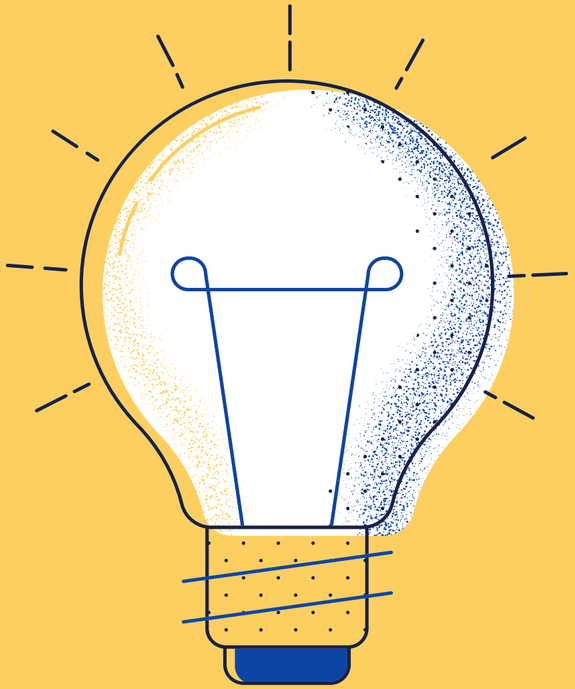
Pancakes & Posters

3/12/2020

Presented by

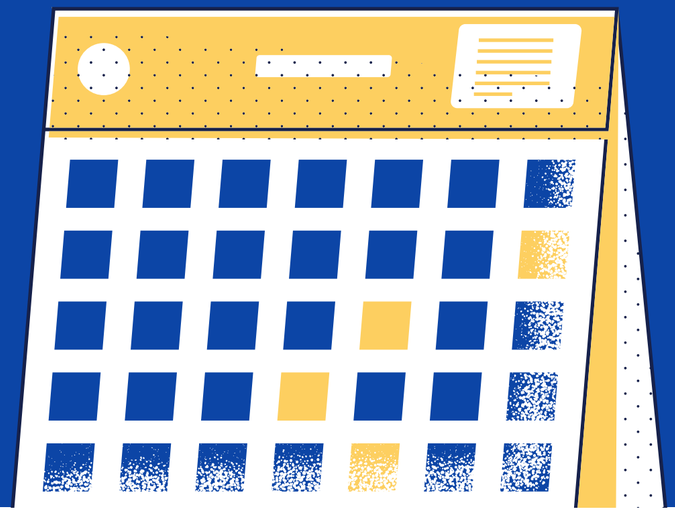


Before making the poster

Poster Content/Style

Presentation tips

BEFORE MAKING THE POSTER



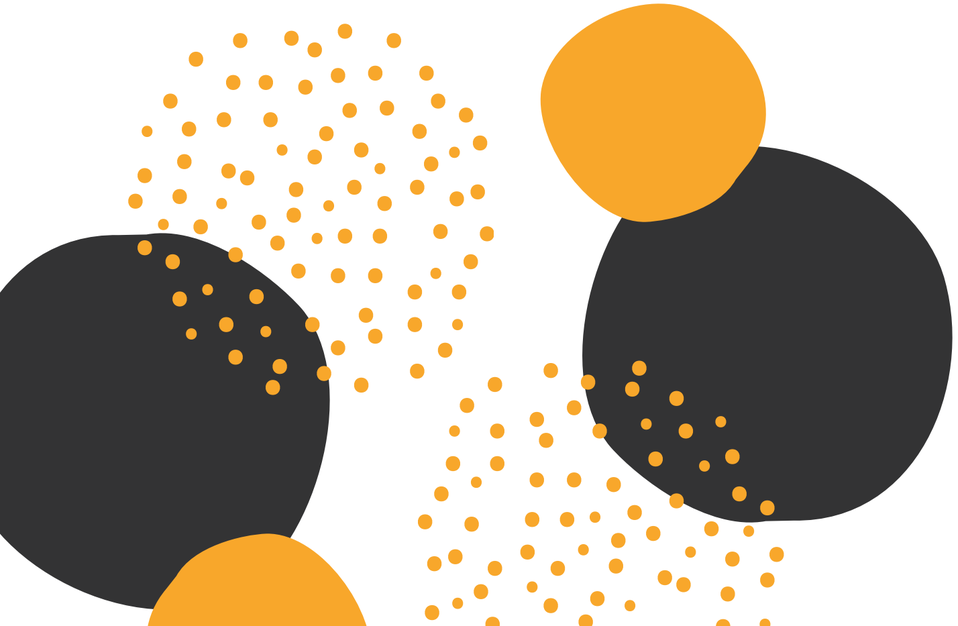
- Check poster size requirements
- Consider the context for your poster
- Use appropriate terminology for audience’s knowledge level

STYLE

- Color
  - No red/green
  - No distracting colors
- Font
  - Check font sizes by printing sample sheet
  - Same font throughout
- Layout
  - Pick a direction (up/down, left/right)

CONTENT

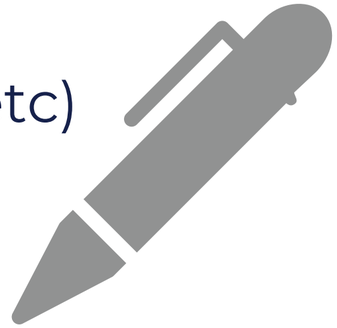
What to Include



- Title
- Introduction/Background
- Methodology
- Results
- Discussion
- Conclusion
- References
- Acknowledgements

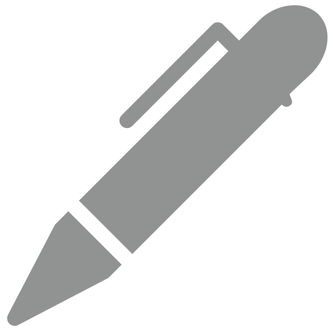
CONTENT

Title	• Descriptive and encompasses the scope of the project
Abstract	• A brief summary of the research project
Intro/Background	• Purpose or objective of the study • Significance of the study
Methodology	• Describe procedures and methods used for the study
Results	• Report results of the study (statistics, figures, etc)



CONTENT

Discussion	• Interpret the meaning of results
Conclusion	• Major findings • Suggestions for future studies • Relevance of study
References	• Sources/resources referenced on poster
Acknowledgements	• Faculty and staff who supported you



CONTENT: PROTOCOL

Bad Example

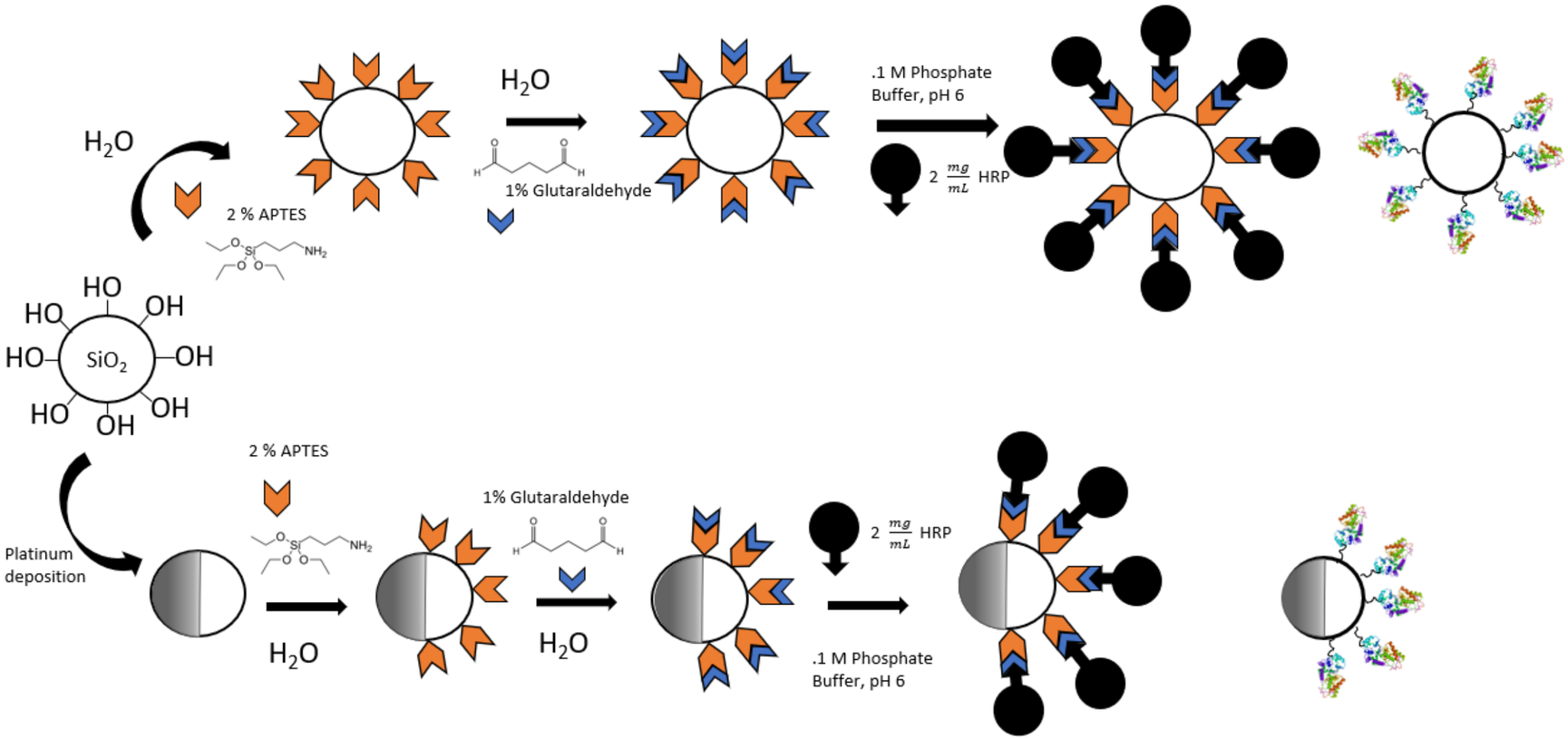
Lisiting out protocol

1. Add 2% APTES, mix for 12 hours
2. Wash 5x with DI water
3. Add 1% glutaraldehyde, mix for 30 minutes
4. Wash 3x with DI water
5. Mix with 2 mg/mL enzyme for 4 hours
6. Wash 3x with DI water

CONTENT: PROTOCOL

Good Example

Using a graphic



HOW TO PRINT

Multimedia Studio Library

- 4th floor Crossland

Paper and Clay

- 3rd floor Student Center

Department

- Petit
- BME

Presenting

PRESENTING

- Dress professionally
  - Can even match your poster
- Practice pitch
  - 1-2 min max
- How to start a pitch
  - Bullet point list
  - Major takeaways
  - Relevance

Poster Examples

Poster 1

Elevated levels of Interleukin-8 in non-small cell lung cancers induce cell survival during chemotherapy

Andrew Pan [1,2], Jonathan Chen [2], Rong Fan [2]

[1]Old Saybrook Senior High School [2]Yale University, Department of Biomedical Engineering

Abstract

Non-small cell lung cancer (NSCLC) is a particular variant of lung cancer that equally affects smokers and non-smokers. Typical treatment of NSCLC involves the use of tyrosine kinase inhibitors. Whilst initial treatment is successful, drug resistance is inevitable in the majority of cases[1]. Several studies have implicated elevated levels of Interleukin-8 to be linked to cancer. This project investigated how Interleukin-8 could be instigating drug resistance in NSCLC cell lines. The data gathered showed evidence that Interleukin-8 was linked to greater cell viability and health, as well as showed higher levels of Interleukin-8 in drug treated cell lines. In conclusion, Interleukin-8 is most likely a factor in inducing drug resistance, by promoting cell survival, enabling the cells to develop mutations and become resilient to chemotherapeutic agents.

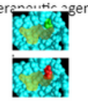


Figure 1.1 Mutation in the EGFR domain inhibits drug binding [2]

Introduction

Interleukin-8 (IL-8), also known as CXCL8, is a chemokine produced by macrophages and other cell types such as epithelial cells and endothelial cells. Interleukin-8 normally functions as a pro-inflammatory agent, that is also involved in chemotaxis, initiating the migration of immune cells toward the site of an infection. Interleukin-8 activates multiple intracellular signal transduction pathways, primarily through G-protein linked cell receptors CRCX1 and CRCX2. Recent studies have shown however, that these pathways may be interacting with the MAPK-ERK pathways to promote tumor growth and angiogenesis [3]. Interluekin-8 has also been linked to enabling drug resistance in tumors exposed to chemotherapeutic agents. Despite its role as an inflammatory agent, the evidence from this study and several others suggests that elevated levels of IL-8 in cancer plays a role in sustaining and spreading the tumor microenvironment

Hypothesis

Non-small cell lung cancer cells will raise Interleukin-8 levels in response to drug treatment in an effort to induce cell survival.

Methods

- Two NSCLC Cell Lines ,PC-9 and its drug-resilient derivative, PC-9BR, were obtained from Vanderbilt University. They were split into two groups, a control group, and a drug-treated group. Each group was cultured in a 10% RPMI cell medium and incubated at 37° Celsius. The drug treated groups were treated with 3 um of the tyrosine kinase inhibitor erlotinib.

- A unique single cell capture device designed by Professor Rong Fan was used to isolate individual cells in a population and analyze their unique proteome [4]. This chip was a two part system involving an anti-body barcode and a PDMS capture chip, containing 14 columns with 220 wells per column. The antibody barcode contained up to twenty antibodies running in parallel to capture and read proteins. Over 2,000 individual cells were analyzed.

- Cells were loaded in the chip via micropipette injection. The chips were then imaged in the chip at 0h and 24h stages after loading them into the chip. This allowed us to observe physiological changes that occurred and match them to protein secretions. IsoPlexis Software was used to analyze the data.

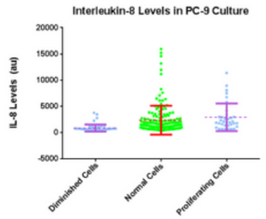
Qualitative Observations

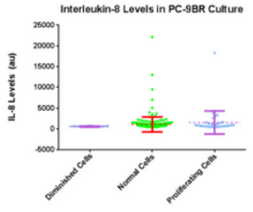
Normal Cells-Characterized with no major deformities or shape changes.

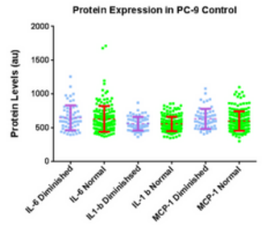
Diminished Cells– Characterized as having a more unhealthy appearance, with less-defined membranes and a shriveled look.

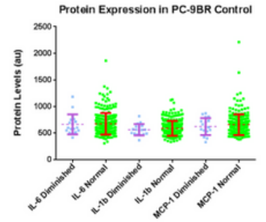
Results

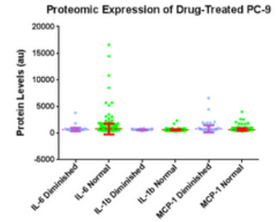
Each point is a single cell

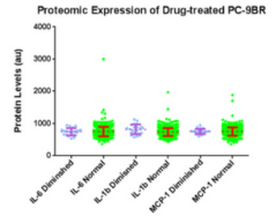


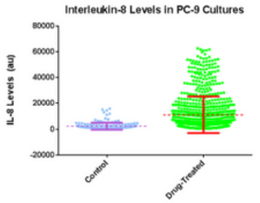


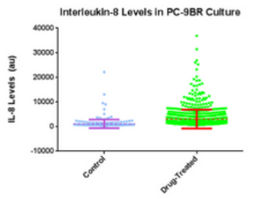


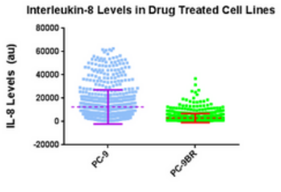












Determining High Producers : High-producers are cells that are expressing the highest levels of IL-8 in a given population. To determine high-producers, the following equation was used

(Given value)-(Control group mean + 2 Standard deviations) > 0

Drug Treated PC-9	Drug Treated PC-9BR	
833	773	N
311	136	High Producers
37.334%	17.594%	% High Producers

Discussion

1. **Increased IL-8 levels in healthier cells indicate that IL-8 plays a role in maintaining cell viability and health**. IL-8 Levels taken from diminished cells in control groups were significantly lower than from normal cells. Control groups were used due to the possibility of erlotinib affecting IL-8 expression. This pattern of decreased expression in diminished cells was not found in other protein that were also measured. Expression levels of other proteins remained relatively stable between the diminished cells and the normal cells.IL-8 was also expressed more highly in cells that divided into two. Cell proliferation is a major factor in cell viability, and healthier cells will tend to divide more.
2. **Cells higher levels of IL-8 to induce cell survival in the presence of the tyrosine kinase inhibitors.** Cells will express higher levels of IL-8 because the presence of drug compounds places stress on the tumor population and reduces cell viability. Because IL-8 appears to be linked to cell viability, high IL-8 levels can possibly mitigate the effect of chemotherapy on tumors. This can be further supported by the higher levels of IL-8 in drug-treated non resistant strains when compared to drug-treated resistant strains. Non-resistant strains are more susceptible to treatment, which is why they would require higher levels of IL-8 to survive. It is probable that IL-8 does not directly cause drug resistance as supported by other papers, but instead facilitates it by enabling cell survival.
3. **Only certain cells in the culture that are secreting very high levels of IL-8**. In the drug-treated cultures, only a select amount of cells were expressing significantly high levels of IL-8. These "high-producers" were determined by subtracting the mean level of expression plus two standard deviations of the control group from the given expression level of a single cell in the drug-treated group. These cells are most likely the ones that are responsible for reseeding and restarting relapsed tumors after treatment. Additionally, more high producers are found in the non-resilient strain, which again supports that IL-8 is enabling cell survival. While Waugh proposed the use of IL-8 blockers in individualized treatments of cancer [2] we propose that this treatment would need to be specialized and targeted toward "high-producers" in order to be effective

References

1. Godin-Heymann, N., Ulkus, L., Brannigan, B. W., McDermott, U., Lamb, J., Maheswaran, S., . . . Harber, D. A. (2008). **The T790M "gatekeeper" mutation in EGFR mediates resistance to low concentrations of an irreversible EGFR inhibitor.** *Molecular Cancer Therapeutics*.

2. Clark, Jennifer; Cools, Jan; Gilliland, D. Gary (2013): **Structural Models of EGFR Showing the T790M Resistance Mutation** Figure_2.tif. PLOS Medicine.10.1371/journal.pmed.0020075.g002.

3. Waugh, D. J., & Wilson, C. (2008).**The Interleukin-8 Pathway in Cancer.** *Clinical Cancer Research*.

4. Fan, R., Heath, J., Wei, W., Shin, Y. S., Ma, C., Wang, J., & Elitas, M.(2013).**Microchip platforms for multiplex single-cell functional proteomics with applications to immunology and cancer research.** *Genome Medicine*

Acknowledgements

I would like to thank Dr. Fan for hosting me in his lab, and Jonathan Chen, a graduate student and PhD candidate of Yale University, for serving as my mentor and guiding me throughout my project.

Poster 2

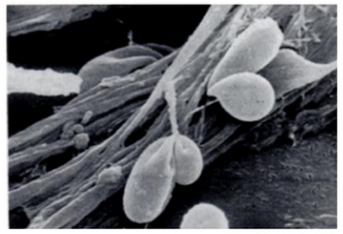
Real-time Visualization of Shear-dependent Erythrocyte Deformation into Schistocytes using Single Micron Microfluidics

[1]Wallace H. Coulter Department of Biomedical Engineering, Georgia Institute of Technology and Emory University, Atlanta, GA; [2]Department of Pediatrics, Aflac Cancer Center and Blood Disorders Service of Children's Healthcare of Atlanta, Emory University School of Medicine, Atlanta, GA; [3]School of Electrical and Computer Engineering, Georgia Institute of Technology, Atlanta, USA

Background

Dynamic Mechanical Microenvironment: Red blood cells (RBC) experience a wide variety of shear stresses and pressures, especially in prothrombotic conditions in the microvasculature. This introduces pathologic fibrin matrices that affect the fluidic microenvironment and create physical obstacles in the blood stream. This introduces mechanical forces that act as biophysical cues affecting the function and form of cells, specifically forming abnormal RBC morphologies.

Clinical Relevance: The presence of mechanically damaged erythrocytes like schistocytes in blood smears are used to detect prothrombotic conditions, such as disseminated intravascular coagulation. However, the underlying biophysical mechanisms of deformation remain largely unknown.



Bull and Kuhn, Blood, 1970

Figure 1. Traditional "hanged red blood cell" mechanism of schistocyte production.

Traditional Explanation: A widely cited 1970 SEM study describes schistocyte formation occurring when "rapidly moving red cells encounter fine fibrin strand" resulting in a "hanged red blood cell" and eventual schistocyte formation (**Figure 1**).

Ciciliano, Abbaspour Woodall, Wu, Bakir, Lam, Lab on a Chip, 2017

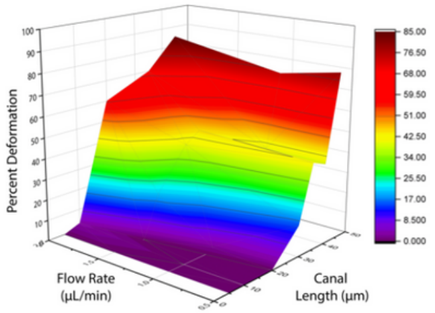
Figure 2. Microfluidic canal constricting RBC resulting in schistocyte-like formation identified with green arrow.

Potential Additional Mechanism: Recent work suggests that increasing RBC microconstriction in microfluidics increases the plastic deformation of RBC (**Figure 2**). **We hypothesized that increasing shear rate in conjunction with canal length would increase the formation of altered erythrocytes including schistocytes.**

Erythrocytic Deformation Increases with Shear

Erythrocytes emerge progressively fragmented as shear rate increases at controlled canal lengths. Degree of plasticity and severity also increases (Figure 4).

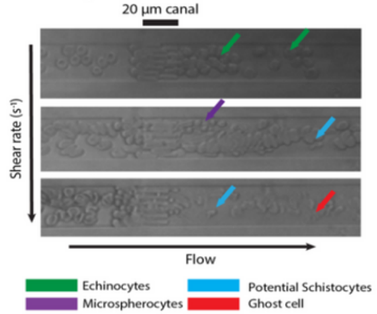
This supports our hypothesis that increasing shear rate results in the increase of altered erythrocytes formed, real-time results will be confirmed via RBC staining of device effluent moving forward.



Potential Schistocyte Formation Region

Figure 5. 3D graph of percent plastic deformation of erythrocytes (z-axis and color spectrum) as a function of canal lengths in µm (x-axis) and flow rate in µL/min (y-axis). Flowrate is directly related to shear rate (s^{-1}).

Figure 4. Images of real-time deformation of RBC at increasing shear rates as canal length is maintained.



Increasing shear rate in conjunction with canal length increases the percent of RBC deformation (Figure 5).

Increasing shear rate at lower canal lengths of 5 µm, 10 µm and 15 µm resulted in little or no erythrocyte fragmentation. However increasing shear rates at canal lengths of 20 µm resulted in reversible burr cell formation at low shear rates, and then increased fragmentation to potential schistocyte and ghost cell formation at the highest shear rates.

Microfluidic System and Experimental Methods

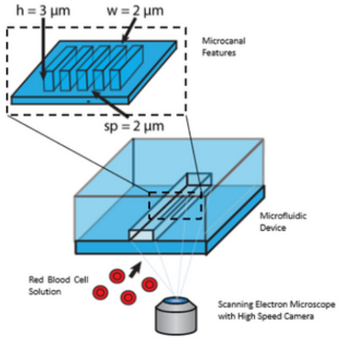


Figure 3. Model of experimental microfluidic system. Each device is comprised of five columns that form "microcanals" that are 2 µm wide and 3 µm tall with lengths varying between 5 µm and 45 µm.

Microfluidic Device: Our microfluidic devices are fabricated via electron beam lithography and contain "microcanals" within a long straight channel, simulating the physical dimension of in vivo microvascular constrictions (**Figure 3**).

Altering Shear Rate: Shear rate was varied between 30,000 to 120,000 dyne/cm^2 at canals.

Video Microscopy: RBC deformation was observed and recorded in real-time using high speed video microscopy.

Novelty: To our knowledge, there are no other systems allowing for visual analysis of erythrocyte fragmentation through single micron microfluidic constrictions.

Our microfluidic platform also decouples biochemical from biophysical cues, allowing us to study only physical interactions without confounding variables.

Conclusions and Future Work

Our results suggest that shear rate and constriction time work synergistically to affect plastic erythrocyte deformation into a variety of abnormal morphologies typical in thrombotic microangiopathic disorders.

Moving forward, we will study how introducing biochemical cues affect erythrocyte fragmentation, as well as develop novel microfluidic features to create an artificial fibrin mesh to further simulate prothrombotic mechanical conditions (**Figure 6**).

These findings will lead to an improved understanding of microangiopathic pathological processes and aid in developing diagnostic assays in the future.

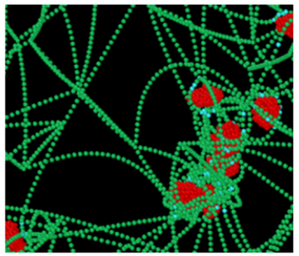


Figure 6. Model of artificial fibrin network based on fibrin crosslink location derived from confocal images of in vitro fibrin networks. Developed in collaboration with Alexander Alexeev in the Georgia Tech Complex Fluids Modeling and Simulation Group.

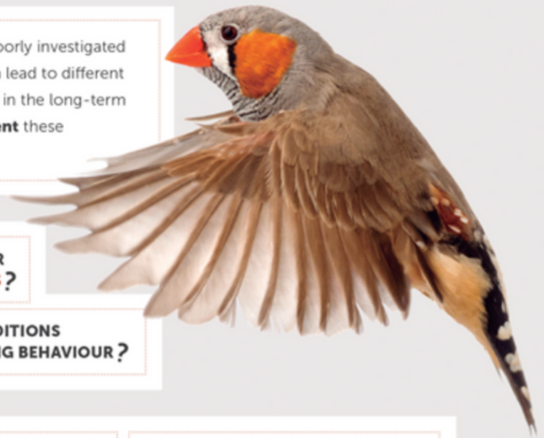
Poster 3

FLEXIBLE FORAGING BEHAVIOUR IN WILD ZEBRA FINCHES AND ITS RELATION WITH TEMPERATURE

CATERINA FUNGHI, LUKE MCCOWAN, WIEBKE SCHUETT AND SIMON GRIFFITH

INTRODUCTION

- **Foraging as behavioural trait** has been poorly investigated
- **Fluctuation** in enviromental contitions can lead to different behavioural strategies being equal in fitness in the long-term
- In **extreme and unpredictable environment** these fluctuations are more pronounced



QUESTIONS

IS INDIVIDUAL FORAGING BEHAVIOUR CONSISTENT IN WILD ZEBRA FINCHES?

HOW DO THE ENVIRONMENTAL CONDITIONS (TEMPERATURE) INFLUENCE FORAGING BEHAVIOUR?

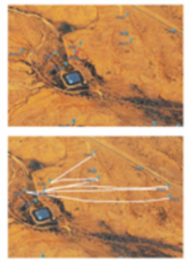
MATERIALS AND METHODS

16 FEEDERS RANDOMLY LOCATED (I.E. 2 TRIALS)

Zebra finches were PIT - tagged and foraging behaviour monitored using a decoder-antenna-feeder system over 3 weeks.

PC ANALYSIS ON DAILY FORAGING BEHAVIOUR OF 72 ADULTS

52% variation	PC1 foraging
Visits	0.3
Distance travelled	0.58
Nb. feeders	0.6
Feeder fidelity	-0.46



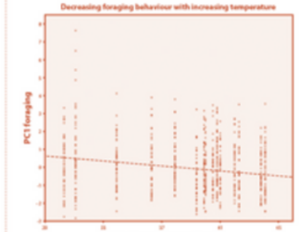
Example of 2 individuals showing different foraging behaviour. Dots represent feeders located 800m around a dam

LOW PC1 foraging:
- visits per feeder
- distance travelled
- feeders visited
+ fidelity

HIGH PC1 foraging:
+ visits per feeder
+ distance travelled
+ feeders visited
- fidelity

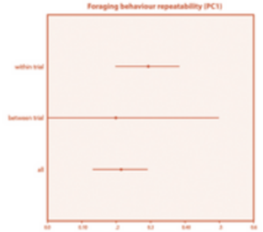
RESULTS AND DISCUSSION

IN EXTREME ENVIRONMENTS FORAGING BEHAVIOUR WAS INFLUENCED BY TEMPERATURE



GLMM: P<0.001 , N=68. Negative relation between foraging behaviour (PC1) and Temperature (°C)

CHANGED ENVIRONMENT LED TO FLEXIBLE FORAGING BEHAVIOUR > OPPORTUNISM?



Repeatability (LMM) calculated considering all days of all trials, between and within trials.

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MACQUARIE University

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