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Apoptosis induced by paclitaxel is dependent of BimEL interactome in triple negative breast cancer cells

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Microtubule disrupting agents (MTAs) are frequently used in triple-negative cancer chemotherapy. These compounds induce cell death by regulating the function of proteins involved in mitochondrial outer membrane permeabilization, cytochrome c release and caspase activation. An *in silico* analysis of the protein interaction network associated to BimEL and MTAs (paclitaxel, vincristine and combretastatin A-4) interactome was done. The network had 153 nodes and 2760 connections, and revealed that p53; Bax, Casp-3 and Bcl-2 proteins are essential topological nodes essential nodes. *In vitro* experimental interactome validation was performed on MDA-MB-231 breast cancer cells treated with IC50 of paclitaxel. Paclitaxel induced apoptosis with a significant increase in the BimEL, p53, and Bax proteins concentration, nevertheless, the concentration of Bcl-2, cleaved -Casp-3 and pro-Casp-3 proteins remain constant during the time of treatment. The complex formation of BimEL with p53, Bax or cleaved-Casp-3 increased even at 48 hour of treatment, and a favorable decrease between Bax and Bcl-2 complex was observed. We conclude that apoptosis induced by paclitaxel in triple negative breast cancer cells involve interactions between BimEL and topological nodes, some of them non-reported before, such as BimEL/p53 and BimEL/cleaved-Casp-3. The study suggests that the effectiveness of MTAs treatments in resistant types of cancer cells could be determinate monitoring those complexes occurrence.

Biography

Gina Marcela Méndez-Callejas has completed her PhD in the Biology Applied to Human Health Section from Roma Tre University. She is a member of Biomedical and Human Applied Genetics Research Group from UDCA in Colombia. Currently, she leads the research studies about the basis of molecular interactions induced by anti-cancer drugs of natural and synthetic origin.

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