

第1回 代謝病態学セミナー

Physiological Importance of Phosphatidylserine and Phosphatidylethanolamine in Mammalian Cells

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場所：山崎記念館 1F (Yamasaki Memorial Hall 1F)

Abstract:

All mammalian cell membranes contain the two metabolically-related phospholipids phosphatidylserine (PS) and phosphatidylethanolamine (PE). The exposure of PS on the cell surface is a signal for removal of apoptotic cells and for initiation of the blood-clotting cascade. PE is abundant in mitochondrial membranes, is utilized for synthesis of glycosylphosphatidylinositol anchors of proteins, and appears to be required during cytokinesis. In mammalian cells, PS is synthesized on the endoplasmic reticulum (ER) and mitochondria-associated membranes (MAM) by phosphatidylserine synthase-1 (PSS1) and PSS2. We have generated viable mice deficient in PSS1 and/or PSS2. Newly-made PS is imported into mitochondria via membrane juxtaposition between ER/MAM and mitochondria. In mitochondria, PS is decarboxylated to PE by PS decarboxylase (PSD). Although PE is also made from CDP-ethanolamine in the ER, the majority of mitochondrial PE is made in mitochondria. We have generated PSD-deficient mice. Complete elimination of PSD caused embryonic lethality despite doubling in activity of the other PE biosynthetic pathway, indicating that pools of PE can be compartmentalized according to biosynthetic origin. Electron microscopy of embryos lacking PSD, and fluorescence microscopy of embryonic fibroblasts, revealed aberrant mitochondria. We propose that lack of PSD reduces the mitochondrial PE content, thereby causing mitochondrial abnormalities.

本セミナーを、大学院・博士課程講義科目の先端診断学理論の補講といたします。補講としての受講認定を希望する博士課程学生の皆様方には、会場の受講者記名リストに署名ください。

また、このセミナーは、大学院教育改革支援プログラム「臨床・基礎・社会医学一体型先端教育の実践」の一環として実施されます。

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