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The 4rd International Conference Of

Non-Lethal Roles of Cell Death Proteins

7 - 10 September 2026 — Madrid, Spain



The Ottawa
Hospital

L'Hôpital
d'Ottawa



מכון ויצמן למדע
WIZMANN INSTITUTE OF SCIENCE



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The Company of
Biologists



The 4rd International Conference Of Non-Lethal Roles of Cell Proteins

- Madrid

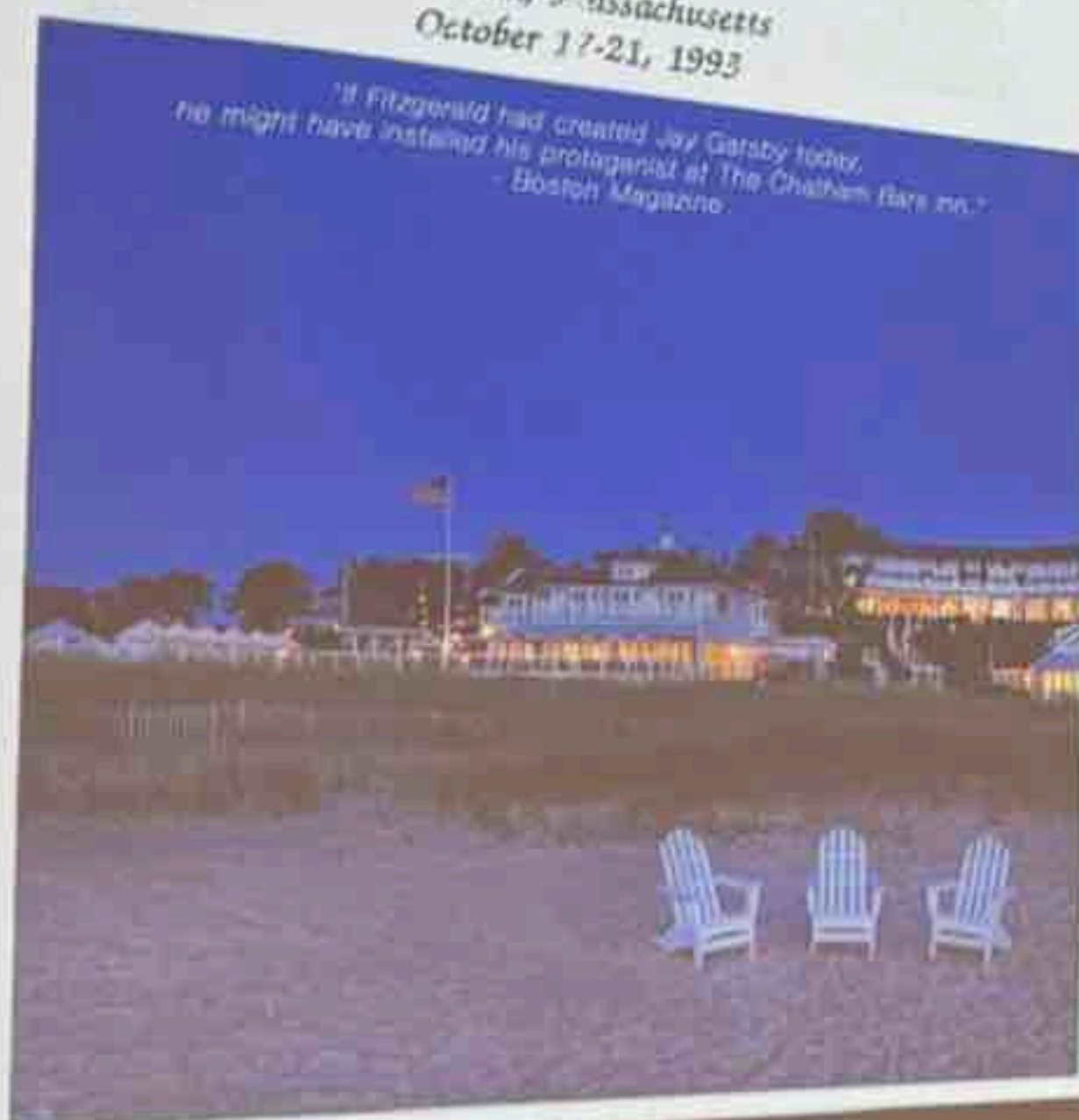




Cell Death in Cancer and Development

Chatham Bars Inn
Chatham, Massachusetts
October 17-21, 1993

"If Fitzgerald had created Jay Gatsby today,
he might have installed his protagonist at the Chatham Bars Inn."
- Boston Magazine



CELL DEATH IN CANCER AND DEVELOPMENT

CONFERENCE CHAIRPERSON

Alan H. Eastman
Dartmouth Medical School
Hanover, NH

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University of Vermont Medical School
Burlington, VT

John T. James
Johns Hopkins Oncology Center
Baltimore, MD

Andrew H. Wyllie
University of Edinburgh
Edinburgh, Scotland

SCIENTIFIC PROGRAM AND CONFERENCE SCHEDULE

(All slide sessions will be held in Dight Hall; poster sessions will be held in the South Lounge and Dight Hall and all events will be in the Main Dining Room unless otherwise indicated)

Sunday, October 17

REGISTRATION

KEYNOTE ADDRESS

Andrew H. Wyllie, University of Edinburgh, Edinburgh, Scotland, "The Regulation of Apoptosis by Cancer Genes."

DINNER

MIXER (South Lounge)

Monday, October 18

BREAKFAST

SESSION I: CELL DEATH AND DEVELOPMENT

Chairperson: H. Robert Horvitz, Massachusetts Institute of Technology, Boston, MA

8:00 a.m. to 9:40 a.m.

8:40 a.m. to 10:20 a.m.

10:20 a.m. to 10:40 a.m.

10:40 a.m. to 11:20 a.m.

11:20 a.m. to 12:00 noon

LUNCH

SESSION II: CELL DEATH IN THE PATHOGENESIS OF CANCER

Chairperson: John T. James, Johns Hopkins University, Baltimore, MD

1:00 p.m. to 4:40 p.m.

4:40 p.m. to 5:20 p.m.

COFFEE BREAK

5:20 p.m. to 5:40 p.m.

5:40 p.m. to 6:20 p.m.

6:20 p.m. to 7:00 p.m.

SELECTED ABSTRACT PRESENTATIONS

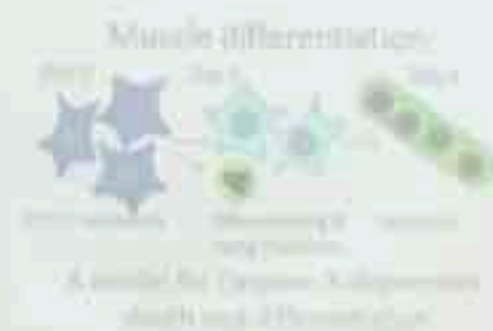
7:00 p.m. to 8:00 p.m.

8:00 p.m. to 9:30 p.m.

DINNER

POSTER SESSION A

How does caspase cleavage cause different fates?



Compare substrate cleavage in death and differentiation and ask: are there fate specific substrates?









Acknowledgements



Olivier Julien
Erik Gomez
Kolman Van Baar
Jasmar Patel

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Bill McManis
Patrick Connelly
Scott Taylor
Kate Vukobratovic
Roman Lambert
Fatemeh Fotouhi



Government of Alberta

Alberta Health Services

Alberta Innovates

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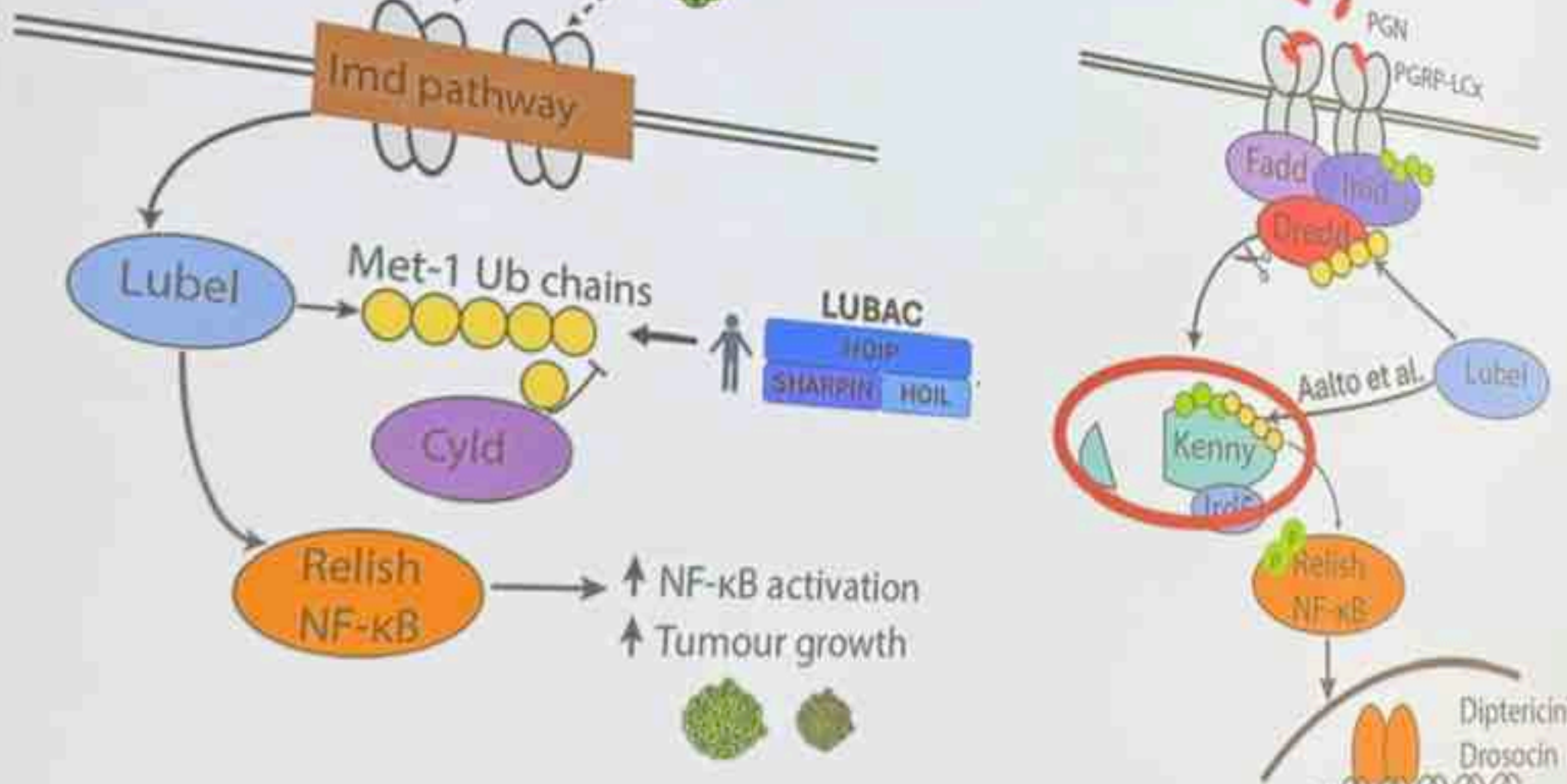
Alberta Innovates











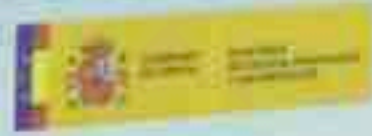
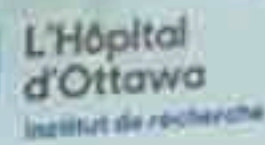
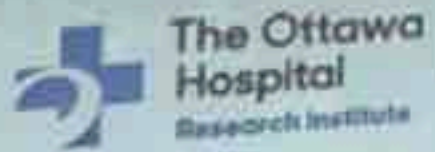






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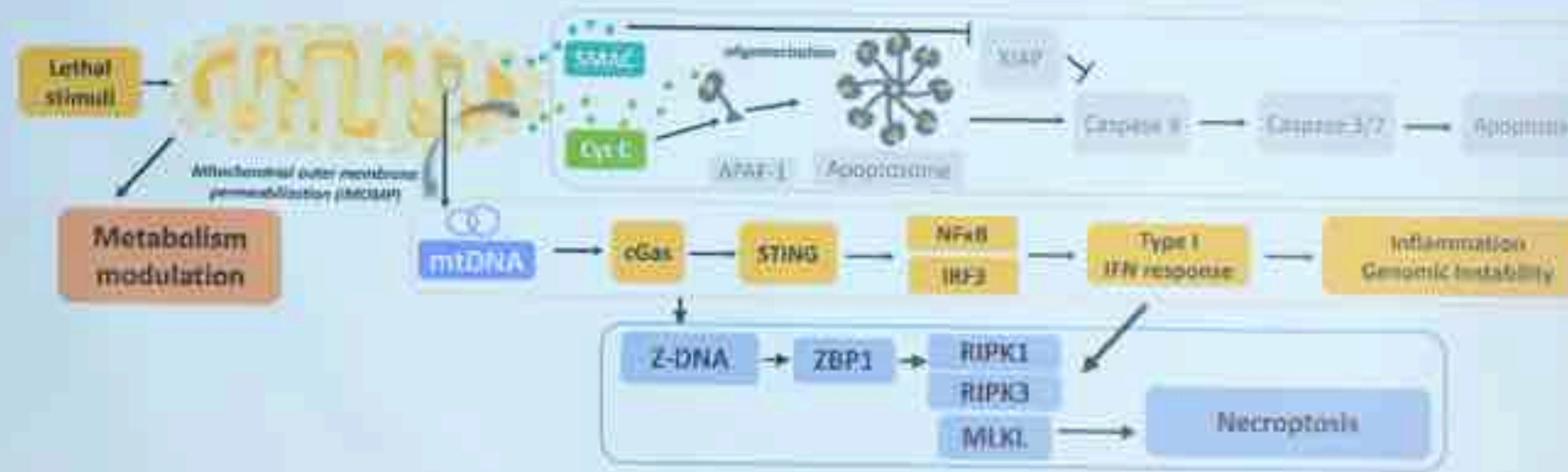
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What's next?

48 / 48



- How can APAF-1 downregulation lead to lymphoma? Direct mechanism or compensatory response?
- How do the cells die? Necroptosis hypothesis with or without Apoptosis?









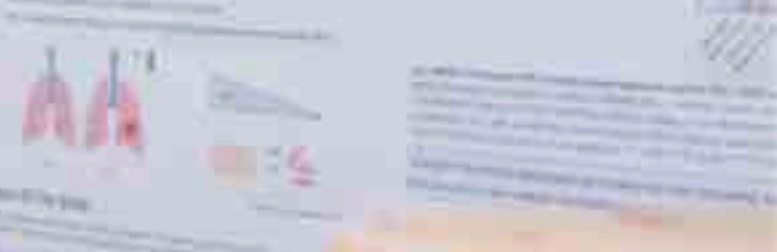




A Novel Chemical Disruptor of MDL-1/SOK Transmembrane Interact Epithelial-to-Mesenchymal Transition and Migration in Lung C

Author: [Name], Advisor: [Name], Institution: [Name]

Background: Epithelial-to-mesenchymal transition (EMT) is a process by which epithelial cells lose their polarity and cell-cell adhesion, and acquire a migratory, invasive phenotype. This process is regulated by various signaling pathways, including the MDL-1/SOK pathway. In this study, we investigated the role of MDL-1/SOK in EMT and migration in lung cancer cells.



Results: We found that MDL-1/SOK interaction is essential for EMT and migration in lung cancer cells. Treatment with a novel chemical disruptor of MDL-1/SOK interaction significantly inhibited EMT and migration, as measured by wound healing assays and transwell invasion assays.



Conclusions & Discussion: Our results demonstrate that MDL-1/SOK interaction is a key regulator of EMT and migration in lung cancer cells. The novel chemical disruptor of MDL-1/SOK interaction is a promising candidate for the treatment of lung cancer.

Future Prospects: Further studies are needed to investigate the mechanism of action of the chemical disruptor and its potential as a therapeutic agent.

Acknowledgments: We thank [Name] for their support and assistance.

References: [List of references]

CIPF



HIDDEN CASPASE 3 SUBSTRATES

Jaschke et al. • Olivier Jullien • Howard Fearnhead

Abstract: Caspase-3 is a key effector of apoptosis. It is activated by various stimuli, including DNA damage, oxidative stress, and growth factor withdrawal. Here, we have identified a set of novel substrates for caspase-3, which are involved in the regulation of cell cycle and DNA replication. These substrates are phosphorylated by cyclin-dependent kinases (CDKs) and are targeted for degradation by the ubiquitin-proteasome system. Our findings suggest that caspase-3 may play a role in the regulation of cell cycle and DNA replication, in addition to its role in apoptosis.



SEQUENCE

Substrate	Sequence
Substrate 1	1-100
Substrate 2	101-200
Substrate 3	201-300
Substrate 4	301-400
Substrate 5	401-500
Substrate 6	501-600
Substrate 7	601-700
Substrate 8	701-800
Substrate 9	801-900
Substrate 10	901-1000



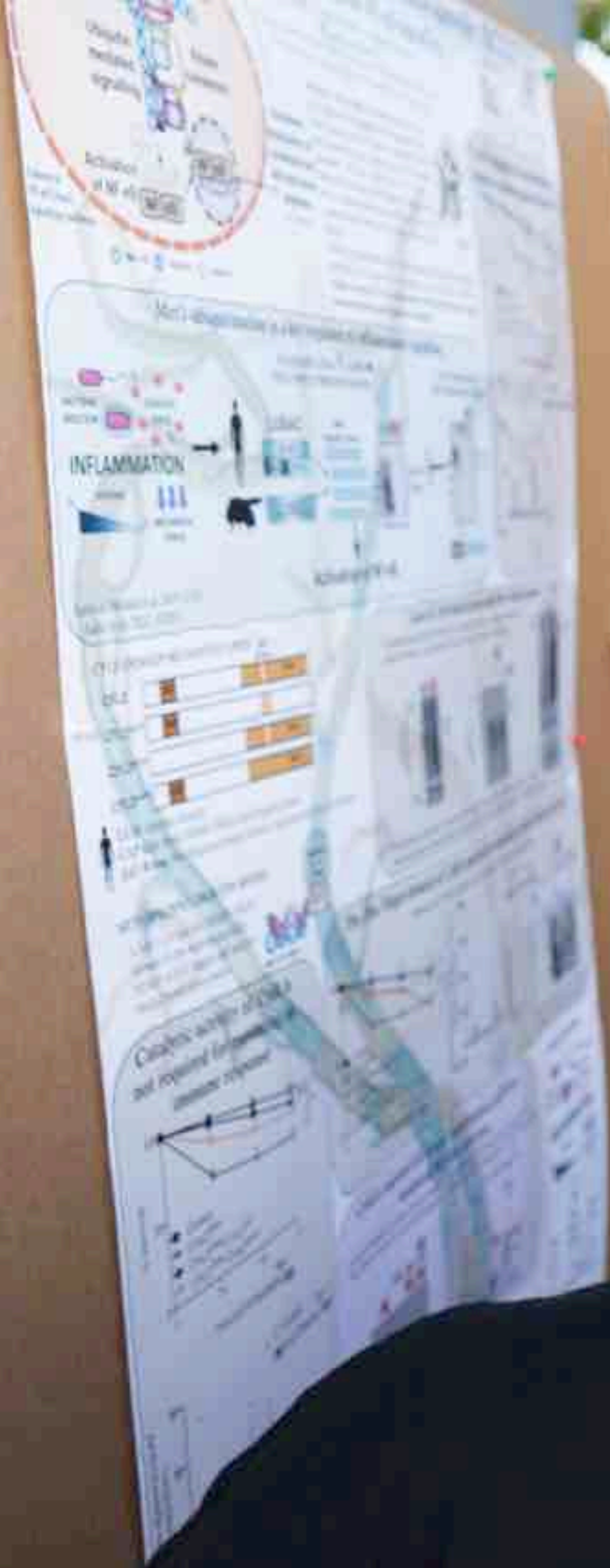
A binary cell-fate decision couples apoptosis to epithelial...

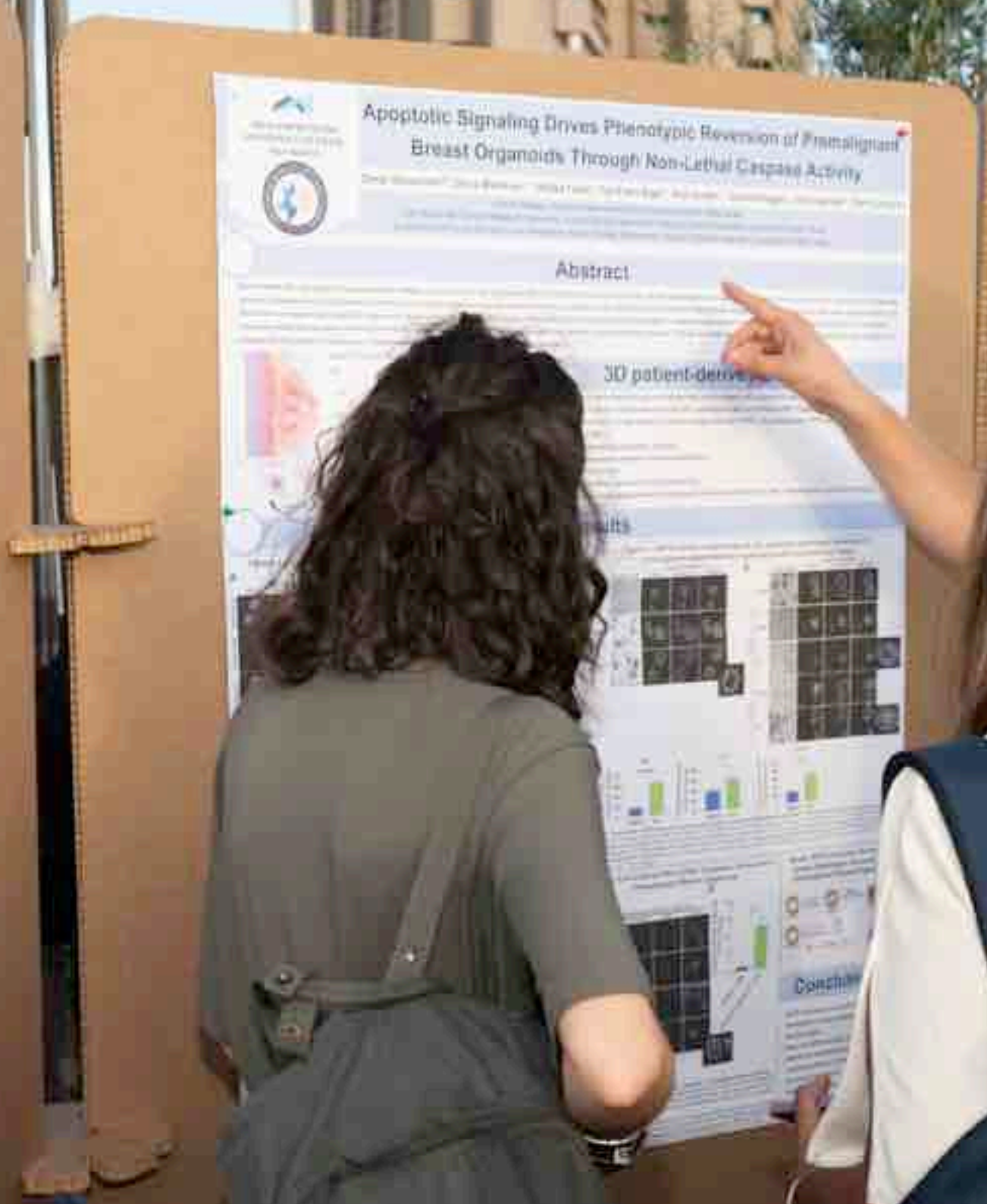
David Smith, University of...

Department of...

Abstract: ...

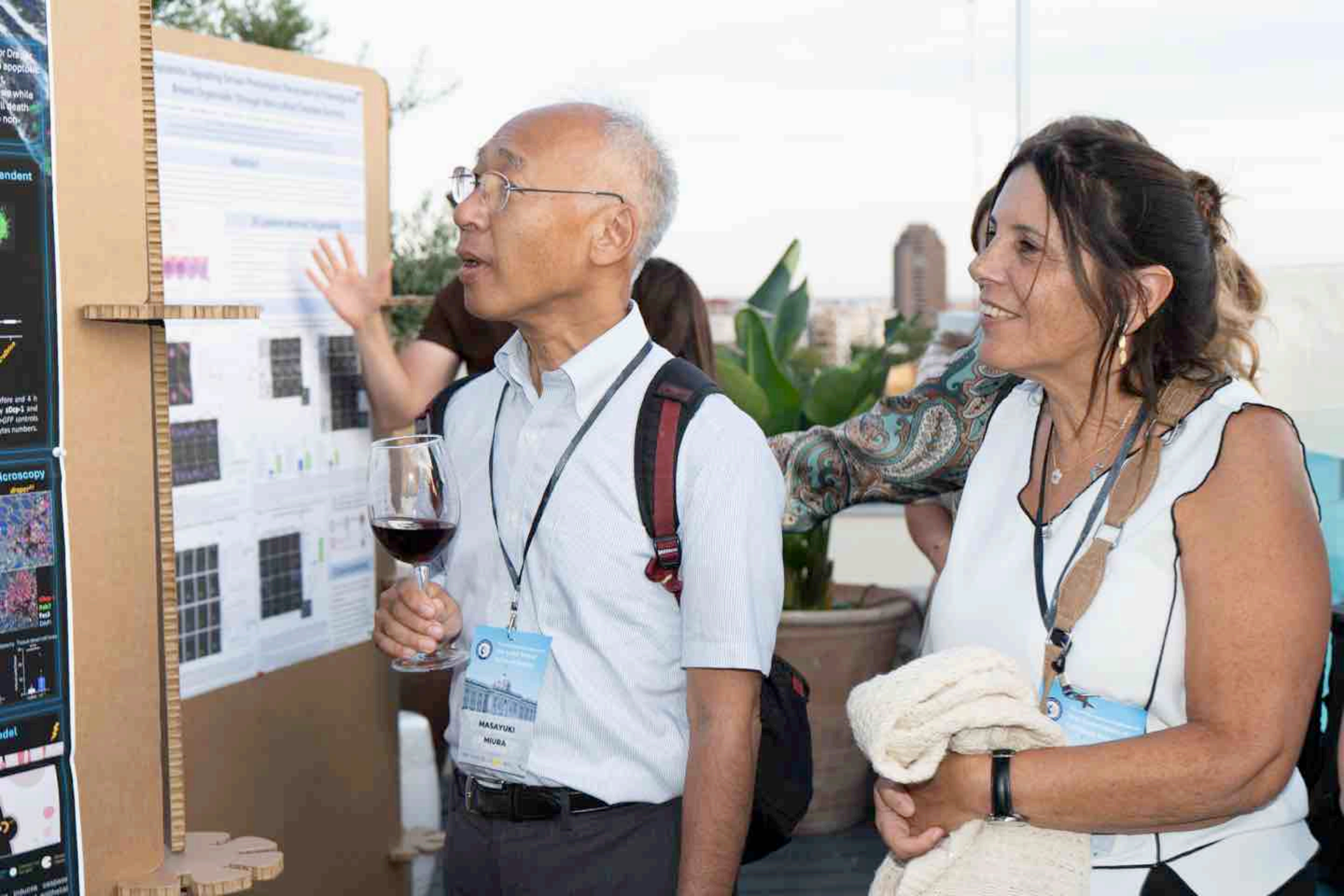
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ERIK GOMEZ-CARDONA

QUESTION

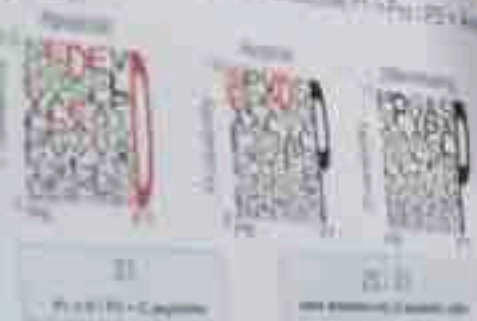
Could secondary proteolysis hide caspase

PRODUCTION

Caspases regulate cell fate through selective protein
covering caspase substrate is important for understanding
covered using NMR spectroscopy. However, secondary
structure caspase substrate is escape detection by NMR

DISCOVERY

Proteolytic cleavage of caspase-3 reveals a selective P1-P2-P3-P4



CASPASE-3 VALIDATION

A

B

C

D

E

F

G

H

I

J

K

L

M

N

O

P

Q

R

S

T

U

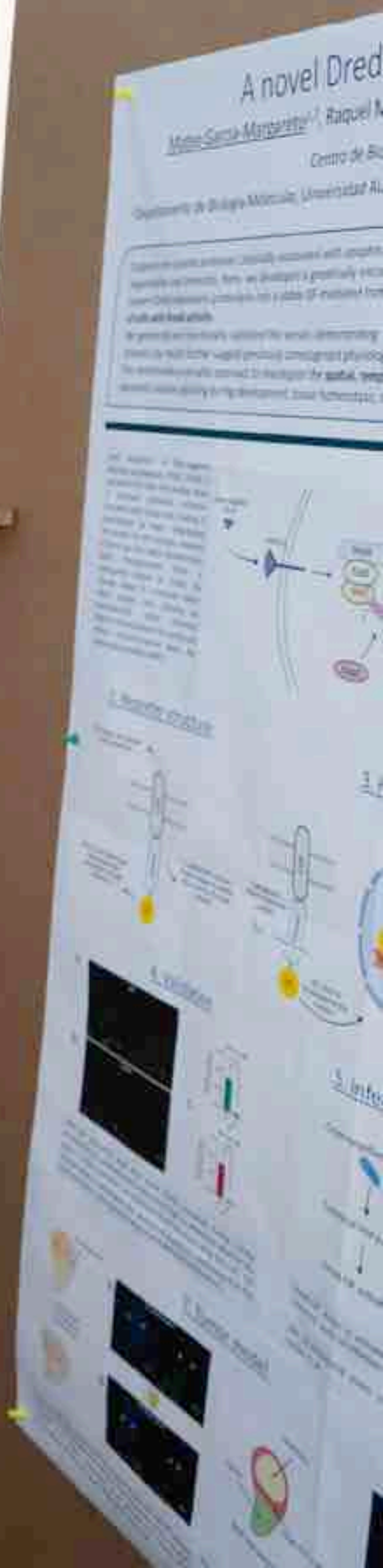
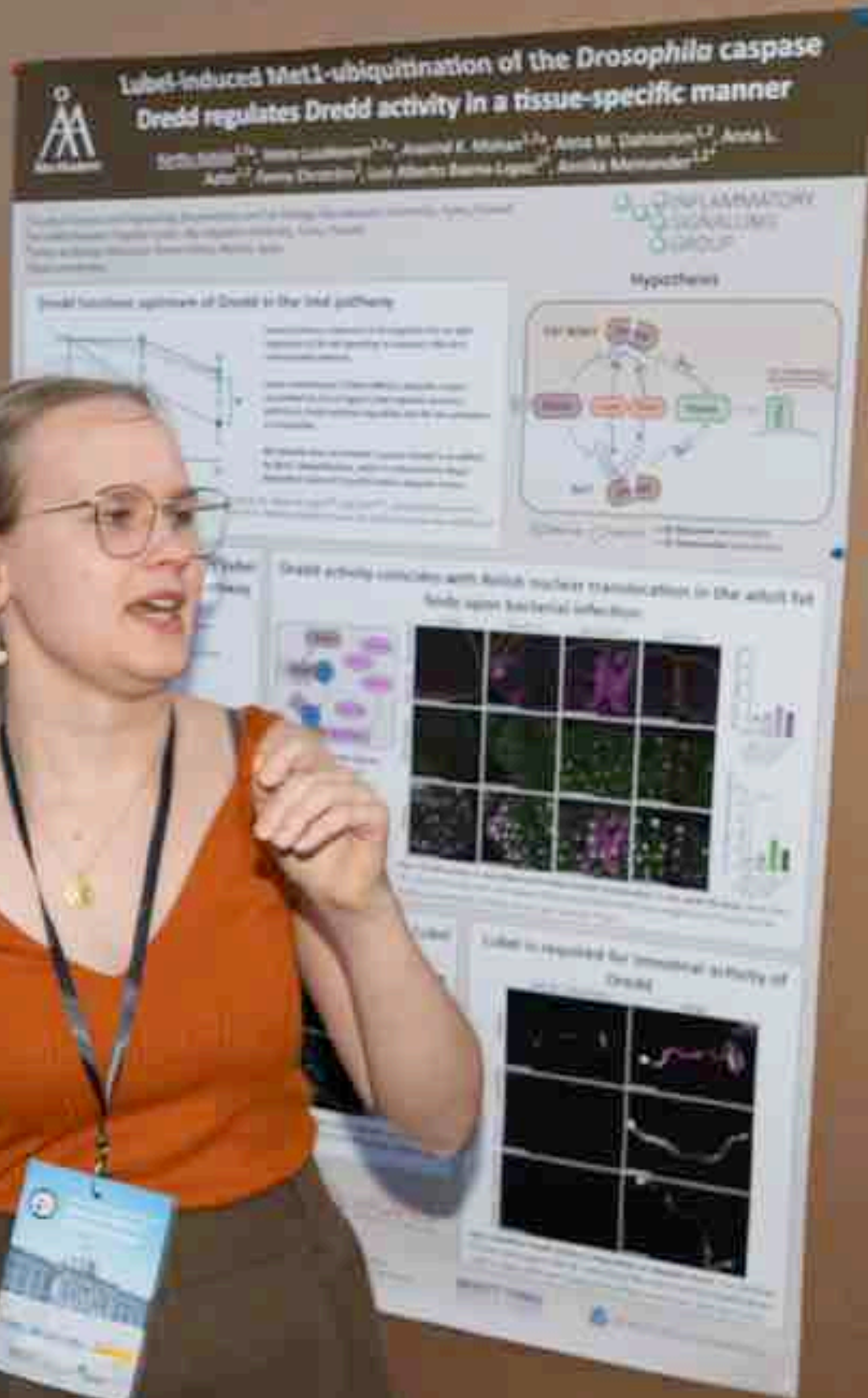
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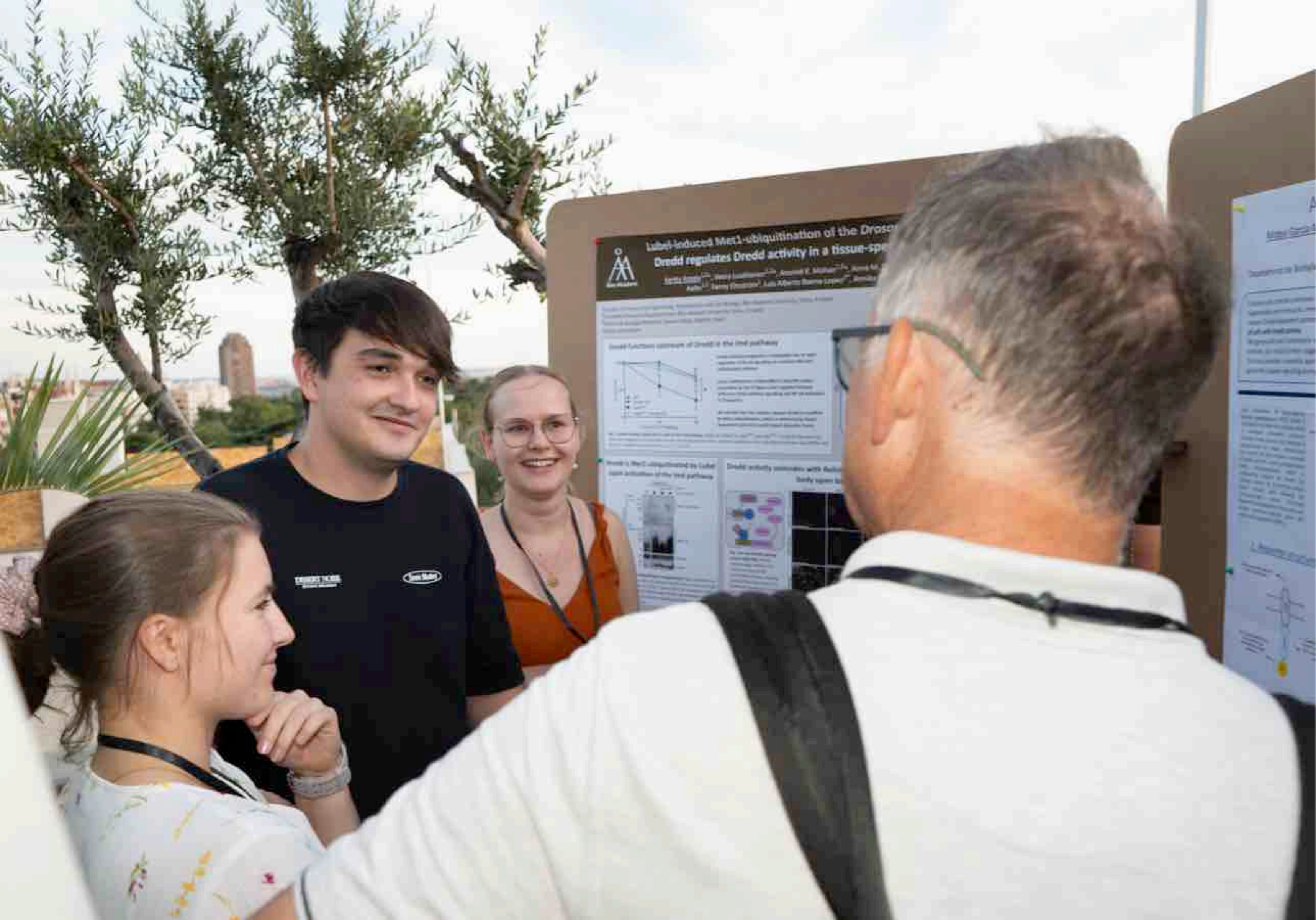
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