

# Academic posters

Your business card

CORSO DELLA LAUREA MAGISTRALE IN BIOLOGIA  
“MALATTIE GENETICHE: DALLA DIAGNOSI ALLA TERAPIA”  
Università degli Studi di Milano-Bicocca  
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**"THE MORE STRIKINGLY VISUAL  
YOUR PRESENTATION IS,  
THE MORE PEOPLE WILL  
REMEMBER IT.  
AND MORE IMPORTANTLY,  
THEY WILL REMEMBER YOU."**

— Paul Arden

Direttore creativo britannico

# Summary

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**02**

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**Presenting your research**

# 01. What is a scientific poster?

**A concise and visual  
summary of your research.**

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**Its purpose is to be  
accessible  
and to drive attention to  
your research.**



# A scientific poster is a visual abstract of your research

## Insekten als Indikatoren für Renaturierungserfolge

**Vasco Elbrecht** (Vasco.Elbrecht@rub.de), **Ralph Tollrian** & **Florian Leese**  
RUB Ruhr-Universität Bochum, Lehrstuhl für Evolutionsökologie und Biodiversität der Tiere

### Hintergrund

Gewässer werden derzeit europaweit renaturiert, um unsere Trinkwasserversorgung auch in Zukunft zu sichern. Oft kehren Stress-sensitive Arten nach den meist kostspieligen Maßnahmen nicht unmittelbar in die Gewässer zurück, wobei nicht klar ist ob: **a)** die Renaturierung unzureichend war oder **b)** die Organismen das renaturierte Gewässer nicht selbständig wiederbesiedeln können. Informationen über die Mobilität von empfindlichen Indikatorarten, wie z. B. der Steinfliege *Dinocras cephalotes*, sind folglich essentiell um den Erfolg von Renaturierungen zu bewerten (Abb. 1).

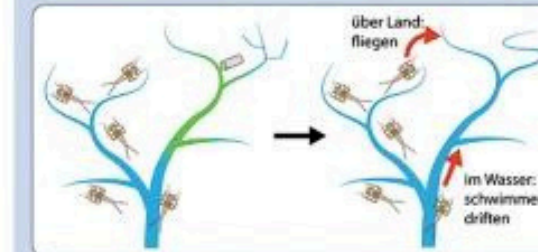


Abbildung 1: Ob und wie Indikatorarten (schematisch in Braun dargestellt) renaturierte Gewässer wiederbesiedeln können ist oft nicht bekannt. Diese Informationen sind jedoch essentiell, um Abschnitte für konkrete Maßnahmen zu identifizieren sowie den Erfolg von Renaturierungen zu bewerten.

**Ziel** dieser Masterarbeit war es, das Ausbreitungspotenzial der Steinfliegenart *D. cephalotes* und somit ihre Eignung als Indikatorart mit molekularen Methoden zu untersuchen.

### Material und Methoden

Die Ausbreitungsfähigkeit und -wege von Insekten können durch reines Beobachten nur schwer bzw. gar nicht ermittelt werden. Mit modernen molekularen, DNA-basierten Methoden, ist dies jedoch präzise und kostengünstig möglich. Zusätzlich können über den sogenannten „genetischen Fingerabdruck“ Aussagen zum Zustand und Bedrohung von Populationen gemacht werden.



Abbildung 2: Die Steinfliege *D. cephalotes*. 316 Tiere von 29 Probestellen im Hochsauerland wurden in der Arbeit molekular untersucht.

In diesem Projekt genutzte genetische „Fingerabdrücke“:

 <b>„CO1“ Gen</b> • Mitochondriales Gen • Historische Prozesse (Tausende von Jahren)	 <b>Mikrosatelliten</b> • Variable Kern DNA • Vergleichbar mit Vaterschaftstest • Rezente Prozesse (wenige Jahre)
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### Ergebnisse und Diskussion

Analysen des CO1 Gens zeigen, dass *D. cephalotes* aus zwei genetisch diversen Gruppen besteht (A und B in Abb. 3). Vertreter beider Gruppen sind über das Untersuchungsgebiet relativ gleichmäßig verteilt, was eine gute Ausbreitungsfähigkeit der Individuen belegt. Die Analyse der hochauflösenden Mikrosatelliten bestätigt dies, da alle Populationen genetisch sehr ähnlich sind (Abb. 4).

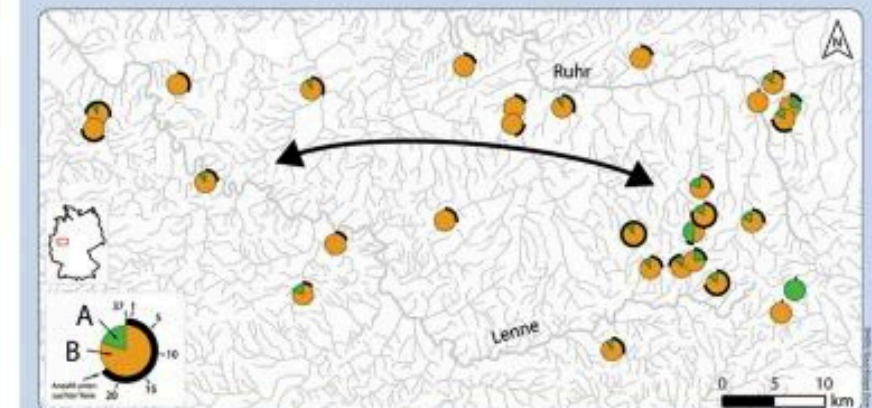


Abbildung 3: Karte der 29 untersuchten *D. cephalotes* Populationen. Mit dem CO1 Gen wurden zwei genetisch diverse Gruppen gefunden: A (grün) und B (orange) deren relative Häufigkeit im Kreisdiagramm dargestellt ist. Beide genetischen Gruppen sind im Untersuchungsgebiet gleichmäßig verteilt und in den Populationen sind oft beide Gruppen vertreten. Dies deutet auf einen genetischen Austausch zwischen den Populationen hin, also dass ausgewachsene Steinfliegen zu anderen benachbarten Populationen fliegen können (mit einem Pfeil schematisch dargestellt). Ohne Austausch zwischen Populationen wären deutlichere Muster zu erwarten, d.h. die Kreise hätten entweder nur die Farbe Orange oder Grün.

- CO1 und Mikrosatelliten-Untersuchungen zeigen, dass alle Populationen genetisch sehr ähnlich sind. Dies belegt, dass *D. cephalotes* eine **gute Ausbreitungsfähigkeit** hat.
- Über adulte fliegende Tiere kann *D. cephalotes* innerhalb weniger Jahre renaturierte Gewässerabschnitte von benachbarten Bächen wiederbesiedeln.
- Somit eignet sich die Art als **guter Indikator**, um den Erfolg von Renaturierungen zu überprüfen.
- Diese Arbeit zeigt das - bislang weitgehend ungenutzte - Potenzial genetischer Methoden.

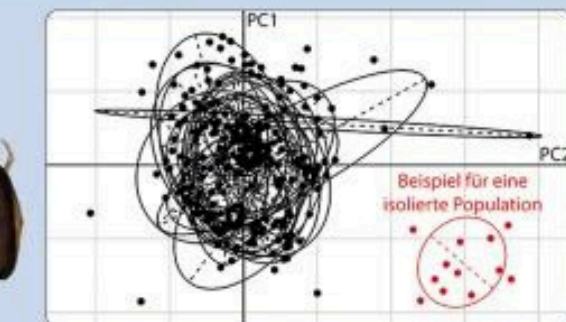


Abbildung 4: Hauptkomponentenanalyse (PCA) für die Mikrosatelliten-Daten, gruppiert nach Sammelorten. Alle Punkte (= untersuchte Individuen) überlagern sich, was auf eine große genetische Ähnlichkeit und somit eine gute Ausbreitungsfähigkeit der Organismen hindeutet.

Hätte die Steinfliege eine schlechte Ausbreitungsfähigkeit, so wären genetisch distinkte Populationen (rote Gruppe) zu erwarten.

### Wirtschaftliche Bedeutung

Renaturierungen sind fast immer **teuer** jedoch oft **nicht direkt erfolgreich**. Ob eine Wiederbesiedlung an ungenügenden Maßnahmen oder der fehlenden Nähe von Quellpopulationen scheitert, kann ohne Kenntnis über die Ausbreitungsfähigkeit der Arten nicht eindeutig bestimmt werden. Die Ergebnisse dieser Masterarbeit zeigen, dass:

- sich über die genetische Diversität die **Gefährdung von Populationen** (und somit auch die Gefährdung ihrer Funktion im Ökosystem) durch anthropogene Einflüsse feststellen lässt,
- genetische Daten entscheidende **Hinweise über die Isolation und Wiederbesiedelbarkeit** von Gewässerabschnitten geben können,
- anhand von genetischen Landkarten geeignete **Stellen mit hohem Wiederbesiedlungspotenzial identifiziert** werden können,
- diese wichtigen Informationen durch molekulare Methoden mit vergleichsweise **geringem Aufwand und kostengünstig** gewonnen werden können.

### Video-Poster

Dreiminütiges Video zu der Masterarbeit (auf Englisch). [www.goo.gl/4K7Pbt](http://www.goo.gl/4K7Pbt)

### Veröffentlichungen aus der Masterarbeit

Elbrecht, Feld, Gao, Hering, Sørensen, Tollrian & Leese (2014): Genetic diversity and dispersal potential of the stonefly *Dinocras cephalotes* in a central European low mountain range. *Parasitology*, 144, 1001-1010. doi:10.1017/S0022268913001001

### Über den Autor

Vasco Elbrecht ist Doktorand im „GeneStream“ Projekt. Im Rahmen der Arbeit entwickelt und testet er modernste genetische Methoden zur Fließgewässerbewertung. [www.GeneStream.de](http://www.GeneStream.de)



### Projektpartner

Wir danken Christian K. Feld, Maria Gies, Daniel Hering und Martin Sondermann (Universität Duisburg-Essen) für die Zusammenarbeit in diesem Projekt.

# 02. Why present a scientific poster?



# Communication and connection



## Share key results

**It's not** to summarize every detail of your research



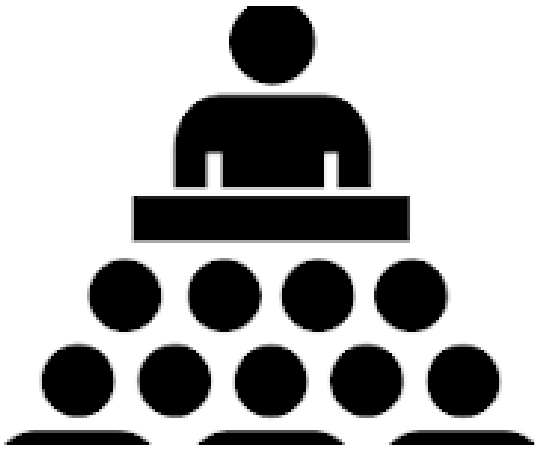
## Networking tool

It serves as a conversation starter



## Communication tool

It should use visuals to draw people from a distance.



## **Practice public speaking**

Present research in a formal setting



## **Direct interactions**

Get feedback from peers; share ideas and create collaborations



## **Enhance your resume**

Conference poster abstract are searchable; poster award with cash price

# It enhances your research and CV



# Why are academic posters important? Responsibility!

**01.**  
**Represents  
you and  
your lab**

at conferences,  
symposia, hallways,  
informational days

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**03.**  
**Demonstrate  
expertise**

**02.**  
**Demonstrate  
attention  
to detail**

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**04.**  
**Enhance  
your  
resume**

# **IMPORTANT: Work with your research supervisor**

Vital for your research group/University: it represents their laboratory.  
Should be involved in editing and final approval.

## **Other experts in your field or a related field?**

They will read it, even if it's bad

## **People who don't know your research?**

A good poster can attract previously uninterested passersby and may open new contacts, at the intersections with other disciplines

## **The broader public?**

Increases your potential audience and impact

# **03. Who is your audience?**



## 04. Structure and content

# **CLEAR ORGANIZATION AND A CONCISE CENTRAL ARGUMENT.**

Focus on findings that support your argument and validate your conclusion.

# Main elements of a poster

## -Hierarchy and the 'must haves' -



### **1. Title**



### **2. Names of people involved and affiliations**



### **3. Abstract (max 200 words)**

What is known; what is not known; what you set out to do; how you have done it and the key findings and results; conclusions and significance.

# Main elements of a poster



## 4. Introduction

background knowledge  
in the field; open questions;  
project aim(s);  
a couple of literature references



## 5. Methods/Approach

basis of techniques used,  
keep it short;



## 6. Results (3-4 Figures)

Show examples  
of key experiments  
with Figure legends  
specifying experimental  
details (type of exp.,  
samples, etc..)

# Main elements of a poster



## 7. Conclusions/future perspectives (bullet points)

summarize the research findings and impact/significance



## 8. Acknowledgements

grant funding, research programs, laboratory, mentors, collaborators  
(smaller at the bottom)



## 9. References

A few literature references  
(smaller at the bottom)



## 10. Contact

your contact email; lab website; QR code; foto  
(smaller at the bottom)



# 05. Poster design

# Concept - first draft

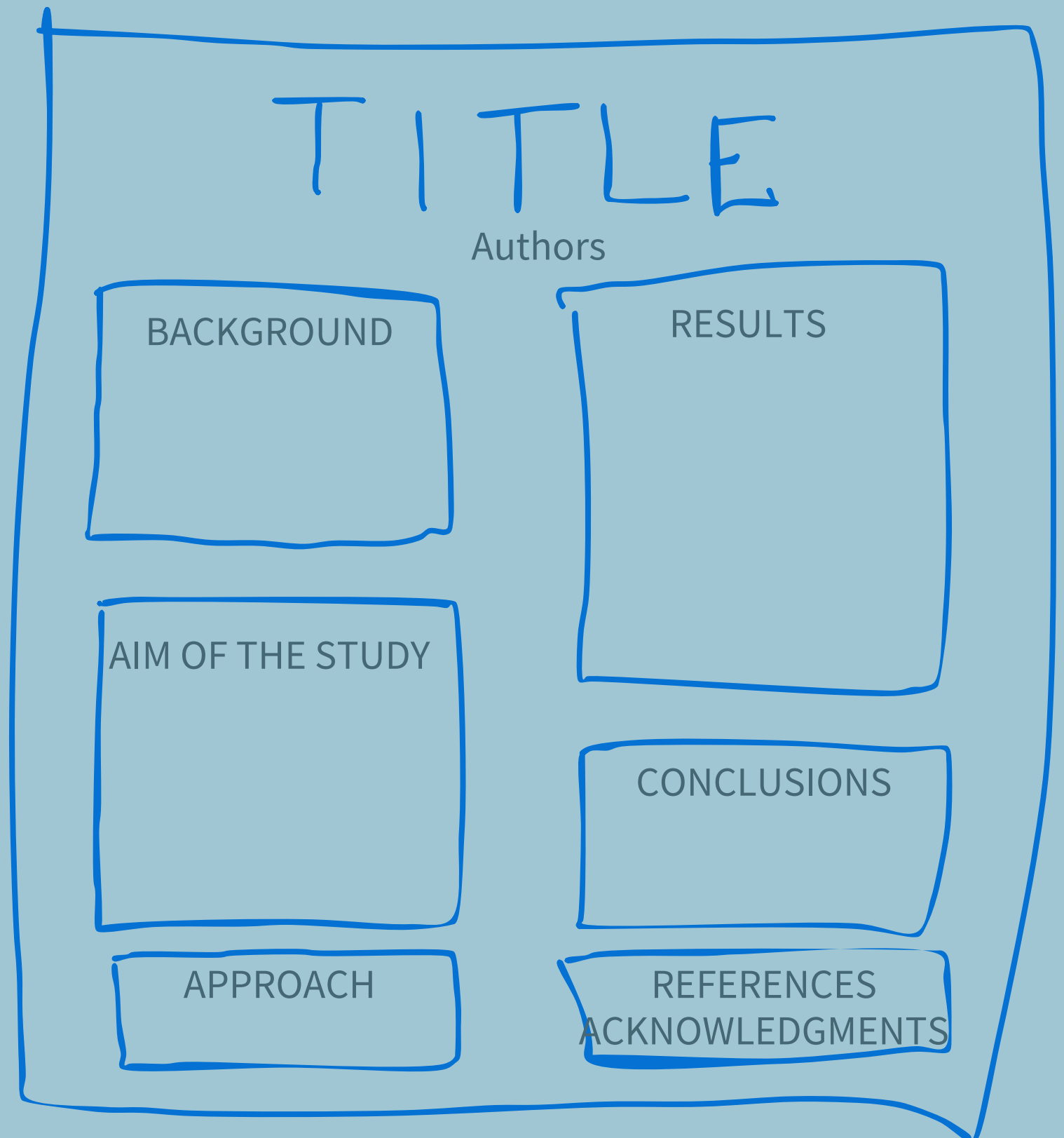
## Ask yourself questions:

- What findings I want people to focus on?
- What are two or three background knowledge items to know?
- What knowledge is missing?
- What is the main goal of my research?
- How did I approach the research?
- What are the key results I obtained?
- What is the impact/relevance of my results in the field?

# Concept - first draft

## Create storyboard:

- Headings, sections
- Experiments to present (3-4)
- Prepare visuals (photos, graphs...)
- Prepare text; concise; figure legends



## **Layout and size**

Check with organizers

(A1 vertical: W594 x L841 cm  
W23.4 x L33.1 Inch)

## **Panels**

Use numbered headings

## **Negative space**

Leave space at the edges;  
leave clear space between  
sections

**Poster  
must be  
“stand  
alone”**

## Eye-catching visuals

One visual related to your research visible from far

## Colors

Use a limited number (3-4).  
Background color; accent color to draw attention

## Alignment

Align sections

## Font

- legible fonts like Times New Roman, Arial..
- use same fonts throughout the poster
- visible from 2-3 meters:
  - 72 points font for title
  - 48 points font for headings
  - 24 points font for body text



# Which would you choose?

1.

### If you can read this you must be nocturnal...

**Abstract**  
The study aims to develop vertebrate models to elucidate molecular mechanisms by which known mutations in the MYH9 gene disrupt development. There are five clinical disorders that result from mutations in MYH9 that are classified as MYH9-related disease: May-Hegglin anomaly, Sebastiani, Fabry and Epstein syndromes, and non-syndromic deafness (DFNA17). MYH9 encodes for the highly conserved non-muscle myosin IA protein (NMLIA), which has essential roles in cell division, cell migration and cell shape changes. However, there is a critical gap in the understanding of how MYH9 mutations found in the human population contribute to the etiology of MYH9-related diseases. Current studies involve the investigation of a zebrafish mutant line containing a stop codon in Exon 12, which results in a truncated NMLIA protein. In addition, we are developing zebrafish models of the most common human MYH9 mutations, specifically of the conserved Arginine 1027/1028 locus to examine organ development in MYH9-related disease using CRISPR/Cas9 genome editing.

**Introduction**  
The MYH9 gene is located on chromosome 15q21.1 and encodes for the non-muscle myosin IA protein (NMLIA). Mutations in MYH9 are associated with several clinical disorders, including May-Hegglin anomaly, Sebastiani syndrome, Fabry and Epstein syndromes, and non-syndromic deafness (DFNA17). MYH9 is highly conserved across species, and its protein product, NMLIA, is essential for cell division, cell migration, and cell shape changes. The zebrafish (*Danio rerio*) is an excellent model organism for studying MYH9-related diseases because of its genetic tractability and the availability of CRISPR/Cas9 genome editing technology.

**Methods & Materials**  
We generated zebrafish mutant lines using CRISPR/Cas9 genome editing. The MYH9 gene was targeted using a pair of guide RNAs (gRNAs) designed to target specific exons of the gene. The mutant lines were screened for the presence of the desired mutations using Sanger sequencing. The mutant lines were then analyzed for phenotypic changes using various assays, including RT-PCR analysis, in situ hybridization, and histological analysis.

**Results**  
We successfully generated zebrafish mutant lines for the MYH9 gene. The mutant lines were screened for the presence of the desired mutations using Sanger sequencing. The mutant lines were then analyzed for phenotypic changes using various assays, including RT-PCR analysis, in situ hybridization, and histological analysis. We found that the mutant lines exhibit various phenotypic changes, including growth retardation, skeletal abnormalities, and hearing loss.

**Discussion**  
The results of this study demonstrate that zebrafish is a suitable model organism for studying MYH9-related diseases. The mutant lines generated using CRISPR/Cas9 genome editing exhibit various phenotypic changes that are similar to those observed in human patients with MYH9-related diseases. This study provides a valuable resource for studying the molecular mechanisms of MYH9-related diseases and for developing potential therapies.

**Conclusion**  
Zebrafish is a suitable model organism for studying MYH9-related diseases. The mutant lines generated using CRISPR/Cas9 genome editing exhibit various phenotypic changes that are similar to those observed in human patients with MYH9-related diseases. This study provides a valuable resource for studying the molecular mechanisms of MYH9-related diseases and for developing potential therapies.

**References**  
1. May-Hegglin anomaly. J Clin Invest. 1952;31:1-10.  
2. Sebastiani syndrome. J Clin Invest. 1952;31:1-10.  
3. Fabry and Epstein syndromes. J Clin Invest. 1952;31:1-10.  
4. Non-syndromic deafness (DFNA17). J Clin Invest. 1952;31:1-10.

**Acknowledgements**  
This work was supported by the National Institutes of Health (NIH) Grant R01NS091111 and the University of Wisconsin-Milwaukee (UWM) Graduate Research Assistantship Program.

2.

### miR-218 is Essential for Spinal Cord Motor Neuron Development

Karen P. Thiebets<sup>1</sup>, Heejin Nam<sup>2</sup>, Xiaoku A. Cambromne<sup>3</sup>, Rongkun Shen<sup>2</sup>, Stacey M. Glasgow<sup>4</sup>, Richard H. Goodman<sup>1</sup>, Jae W. Lee<sup>1,3</sup>, Seunghae Lee<sup>1,3</sup>, & Soo-Kyung Lee<sup>1,2,3\*</sup>

**Abstract**  
MicroRNAs have emerged as an important component of gene regulatory networks, but it is still unclear how they collaborate with transcription factors in the gene network to determine neuronal cell fate. Here we show that in the developing spinal cord, the expression of miR-218 is highly induced by the *Isl1-Lhx3* complex, which directs motor neuron fate. Inhibition of miR-218 suppresses the generation of motor neurons in chick mouse and mouse embryonic stem cells, suggesting that miR-218 plays a crucial role in motor neuron differentiation. Our unbiased RISC-drop screens, coupled with in vivo reporter assays, revealed that miR-218 directly represses transcripts that promote developmental programs for interneurons and neural progenitors. In addition, miR-218 activity is required for *Isl1-Lhx3* to effectively induce motor neurons and suppress interneuron fates. Together, our studies uncovered an essential role of miR-218 as a downstream effector of the *Isl1-Lhx3* complex in establishing motor neuron identity.

**Conclusions**  
• miR-218 is expressed and active in developing spinal cord motor neurons.  
• *Isl1-Lhx3* directly binds and upregulates miR-218-1 and miR-218-2 genes.  
• miR-218 is essential for the generation of motor neurons from ESCs.  
• miR-218 inhibits expression of genes that are important for neural progenitors and interneurons.

**Figure 1. miRNA Upregulation**  
Figure 1 shows the upregulation of miR-218 in the developing spinal cord. Panel A shows a schematic of the spinal cord and the expression of miR-218 in motor neurons. Panel B shows a bar graph of miR-218 expression levels in motor neurons compared to other cell types. Panel C shows a heatmap of miR-218 expression levels in various tissues.

**Figure 2. miR-218 Expression**  
Figure 2 shows the expression of miR-218 in the developing spinal cord. Panel A shows a schematic of the spinal cord and the expression of miR-218 in motor neurons. Panel B shows a bar graph of miR-218 expression levels in motor neurons compared to other cell types. Panel C shows a heatmap of miR-218 expression levels in various tissues.

**Figure 3. miR-218 Inhibition In Vivo**  
Figure 3 shows the effect of miR-218 inhibition on motor neuron development in vivo. Panel A shows a schematic of the spinal cord and the expression of miR-218 in motor neurons. Panel B shows a bar graph of miR-218 expression levels in motor neurons compared to other cell types. Panel C shows a heatmap of miR-218 expression levels in various tissues.

**Figure 4. miR-218 Inhibition in ESCs**  
Figure 4 shows the effect of miR-218 inhibition on motor neuron development in ESCs. Panel A shows a schematic of the spinal cord and the expression of miR-218 in motor neurons. Panel B shows a bar graph of miR-218 expression levels in motor neurons compared to other cell types. Panel C shows a heatmap of miR-218 expression levels in various tissues.

**Figure 5. miR-218 Target Screen**  
Figure 5 shows the results of a miR-218 target screen. Panel A shows a heatmap of miR-218 expression levels in various tissues. Panel B shows a bar graph of miR-218 expression levels in motor neurons compared to other cell types. Panel C shows a heatmap of miR-218 expression levels in various tissues.

**Figure 6. miR-218 Target Validation**  
Figure 6 shows the validation of miR-218 targets. Panel A shows a schematic of the spinal cord and the expression of miR-218 in motor neurons. Panel B shows a bar graph of miR-218 expression levels in motor neurons compared to other cell types. Panel C shows a heatmap of miR-218 expression levels in various tissues.

3.

### Zebrafish as a Model for MYH9-Related Disease

Laura A. Rolfs, Elizabeth J. Falat, and Jennifer H. Gutzman  
Department of Biological Sciences, University of Wisconsin-Milwaukee

**Abstract**  
This study aims to develop vertebrate models to elucidate molecular mechanisms by which known mutations in the MYH9 gene disrupt development. There are five clinical disorders that result from mutations in MYH9 that are classified as MYH9-related disease: May-Hegglin anomaly, Sebastiani, Fabry and Epstein syndromes, and non-syndromic deafness (DFNA17). MYH9 encodes for the highly conserved non-muscle myosin IA protein (NMLIA), which has essential roles in cell division, cell migration and cell shape changes. However, there is a critical gap in the understanding of how MYH9 mutations found in the human population contribute to the etiology of MYH9-related diseases. Current studies involve the investigation of a zebrafish mutant line containing a stop codon in Exon 12, which results in a truncated NMLIA protein. In addition, we are developing zebrafish models of the most common human MYH9 mutations, specifically of the conserved Arginine 1027/1028 locus to examine organ development in MYH9-related disease using CRISPR/Cas9 genome editing.

**Zebrafish *myh9a* and *myh9b* Expression During Development**  
Figure 1 shows RT-PCR analysis of *myh9a* and *myh9b* expression during zebrafish development. Panel A shows a gel image of RT-PCR products. Panel B shows a bar graph of *myh9a* and *myh9b* expression levels at different stages of development.

***myh9a* is Not Required For Zebrafish Development**  
Figure 2 shows that *myh9a* is not required for zebrafish development. Panel A shows a schematic of the zebrafish embryo and the expression of *myh9a*. Panel B shows a bar graph of *myh9a* expression levels in embryos. Panel C shows a heatmap of *myh9a* expression levels in various tissues.

**CRISPR Screening**  
Figure 3 shows the results of CRISPR screening for *myh9a* loss of function. Panel A shows a schematic of the zebrafish embryo and the expression of *myh9a*. Panel B shows a bar graph of *myh9a* expression levels in embryos. Panel C shows a heatmap of *myh9a* expression levels in various tissues.

**MYH9 Gene and NMLIA Protein**  
Figure 4 shows the structure of the MYH9 gene and the NMLIA protein. Panel A shows a schematic of the MYH9 gene and the NMLIA protein. Panel B shows a bar graph of MYH9 expression levels in various tissues. Panel C shows a heatmap of MYH9 expression levels in various tissues.

**Human Zebrafish Homology**  
Figure 5 shows the homology between human and zebrafish MYH9 genes. Panel A shows a schematic of the MYH9 gene and the NMLIA protein. Panel B shows a bar graph of MYH9 expression levels in various tissues. Panel C shows a heatmap of MYH9 expression levels in various tissues.

**Common and Conserved Amino Acid Mutations**  
Figure 6 shows common and conserved amino acid mutations in the MYH9 gene. Panel A shows a schematic of the MYH9 gene and the NMLIA protein. Panel B shows a bar graph of MYH9 expression levels in various tissues. Panel C shows a heatmap of MYH9 expression levels in various tissues.

**CRISPR-Cas9 Mediated Mutagenesis**  
Figure 7 shows the results of CRISPR-Cas9 mediated mutagenesis. Panel A shows a schematic of the zebrafish embryo and the expression of *myh9a*. Panel B shows a bar graph of *myh9a* expression levels in embryos. Panel C shows a heatmap of *myh9a* expression levels in various tissues.

**Future Directions and References**  
This study provides a valuable resource for studying the molecular mechanisms of MYH9-related diseases and for developing potential therapies. Future directions include studying the role of miR-218 in motor neuron development and the role of MYH9 in other tissues. References are provided for further reading.

4.

### Salvage Archaeology at the Snake River Sandspit Site in Nome, Alaska

This poster presents the findings of a salvage archaeology project at the Snake River Sandspit site in Nome, Alaska. The project was conducted to recover and document archaeological resources that were threatened by a proposed development. The findings include a variety of artifacts, including tools, weapons, and personal items, which provide insight into the lives of the people who lived at the site. The poster also includes a map of the site and a list of the artifacts recovered.

**Introduction**  
The Snake River Sandspit site is located in Nome, Alaska, and is a significant archaeological site. The site was discovered in 1980 and has since been the focus of several archaeological excavations. The most recent excavation was conducted in 2010 and was a salvage archaeology project. The project was conducted to recover and document archaeological resources that were threatened by a proposed development.

**Methods**  
The salvage archaeology project was conducted using a variety of methods, including excavation, surveying, and artifact recovery. The project was conducted in a systematic and thorough manner, and all artifacts were carefully documented and preserved.

**Results**  
The salvage archaeology project recovered a variety of artifacts, including tools, weapons, and personal items. These artifacts provide insight into the lives of the people who lived at the site. The artifacts include a variety of items, including a knife, a spearhead, and a bone tool.

**Conclusion**  
The salvage archaeology project was a successful one, and it has provided a valuable resource for the study of the Snake River Sandspit site. The artifacts recovered provide insight into the lives of the people who lived at the site, and they are a valuable part of the site's archaeological heritage.

5.

### Developing epigenetic synergistic drug combinations with albendazole in paediatric acute myeloid leukaemia

ABZ shown to have anti-leukaemic effects as a single agent and in combination with AZA

This poster describes the development of synergistic drug combinations with albendazole (ABZ) in paediatric acute myeloid leukaemia (AML). The study shows that ABZ has anti-leukaemic effects as a single agent and in combination with azacitidine (AZA). The results of the study are presented in a series of graphs and tables, which show the effects of ABZ and AZA on AML cell viability and proliferation. The study also includes a discussion of the mechanisms of action of ABZ and AZA, and the potential for these drugs to be used in combination to improve the treatment of AML.

**Introduction**  
Acute myeloid leukaemia (AML) is a common type of cancer that affects the bone marrow. The disease is characterized by the presence of abnormal white blood cells that interfere with the normal function of the bone marrow. The standard treatment for AML is chemotherapy, but there is a need for more effective and less toxic treatments. The development of synergistic drug combinations is a promising approach to improve the treatment of AML.

**Methods**  
The study was conducted using a series of experiments to evaluate the effects of ABZ and AZA on AML cell viability and proliferation. The experiments included cell viability assays, proliferation assays, and gene expression analysis. The results of the experiments are presented in a series of graphs and tables.

**Results**  
The results of the study show that ABZ has anti-leukaemic effects as a single agent and in combination with AZA. The combination of ABZ and AZA significantly reduces AML cell viability and proliferation, and it also increases the expression of pro-apoptotic genes. These results suggest that ABZ and AZA may be used in combination to improve the treatment of AML.

**Conclusion**  
The study shows that ABZ has anti-leukaemic effects as a single agent and in combination with AZA. The combination of ABZ and AZA significantly reduces AML cell viability and proliferation, and it also increases the expression of pro-apoptotic genes. These results suggest that ABZ and AZA may be used in combination to improve the treatment of AML.

6.

### Negative regulation of the protein kinase Raf

Augustin Deniaud<sup>1</sup>, Robert Townley, Claire de la Cova  
University of Wisconsin-Milwaukee Department of Biological Sciences

**Abstract**  
Raf is a key component of the mitogen-activated protein kinase (MAPK) signaling pathway. It is activated by growth factors and plays a central role in cell proliferation and differentiation. The regulation of Raf activity is therefore a critical step in the signaling pathway. In this study, we have investigated the negative regulation of Raf activity by the protein kinase Lhx3. We show that Lhx3 binds to Raf and inhibits its activity, thereby preventing the activation of the MAPK pathway. This finding provides a new insight into the regulation of Raf activity and the signaling pathway.

**Introduction**  
The mitogen-activated protein kinase (MAPK) signaling pathway is a central component of the cell signaling network. It is involved in a wide range of cellular processes, including cell proliferation, differentiation, and survival. The pathway is initiated by growth factors, which bind to and activate the receptor tyrosine kinase (RTK). This leads to the activation of the Ras protein, which in turn activates the Raf protein. Raf then activates the MEK protein, which activates the ERK protein. The ERK protein then enters the nucleus, where it phosphorylates and activates transcription factors, leading to the expression of target genes.

**Methods**  
We used a variety of methods to investigate the negative regulation of Raf activity by Lhx3. These methods include co-immunoprecipitation assays, pull-down assays, and in vitro kinase assays. We also used a luciferase reporter assay to measure the activity of the MAPK pathway in cells expressing Lhx3.

**Results**  
Our results show that Lhx3 binds to Raf and inhibits its activity. This inhibition is mediated by the interaction of the Lhx3 protein with the Raf protein. We also show that Lhx3 inhibits the activation of the MAPK pathway in cells expressing Lhx3. These findings provide a new insight into the regulation of Raf activity and the signaling pathway.

**Conclusion**  
The results of this study show that Lhx3 binds to Raf and inhibits its activity. This inhibition is mediated by the interaction of the Lhx3 protein with the Raf protein. We also show that Lhx3 inhibits the activation of the MAPK pathway in cells expressing Lhx3. These findings provide a new insight into the regulation of Raf activity and the signaling pathway.

7.

### PIGS IN SPACE: EFFECT OF ZERO GRAVITY AND AD CARMINUM FEEDING ON WEIGHT GAIN IN CAVIA PORCELLIUS

Colin B. Purnell  
6673 College Avenue, Swardsmore, PA 15081 USA

**ABSTRACT**  
The purpose of this study was to determine the effect of zero gravity and ad carminum feeding on weight gain in *Cavia porcellus*. The study was conducted in a space station simulator, and the results are presented in a series of graphs and tables. The results show that zero gravity and ad carminum feeding significantly reduce weight gain in *Cavia porcellus*. These findings provide a new insight into the effects of zero gravity and ad carminum feeding on weight gain in *Cavia porcellus*.

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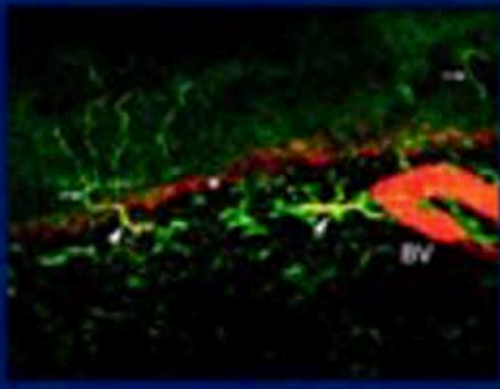
**Conclusions**  
The results of this study show that zero gravity and ad carminum feeding significantly reduce weight gain in *Cavia porcellus*. These findings provide a new insight into the effects of zero gravity and ad carminum feeding on weight gain in *Cavia porcellus*.

# Examples

What do you think should be fixed about this poster?

## If you can read this you must be nocturnal...

*Your eyes are not needed to finish.  
They are used of your audience here.*

<h3>Abstract</h3> <p>Placeholder text for the abstract section.</p>	<h3>Results</h3> 	<h3>Results</h3> 	<h3>Discussion</h3> <p>Placeholder text for the discussion section.</p>
<h3>Introduction</h3> <p>Placeholder text for the introduction section.</p>	<h3>Methods &amp; Materials</h3> <p>Placeholder text for the methods and materials section.</p>	<h3>Methods &amp; Materials</h3> <p>Placeholder text for the methods and materials section.</p>	<h3>Conclusion</h3> <p>Placeholder text for the conclusion section.</p>
<h3>Questions</h3> <p>Placeholder text for the questions section.</p>	<h3>References</h3> <p>Placeholder text for the references section.</p>	<h3>Acknowledgements</h3> <p>Placeholder text for the acknowledgements section.</p>	

# Examples

What do you think should be fixed about this poster?

**PIGS IN SPACE**  
*EFFECT OF ZERO GRAVITY AND AD LIBITUM FEEDING ON WEIGHT GAIN IN CAVIA PORCELLIUS*

Colin B. Purrington\*  
6673 College Avenue, Swarthmore, PA 19081 USA

**ABSTRACT:**  
One hundred twenty Cavia porcellus (Guinea pigs) were transported to the International Space Station (ISS) in 2002. Each pig was housed individually and provided with ad libitum access to food and water. The effect of zero gravity and ad libitum feeding on weight gain was determined. The results show that the effect of zero gravity on weight gain was not significant. The effect of ad libitum feeding on weight gain was significant. The results show that the effect of zero gravity and ad libitum feeding on weight gain was not significant. The results show that the effect of zero gravity and ad libitum feeding on weight gain was not significant.

**INTRODUCTION:**  
The human space program began in the early 1960s with the launch and proliferation of satellites and orbital stations. Since then, numerous experiments have been conducted in space to study the effects of zero gravity on various biological systems. One of the most interesting areas of research has been the study of weight gain in space. The purpose of this study was to determine the effect of zero gravity and ad libitum feeding on weight gain in Cavia porcellus. The results show that the effect of zero gravity and ad libitum feeding on weight gain was not significant.

**MATERIALS AND METHODS:**  
One hundred twenty Cavia porcellus (Guinea pigs) were transported to the International Space Station (ISS) in 2002. Each pig was housed individually and provided with ad libitum access to food and water. The effect of zero gravity and ad libitum feeding on weight gain was determined. The results show that the effect of zero gravity and ad libitum feeding on weight gain was not significant.

**RESULTS:**  
Mean weight of pigs in space was 0.800 ± 0.050 g. Some individuals weighed less than others, but these variations were due to differences in the diet. The results show that the effect of zero gravity and ad libitum feeding on weight gain was not significant. The results show that the effect of zero gravity and ad libitum feeding on weight gain was not significant.

**CONCLUSIONS:**  
The effect of zero gravity and ad libitum feeding on weight gain in Cavia porcellus was not significant. The results show that the effect of zero gravity and ad libitum feeding on weight gain was not significant. The results show that the effect of zero gravity and ad libitum feeding on weight gain was not significant.

**ACKNOWLEDGEMENTS:**  
I am grateful for generous support from the National Research Foundation, Space and Air Force, and the High Frontier Space Foundation. Technical staff were funded by NASA/ESA. The construction of space hardware was supported by the University of Cambridge. I am also grateful for comments on early drafts by Myriam Adams, CUB, Corpus Christi, USA. Finally, special thanks to the CUB Foundation for generous funding of my research after the completion of the study.

**LITERATURE CITED:**  
MANN, 1962. Protein Synthesis in Space. J. Appl. Phys. 33: 200-201.  
MANN, G.B., G. L. LLOYD, and N. M. MANNING. 1965. The Effect of Space Flight on the Growth of Guinea Pigs. J. Appl. Phys. 36: 200-201.  
MANN, M. 1965. Protein Synthesis in Space. J. Appl. Phys. 36: 200-201.



# Examples


What do you think should be fixed about this poster?

## Salvage Archaeology at the Snake River Sandspit Site in Nome, Alaska


**Concurrence of No Historic Properties:**

- March 10, 1998 – The Corps sent a letter to the SHPO requesting concurrence that their project to improve the harbor at Nome, Alaska “does not have the potential to affect cultural resources.”
- April 29, 1998 – The Corps received a letter from the SHPO, in which she concurred that “there are no historic properties in the area of potential effect.”

Despite this, the Corps thought it was a good idea to have an archaeological monitor on site during the groundbreaking. A private archaeologist familiar with the area was sub-contracted to monitor the initial construction during May 2005.



First evidence of the second house pit (Locus B), discovered by Corps archaeologist Margan Grover and bulldozer operator Mike Itano



**Proposed Mitigation (as agreed upon in the draft MOA):**

- 1) Write a site report (Data Recovery Report)
- 2) Provide for an accredited museum conservator to visit the City’s Carrie M. McLain Memorial Museum and assist in the conservation and curation of the site artifacts on display
- 3) Assist with the accessioning of site artifacts and archaeofauna (bagging, cataloging, and if appropriate photographing)
- 4) Provide a museum-quality display case to the City’s Carrie M. McLain Memorial Museum
- 5) Present information learned from the site in a series of public lectures in Nome
- 6) Prepare a manuscript on information learned from the site that can be utilized by Nome teachers (grades 5-12)
- 7) Present information learned from the site to a conference of peers
- 8) Submit an article about the site for publication in a peer-reviewed journal (if not accepted, publish elsewhere)

**Discovery of the Site (Locus A):**

- 1<sup>st</sup> week of May, 2005 – The sub-contracted archaeologist identified the remnants of a semi-subterranean house pit while monitoring the construction.
- The archaeologist took photographs and recovered approximately 25 artifacts, then decided that the house pit was ineligible for inclusion on the National Register of Historic Places and allowed the bulldozers to push the remains into the ocean.
- May 14, 2005 – The Corps received a letter from the sub-contracted archaeologist mentioning the discovery and subsequent destruction of the semi-subterranean house pit.
- May 26, 2005 – The Corps sent a letter to the SHPO stating that the house pit is “not eligible for the National Register for Historic Places” because it “has lost integrity of design, materials, workmanship, and association.”
- September 27, 2005 – The Corps sent a letter to Nome Eskimo Community (tribe), apologizing for not consulting after the discovery of the site and stating that they will continue to work with the tribe to mitigate the damage done.
- October 28, 2005 – The SHPO sent a letter to the Corps in which she concurred with the “finding that the house pit no longer retains sufficient integrity to be eligible” and agreed that “appropriate mitigation would include the development of interpretive signs that discuss the Native history of the Nome area.”

**The Excavation:**

- Occurred from July 26, 2006 to August 26, 2006.
- Involved over 25 community volunteers, including:
  - City of Nome employees
  - Nome Eskimo Community (tribe) employees, members, and tribal Elders
    - Mr. Karlin Itchok, the tribe’s Historic Preservation Representative, participated in the excavation every day
  - Kawerak, Inc. (regional non-profit Native corporation) employees
  - Interested Nome citizens
- Involved 6 Corps employees, including biologists and chemists as well as archaeologists and archaeology interns

**Continued Discovery of the Site (Locs B and C):**

- July 2006 – the Corps sent one of its own archaeologists, Margan Grover, to monitor the continued project construction.
- July 26, 2006 – Margan identified the remains of a second semi-subterranean house pit. She called the SHPO and left a telephone message about the discovery of the house pit, along with her contact information. She also contacted the City of Nome, Nome Eskimo Community (tribe), and Dering Straits Native Corporation. She called the SHPO again and spoke with a Review and Compliance Archaeologist at the SHPO’s office, who agreed that she should excavate a test pit and do some shovel digging to identify the boundaries of the feature.
- July 27, 2006 – Margan called the SHPO again and left another telephone message about the site.
- July 28, 2006 – Margan called the SHPO again and talked with a Review and Compliance Archaeologist at the SHPO’s office. Margan told the SHPO archaeologist that she was assuming the site was eligible for the National Register, and that she was going to excavate at least 50% of the site.
- August 3, 2006 – A meeting was held in Nome between the Corps, the Nome Eskimo Community, and the City of Nome, with the SHPO participating via teleconference, to discuss the discovery of the site and what to do about it.

**Public Outreach in Nome:**

- Public viewing at Old St. Joe’s Cathedral (August 10, 2006).
  - Over 200 people attended
- Viewing of artifacts at Nome Eskimo Community’s building, for tribal members (August 2006)
- Viewing of artifacts at Kawerak’s building during the regional shareholders meeting (August 2006).
- Another public viewing event at Old St. Joe’s Cathedral (September 16, 2006)
  - Over 150 people attended
- Margan Grover gave a public lecture at the National Park Service’s building (November 2006)

**Where We Are Today:**

- Multiple drafts of the MOA have been sent out to signatories and concurring parties (on the following dates):
  - November 22, 2006
  - September 22, 2008
  - April 13, 2009
  - August 10, 2009
  - December 14, 2009
- After a stalemated meeting among the signatories to the MOA on December 15, 2009, and numerous unproductive meetings afterwards, advice was informally requested from the Advisory Council on Historic Preservation. On March 19, 2010, the ACHP sent the Corps an edited draft of the MOA.
- A new draft of the MOA is currently under discussion.
- Artifact and faunal analyses are being undertaken by Corps archaeologist Kelly Itchok, and the Data Recovery Report is being drafted.

# Examples of better posters

What do you think about this poster?



## miR-218 is Essential for Spinal Cord Motor Neuron Development

Karen P. Thiebess<sup>1</sup>, Heejin Nam<sup>4</sup>, Xiaolu A. Cambronne<sup>2</sup>, Rongkun Shen<sup>2</sup>, Stacey M. Glasgow<sup>5</sup>, Richard H. Goodman<sup>2</sup>, Jae W. Lee<sup>1,3</sup>, Seunghye Lee<sup>4,\*</sup>, & Soo-Kyung Lee<sup>1,2,3,\*</sup>

<sup>1</sup>Pediatric Neuroscience Research Program, Papé Family Pediatric Research Institute, Department of Pediatrics, <sup>2</sup>Vollum Institute, <sup>3</sup>Department of Cell and Developmental Biology, Oregon Health & Science University, Portland, OR 97239, USA; <sup>4</sup>College of Pharmacy and Research Institute of Pharmaceutical Sciences, Seoul National University, Seoul 151-742, Korea; <sup>5</sup>Center for Cell and Gene Therapy, Baylor College of Medicine, One Baylor Plaza, Houston, TX 77030, USA

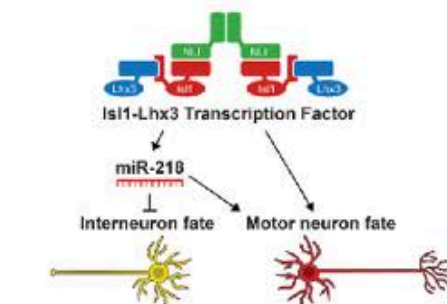


### Abstract

microRNAs have emerged as an important component of gene regulatory networks, but it is still unclear how they collaborate with transcription factors in the gene network to determine neuronal cell fate. Here we show that in the developing spinal cord, the expression of miR-218 is highly induced by the Isl1-Lhx3 complex, which directs motor neuron fate. Inhibition of miR-218 suppresses the generation of motor neurons in chick neural tube and mouse embryonic stem cells, suggesting that miR-218 plays a crucial role in motor neuron differentiation. Our unbiased RISC-trap screens, coupled with *in vivo* reporter assays, revealed that miR-218 directly represses transcripts that promote developmental programs for interneurons and neural progenitors. In addition, miR-218 activity is required for Isl1-Lhx3 to effectively induce motor neurons and suppress interneuron fates. Together, our studies uncovered an essential role of miR-218 as a downstream effector of the Isl1-Lhx3 complex in establishing motor neuron identity.

### Conclusions

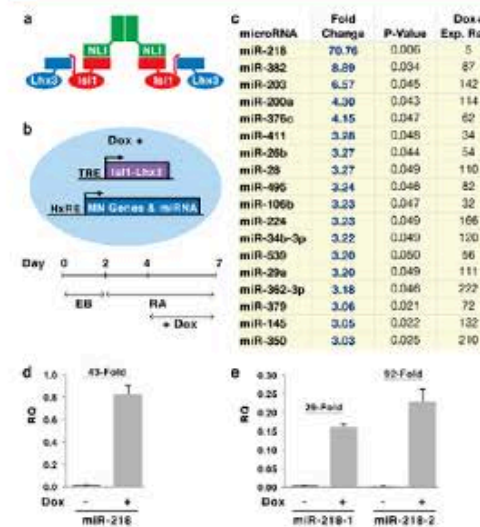
- miR-218 is expressed and active in developing spinal cord motor neurons.
- Isl1-Lhx3 directly binds and upregulates miR-218-1 and miR-218-2 genes.
- miR-218 is essential for the generation of motor neurons from ESCs.
- miR-218 inhibits expression of genes that are important for neural progenitors and interneurons.



### Acknowledgements

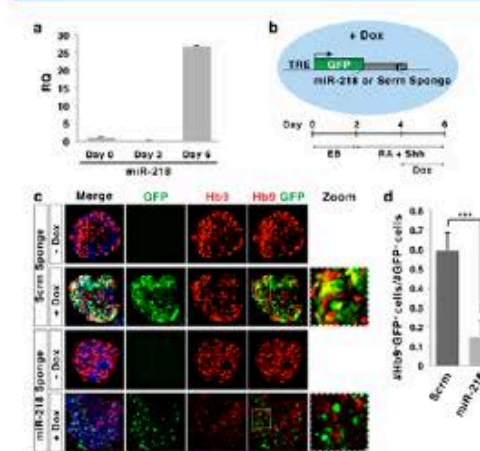
We are grateful to Drs. Fred H. Gage and Xishei Cao for the miRNA sensor vector; to Dr. Greg Smith for creating the image2 GFP/RFP pixel intensity analysis script; Younsung Park for his excellent technical support; to Lee laboratory members for discussions. This research was supported by grants from NIH/NINDS (R01 NS054941) (to S.-K.L.), NIH/NINDS (R01 DK094678) (J.W.L.), NIH/NIMH (R01 MH094418) (to R.H. Goodman) and Research Institute of Pharmaceutical Sciences, POSCO TJ Park Science Fellowship, Basic Science Research Program (2012R1A1A1011749) and Bio 2 Medical Technology Development Program (2012M3A5C0005078) of the National Research Foundation (NRF) funded by the Korean government (MEST) and National R&D Program for Cancer Control, Ministry of Health & Welfare, Republic of Korea (1220120).

### Figure 1. miRNA Upregulation



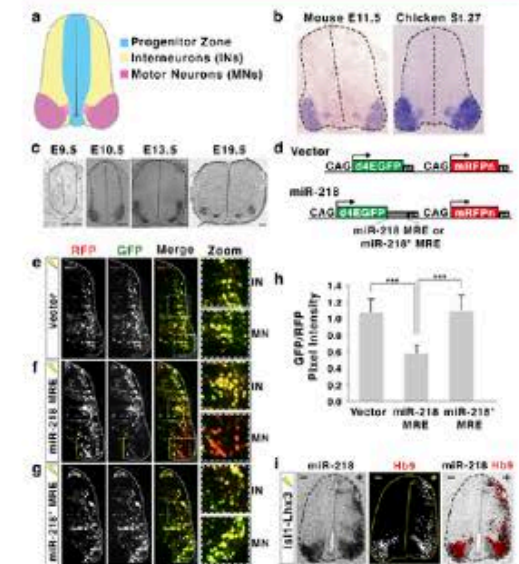
(a) Illustration of the Isl1-Lhx3 transcription factor. (b) Isl1-Lhx3-ESC motor neuron differentiation model. (c) A list of miRNAs that exhibit a significant induction in expression by Dox treatment ( $> 3$ -fold,  $p < 0.05$ ), as determined by TaqMan miRNA arrays. Expression rank (Exp. Rank) describes the rank of relative expression levels of each miRNA in Dox-treated conditions. (d-e) Isl1-Lhx3 triggered the expression of miR-218, miR-218-1, and miR-218-2, as determined by qPCR using TaqMan probes. Error bars represent the standard deviation, RQ, Relative quantity.

### Figure 4. miR-218 Inhibition in ESCs



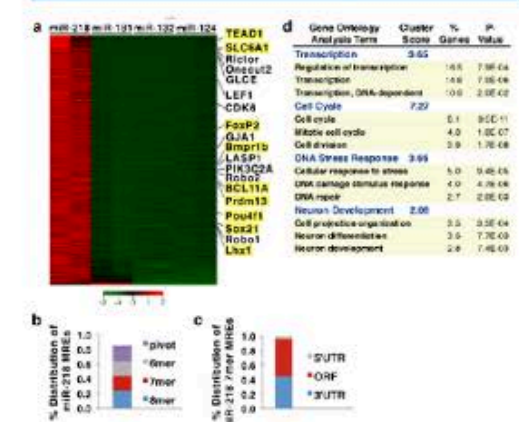
(a) Relative miR-218 expression in monolayer ESCs (day 0), embryoid bodies (day 2) and ESC-derived motor neurons (day 6) as quantified by qPCR. (b) Illustration of Dox-induced sponge-ESC lines. (c) Immunohistochemical analyses in ESC-derived motor neurons at differentiation day 6. (d) Inhibitors were quantified by the ratio of Hb9+GFP+ double motor neurons over the number of GFP+ cells. Error bars represent the standard deviation.  $***p < 0.0001$  in two-tailed student's t-test.

### Figure 2. miR-218 Expression



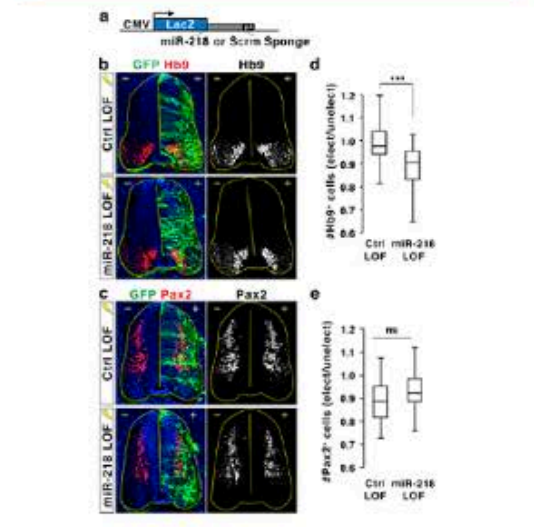
(a) Illustration of developing spinal cord. (b-c) Developing spinal cord miR-218 *in situ* hybridization. (d) Illustration of microRNA sensor. (e-g) Chicken spinal cord electroporation of miRNA sensor. (h) Quantification of relative pixel intensity of GFP/RFP+ in motor neurons. Error bars represent standard deviation.  $***p < 0.0001$  in two-tailed student's t-test,  $n = 5$  embryos. (i) miR-218 expression is highly induced by Isl1-Lhx3 overexpression.

### Figures 5. miR-218 Target Screen



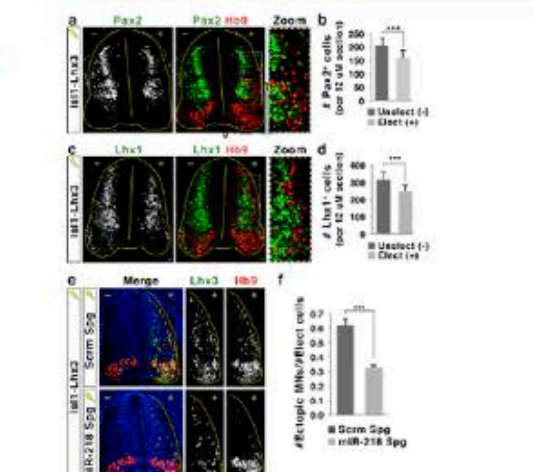
(a) RISC-trap screens identified direct target genes for miR-218. When analyzed against RISC-trap screens for miR-181, miR-132, miR-124. All identified miR-218 targets are sorted in a heatmap by fold-change and biological replicates, compared to miR-181 RISC-trap (Dox +0.05, fold enrichment  $> 4$ ). Previously published miR-218 targets identified in the RISC-trap screen are labeled and selected previously unknown miR-218 targets are highlighted in yellow. (b-c) RISC-trap analysis of miR-218 MREs in 1178 genes targeted by miR-218. (d) Gene ontology cluster analysis.

### Figure 3. miR-218 Inhibition *In Vivo*



(a) Illustration of the sponge inhibitor. (b-c) Loss of function (LOF) analyses in chicks electroporated with sponge inhibitors and 2'Ome RNA inhibitors. (d-g) The effect of LOF conditions were quantified by the ratio of Hb9+ cells or Pax2+ cells on the electroporated (elect, +) vs. unelectroporated (unelect, -) side.  $***p < 0.0001$  in two-tailed student's t-test, n.s., non significant,  $n = 18-21$  embryos.

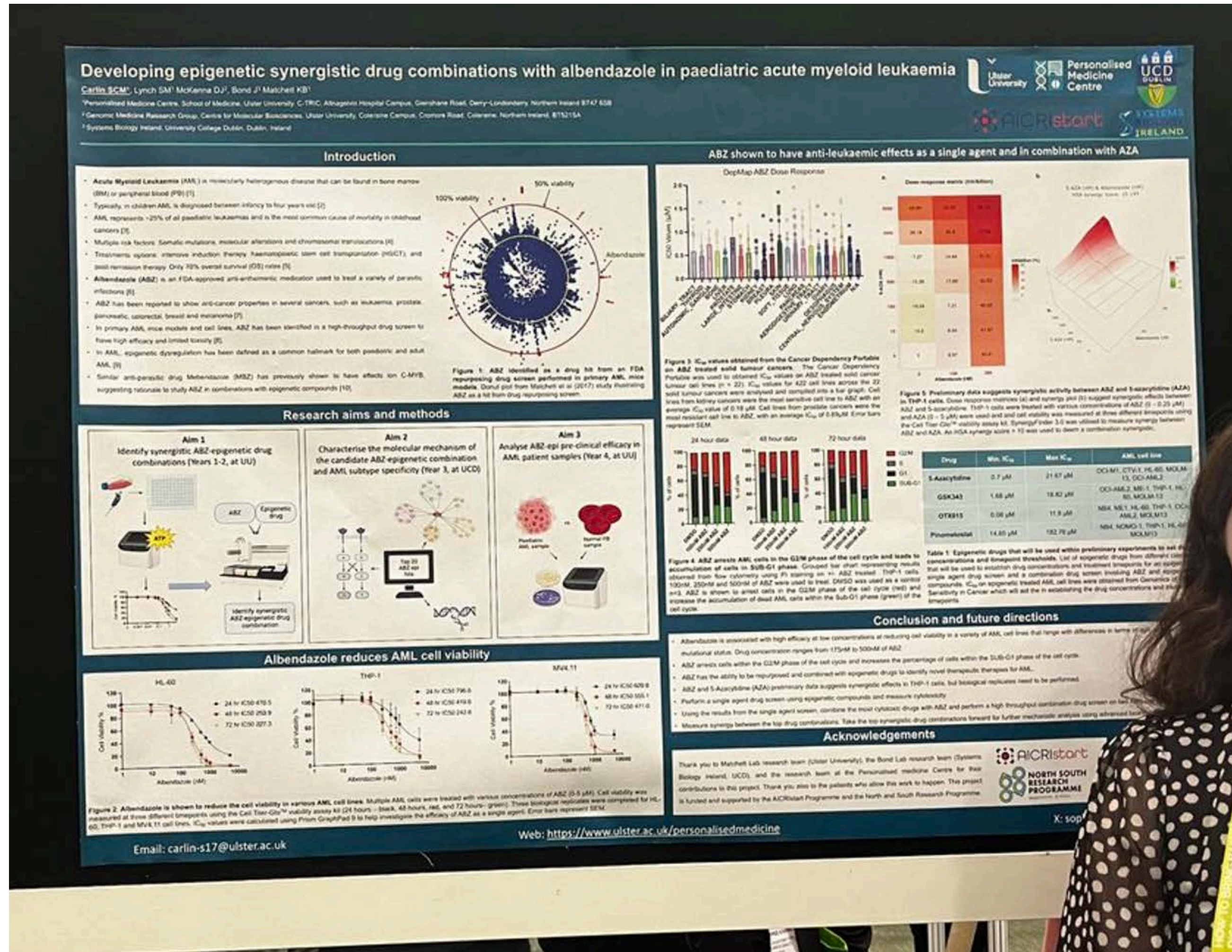
### Figure 6. miR-218 Target Validation



(a-d) Immunohistochemical analyses with Hb9, Pax2 and Lhx1 in the chick neural tube electroporated with Isl1-Lhx3. (b-d) Quantification of Pax2+ or Lhx1+ interneurons. Error bars are standard deviation.  $***p < 0.0001$  in two-tailed student's t-test,  $n = 3$  embryos. (e-f) Electroporation of motor neuron formation by Isl1-Lhx3 in the presence of either miR-218 sponge inhibitor (miR-218 Spp) or scrambled sponge inhibitor (Scrm Spp) in the chick neural tube. (g) The effect of inhibition of miR-218 quantified by the ratio of ectopic Hb9+ motor neurons (MN) over Lhx3-expressing transacted cells (Elect cells). Error bars are standard error of the mean.  $***p < 0.0001$  in two-tailed student's t-test,  $n = 5$  embryos.


# Examples of better posters

What do you think about this poster?




# Examples of good posters

What do you think about this poster?



## Zebrafish as a Model for MYH9-Related Disease

Laura A. Rolfs, Elizabeth J. Falat, and Jennifer H. Gutzman  
Department of Biological Sciences, University of Wisconsin-Milwaukee



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### Abstract

This study aims to develop vertebrate models to elucidate molecular mechanisms by which known mutations in the MYH9 gene disrupt development. There are five clinical disorders that result from mutations in MYH9 that are classified as MYH9-related diseases: May-heggin anomaly, Sebastian, Fetchner and Epstein syndrome; and non-syndromic deafness DFNA17. MYH9 encodes for the highly conserved non-muscle myosin IIA protein (NMIIA), which has essential roles in cell division, cell migration and cell shape changes. However, there is a critical gap in the understanding of how MYH9 mutations found in the human population contribute to the etiology of MYH9-related disease. Current studies involve the investigation of a zebrafish mutant line containing a stop codon in Ex12Δ37, which results in a truncated NMIIA protein. In addition, we are developing zebrafish models of the most common human MYH9 mutations, specifically at the conserved Arginine 702/705 locus to examine organ development in MYH9-related disease using CRISPR/Cas genome editing.

### Zebrafish myh9a and myh9b Expression During Development

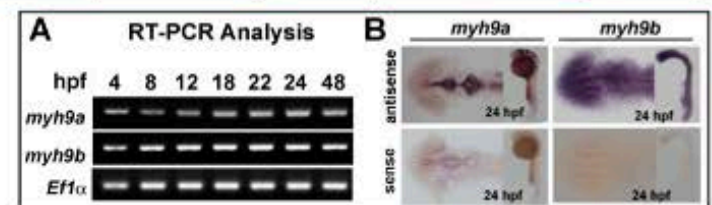


Figure 3: Zebrafish *myh9* gene expression. A) RT-PCR from whole embryo at time points indicated, hours post fertilization (hpf). B) *In situ* hybridization with sense controls and non-specific ventricle staining in *myh9a* samples. Adapted from Gutzman *et al.*, 2015.

### CRISPR Screening

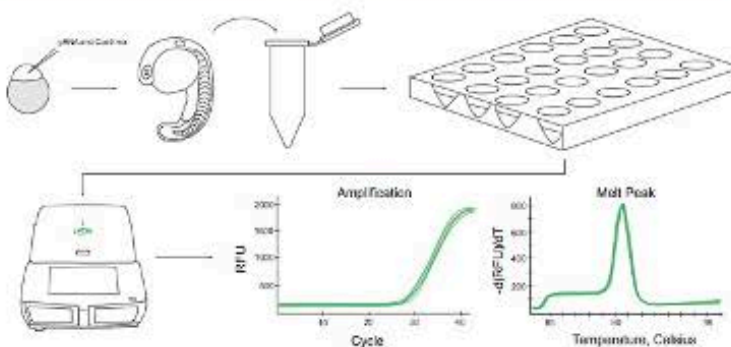
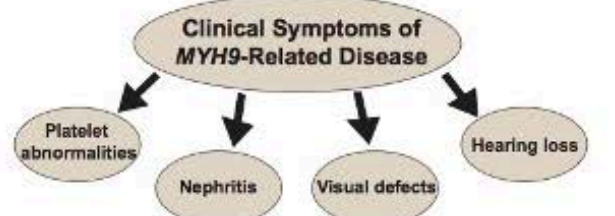


Figure 6: HRMA protocol. Embryos are injected with sgRNA/Cas-9 mix and allowed to develop ~24 hr. DNA is extracted and samples, along with primers and HRMA reagent, are loaded into a 96-well plate for reading. Thermocycler runs CRISPR specific protocol and produces amplification and melt peak charts which then can be used for genotyping.

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### Clinical Symptoms of MYH9-Related Disease



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### MYH9 Gene and NMIIA Protein

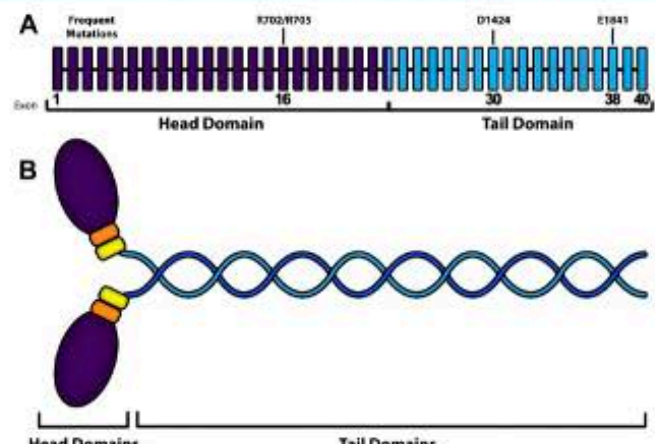


Figure 1: MYH9 gene and NMIIA protein. A) Most frequent disease causing amino acid mutations in the human MYH9 gene. B) NMIIA protein.

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### Human:Zebrafish Homology

Organism	Gene	Protein
Human	MYH9	NMIIA
Zebrafish	<i>myh9a</i>	NMIIA
	<i>myh9b</i>	NMIIA

Table 1: Human MYH9 gene and corresponding zebrafish *myh9* genes. Zebrafish contain two *myh9* genes likely due to the teleost genome duplication.

	Gene	Protein
<i>myh9a</i>	75%	78%
<i>myh9b</i>	80%	82%

Table 2: Zebrafish *myh9* gene and protein homology compared to human MYH9.

### Common and Conserved Amino Acid Mutations

**A R702/R705**

MYH9 LKNDKLVLELIDILKSCOFFENHY  
*myh9a* LKNDKLVLELIDILKSCOFFENHI  
*myh9b* LKNDKLVLELIDILKSCOFFENHI

**B D1424**

MYH9 TKTHTLQQEELDILVLDLHQKQK  
*myh9a* TKTHTLQQEELDILVLDLHQKQK  
*myh9b* TKTHTLQQEELDILVLDLHQKQK

**C E1841**

MYH9 QVACIKQVRRTEKRLKLVLLQV  
*myh9a* QQSTRQVRRVRSKLVKLVLLQV  
*myh9b* QQASHLVRRVRSKLVKLVLLQV

Figure 2: Amino acid sequence homology between human MYH9 and zebrafish *myh9* genes. A) Arginine 702/705 locus. B) Aspartic acid 1424 locus. C) Glutamic Acid E1841 locus.

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### myh9a Is Not Required For Zebrafish Development

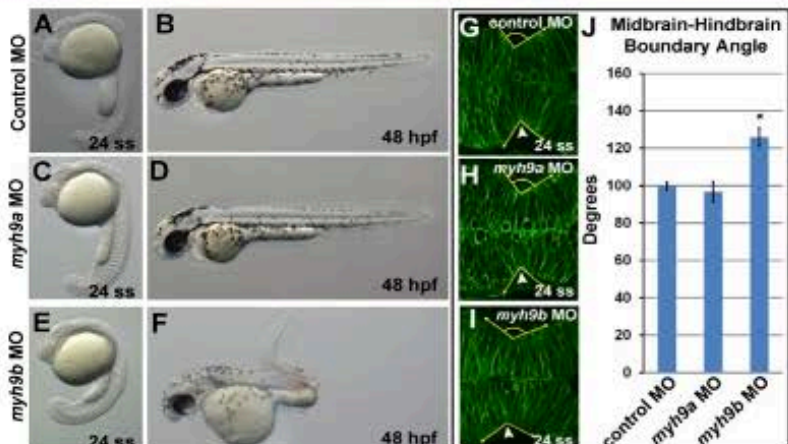


Figure 4: *myh9b* is required for normal zebrafish development. A-F) Brightfield images of live embryos injected with *myh9* morpholino (MO) indicated. G-I) Live confocal imaging of midbrain-hindbrain boundary (MHB) cell shape. J) Morphogenetic analysis of MHB development angle. One-way ANOVA with multiple t-test comparisons, asterisks indicate p<0.001. Adapted from Gutzman *et al.*, 2015.

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### CRISPR Workflow

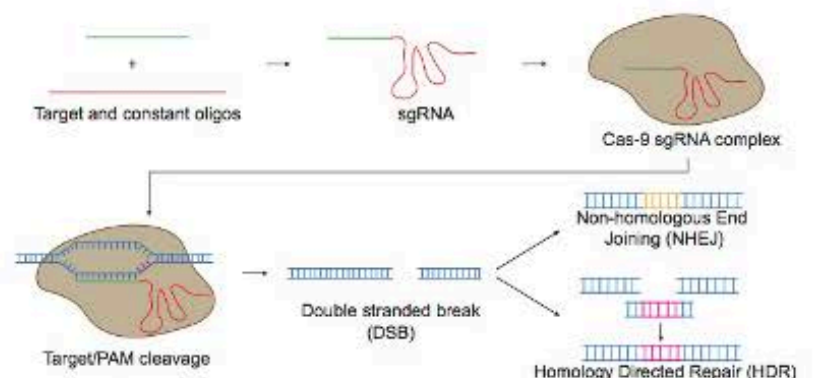


Figure 5: CRISPR-Cas9 Mediated Mutagenesis. Mutation generation initiates by annealing target and constant oligos followed by sgRNA transcription. Recognition of the constant region results in the formation of the Cas-9 sgRNA complex, which allows for cleavage at the target site and the induction of a double stranded break. Endogenous cellular repair machinery can produce insertions or deletions through NEJ or HDR.

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### myh9b loss of function and Heart Edema Development

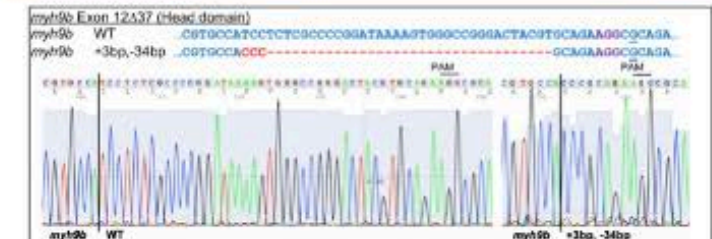


Figure 7: *myh9b* Exon12Δ37 CRISPR line generated in our lab. Sequence of wild type and mutant allele shown. Red indicates mutant base pairs or deletions. Purple indicates PAM sequence.

### myh9b loss of function and heart edema development.

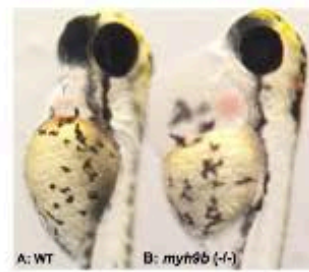


Figure 8: *myh9b* loss of function and heart edema development. A) Wild type fish B) *myh9b* (-/-) mutant showing heart string and edema development at 4 days post fertilization. Previous literature suggests this edema may be due to a kidney defect (Mueller *et al.*, 2011).

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### Future Directions and References


- Generate *myh9a* deletion line
- Generate CRISPR *myh9b* human mutation mimics within the *myh9a* deletion line
- Examine mutant development for other potential clinical defects
- Examine whether heart edema developed in current *myh9b* mutant line results in decreased lifespan

Gutzman, J. H., Sahu, S. U. and Kwas, C. (2015). Non-muscle myosin IIA and IIB differentially regulate cell shape changes during zebrafish brain morphogenesis. *Dev Biol* 397, 103-115.

Müller, Tobias, *et al* (2011). "Non-Muscle Myosin IIA is Required for the Development of the Zebrafish Glomerulus." *Kidney International*, U.S. National Library of Medicine.

# Examples of good posters

What do you think about this poster?



## Negative regulation of the protein kinase Raf

Augustin Deniaud\*, Robert Townley, Claire de la Cova  
University of Wisconsin-Milwaukee Department of Biological Sciences

\*presenting author

### Abstract

Activating mutations in the Ser/Thr protein kinase Raf are common in cancers, especially melanomas. Raf is activated by the EGF Receptor and Ras, and signaling transduction occurs through activation of a kinase cascade involving Raf, MEK, and ERK. However, relatively little is known about cellular regulators that inhibit signaling by activated Raf. Using the nematode *Caenorhabditis elegans*, we performed a genetic screen to identify candidate negative regulators of Raf. In wild-type *C. elegans*, Raf/LIN-45 protein degradation is triggered by activation of the downstream kinase ERK in an epithelial cell type called vulval precursor cells (VPCs). LIN-45 degradation is mediated by the E3 ubiquitin ligase SEL-10 and requires a conserved Cdo4-phosphodegron (CPD) sequence located in Raf proteins. In our genetic screen, we identified a mutation, *cov19*, that causes stabilization of LIN-45 protein in VPCs. We will present evidence that *cov19* is a loss-of-function mutation in *ufd-2*, which encodes a conserved ubiquitination factor UFD-2/UBE4B. We find that UFD-2 acts cell-autonomously to promote LIN-45 protein degradation. However, unlike SEL-10, UFD-2 may not regulate LIN-45 protein via the CPD motif. Because UFD-2 is known to act in a chaperone-mediated cytoplasmic unfolded protein response, we are now using a candidate mutant approach to determine whether LIN-45 is regulated by this mechanism. Finally, we find that loss of *ufd-2* enhances phenotypes caused by a mutant, activated form of LIN-45, strongly suggesting that a UFD-2-mediated mechanism may be important to inhibit mutant forms of Raf found in human cancers.

### Introduction

- In *C. elegans*, vulva formation is controlled by the Ras-Raf-MEK-ERK kinase cascade in vulval precursor cells (VPCs).

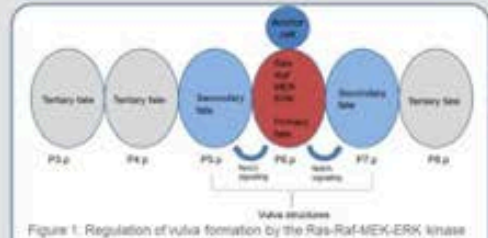


Figure 1. Regulation of vulva formation by the Ras-Raf-MEK-ERK kinase cascade. Circles indicate VPCs.

- In wild type, the *C. elegans* Raf ortholog LIN-45 is degraded in P6.p and not degraded in P5.p and P7.p during the L3 stage.
- In a mutant lacking SEL-10 (a conserved E3 ubiquitin ligase), the LIN-45 protein is stabilized (de la Cova and Greenwald 2012).




Figure 2. Degradation of YFP-LIN-45. These images show VPCs in the L3 stage after one cell division. A: LIN-45 degraded in wild type. B: LIN-45 stabilized in a SEL-10(0) mutant.

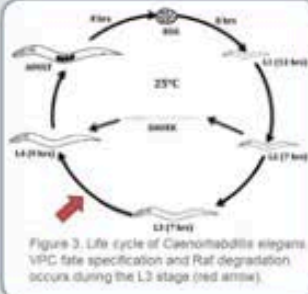


Figure 3. Life cycle of *Caenorhabditis elegans*. VPC fate specification and Raf degradation occurs during the L3 stage (red arrow).

### Methodology

#### Mutagenesis screening

- We generated a transgenic *C. elegans* strain which expresses an activated mutant form of the *C. elegans* Raf ortholog LIN-45(V627E).
- The activated LIN-45(V627E) mutation results in overactivation of the ERK signaling pathway. In humans, this can result in cancer. In *C. elegans*, it can cause ectopic ERK activation and the multivulva (Muv) phenotype.
- We performed a mutagenesis screen on the LIN-45(V627E) strain.
- We screened for mutants with enhancement of the multivulva phenotype, an indicator of highly activated Raf.

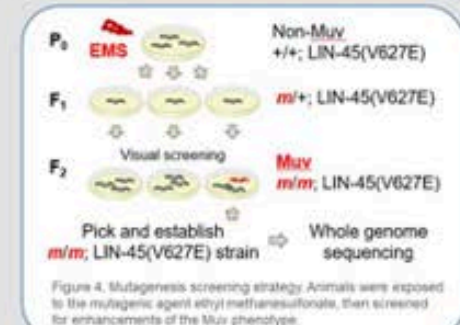


Figure 4. Mutagenesis screening strategy. Animals were exposed to the mutagenic agent ethyl methanesulfonate, then screened for enhancements of the Muv phenotype.

#### Variant mapping

- One of the variants identified was mapped to chromosome II using a SNP mapping technique with a polymorphic strain (Dotsidou, Jemault, and Poole 2016).
- Within this region, we found a premature stop in E3/E4 ubiquitin ligase *ufd-2*. We named this allele *cov19*.
- We also acquired an independent deletion allele of *ufd-2* from the *Caenorhabditis* Genome Center named *tm1380*.

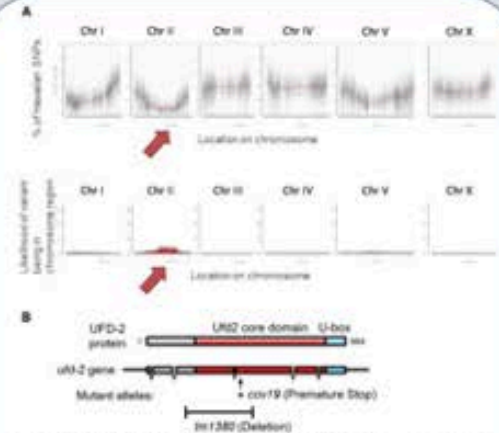


Figure 5. Mapping of *ufd-2*. Arrows indicate region on chromosome 2 where the SNP mapping showed linkage to the mutant phenotype. A: Top graphs indicate percentage of Hawaiian SNP. Bottom graphs indicate the likelihood of the mutant being in the region. B: Characterization of the UFD-2 locus with our *cov19* allele and the CGC tm1380 allele.

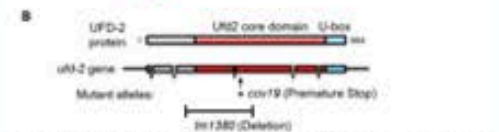


Figure 6. Schematic of known interaction between UFD-2 and CHN-1 in the role of myosin degradation.

### Results

#### ufd-2 is required for degradation of the Raf ortholog LIN-45

- In wild type, LIN-45 protein is degraded in P6.p post-transcriptionally (de la Cova and Greenwald 2012).
- Loss of function of *ufd-2* results in stabilization of LIN-45 protein in P6.p.
- The deletion allele and our premature stop mutant both stabilized LIN-45.
- We generated a transgenic strain that expresses UFD-2(+) specifically in VPCs.
- UFD-2(+) rescues the *ufd-2(tm1380)* mutant.

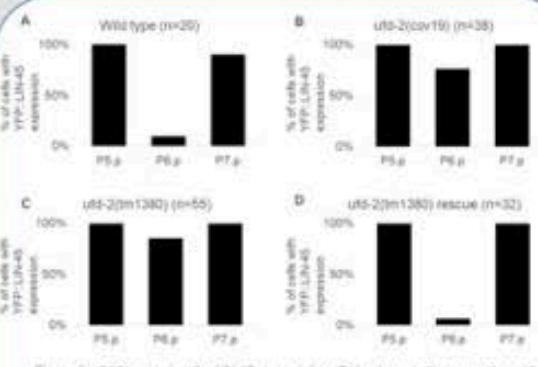


Figure 6. *ufd-2* is required for LIN-45 degradation. Data shown is the percentage of cells that are positive for YFP-LIN-45. A: wild-type control where LIN-45 protein is degraded in P6.p. B: *ufd-2(tm1380)* premature stop mutant. C: *ufd-2(tm1380)* deletion allele. D: *ufd-2(tm1380)* with rescuing transgene.

#### LIN-45 degradation does not require CHN-1/CHIP

- The role of CHN-1/CHIP, a protein known to interact with UFD-2 in the degradation of unfolded myosin chains, was investigated.
- We hypothesized that CHN-1 might be required for LIN-45 protein degradation.

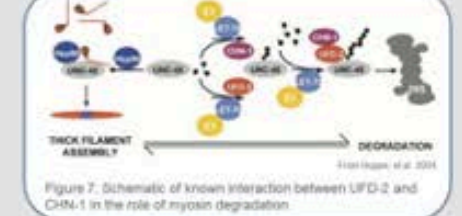


Figure 7. Schematic of known interaction between UFD-2 and CHN-1 in the role of myosin degradation.

- The deletion allele *chn-1(by155)* did not result in increased stabilization of LIN-45.
- A double mutant of *chn-1(by155)* and *ufd-2(tm1380)* did not significantly increase the frequency at which LIN-45 is stabilized (p=0.2580, Fisher's exact test).

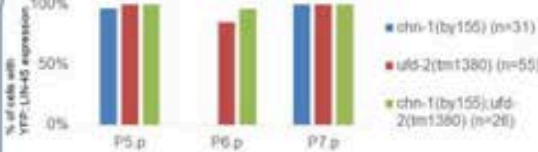


Figure 8. Raf/LIN-45 degradation does not require CHN-1/CHIP. LIN-45 degradation in VPCs in *ufd-2(tm1380)*, a known deletion of *chn-1(by155)*, and a *ufd-2(tm1380);chn-1(by155)* double mutant.

### Conclusion

- ufd-2* is required for Raf/LIN-45 degradation.
- Loss of function of *ufd-2* results in stabilization of Raf/LIN-45 protein in P6.p.
- Loss of function of *chn-1* does not result in increased Raf/LIN-45 stabilization in P6.p.

### Future directions

- We will perform a deletion analysis within the Raf/LIN-45 protein to determine where *ufd-2* interacts with Raf.
- We will test whether UFD-2 requires the same degron sequence that is required by SEL-10.
- We will obtain mutants in other pathways known to degrade proteins. These include chaperone-mediated autophagy, general autophagy, selective autophagy.

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Show examples of key experiments.

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What is the main goal of the research and the research  
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Figure legends with specifications of type of experiment,  
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In a nutshell (graphical model)

Acknowledgements

Grant funding, research programs, laboratory, mentors

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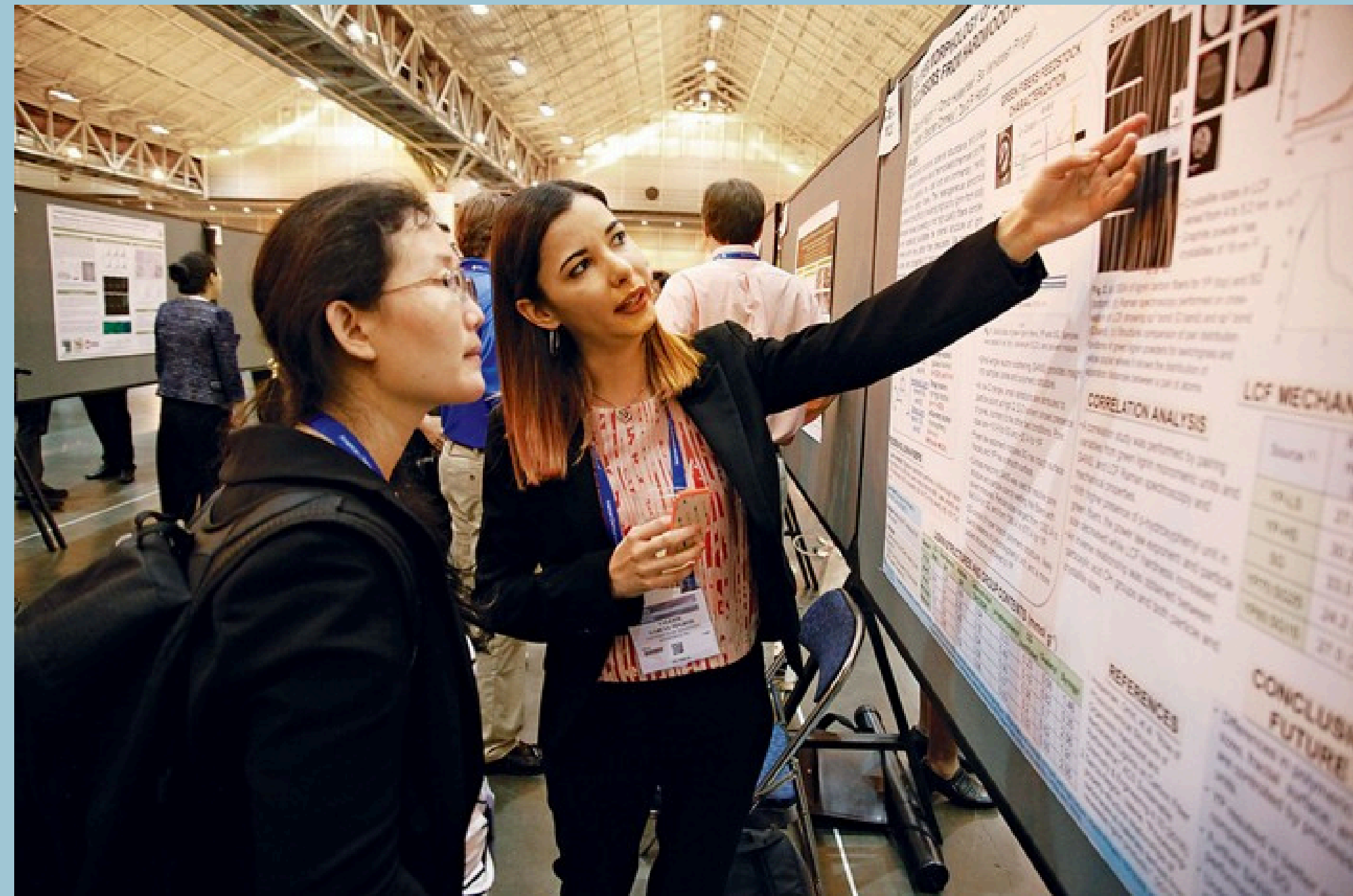


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# 06. Presenting your research



# 01

**Know what to say**

for each figure and  
**transitions** between  
sections

# 02

**Know all the details of  
your research**

# Knowledge



## 03

### **Practice for your audience**

Walk people through your poster in about 1 min; 5 min; 10 min

## 04

### **Master questions (specific and open ended)**

Tell me about your research; how does this relate to findings in the field; what are the implications for the future

# Practice

**Be enthusiastic about your work**

**Start a conversation asking questions**

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# Resources

# Thank you!

## Questions?



