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Commentary

Mechanism of Ceramide in Lipid Signalling

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DESCRIPTION

Lipid signalling, in its broadest sense, refers to any biological signalling process that involves a lipid messenger binding to a protein target, such as a receptor, kinase, or phosphatase, and then mediating the effects of these lipids on specific cellular responses. Because lipids can easily flow through membranes, lipid signalling is regarded to be qualitatively distinct from other conventional signalling paradigms. Because lipid messengers can't be stored in vesicles before being released, they're generally biosynthesized "on demand" at their target site of action. As a result, many lipid signalling molecules cannot freely circulate in solution and must instead be linked to specific carrier proteins in serum.

Ceramide (Cer) is produced when SphingoMyelinases (SMases), enzymes that hydrolyze the phosphocholine group from the sphingosine backbone, break down SphingoMyelin (SM). Alternatively, the enzymes Serine Palmitoyl Transferase (SPT) and ceramide synthase in organelles such as the Endoplasmic Reticulum (ER) and possibly in Mitochondria-Associated Membranes (MAMs) and perinuclear membranes can synthesise this sphingosine-derived lipid (sphingolipid) from scratch. Ceramide's location in the metabolic hub causes it to produce other sphingolipids, with the C1 hydroxyl (-OH) group being the most common modification site. The enzymes glucosyl or galactosyl ceramide synthases can attach a sugar to ceramide. Ceramide can also be broken down by ceramidases enzymes, resulting in the creation of sphingosine. Ceramide kinase can also attach a phosphate group to ceramide (phosphorylation). The enzyme sphingomyelin synthase may also regenerate sphingomyelin from ceramide by receiving a phosphocholine headgroup from Phosphatidyl Choline (PC) diacylglycerol is formed.

Ceramide has a neutral headgroup and two hydrophobic ("water-fearing") chains. As a result, it has a low water solubility and is only found within the organelle where it was created. Ceramide also rapidly flip-flops across membranes due to its hydrophobic character, as evidenced by experiments in membrane models and red blood cell membranes (erythrocytes). Ceramide, on the other hand, may interact with other lipids to generate larger regions known as microdomains, which limit its ability to flip-flop. Because it is known that ceramide produced by acidic SMase enzymes in the outer leaflet of an organelle membrane may have distinct responsibilities than ceramide produced by neutral SMase enzymes in the inner leaflet, this could have a huge impact on the signalling activities of ceramide.

Ceramide regulates several cell-stress responses, including programmed cell death (apoptosis) and cell ageing. Defining the direct protein targets of action of ceramide has piqued researchers' curiosity in a number of studies. These enzymes, known as Ceramide-Activated Ser-Thr PhosPhatases (CAPPs), such as protein phosphatase 1 and 2A (PP1 and PP2A), have been observed to interact with ceramide in investigations conducted outside of a living organism (in vitro). Ceramide-inducing drugs such as Tumour Necrosis Factor-alpha (TNF) and palmitate, on the other hand, have been demonstrated to cause the ceramide-dependent removal of a phosphate group (dephosphorylation) of the retinoblastoma gene product RB in cell. protein kinases B (AKT protein family) and C (PKB and PKC) enzymes. Ceramide is also linked to the activation of the kinase Suppressor of Ras (KSR) PKC and cathepsin D, according to substantial evidence. Cathepsin D has been proposed as the primary target for ceramide produced in lysosomes, making lysosomal acidic SMase enzymes one of the essential participants in the mitochondrial apoptosis process. Ceramide was also shown to activate PKC, suggesting that it is involved in the inhibition of AKT, the modulation of the voltage differential between the interior and outside of the cell (membrane potential), and apoptosis-promoting signalling actions. Daunorubicin and etoposide are examples of chemotherapeutic agents. In investigations on mammalian cells, increase the *de novo* synthesis of ceramide. Certain inducers of apoptosis, notably receptor stimulators, yielded similar results.

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