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STRATEGIES FOR TARGETING METABOLIC PATHWAYS IN TRIPLE NEGATIVE BREAST CANCER

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Abstract
TNBC is negative for all ER, PR and HER-2, there are no efficient targeted therapies at present. Triple negative breast cancer (TNBC) refers to any breast cancer that does not express the genes for estrogen receptors (ER), progesterone receptors (PR) and HER2/neu. This is what makes it more difficult to treat since most hormone therapies target one of the three receptors. There are 2 subtypes: basal-like and non-basal-like.OXPHOS (Oxidative Phosphorylation) is the metabolic pathway in which cells use enzymes to oxidize nutrients, thereby releasing energy which is used to produce ATP. It mostly takes place in the mitochondria.TNBC shows the expression of both glycolysis- and mitochondrial metabolism-related proteins in tumor cells with higher ratios of glycolysis type in basal-like type tumors and non-glycolysis type in non-basal-like type tumors. It has been found that in Mantle Cell Lymphoma (MCL) resistant to Ibrutinib exhibits increased OXPHOS. MD Anderson Therapeutics team discovered a potent OXPHOS inhibitor, IACS-010759, in order to inhibit oxidative phosphorylation. TNBC cells have high glucose uptake, increase lactate production, and low mitochondrial respiration was related with activation of mTOR pathway and decreased expression of P70S6K gene is reexpressed and their glycolytic pathway is reversed to oxidative phosphorylation. But in certain cases it has been found that lowered OXPHOS activity in TNBC cells renders them highly dependent on glycolysis (Warburg effect). In this review article, we are discussing the possibilities of targeting both glycolysis and oxidative phosphorylation in TNBC to bring out some effective treatments strategies.

Key words: OXPHOS, Glycolysis, TNBC, Warburg effect

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BIOLOGY, CHEMISTRY AND GENETICS OF LIPOPROTEIN DISORDERS: HYPOLIPIDEMIA

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Abstract
Hyperlipidemia and thereby artherosclerosis is the leading cause of cardiac illness and deaths. World wide, it causes deaths almost twice as many as those caused by cancer as 10 times as many as those caused by accident. Hyperlipidemia is an elevation of lipids in the blood stream and these lipids include fats, fatty acids, cholesterol, cholesterol esters, phospholipids and triglycerides. At 1% drop in serum cholesterol reduces the risk for CHD by 2%. Well delineated metabolic abnormalities owing to a single gene defect are rare, whereas multifactorial disorders influenced by the interaction of many genes and environmental factor predominant. This is especially true regarding lipoprotein disorders, which often have a complex etiology. The phenotype of these disorder often modulated by gene-gene and gene-environment interactions. Dietary changes ,gender, hormonal influences and variation of other gene loci are among the major sources of variation in phenotypic expression. Lipoprotein disorders of genetic etiology are the major interest because of their major impact on health. This review focuses on major forms of primary and familial dyslipoproteinemia with an emphasis on their atherogenic potential.

Keywords: Hyperlipidemia, artherosclerosis, cardiac illness and deaths.

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