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Molecular iodine impairs chemoresistance mechanisms, enhances doxorubicin retention and induces downregulation of the CD44+/CD24+ and E-cadherin+/vimentin+ subpopulations in MCF-7 cells resistant to low doses of doxorubicin.

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Abstract

One of the most dreaded clinical events for an oncology patient is resistance to treatment. Chemoresistance is a complex phenomenon based on alterations in apoptosis, the cell cycle and drug metabolism, and it correlates with the cancer stem cell phenotype and/or epithelial-mesenchymal transition. Molecular iodine (I2) exerts an antitumor effect on different types of iodine-capturing neoplasms by its oxidant/antioxidant properties and formation of iodolipids. In the present study, wild-type breast carcinoma cells (MCF-7/W) were treated chronically with 10 nM doxorubicin (DOX) to establish a low-dose DOX-resistant mammary cancer model (MCF-7/D). MCF-7/D cells were established after 30 days of treatment when the culture showed a proliferation rate similar to that of MCF-7/W. These DOX-resistant cells also showed increases in p21, Bcl-2 and MDR-1 expression. Supplementation with 200 µM I2 exerted similar effects in both cell lines: it decreased the proliferation rate by ~40%, and I2 co-administration with DOX significantly increased the inhibitory effect (to ~60%) and also increased apoptosis (BAX/Bcl-2 index), principally by inhibiting Bcl-2 expression. The inhibition by I2 + DOX was also accompanied by impaired MDR-1 induction as well as by a significant increase in PPARγ expression. All of these changes could be attributed to enhanced DOX retention and differential down-selection of CD44+/CD24+ and E-cadherin+/vimentin+ subpopulations. I2 + DOX-selected cells showed a weak induction of xenografts in Foxn1nu/nu mice, indicating that the iodine supplements reversed the tumorogenic capacity of the MCF-7/D cells. In conclusion, I2 is able to reduce the drug resistance and invasive capacity of mammary cancer cells exposed to DOX and represents an anti-chemoresistance agent with clinical potential.

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