Peroxisome in animal cell

Science, Biology

Peroxisomes have an essential function in cell metabolism. [1] They are small, membrane bounded organelles that have at least fifty different enzymes for several metabolic reactions. [2] Their appearance is like lysosomes; however, their main function is oxidation reactions especially oxidation of hydrogen peroxide and producing oxygen and water. There are diseases related and caused by this organelle that can be classified in three groups.

First group is a disorder duo to overall dysfunction of peroxisomes, like zellweger syndrome, second group duo to dysfunction of only some peroxisomal functions, Example for disorder of this group is rhizomelic chondrodysplasia punctata and third group because of dysfunction of a single peroxisomal function, Example of this group is acatalasemia. In this review I will report the function and dysfunction of peroxisomes, their appearance, related diseases future plans as well as techniques for isolation and visualization of them.

From the literature it is known that beta oxidation of long chain and very long chain fatty acids is a lake in group one disorders like infantile refsum's. On the other hand in x-linked adrenoleukodystrophy the lake of beta oxidation of very long chain fatty acids can be seen. Introduction Peroxisome is a core shape organelle in both animal and plant cells bounded by a single membrane. [3] The main function is oxidation in other word it contain enzymes that transfer hydrogen from various substrate to oxygen making hydrogen peroxide as a by-product.

The reactions inside peroxisomes have different functions such as: breaking down the fatty acids to smaller molecules for further reactions in

mitochondria, detoxifying the harmful compounds in cells especially liver cells. Peroxisomes grow larger and after reaching a certain size they will split in two. The problems with dysfunctioning of peroxisomes are mostly a group of genetically heterogeneous metabolic diseases. [4] There are enzymatic abnormalities that can be single or multiple. One of the features of most peroxisomal dysfunctions is neurological disorders.

There are medical care and treatments which depends on several items such as level of disorder, age of patient, risks and benefits of treatment etc. Appearance and location Proxisomes are small vesicles found around the cell and have a single membrane of lipid bilayer that contains digestive enzymes in order to break down the toxic materials in cell. [5] They also have a dense crystalline core in the middle that is the place for oxidative enzymes. [6] Fig 1. Peroxisome and its crystaline core. [7]

They sometimes appear spherical in transmission electron micrographs and have a varying size of 0. 1 fm to 1. 0 fm. [8] Functions Peroxisomes take the required nutrients for cells. [5] They digest fatty acids, amino acids and do cholesterol synthesis and many of them exist in liver cells to take part in ethanol digestion. The peroxisomal enzymes affect complex molecules and break theme down into smaller molecules; one of the most common side products of digestion is Hydrogen peroxide that is broken down to water and oxygen. Oxygen can be used for next digestion reaction.

For example white blood cells produce hydrogen peroxide in order to fight bacteria. [9] This compound will break down by peroxisomal enzymes to oxygen and water. These enzymes (peroxisomal enzymes) are usually created by lysosomes and finally inserted into the peroxisome. [5] The main

function of peroxisome is to help cell remove the toxic material such as hydrogen peroxide; so they contain oxidation enzymes like D-amino acid oxidase, ureate oxidase and catalase. [10] Peroxisomes are a major place of oxygen production thanks to hydrogen peroxide oxidation activity.

Isolation There are different types of isolation for peroxisomes such as, preparation of crude peroxisomal fraction (CPF) from animal tissue, preparation of crude peroxisomal fraction (CPF) from cell cultures and isolation of peroxisomes on a density gradient. [11] The main procedure of peroxisome isolation with different degree of purity is by using a simple method of homogenization plus centrifugation that removes other cell materials like organelles and lipids in order to get a crude peroxisomal fraction for further purification in case it is required.

A highly purified CPF is obtained by density gradient based on differential centrifugation that in some steps longs for 1. 5 hours at 10, 000 g or 3. 5 hours at 45, 000 g. In all of these protocols the procedure is performed at 4 ? C plus all the solutions and equipment should be cooled. Visualisation One way of peroxisome visualization is by attachment of Green fluorescent protein to this organelle and observation via fluorescent microscope. [12] (GFP-PTS1) is a C-terminus peroxisomal targeting signal that will be added to peroxisomes during the transfection of cells by GFP expression plasmid (pGFP).

Peroxisomes are usually visualize as one membrane-bounded vesicles that shows two types of movement, most of them have a slow moves in comparison with the other group that have a fast movement. During experimenting it was appeared that only the second group's movements is

energy dependent. Moreover, high resolution analysis proved the association of peroxisomes with microtubules. What is more is the random distribution of peroxisomes in daughter cells during mitosis. Division of peroxisomes Peroxisomes just like other parts of cell must be divided during cell division.

This organelle has the ability of division, proliferation and degradation in response to environment. There are two types of divisions, constitutive and regulated: During cell division, mitosis, or when the organelle is old constitutive division of peroxisomes which is for increasing the number or volume of the organelle will happen. On the other hand when there is an external signal for proliferation of peroxisomes, regulated division will happen which is not connected with mitosis. Illnesses related to dysfunction of proxisomes in humans

The human peroxisomal disorders are explained as genetic heterogeneous autosomal recessive diseases that are classified in three groups. [13] First one (group A) contain diseases like Zellweger syndrome, infantile Refsum disease and neonatal adrenoleukodystrophy which are characterized by severe neurological and hepatic dysfunction, craniofacial abnormalities and hypotonia, ending to an early death in this disease patients accumulate phytanic acid and very long chain fatty acids in circulation.

The patients in group B, rhizomelic chondrodysplasia punctata, rhizomelia, cataracts, epiphysial calcifications and ichthyosis accumulate higher levels of phytanic acid and in spite of group A, have a normal amount of very long chain fatty acids. Group C shows a milder symptoms based on the mutated gene that as a result will change the activity or localization of single enzyme. Disorder in other groups affects on location of multiple proteins. In

conclusion, although the high number of peroxisomal disorders in an individual can lead to death, being able to propagate their cells in lab environment has provided lots of useful information.

Dysfunctions Proteins of peroxisomes are not made inside but come from cytoplasm after they are synthesized. There should be a kind of signal or sign in order to direct these proteins to peroxisomes. This targeting process occurs with interaction between specific peroxisomal targeting signals (PTS) and their receptors (peroxisome proliferator-activated receptor). In Zellweger disorder in patient cell line an absence of multiple matrix proteins were observed.

Walton et al, Wendland and Subramani by microinjection and in vitro assays showed a defect in the import of PTS1-containing proteins in these cell lines, these researches were extended further by Motley et al and Slawecki et al within both PTS1- and PTS2-containing proteins. These studies provided that the human cells could be divided into four groups from the peroxisomal disorder perspective of view, just like the yeast pex mutants, 1)Only deficiency in PTS1 import; 2)Only deficiency in PTS2 import; 3) deficiency in both PTS1 and PTS2; 4) problem with biogenesis of peroxisomal membrane.

In conclusion there is a high homology between the cellular import deficiency phenotypes of the yeast and humans mutated cells and that the yeast cells are a good model of human peroxisomal disorders. Treatment An improve in number of peroxisomes can be triggered by being treated with pharmaceutical chemicals named peroxisome proliferators that are from fibrate class. [8]The system in which the fibrates cause an improve in peroxisomes number starts with binding of them to peroxisome proliferator-

activated receptor-alpha there are two other isotopes as well beta and gamma.

The alpha receptor is a ligand-dependent proliferators response element in enhancer place of the genes coding for proteins that are participating in uptake, activation and beta oxidation of fatty acids. The activation of alpha receptor makes an increase in the transcription of the genes coding for these peroxisomal enzymes only in some parts and not others. The study in field of peroxisomes has been done in both vitro and vivo by analyzing the expression of genes coding for peroxisomal proteins as well as measurements of enzyme activity.

Conclusion

It is great that in the last decade, our understanding of peroxisomal protein import has been improved rapidly. [13] Without any discussion this organelle has lots of features making it unique. As a future promises are more information on roles of ER in peroxisomes biogenesis and more details in protein import mechanism. Moreover, it is likely that in the next few years the molecular mutations in lots of human peoxisomal disorders will be defined. It is an interesting area for scientists to find out about peroxisome proliferation, homeostasis, movements and why these movements are necessary.