Studies of Bioenergetics Alterations in Breast Cancer Lines Induced by Sodium Butyrate

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Tumor cells are characterized by a different bioenergetic phenotype compared to normal cells. This phenotype can change with the microenvironment, tumor progression and tumorigenicity. Classically, cancer cells are characterized by enhanced glycolysis even if the oxygen tension is normal (aerobic glycolysis or Warburg effect). Lately, it has been highlighted that mitochondrial function can be important for tumor development [1]. Results in the literature show various effects caused by histone deacetylase inhibitors (HDACis), which induce accumulation of acetylated substrates, generating cell cycle arrest, differentiation and cell death [2]. However, the HDACis effects in the energy metabolism modulation remain elusive. Recent studies have shown that HDACis are able to modulate the glycolytic metabolism and mitochondrial function from highly glycolytic lung tumor cells [3]. In this context we have investigated how sodium butyrate (NaB), a histone deacetylase inhibitor, alters the energy metabolism in breast tumor cell lines with different stages of tumorigenicity. We observed that NaB treatment induced an attenuation of glycolysis, reflected by a decrease in lactate release in MCF-7 and T47D lines. Furthermore, the treatment induced an increase in ROUTINE, LEAK and ETS in T47D, while no change was observed in MCF-7. Interestingly, we observed an increase in ROX of MDA-MB-231, suggesting that NaB can induce the activity of other oxidases. Taken together, these results showed that cells in different stages of tumorigenic progression react differently to NaB which highlight the importance of tumor cells metabolic profile identification for drug treatments.

References:


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