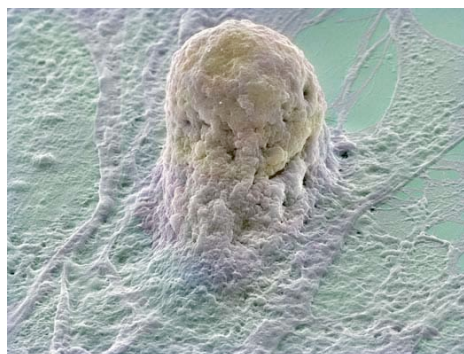


Markus Manz Research Funded by OncoSuisse

A research proposal made by Markus Manz covering disease initiating stem cells in human myeloproliferative disease together with Dr. Radek Skoda of the University of Basel has been approved for funding by Oncosuisse to cover a post-doc and consumables for 2 years.



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Italian Industrial Alliance visits IRB

The ISPE is the world's largest non-profit society serving the pharmaceutical science and manufacturing industries. A group of members led by Dr Claudio Rolandi organized a site visit to the IRB. Luca Varani presented his project on Computational Biology.



ISPE

Affiliata Italiana

ENGINEERING
PHARMACEUTICAL
INNOVATION

New member of the IRB Staff

Miriam Miladic has joined the IRB as Secretary to the Direction replacing Susanna Memoli. Miriam has been working for 7 years at General Electric of Riazzino, enjoys wakeboarding and other outdoor sports.



Siro Bianchi e Riccardo Bernasconi officially joined the lab of Maurizio Molinari

Tatiana Soldà publishes in Molecular Cell

Substrate-specific requirements for UGT1-dependent release from calnexin. 20.07.2008

Newly synthesized glycoproteins displaying monoglucosylated N-glycans bind to the endoplasmic reticulum (ER) chaperone calnexin, and their maturation is catalyzed by the calnexin-associated oxidoreductase ERp57. Folding substrates are eventually released from calnexin, and terminal glucoses are removed from N-glycans. The UDP-glucose:glycoprotein glucosyltransferase (UGT1, UGGT, GT) monitors the folding state of polypeptides released from calnexin and adds back a glucose residue on N-glycans of nonnative polypeptides, thereby prolonging retention in the calnexin chaperone system for additional folding attempts. Here we show that for certain newly synthesized glycoproteins UGT1 deletion has no effect on binding to calnexin. These proteins must normally complete their folding program in one binding event. Other proteins normally undergo multiple binding events, and UGT1 deletion results in their premature release from calnexin. For other proteins, UGT1 deletion substantially delays release from calnexin, unexpectedly showing that UGT1 activity might be required for a structural maturation needed for substrate dissociation from calnexin and export from the ER.

