

CANCER CENTER SEMINAR SERIES



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Dr. Maria Hatzoglou is a world leader in understanding how translation (from the IRES) is regulated during stress. She studies both pro-apoptotic and anti-apoptotic mechanisms. These studies have exciting relevance for several diseases including diabetes and cancer.

When cells undergo stress, say, starvation, inflammation, infection, cancer, or something else, the response is often a global (but temporary) shut-down of cell gene expression (they stop making most proteins) as an apparent way to survive in the long-run. The major mechanism for shut-down is blockage of translation of mRNAs (rather than turning off transcription from DNA).

Yet, approximately 5% of messenger RNAs keep on translating, and the protein products from these are mostly ones that counteract the effects of cell stress. Depending on conditions, some proteins made during stress hasten cell death (apoptosis) rather than aid in cell survival (all to help the animal host survive in the long-run).

How can this happen? How can translation of most (95%) of mRNAs be stopped, but some (5%) allowed? The answer: the majority of mRNAs are translated by a canonical 5'-end cap-dependent translation mechanism where the ribosomes enter at the 5' end of the mRNA and can be stopped by phosphorylation of certain translation initiation factors, whereas the minority that are translated during stress use an internal ribosomal entry site (an IRES) for ribosomal entry and respond quite differently.

“Signaling Pathways in Cell Fate Decisions During Stress”

Friday, January 13, 2012

Frohring Auditorium - BRB 105

12 - 1:00 pm

*Hosted by the Cancer Cell Signaling Program, led by Drs. Clark Distelhorst, Nancy Oleinick and Alex Almasan