Molecular Medicine

Podcast Transcript
Episode 37

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 your host for this week's podcast. This week we're going to go over two research papers: "EPO Cross-Talk In Multiple Sclerosis Model" and "Estrogen Receptor In Trauma-Hemorrhage"; and, then we'll look at a Review and Assess paper which appeared in our November-December issue, "Pharmacology of Traumatic Brain Injury".

But, before we do that let me take a minute to remind you about our goal at *Molecular Medicine*. Our mission is to publish novel work 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 website for information, www.molmed.org.

EPO Cross-Talk In Multiple Sclerosis Model
I guess I should start with defining "EPO", E-P-O – that's the abbreviation I'm going to use for erythropoietin. Systemically administered EPO crosses the blood brain barrier and it's protective in several animal models of disease. Endogenous EPO is produced by hypoxic or ischemic injury, but little is known regarding the expression of endogenous EPO in central nervous system [CNS] diseases. Dr. Manuela Mengozzi and her colleagues investigated the expression of EPO in the spinal cord, and to do this, she used two models of experimental autoimmune encephalomyelitis, or EAE for short. These EAE models are representative of multiple sclerosis. Dr. Mengozzi's findings indicate that EPO is induced in EAE and is negatively regulated by interferon gamma and tumor necrosis factor. This cross-talk between EPO and inflammatory cytokines in the central nervous system may have important implications in disease pathogenesis. Therapeutic exploitation strategies could be aimed at upregulating the endogenous EPO-mediated protective response.

The next primary research paper we'll look at deals with the:
Estrogen Receptor In Trauma-Hemorrhage
Sex hormones are known to modulate immune function in animals and humans under normal conditions, but also under stress. Administration of estrogen following trauma-hemorrhage attenuates the elevation of cytokine production and mitogen-activated protein kinase. What we don't know is whether the beneficial effects of estrogen are mediated by estrogen receptor ER-α or ER-β. In this work, Dr. Moeinpour and colleagues, both in Alabama and Brazil, sought to determine which estrogen receptor was responsible for the salutary effects in a model of trauma-hemorrhage. Using an agonist strategy to isolate the receptor subtypes, Dr. Moeinpour determined that both ER-α and ER-β both contribute to the beneficial effects of estrogen on keratinocytes after trauma-hemorrhage.

Now we're going to switch gears a little bit and talk about the Review and Assess paper we have from our November-December issue, and this deals with:
Pharmacology Of Traumatic Brain Injury or TBI
Traumatic brain injury is a major health care problem and significant socioeconomic challenge it's the leading cause of death and disability in young people. Approximately 1.5 million patients in the United States are af-

fected each year, and despite advances in research and improved neurointensive care, no specific pharmacological therapy for traumatic brain injury patients is currently available. Dr. Kathryn Beauchamp and her colleagues review the published prospective clinical trials on pharmacological treatment modalities for traumatic brain injury patients and outline future promising therapeutic options in this field.

That's it for this week's episode of *Mollie Medcast*. You can find all these papers and many more of them on our website, www.molmed.org that's www.m-o-l-m-e-d.org. For questions or comments regarding this podcast, please feel free to send me an email at: margot@molmed.org.

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