



PRESENTING YOUR RESEARCH

Dimitra Bourboulia, PhD Assistant Dean for UME and GME, Director, Office of Research for Medical Students, July 08 2020





SUMMER REPORT – September

SUMMARY includes

- One abstract

(two formats depending on the science),

and <u>- One visual</u> (email us and we add it to your file)

POSTER PREPARATION

* Powerpoint slides put together





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SUMMARY – Abstract – 300 – 350 words:

Title (10 – 15 words) **Author information** (Spell out names)

Background/Intro Experimental Procedures Results Conclusions/Future Directions

No images/tables/references/links in the abstract





MAIN TEXT – Format (1)

https://cbs.umn.edu/sites/cbs.umn.edu/files/public/downloadsAnnotated Nature abstract.pdf

One or two sentences providing a basic introduction to the field, comprehensible to a scientist in ______ any discipline.

Two to three sentences of more detailed background, comprehensible to scientists in related disciplines.

One sentence clearly stating the general

problem being addressed by this particular

study.

One sentence summarising the main result (with the words "here we show" or their equivalent).

Two or three sentences explaining what the main result reveals in direct comparison to what was thought to be the case previously, or how the main result adds to previous knowledge.

One or two sentences to put the results into a more general context.

Two or three sentences to provide a broader perspective, readily comprehensible to a scientist in any discipline, may be included in the first paragraph if the editor considers that the accessibility of

During cell division, mitotic spindles are assembled by microtubule-based motor proteins^{1.2}. The bipolar organization of spindles is essential for proper segregation of chromosomes, and requires plus-end-directed homotetrameric motor proteins of the widely conserved kinesin-5 (BimC) family³. Hypotheses for bipolar spindle formation include the 'push-pull mitotio muscle' model, in which kinesin-5 and opposing motor proteins act between overlapping microtubules^{2,4,5} However, the precise roles of kinesin-5 during this process are unknown. Here we show that the vertebrate kinesin-5 Eg5 drives the sliding of microtubules depending on their relative orientation. We found in controlled in vitro assays that Eg5 has the remarkable capability of simultaneously moving at "20 nm s" towards the plus-ends of each of the two microtubules if crosslinks. For anti-parallel microtubules, this results in relative sliding at "40 nm s⁻¹, comparable to spindle pole separation rates in vivo². Furthermore, we found that Eg5 can tether microtubule plus-ends, suggesting an additional microtubule-binding mode for Eg5. Our results demonstrate how members of the kinesin-5 family are likely to function in mitosis, pushing apart interpolar microtubules as well as recruiting microtubules into bundles that are subsequently, polarized by relative sliding. We anticipate our assay to be a starting point for more sophisticated in vitro models of mitotic spindles. For example, the individual and combined action of multiple mitotic motors could be tested, including minus-enddirected motors opposing Eg5 motility. Furthermore, Eg5 inhibition is a major target of anti-cancer drug development. and a well-defined and quantitative assay for motor function will be relevant for such developments.

the paper is significantly enhanced by their inclusion. Under these circumstances, the length of the paragraph can be up to 300 words. (The above example is 190 words without the final section, and 250 words with it).





MAIN TEXT – *Format* (2) http://clincancerres.aacrjournals.org/content/24/17 Supplement/PR04

Background: Tyrosine kinase inhibitors (TKI) have yielded promising responses in non-small cell lung cancer (NSCLC) with EGFR mutations and ALK translocations. However, these and other targeted therapies are limited by intrinsic and acquired drug resistance. The previous study from our group investigated tumor autonomous resistance mechanisms by developing patient-derived cancer models (PDCs). In this study, we aimed to decipher the nonautonomous resistance mechanisms via tumor microenvironment by developing patient-derived fibroblast (PDF) models.





MAIN TEXT – Format (2)

Method: Cancer-associated fibroblast cell lines are established directly from individual EGFR mutant NSCLC biopsies. These cell lines, as representative of each patient's tumor microenvironment, are further subjected to functional analysis. An imaging-based high-throughput platform is developed to screen for nonautonomous resistance by co-culturing PDC and PDF models in vitro. In the parallel, two independent approaches are performed to further identify mechanisms underlying the nonautonomous resistance. These include a drug screen to determine the pathway maintaining the cancer cells' survival, and a secretomic analysis on the PDFs to identify the plausible cytokine(s) responsible for the resistance.





MAIN TEXT – Format (2)

Result: By co-culturing screening, nonautonomous resistance can be found in a wide spectrum of models. The subsequent drug screen reveals both a canonical HGFdependent and novel HGF-independent mechanisms contributing to EGFR TKI resistance. Both of these can be explained by the PDF's variable cytokine secretion and can be overcome by specific therapeutic combinations. Moreover, the microenvironment-driven EGFR TKI resistance has also been validated in vivo. The prevalence of the identified cytokine is further tested in clinical specimens.





MAIN TEXT – Format (2)

Conclusion: PDFs provide a new avenue to explore nonautonomous resistance for targeted therapy. Applying this approach, we identified both the canonical HGF-dependent and novel HGF-independent mechanisms that putatively confer EGFR TKI resistance. Taking EGFR TKI therapy as a paradigm, these findings will be valuable to optimize targeted therapy and to inform the design of personalized pharmaceutical interventions.





SUMMER REPORT – Early September

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TITLE

Authors:

KIDNEY CANCER Objective of project Objective of project Objective of project	ResultsEach result will include1 -A brief finding and a short description23-3-4-4-5-5-6-7-8-10-
 Methods – Approach Bullet points describe the methods Example: a cohort of patients Samples collected ELISA, WB, Protein extraction Statistics 	Conclusion/Future DirectionsImage is a good ideaKIDNEY CANCER







SUMMER REPORT - Submission

- <u>Medhub Evaluation report</u>
 - Copy/paste your Abstract
 - Email Abstract+visual to our office
 - Early September





POSTER PREPARATION

* Powerpoint slides put together







POSTER BASICS (1)

- Provide a visual description of your work
- Focus on one message only
- Highlight key points





POSTER BASICS (2)

- Title is attention grabbing short, sharp and compelling
- Keep text minimal; use bullet points instead of whole sentences
- Use column format
- Use visuals
- Use charts for tabulated data
- Ensure is readable from 4-6 feet away.
- Text size at least 24 points and 36 for headings
- Always check the sponsor's specifications
- Light background with dark letters is a safe choice
- Include results, conclusions, future directions
- Provide acknowledgements and references (smallest fonts)
- Obtain feedback when presenting your poster





POSTER PRESENTATION

- Make connections with peers and experts
- Obtain feedback when presenting your poster
- Promote your poster in advance
- Prepare and PRACTICE a concise, focused, 2-3 minute 'elevator pitch' summing up your work's key points and the importance
- Practice a 5-10 talk
 - What was the questions you are addressing
 - Data you generated
 - Conclusions and their meaning
- Handouts are useful for those who show interest
- When speaking look at the viewer not the poster





Preparing Your Summer Research Poster

Debbie Rexine & Sabra Snyder IMT Educational Communications