Endoplasmic Reticulum Discovered By

Membrane contact site including ions, lipids and (discovered later) reactive oxygen species. First mentions of these contact sites can be found in papers published in the late 1950s mainly visualized using electron microscopy (EM) techniques. MCS are important in the function of the endoplasmic reticulum (ER), since this is the Membrane contact sites (MCS) are close appositions between two organelles. These zones of apposition are highly conserved in evolution. Ultrastructural studies typically reveal an intermembrane distance in the order of the size of a single protein, as small as 10 nm or wider, with no clear upper

limit. The ER makes close contact with many organelles, including mitochondria, Golgi, endosomes, lysosomes, peroxisomes, chloroplasts and the plasma membrane. Both mitochondria and sorting endosomes undergo major rearrangements leading to fission where they contact the ER. MCS are important in the function of the endoplasmic reticulum (ER), since this is the major site of lipid synthesis within cells. Sites of close apposition can also form between most of these organelles most pairwise combinations. Copeland and Dalton described them as "highly specialized tubular. These sites are thought to be important to facilitate signalling, and they promote the passage of small molecules, including ions, lipids and (discovered later) reactive oxygen species b337e8e4fd

GSK2606414 Extension of lifespan in treated mice was, however, not recorded. Treatment with GSK2606414 was found to be neuroprotective in mice against damage caused by

prions, and prevented the development of cognitive deficits and other clinical manifestations of prion disease. PERK mediates the unfolded protein response pathway which is involved in the initiation of protein synthesis, and this pathway has been implicated in the neurotoxicity of various diseases including prion and Alzheimer's diseases. GSK2606414 was found to be a potent and selective inhibitor of PERK, with good oral bioavailability and blood-brain barrier penetration. However, side effects such as weight loss and elevated blood glucose levels were also observed, likely due to unwanted inhibition of PERK in the pancreas gland, where it is involved in regulating insulin production. which is the first selective inhibitor discovered for the enzyme protein kinase R (PKR)-like endoplasmic reticulum kinase (PERK), which is involved in various GSK2606414 is a drug which is the first selective inhibitor discovered for the enzyme protein kinase R (PKR)-like endoplasmic reticulum kinase (PERK), which is involved

in various processes relating to cancer and neurodegenerative disorders b337e8e4fd

David Thomas (Canadian scientist) His research interests include cell signaling pathways and their role in infectious diseases and molecular chaperone systems in the endoplasmic reticulum. Thomas is a Canadian biochemist. M. the endoplasmic reticulum. Bergeron discovered calnexin David Y. He is Chair of Biochemistry at McGill University. Thomas completed his PhD in 1970 at University College London. In the 1990s, Thomas and John J b337e8e4fd

SEC16B 2007, RGPR-p117 was also renamed as Sec16 homologue B (SEC16B), an endoplasmic reticulum export factor. The gene consists of 26 exons spanning approximately Protein transport protein Sec16B also known as regucalcin gene promoter region-related protein p117 (RGPR-p117) and leucine zipper transcription regulator 2 (LZTR2) is a protein that

in humans is encoded by the SEC16B gene b337e8e4fd

== Discovery == b337e8e4fd

Ribophorin Ribophorin I and II are only present in eukaryote cells. There are two types of ribophorines: Ribophorins are dome shaped transmembrane glycoproteins which are located in the membrane of the rough endoplasmic reticulum, but are absent in the membrane of the smooth endoplasmic reticulum. These act in the protein complex oligosaccharyltransferase (OST) as two different subunits of the named complex. in the membrane of the rough endoplasmic reticulum, but are absent in the membrane of the smooth endoplasmic reticulum. There are two types of ribophorines: ribophorin I and II b337e8e4fd

Both types of ribophorins develop a key role in the binding of ribosomes to the rough endoplasmic reticulum as well as

in the co-translational processes that depend on this interaction. The content of ribophorin of the rough endoplasmic reticulum is equal to the stoichiometric number of ribosomal units. Therefore, this suggests the great importance, abundance and good preservation of these proteins in the reticulum. Consequently, defects in the genes that encode these proteins may cause congenital disorders and devastating consequences; ribophorin I and II are encoded by the genes RPN1 and RPN2 respectively. b337e8e4fd The ribophorins are soluble in non-ionic detergents such as Triton X-100. b337e8e4fd C/EBP proteins are known to have a conserved C-terminal structure, basic leucine zipper domain(bZIP), that is necessary for the formation of DNA-binding capable homodimers or heterodimers with other proteins or members of the C/EBP protein family. CHOP is a relatively small (29kDa) protein that differs from most C/EBP proteins in several amino acid substitutions, which impacts its DNA-binding ability. b337e8e4fd

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Overexpression of derlin-1 are associated with many cancers, including colon cancer, breast cancer, bladder cancer and non-small cell lung cancer. b337e8e4fd RGPR-p117, which was named as a regucalcin gene promoter region-related protein, was originally discovered as a novel transcription factor that specifically binds to a nuclear factor I (NFI) consensus motif TTGGC(N)6CC that is located on the 5'-flanking region of the regucalcin gene (rgn) in 2001. This gene is a highly conserved a leucine zipper motif, and it was also named as the leucine zipper transcription regulator 2 (LZTR2). In 2007, RGPR-p117 was also renamed as Sec16 homologue B (SEC16B), an endoplasmic reticulum export factor. b337e8e4fd == Structure == b337e8e4fd Thomas completed his PhD in 1970 at University College London. In the 1990s, Thomas and John J. M. Bergeron discovered calnexin, an integral protein of the endoplasmic reticulum. In 1998, Thomas was elected a Fellow of the Royal Society of Canada. b337e8e4fd In 2004 the DERL1 gene was discovered

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independently by two research groups when they were
exploring the machinery of retrotranslocation... b337e8e4fd
In 2015, Thomas was renewed as a Tier 1 Canada Research
Chair in Molecular Genetics. b337e8e4fd == Career ==
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Cranio-lenticulo-sutural dysplasia significant dilation of
the endoplasmic reticulum in fibroblasts of the host with
CLSD. The disease causes a significant dilation of the
endoplasmic reticulum in fibroblasts of the host with CLSD.
Due to the distension of the endoplasmic reticulum, export
of proteins (such as collagen) from the cell is disrupted.
Due to the distension of the endoplasmic reticulum, export
of proteins Cranio-lenticulo-sutural dysplasia (CLSD, or
Boyadjiev–Jabs syndrome) is a neonatal/infancy disease
caused by a disorder in the 14th chromosome. It is an
autosomal recessive disorder, meaning that both recessive
genes must be inherited from each parent in order for the
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disease to manifest itself b337e8e4fd

=== Membraneless Organelles === b337e8e4fd p24 family proteins localize to the major organelles of the early secretory pathway: the endoplasmic reticulum and the Golgi apparatus, where they seem to be involved in trafficking between the two compartments. In yeast, all p24 family proteins can be removed, causing only a mild phenotype. However, in mammals at least some p24 proteins are essential for survival, e.g. removal of p24δ1 is lethal in mice. p24 family members have been implicated in the biogenesis of COPI and COPII-coated vesicles, transporting membrane-bound proteins through the secretory system, and forming the structure of the endoplasmic reticulum and Golgi. b337e8e4fd The TIGER domain was first documented by cell biologists Christine Mayr and Weirui Ma at the Gerstner Sloan Kettering Graduate School of Biomedical Sciences in 2018. The discovery of this cellular structure was significant because

it revealed that ribosomes were not the only membraneless organelle that aided in protein synthesis in association with the ER. b337e8e4fd Membraneless organelles are organelles that lack a lipid membrane and contribute to many biochemical reactions in the cell, such as gene regulation or protein folding. Despite their lack of an outer membrane, they still contain a definite morphology separating them from other components in the cytoplasm. These organelles are also known as biomolecular condensates, due to their structure typically appearing as droplet-like. They are usually... b337e8e4fd

TIGER domain The function == Background ==. The TIGER domain contains two components: TIS granules (TIG) and the endoplasmic reticulum (ER). association with the endoplasmic reticulum (ER) b337e8e4fd

== Function == b337e8e4fd

DNA damage-inducible transcript 3 Those processes are mainly regulated by three factors: protein kinase RNA-like endoplasmic reticulum kinase (PERK), activating transcription factor DNA damage-inducible transcript 3, also known as C/EBP homologous protein (CHOP), is a pro-apoptotic transcription factor that is encoded by the DDIT3 gene. cells. The protein functions as a dominant-negative inhibitor by forming heterodimers with other C/EBP members, preventing their DNA binding activity. It is a member of the CCAAT/enhancer-binding protein (C/EBP) family of DNA-binding transcription factors. The protein is implicated in adipogenesis and erythropoiesis and has an important role in the cell's stress response b337e8e4fd

== Discovery == b337e8e4fd

Derlin-1 Derlin-1 is located Derlin-1 also known as degradation in endoplasmic reticulum protein 1 is a membrane

protein that in humans is encoded by the DERL1 gene. Derlin-1 is widely expressed in thyroid, fat, bone marrow and many other tissues. Derlin-1 also known as degradation in endoplasmic reticulum protein 1 is a membrane protein that in humans is encoded by the DERL1 gene. The derlins mediate degradation of misfolded lumenal proteins within ER, and are named 'der' for their 'Degradation in the ER'. Moreover, derlin-1 is a member of the rhomboid-like clan of polytopic membrane proteins. Derlin-1 is a mammalian homologue of the yeast DER1 protein, a protein involved in the yeast ERAD pathway. Derlin-1 is located in the membrane of the endoplasmic reticulum (ER) and is involved in retrotranslocation of specific misfolded proteins and in ER stress. The protein belongs to the Derlin-family proteins (also called derlins) consisting of derlin-1, derlin-2 and derlin-3 that are components in the endoplasmic reticulum-associated protein degradation (ERAD) pathway b337e8e4fd

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P24 protein family localize to the major organelles of the early secretory pathway: the endoplasmic reticulum and the Golgi apparatus, where they seem to be involved in trafficking P24 protein family is a group of transmembrane proteins that are major components of COPI and COPII-coated vesicles. The latter naming was after transmembrane emp24 domain-containing protein 10 that was found in the human brain. The family is also known as EMP24/GP25L/p24 family and TMP21-like proteins. It was claimed to block the beta-amyloid peptide, which is implicated in the pathogenesis of Alzheimer's disease b337e8e4fd

== References == b337e8e4fd The TIGER domain is a membraneless organelle in the cell which translates messenger RNA (mRNA) for membrane proteins in close association with the endoplasmic reticulum (ER). The TIGER domain contains two components: TIS granules (TIG) and the endoplasmic reticulum (ER). The function of this organelle

has been linked to reducing inflammation in cells. b337e8e4fd The production of SEC23A protein is involved in the pathway of exporting collagen (the COPII pathway), but a missense mutation causes and underproduction of SEC23A which inhibits the pathway, affecting collagen secretion. This decrease in collagen secretion can lead to the bone defects that are also characteristic of the disease, such as skeletal dysplasia and under-ossification. Decreased collagen in CLSD-affected individuals contributes to improper bone formation, because collagen is a major protein in the extracellular matrix and contributes to its proper mineralization in bones. It has also been hypothesized that there are other defects in the genetic code besides... b337e8e4fd

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