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Hello *Mollie Medcast* listeners and welcome back! *Mollie Medcast* is the podcast for the biomedical journal, *Molecular Medicine*. My name is Margot Puerta, I'm the Managing Editor here at *Molecular Medicine* and I'll be your host for this podcast episode. In this week's podcast we'll take a look at three primary research papers from our upcoming July-August 2010 issue. They are: "One For The Road: E2 For Trauma-Hemorrhage," "Rho-kinase In Heart Preconditioning" and "ST Segment Elevation In AMI."

We'll start by taking a minute to review our goal here at *Molecular Medicine*. Since 1994, our mission has been to publish novel work that's concerned with understanding the pathogenesis of disease at the molecular level, which may lead to the design of specific molecular tools for disease diagnosis, treatment and prevention. If you're interested in submitting a manuscript to the journal, please visit our Web site for information, [www.molmed.org](http://www.molmed.org). Ok, let's get started with the podcast.

The first paper in this podcast episode is:

**One For The Road: E2 For Trauma-Hemorrhage**

Trauma-hemorrhage followed by resuscitation is often fatal in civilian and military trauma. It affects organs such as the liver, kidney, heart and lung. Deleterious effects of trauma-hemorrhage are influenced by sex hormones, and estrogen (or E2) improves immune and cardiovascular response parameters. However, the precise mechanism by which estrogen and other sex hormones produce beneficial effects has yet to be determined. In order to target the possible early E2-mediated effects, Dr. Kozlov and colleagues from Austria and Alabama investigated whether the following processes could be involved in prolonged hypotension: either oxidative/nitrosylative/endoplasmic reticulum [ER] stress or altered mitochondrial function. Additionally, the authors looked at and whether E2 affects these biochemical processes. The title of the manuscript is, "Effect of Estrogen on Mitochondrial Function and Intracellular Stress Markers in Rat Liver and Kidney following Trauma-Hemorrhagic Shock and Prolonged Hypotension." Results indicate that trauma-hemorrhage followed by prolonged hypotension significantly affects mitochondrial function, ER stress markers and free iron levels, and that E2 ameliorated these changes. E2 appears to be a hormonal adjunct that could slow the progression of trauma-hemorrhage during patient transport to the hospital.

The next paper we'll discuss is:

**Rho-kinase In Heart Preconditioning**

Rho-kinase plays a key role in the pathogenesis resulting from heart ischemia reperfusion (I/R) injury. Ischemic preconditioning, which includes brief periods of repetitive cardiac ischemia reperfusion, protects against subsequent lethal periods of ischemia, decreases Rho-kinase activation, and reduces infarct size. While these results are beneficial, little is known about this mechanism of action. Dr. Zhang and colleagues from China and Texas investigate this in their paper entitled, "ERK-MAPK Signaling Opposes Rho-kinase to Reduce Cardiomyocyte Apoptosis in Heart Ischemic Preconditioning." In it, the authors show that ERK-MAPK signaling is required in ischemic preconditioning in order to oppose the Rho-kinase signaling which leads to apoptosis in vivo. These data may lead to new treatment possibilities for patients suffering from coronary heart disease.

And, the last paper in this podcast episode is:

### **ST Segment Elevation In AMI**

ST segment elevation is used to diagnose acute myocardial infarction (or AMI) and it's the major clinical criterion for committing patients with chest pain to emergent coronary revascularizations. Despite its frequent usage, the mechanism responsible for ST segment elevation remains unclear. Since ST segment elevation is the major criteria for thrombolytic therapy, Dr. Long and colleagues from China examined the role of thrombin and its receptor activation in ST segment elevation during acute myocardial infarction. The title of their manuscript is, "Thrombin and its receptor enhance ST segment elevation in acute myocardial infarction by activating the KATP channel." Their results demonstrate that thrombin and activation of its receptor significantly enhance ST segment elevation during AMI. This observation suggests that more robust and accurate methods than reduced ST segment times may be required to assess efficacy of anti-thrombin therapies.

And that's it for this week's episode of the *Mollie Medcast*. Join us next time when we take a look at: hedgehog signaling in meningiomas, microRNAs in acute kidney injury, and insulin signaling in type 2 diabetes. For questions or comments regarding this podcast, please feel free to send me an e-mail at: [margot@molmed.org](mailto:margot@molmed.org), that's m-a-r-g-o-t(at)m-o-l-m-e-d.org. You can also keep up with the journal by following us on Twitter (@mol\_med).

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From New York, this is [margot@molmed.org](mailto:margot@molmed.org), thanks for listening!

Produced by Margot Puerta  
Managing Editor, *Molecular Medicine*

Written by Robert L Pinsonneault  
Associate Editor, *Molecular Medicine*

Edited by Veronica J Davis  
Communications Editor, *Molecular Medicine*

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