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Modification of mitochondrial DNA in breast cancer

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Alterations in mitochondrial DNA have been identified in breast cancer, however, the definite biomarkers of mitochondrial DNA (mtDNA) for cancer detection and prediction of the biological behavior and outcome of breast tumors still need to be confirmed forwardly. The object of this study is to investigate the influences of mitochondrial metabolism and mtDNA polymorphism correlated to histologic subtypes of breast cancer on a biological marker TP53 gene (p53) and clinicopathological parameters such as distal metastasis and disease-free survival (DFS) which measured at 5 years. The copy number, oxidative stress (formation of 8-OHdG in mtDNA), mtDNA 4,977-bp common deletion ($mtDNA^{4977}$) and somatic mutations in the D-loop region of mtDNA in breast cancer and paired nontumorous breast tissues from Taiwanese—patients were examined. We found that tumor group relative to normal group with a higher **relative copy number** and lower mtDNA oxidation individually displayed high level distal metastasis increase and poorer DFS. High percentage of *TP53* mutation only observed in tumor group with a higher **relative copy number**. For alterations in mtDNA variation in the breast cancer, the mtDNA from tumor tissues with negative $mtDNA^{4977}$ and high-level of D-loop mutation had high-level increase of *TP53* mutation percentage and distal metastasis following the poorer DFS. These results reveled that patients with higher copy number, lower oxidative damage, D-loop mutation of mtDNA and without $mtDNA^{4977}$, the malignant progression of breast cancer could be raised. The related regulation factor could be related with P53-**mediated** cell death for cancer cells.