

# The relationship between vorinostat and breast cancer in women

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## Abstract

**Introduction:** Vorinostat (suberoylanilide hydroxamic acid) is a hydroxamic acid derivative that inhibits both class I and II histone deacetylases. The mechanism for the ant proliferative effect of vorinostat is believed to be the result of inhibition of histone deacetylase activity, resulting in the accumulation of acetylated proteins, including histones. The increase in DNA damage caused by several anticancer drugs has been shown to increase the risk of developing secondary cancer. The response to vorinostat could be improved by combining it with antioxidants such as vitamin E for the treatment of oxidatively stressed human malignancies that are otherwise resistant to vorinosta.

**Materials and methods:** Our statistical population included 100 patient and 100 control samples. Venous blood (5 ml) was individually taken and DNA was extracted by ethanol precipitation method. The designed sequence was then amplified by PCR and the specified fragments were excised via RFLP technique. Finally, data were analyzed by SPSS V.22 and T-test..

**Results:** The results show that vorinostat (10  $\mu$ M) induced significant aberrations in whole blood lymphocytes and that tempol (10  $\mu$ M) significantly reduced the number of vorinostat induced aberrations. Treatment of cultures with vorinostat alone did affect spontaneous levels of CAs in the control group.

**Conclusion:** In this study, we examined the potential protective effect of tempol on Genotoxicity of vorinostat in whole blood lymphocytes using Real Time PCR and RFLP assays. Vorinostat is a genotoxic drug to whole blood lymphocytes. It also induces oxidative DNA damage.

Keywords: vorinostat, breast cancer, polymorphism, oxidative DNA