From Exposure to Intervention:

Determining and Mitigating Airway Injury Following Toxic Inhalation

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Mandatory Disclosure

In compliance with Accredited Continuing Education Standards:

Caroline Cherry, PhD is Founder and holds an ownership interest in Enviora Biosciences.

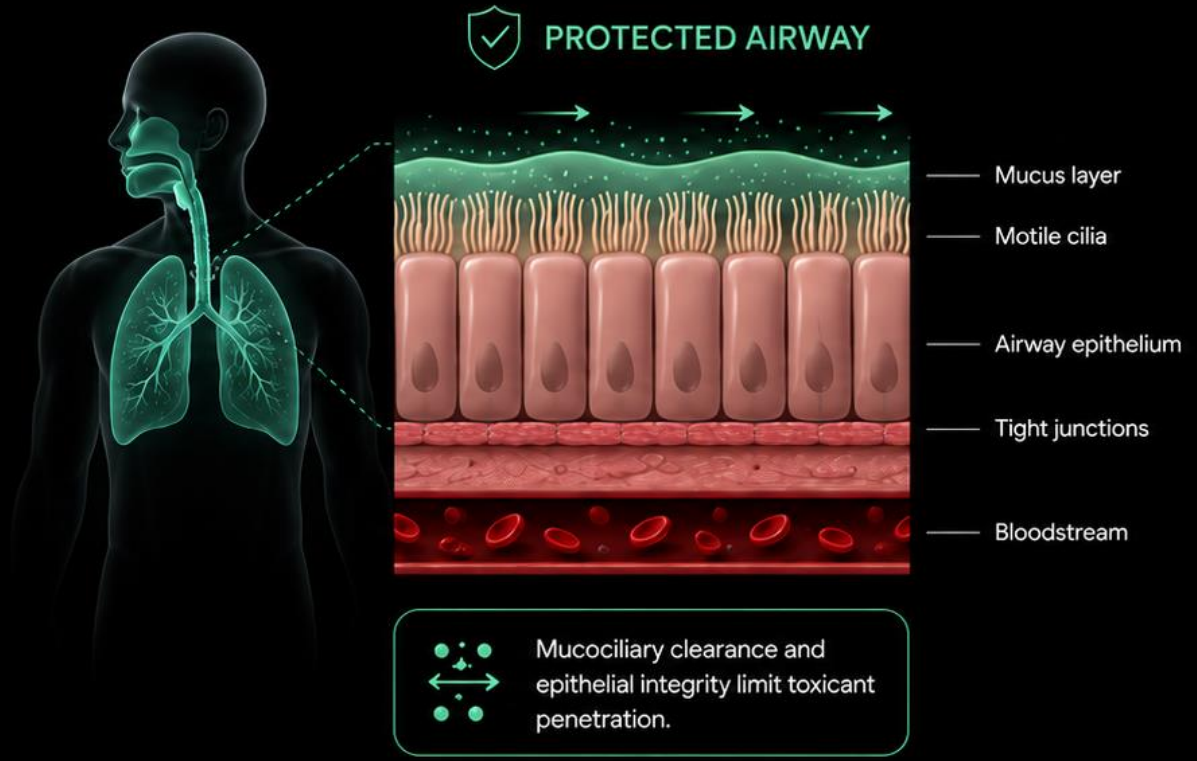
All data presented were generated at the University of California, Los Angeles and are presented for scientific and educational purposes. References to pharmacologic compounds are for research purposes only and do not constitute promotion of any commercial product.

Operational and Environmental Inhalation Exposures






The Airway is the Body's First Line of Defense



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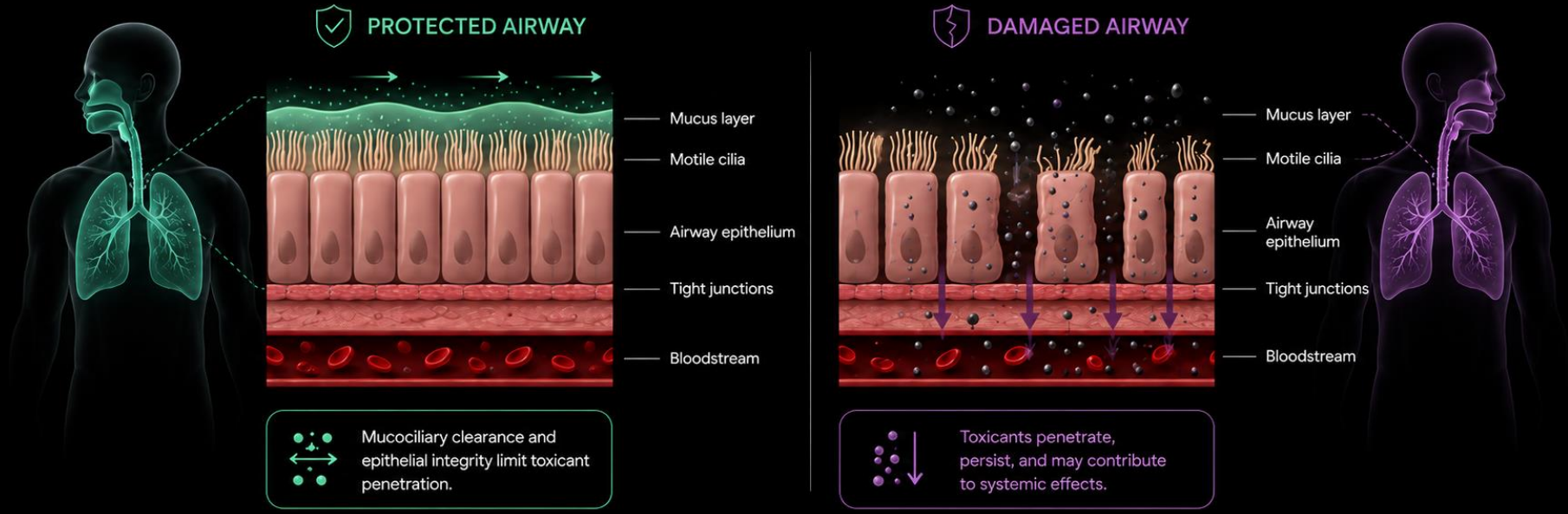
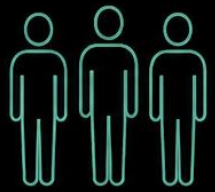


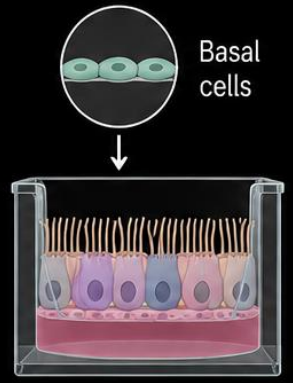
Modeling Toxic Inhalation Injury

1. Human Donors
n = 3



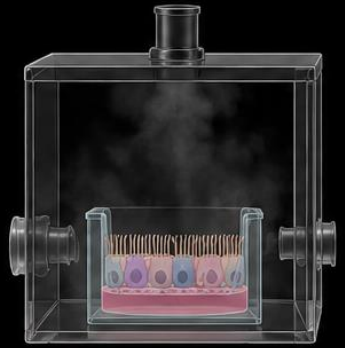
Healthy human lung donors

2. Airway Epithelial Model



Differentiated at air–liquid interface (7 – 14 days)

3. Controlled Smoke Exposure



Programmable exposure system for defined duration and concentration

4. Functional Readouts


Ciliary motion

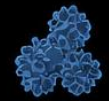

Barrier integrity


TEER


Cytotoxicity

5. Multi-omics Analysis


RNA

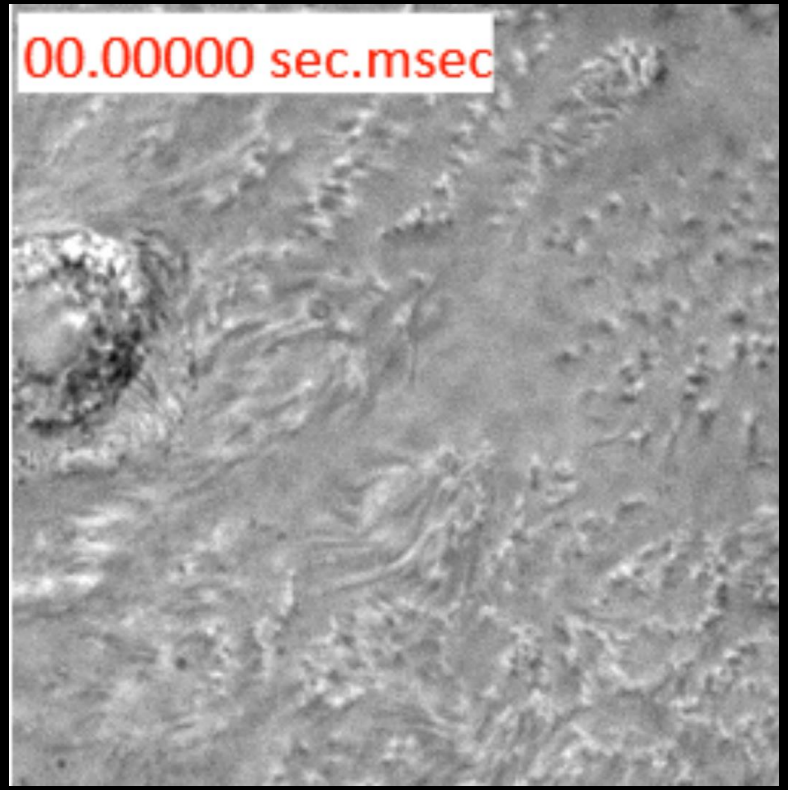

Protein

Phosphoprotein

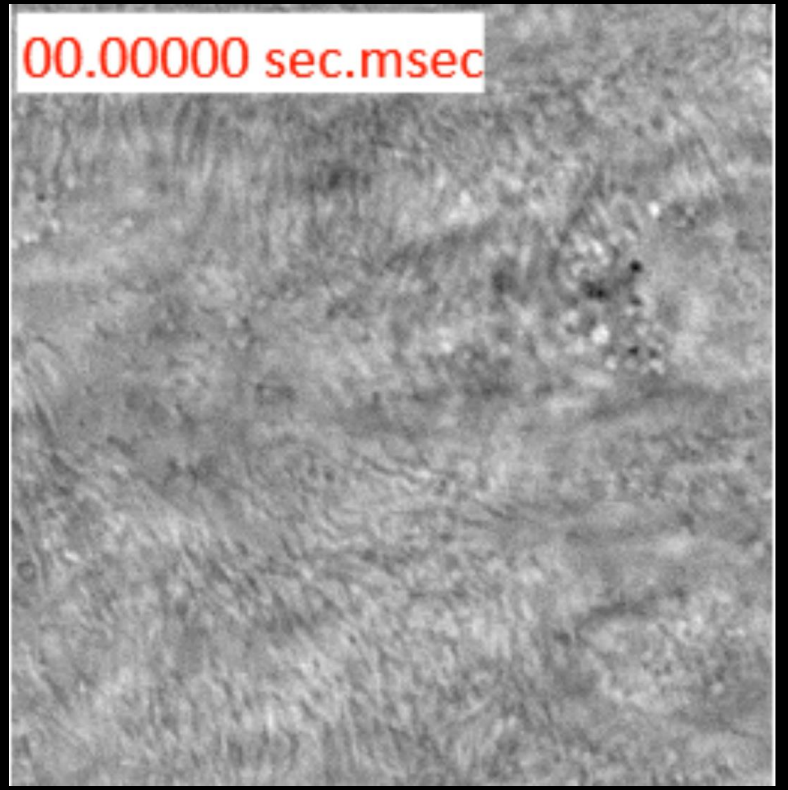
A reproducible human airway model to identify mechanisms of injury and test therapeutic interventions.

Woodfire Smoke Rapidly Impairs Ciliary Movement

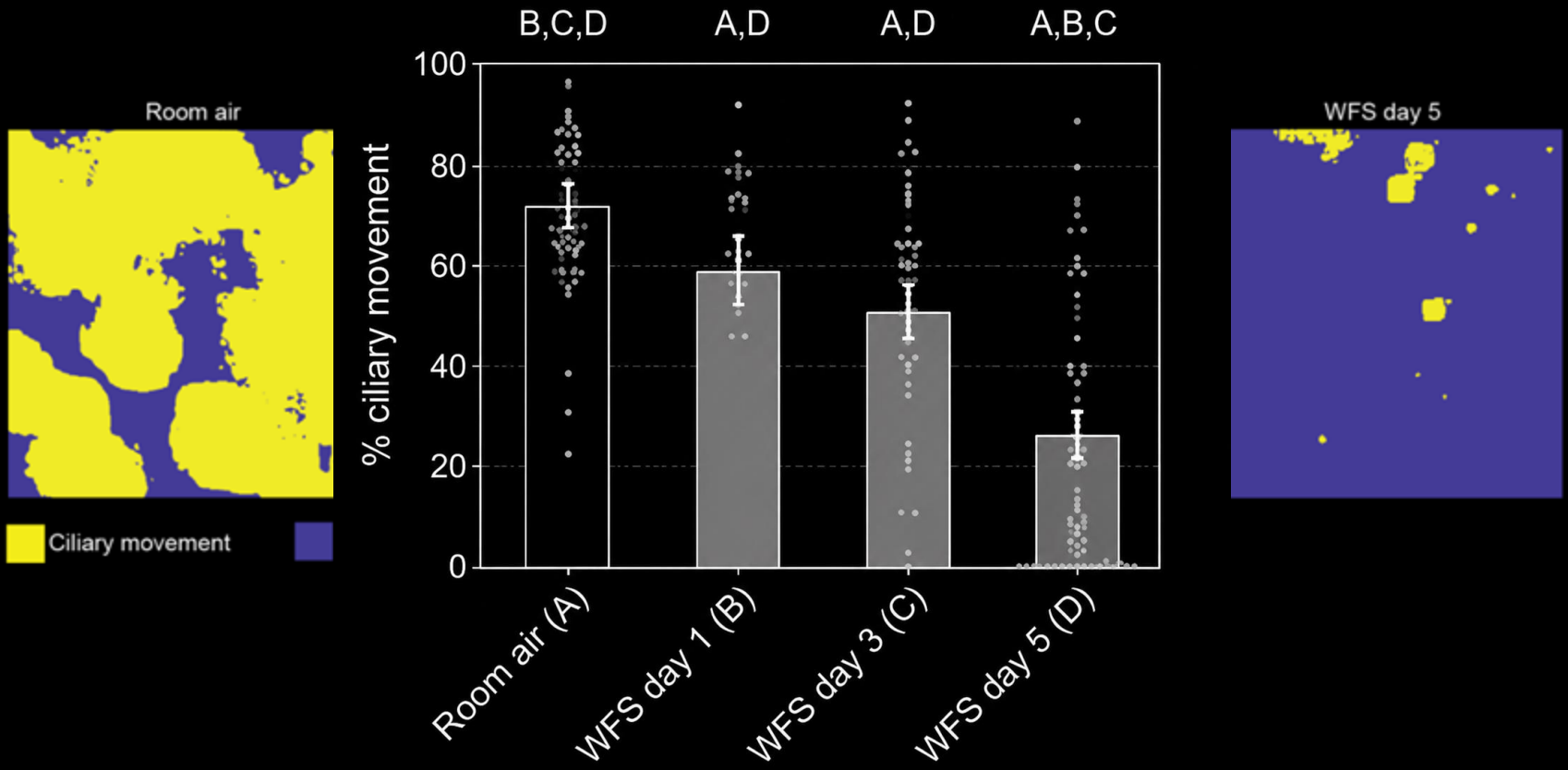
Control



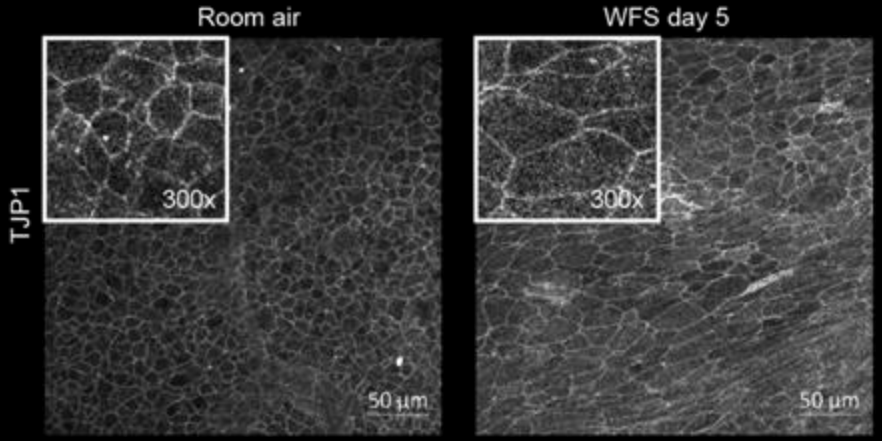
WFS

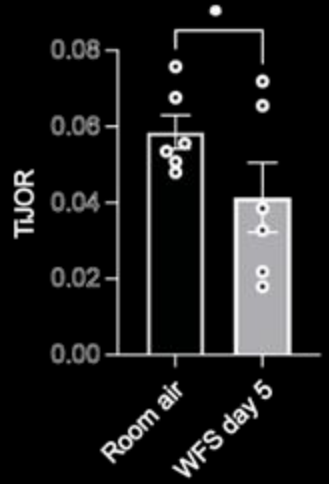


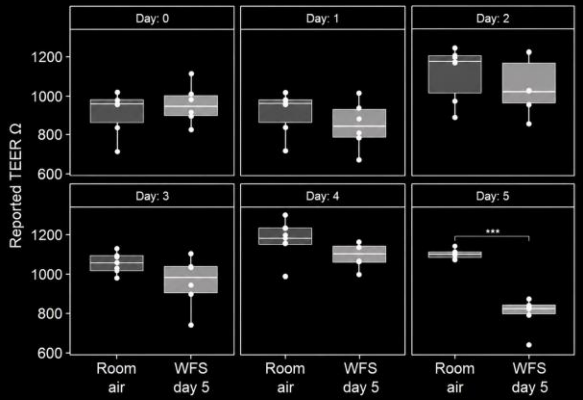
Repeated Exposures Cause Progressive Ciliary Failure



Toxic Inhalation Impairs Airway Defense Mechanisms







What drives this injury?

Why Measure Multiple Layers of Biology?

RNA

The Instructions

What is the cell trying to do?

PROTEIN

The Machinery

What tools does the cell have?

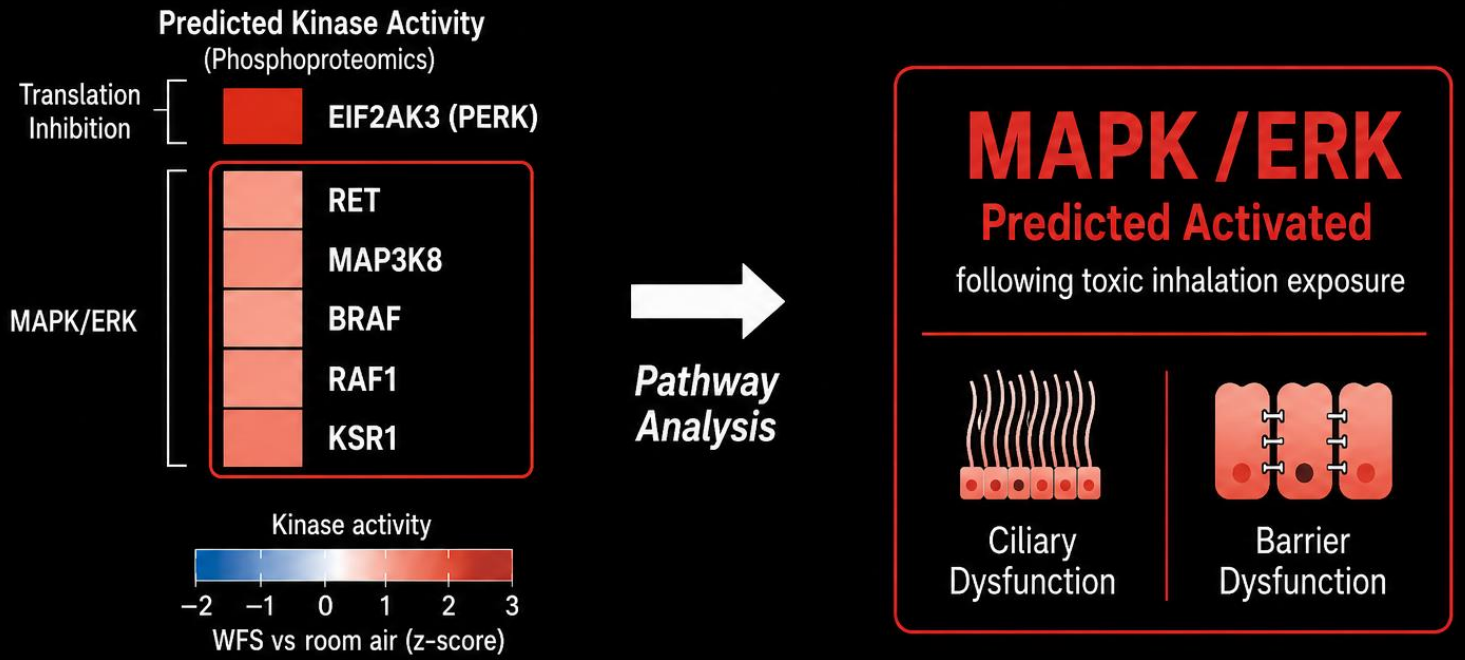
PHOSPHOPROTEIN

The Switches

Which pathways are active?

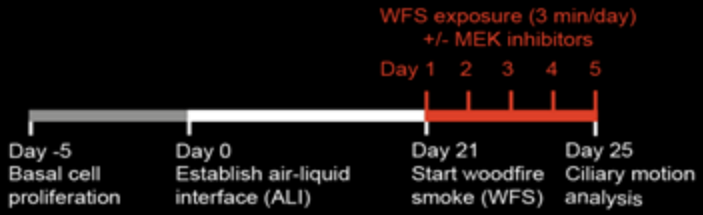
Together, these measurements reveal cellular intent, capability, and activity.

Multi-omics Identifies MAPK/ERK as a Driver of Airway Injury

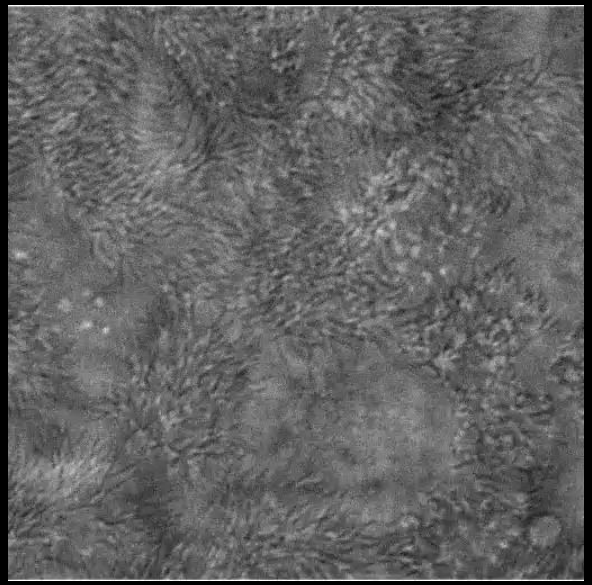


MAPK/ERK signaling emerged as a candidate driver of airway injury following toxic inhalation exposure. → **Test MEK inhibition**

MEK1/2 Inhibition Protects Ciliary Motility up to 80%



Room Air Control



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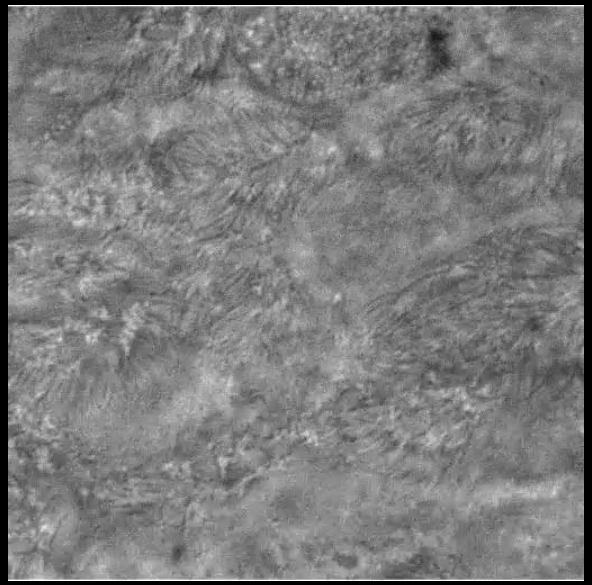
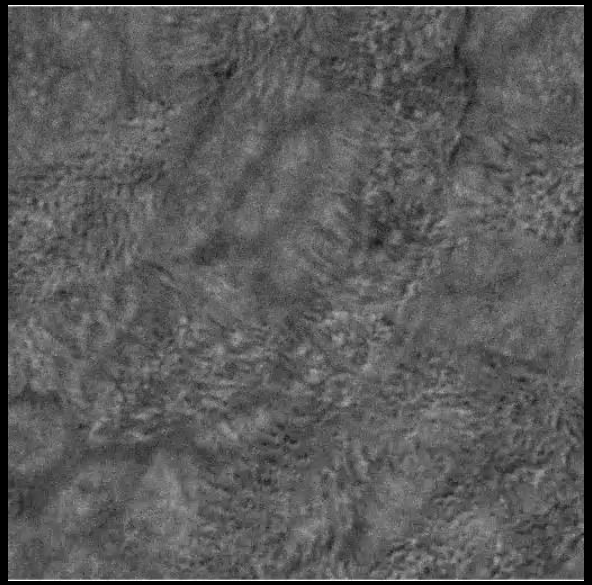
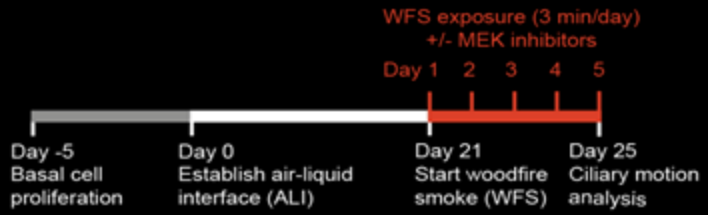


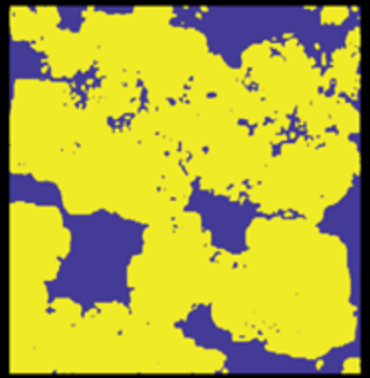
WFS + MEK Inhibitor



MEK Inhibition Preserves Ciliary Function



Room Air Control



WFS

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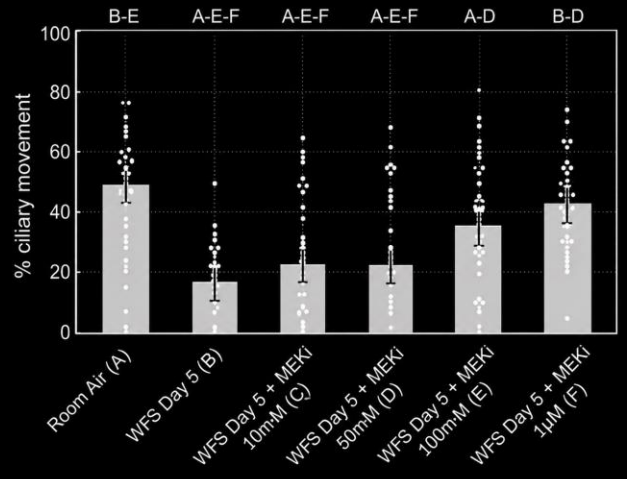
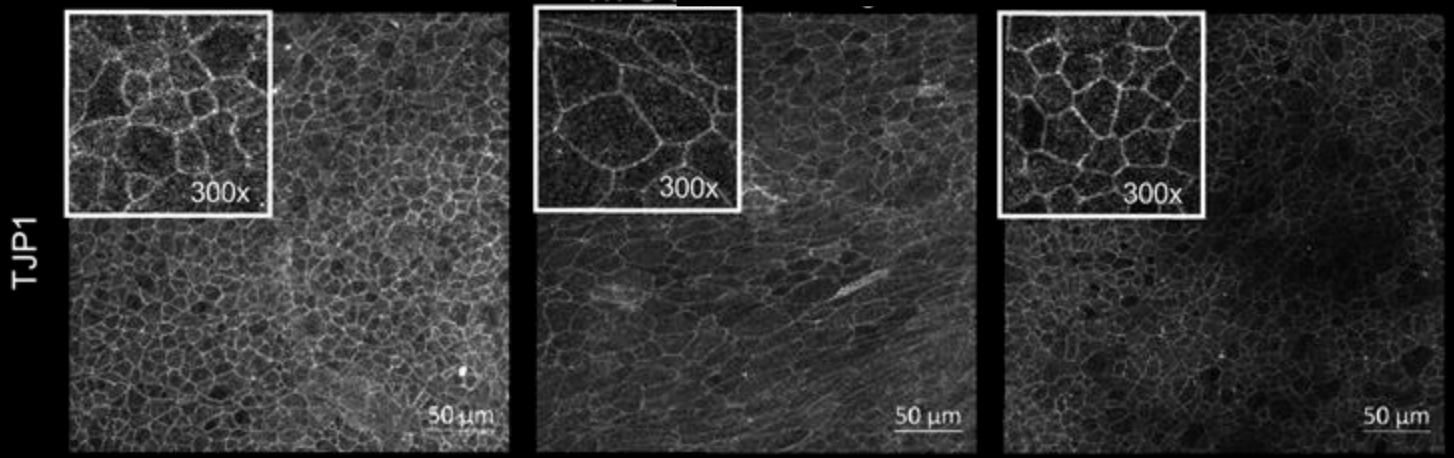


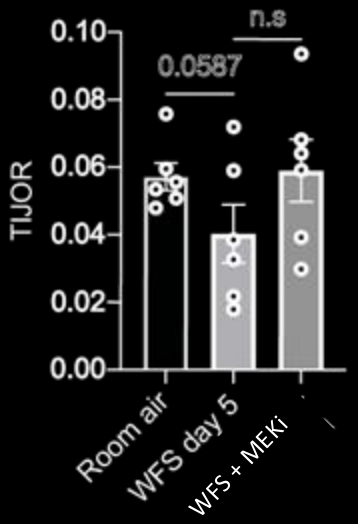
MEK Inhibition Preserves Tight Junction Organization

Room Air Control

WFS

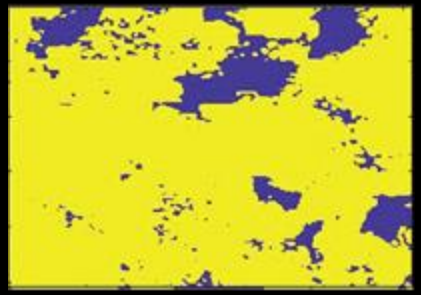
WFS + MEK Inhibitor





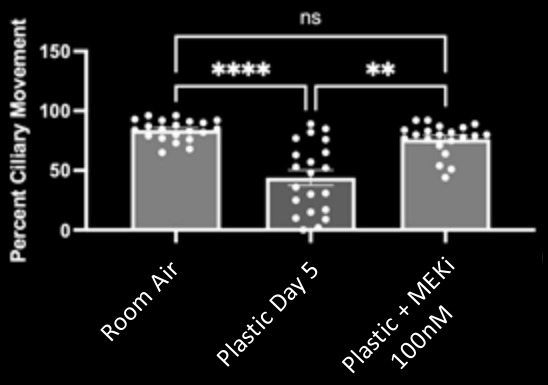
Protective Effects Extend Across Diverse Toxic Inhalation Exposures

Room Air Control



Plastic 5 Day

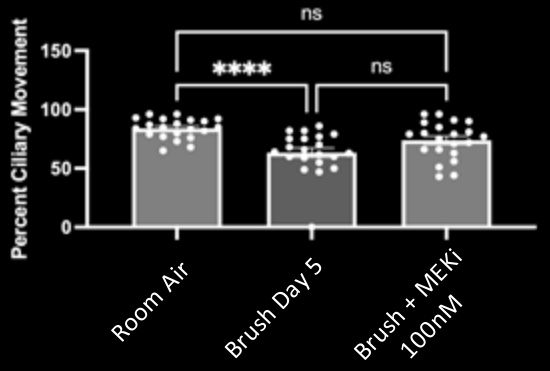
Plastic + MEKi



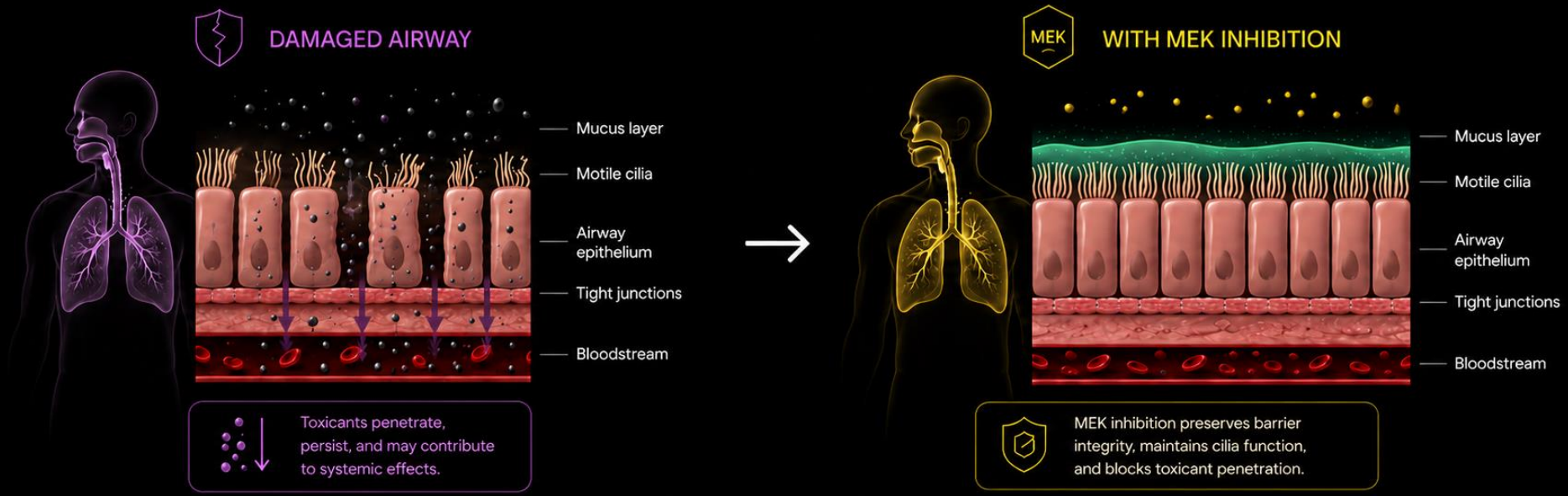
Room Air Control

Brush 5 Day

Brush + MEKi



Proposed Therapeutic Mechanism of Action



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EXPOSURE IS UNAVOIDABLE.
INJURY MAY NOT BE.

Mechanism-based strategies to
preserve airway defense mechanisms
following toxic inhalation exposure.