Edward T. H. Yeh

Current Position: Professor and Chairman of the Department of Cardiology at The University of Texas M. D. Anderson Cancer Center; McNair Scholar at the Texas Heart Institute

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Non-scientific Interests: Reading history and biography

In 1996, my laboratory reported a protein that binds to the death domains of Fas. I named it Sentrin because it functions likes a sentry guarding the cell death pathway and has a conserved ubiquitin domain. The same molecule was discovered by several other laboratories at about same time, and it is now most commonly known as SUMO, small ubiquitin-like modifier. SUMO modification (SUMOylation) turned out to be a major post-translational modification pathway because more than 2,500 SUMO substrates have been reported. Using Sentrin as bait in the yeast two hybrid screen, I discovered that UBC9 is a unique SUMO conjugating enzyme. In 2000, I reported the first human de-conjugating enzyme called SENP1 (Sentrin-specific protease 1) and provided the current classification for other family members. The SUMO pathway was initially thought to be different from the ubiquitination pathway because it does not target a protein for degradation. This view was dramatically altered in 2007 with my discovery of SUMO as an alternative signal for binding of hypoxia-inducible factor 1α to the VHL ubiquitin ligase. In this minireview, I focus on the SENPs and speculate on how a limited number of on and off enzymes is able to regulate such a large universe of biological response.

Read Dr. Yeh's article entitled: SUMOylation and De-SUMOylation: Wrestling with Life's Processes

http://www.jbc.org/cgi/content/full/284/13/8223