

Abstract

DNA repair and DNA damage response are very important biological systems, inevitable to maintain genomic stability and fidelity of the genetic information, for the onset of ovarian cancer. Further, DNA repair is also substantially involved in the response to the therapy, since many chemotherapeutics act as DNA damaging agents.

This literary analysis is intended to survey the relevance of DNA repair to ovarian carcinogenesis. Special emphasis is placed on repair defects, as it is inextricably associated with the onset of cancer and treatment outcome. Apart from well-known alternations in ovarian cancer susceptibility genes, such as *BRCA1* and *BRCA2* involved in homologous recombination repair, ample space will be dedicated to less common gene mutations across different repair pathways.

Research confirms that abnormalities in the proteins responsible for homologous recombination repair are the leading cause of ovarian cancer. The majority of authors also suggested that targeting DNA repair pathways, especially base excision repair, can improve chemotherapy efficiency in a synergic manner. The same applies to nucleotide excision repair, which repairs platinum-DNA adducts and thus contributes to platinum drugs resistance emerging. By way of contrast, mismatch repair in ovarian cancer is rather poorly investigated and its deficiency is frequently related to Lynch syndrome, which predispose people to get colon as well as extracolonic cancers.

Key Words: DNA damage, DNA repair, ovarian cancer, incidence, therapy, resistance towards chemotherapy

