In the current thesis we studied the pten onco- suppressor for loss of heterozygosity in patients with different stages of breast cancer. For this purpose we isolated DNA from human blood and studied heterozygosity by using the primers D10S215, D10S549 and D10S571. The results that came from the Licor analyzer elaborated with the FSTAT program and confirmed a big loss of heterozygosity in the total amount of the samples.

Beyond the pten gene, we studied the expression of the PTEN protein as well as the phosphorelated type P-PTEN. PTEN is a phosphatase with both lipid and protein action. PTEN acts on the phospholipids PIP3 of the membranes and transforms them into PIP2. Thus PTEN acts as an antagonist in the PI3K/AKT signaling pathway.

For this purpose we isolated proteins from human blood using the TRI reagent. We made Western blotting with the PTEN and P-PTEN antibodies and we studied the difference in the expression.

The levels of the expressed active PTEN were very low in healthy people, comparing with the patients with breast cancer. We found that the PTEN active type of the protein was expressed in bigger amounts in the patients with hyperplasia and lobular neoplasia, indicating the need for onco-suppression.

We also found that the cells were expressing the active PTEN, were not expressing the non active P-PTEN type. We confirmed the existence of an on-off switch mechanism that transforms the protein, from the active to the non active type according to the needs of the cells.

Due to the complexity of the disease we need to study simultaneously the action of other molecules of the pathway.