

# 4<sup>th</sup> World Congress on Cancer Science & Therapy

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## Metabolic and redox signaling in breast cancer: Role for PGC-1

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Modifications of the mitochondria in tumors have been presented in the literature, as most cancer cells have to support metabolic transformation in order to promote their proliferation and survival. One of the hallmark alterations of tumor cell metabolism is known as the Warburg Effect, which tumor cells prefer deriving energy through glycolysis, opposed to the more efficient process of oxidative phosphorylation. Defective oxidative phosphorylation will lead to production of reactive oxygen species (ROS), which may enhance cell transformation and ultimately lead to tumor initiation, promotion, and progression. The high metabolic rate of cancer cells drives their intracellular ROS up to an intermediate level, resulting in a shift in redox balance. The peroxisome proliferator-activated receptor- coactivator- 1 (PGC-1) family members are main regulators of several mitochondrial genes. PGC-1 $\alpha$  e  $\beta$  have been considered as main regulators of energy homeostasis of the cell and it has been demonstrated that variations of PGC-1s expression occur in tumor cells in order to promote cell survival. Thus, it is known that breast cancer cells display several mechanisms to promote their survival involving redox-signaling pathways and modifying their metabolism. The mechanisms by which PGC-1s act to control tumor cells proliferations are not completely understood. During this lecture, it will be discussed recent findings regarding redox and metabolic changes in distinct breast cancer subtypes with a special focus in PGC-1s family members.

### Biography

Vanessa Jacob Victorino is a Biologist and has completed her MD from State University of Londrina - Brazil and she is now completing her PhD studies from Sao Paulo University School of Medicine - Brazil. She has published 14 papers in reputed journals and has 10 research awards. Her main research field of interest is metabolic and oxidative alterations in distinct subtypes of breast cancer.

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