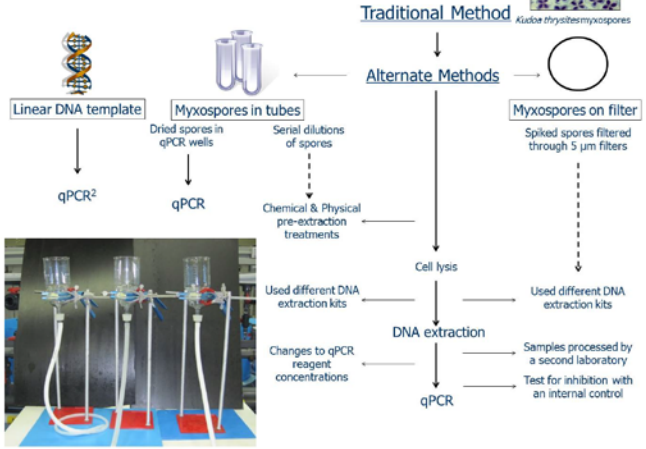
To attempt to detect the parasite in seawater, our objectives were to:

- Develop a practical, field-friendly filtration protocol to trap actinospores for quantification by qPCR.
- Using linear DNA and isolated myxospores, optimize and develop qPCR standard curves for 3 different conditions in order to 1, assess qPCR efficiency; 2, establish thresholds for sensitivity of this assay for use in interpreting raw seawater sample results.
- assess minimum filtration volumes for *Kudoa* detection & diurnal trends in our local waters.

Materials & Methods

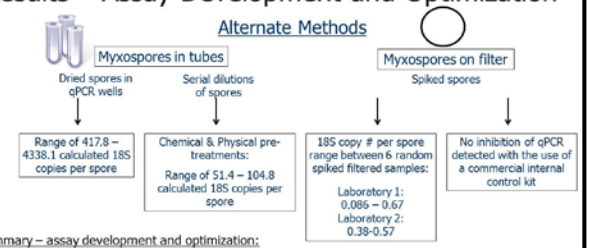

Kudoa thrysites myxospores

DNA extraction and qPCR assay development & optimization:



Gravity filtration unit

Results – Assay Development and Optimization



Results Summary – assay development and optimization:

- Chemical & physical pre-treatments yielded no increase in range of copies of 18S per spore.
- No significant differences between samples run with changes in concentrations of probe or primers in the qPCR reaction
- No significant differences between commercial extraction kits when applied to spores in tubes or myxospores on filters

Results – Assay Application

Relationship between Ct and 18S copy #:

# spores in sample	Ct	18S copies/spore
Spores in tube		
1	41.18	19.8
10,000	25.24	142.25
Spores on filter		
10	37.79	0.214
2500	32.93	2.8

Examples of high and low number spore Ct values in relation to calculated 18S copies per spore.

Minimum water volume to detect *K. thrysites*:

Volume filtered (L)	Average Ct (StDev)	Range of 18S copy # (n=12)
12.5	36.73 (0.73)	18.8-686.5
2.5	37.28 (0.13)	41.4-60.7
0.5	37.58 (0)	One value of 35.4

Volume of filtered raw seawater from Departure Bay, Nanaimo, B.C. to determine a minimum volume required for *Kudoa* detection.

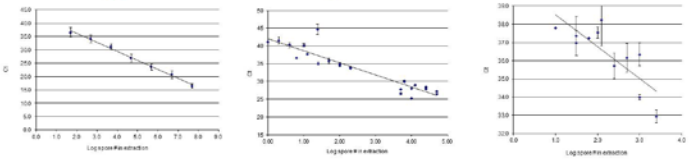
Sequencing of raw SW samples:

Volume filtered (L)	Average Ct (Standard deviation; n=4)	BLAST – best alignment (Accession number; n=1)
12.5	36.91 (1.05)	*Kudoa thrysites* (AF031412.1)
12.5	36.18 (0.78)	*Kudoa thrysites* (AF031412.1)
12.5	37.01 (0.22)	*Kudoa thrysites* (AF031412.1)

Sequencing results from raw seawater (SW) samples to develop a confidence threshold where high Ct's are determined to be a result of *Kudoa* 18S amplification.

Results – Assay Development and Optimization

Traditional Method



R^2 = 0.99; Efficiency = 97.4% | R^2 = 0.87; Efficiency = 97.1% | R^2 = 0.64; Efficiency = 273.7%

Conclusions & Future Directions

- Dried spores in qPCR wells resulted in the highest calculated values of 18S copy number per spore
- Relative to dried sores, spores in tubes and spores on filters resulted in a minimum range of 8-40 & 4000-6000 fold loss of signal, respectively.
- The reason for the loss of qPCR sensitivity is unknown.

- Increase reproducibility for each graph type in order to establish a more clear framework to interpret field samples and refine the relationship between Ct and 18S copy #.
- Further establish Ct threshold for detection of *Kudoa thrysities* by sequencing targeted raw SW samples.

- Once a clear interpretive framework is established, process diurnal and fish farm samples

Acknowledgements

Funds were provided by the Aquaculture Collaborative Research and Development Program (ACRDP) and Marine Harvest Canada.

References

1. Funk VA, Olefson RW, Raap M, Smith D, Aitken L, Haddow JD, Wang D, Dawson-Coates JA, Burke RD, Miller KM. 2008. Comp Biochem Physiol B 149:477-489. 2. Funk V, Raap M, Sojonky K, Jones S, Robinson J, Falkenberg K, Miller KM. 2007. Dis Aquat Org 75:239-249.

Canada

Mike Hawkshaw, UBC Fisheries Center, m.hawkshaw@fisheries.ubc.ca

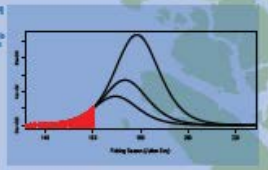
Skeena Salmon

Using Bayesian analysis to improve in-season estimates of salmon run size and timing

Problem

The Skeena River fishery is a mixed stock fishery dominated by a single large sockeye stock. In the case of salmon fisheries management agencies often try to respond to variability in run timing and abundance by varying harvests in accordance with a harvest control rule. Estimating run timing and abundance is very difficult within season and can often lead to less than optimal fishing efforts (Link and Peterman 1998). It is difficult to distinguish between run size and run timing as a fishing season develops because a salmon run will behave in the same way if it is a small run coming early or a large run arriving late in the season. Fear of over-harvest can lead to delays in opening the fishery which concentrates effort and can exacerbate weak stock problems reducing fishing opportunity for the comercial fleet.

Figure 1: An example of how data collected at the beginning of the fishing season could explain three different patterns of run size and timing. The red bars are the observed test fishery catches and the solid lines are three different possible combinations of run size and timing that could explain this data.



Methods

This Bayesian approach builds on methods developed for Bristol Bay salmon stocks by Fried and Hilborn (1988) but takes advantage of advances in Bayesian software to perform full Bayesian inference and evaluate the posterior probability of both the run size and run timing as the fishing season develops. A salmon run is modeled as a logistic curve while run size and run timing are the parameters that shape the curve. The model uses data collected from the commercial fishery and an index fishery and incorporates prior information on run size from precocious male spawners from the year before and escapements from four years previously to infer run size and run timing.

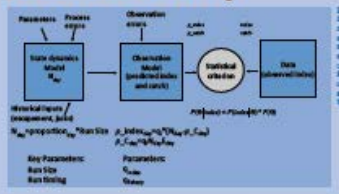


Figure 2: Model schematic explaining how in order to estimate the run size and timing a state model estimates the number of fish returning to the fishery and test fishery in every day of the fishing season. An observation model predicts the test fishery (index) catch and the fishery catch. The predicted index catch and fishery catch are compared to the observed index and fishery

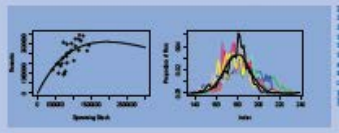


Figure 3: Prior information used in the Bayesian model framework includes the stock recruit function that is used to generate a prior on the run size and the historical run timing pattern that is used to generate a prior for the run timing. In addition the observed back hydroacoustic model components from the previous year are used to provide prior information on run size.

Results

Simulations show the Bayesian model more accurately predicts run size than either of the two non-Bayesian methods tested, in addition it provides an explicit measure of uncertainty in forecasts. The non-Bayesian models fail to accurately distinguish between run timing and run size variation until after the peak of the salmon run and present only point estimates of abundance.

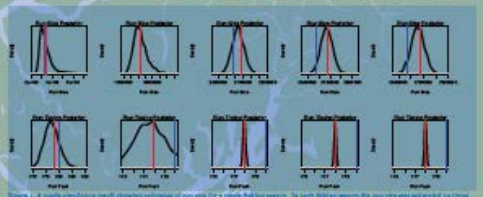


Figure 4: A simple simulation result showing estimates of run size for a single fishing season. In each fishing season the run size was estimated at times in the run passed through a gaussian and an index fishery. The red line shows the simulated run size, the dark line the pre-season estimate of abundance and each plot shows how the posterior predictive estimate of run size changes as the fishing season progresses.

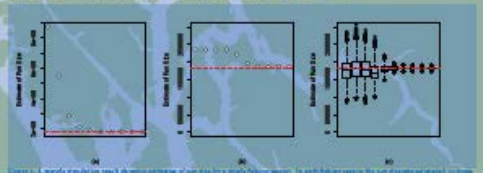


Figure 5: A simple simulation result showing estimates of run size for a single fishing season. In each fishing season the run size was estimated at times in the run passed through a gaussian and an index fishery. The red line shows the simulated run size, which each plot shows how the estimate of run size changes as the fishery progresses. Plot (a) shows a case under the curve estimate, plot (b) shows a weighted combination of pre-season and case under the curve estimates and plot (c) shows the plotted the posterior distributions of the run size estimates using the Bayesian model.

Discussion

Salmon stock management is characterized by high uncertainty within season and managers are forced to make decisions about openings and closings in the face of this uncertainty. A harvest control rule that depends on stock sizes to determine allowable harvest rates or allowable catches requires in-season estimates of abundance. The current methods of in-season run size estimation in use on the Skeena River should be updated. A general Bayesian method for estimating run size and timing should be added to the suite of tools managers available to managers of salmon fisheries. The posterior probability distributions associated with Bayesian estimates of run size and the explicit acceptance of uncertainty that they entail should lead to renewed discussion of appropriate Harvest Control Rules. Revisiting management strategies and embracing uncertainty could be as valuable to the fishery as any marginal improvements in management performance arising from improved in-season estimates.

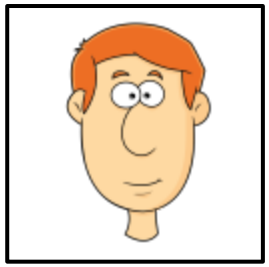
Next Steps

I'm working on a retrospective analysis of run size and timing estimates using historic Skeena data to test the performance of different methods of estimating run size and timing on real data.

References

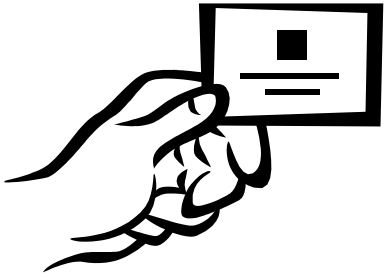
Fried, S.M. and Hilborn, R. Inseason Forecasting of Bristol Bay, Alaska, Sockeye Salmon (Oncorhynchus nerka) Abundance Using Bayesian Probability Theory. Canadian Journal of Fisheries and Aquatic Sciences, 1988, 45(8): 1424-1434, 10.1139/f88-169

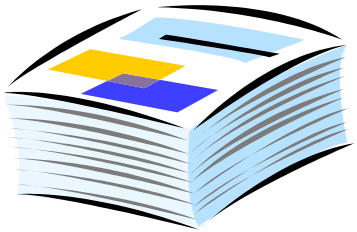
Link, M.R. and R.M. Peterman. 1998. Estimating the value of in-season estimates of abundance of sockeye salmon. Can. J. Fish. Aquat. Sci. 55: 1430-1438.



4. Go the extra mile

- Anticipate questions
- Be accessible
- Provide access to additional information





http:\\mypretendshorturl.ca

5. Proofread and practice

- Proofread!!!
- Mini (test) poster
- Standalone?
- Opening
- Summarize!
- Different versions
- Watch language
- Be engaging

Photo: David Cornejo flickr

SHARC Poster Session

- Assigned judging time – all judges at once
- Format: "Presentation" + questions
- Length: TBC; ~8 minutes (inc. questions)
- Awards: Top Poster
  Honorable Mention
  People's Choice
  Top Overall (poster + lightning)

SHARC Judging Criteria

- Visual appeal (organization, appearance)
- Presentation
    - Flow
    - Comprehension
    - time management
    - handling questions
- Study design
- Study significance

Final Workshop

Preparing/presenting a 1 minute 1 slide lightning talk

March 4th at noon

DHCC 1020 LT, IMP: MSB 160 LT; SMP: RHS 257 LT; NMP: NHSC 9-235 LT.

https://survey.ubc.ca/s/SHARC-workshops/

Resources & Contacts

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a place of mind

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