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Evidence for the implication of human DDC and carbidopa in human cancer cell viability

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L-Dopa decarboxylase (DDC) is a pyridoxal 5-phosphate (PLP)-dependent enzyme that catalyses the decarboxylation of L-Dopa to dopamine (DA). DDC has been identified as a co-activator of Androgen Receptor (AR) and is considered to be a neuroendocrine marker for the detection of prostate cancer. The role of DDC in the pathogenesis of prostate cancer remains unknown. Recent studies have shown that carbidopa inhibits the DDC-dependent activation of AR. In this report, we have investigated the effect of two DDC inhibitors, namely carbidopa (α -methyl-dopahydrazine) and NSD-1015 (3-hydroxybenzyl-hydrazine), on the survival of DU-145 (Androgen Receptor-Independent) cells, as well as on the expression of DDC. The obtained cytotoxicity effects were compared with those obtained for the human neuroendocrine cell line SH-SY5Y, as well as in CHO cells expressing human DDC (CHO/DDC). Both carbidopa and NSD-1015 exerted considerable cytotoxic effects on DU-145 cells. Furthermore, carbidopa was shown to influence the expression of DDC on DU-145 cells. Interestingly, our results indicated that carbidopa and NSD-1015 treated CHO/DDC cells demonstrated an impressive degree of cytotoxicity in the presence of these molecules, as compared to CHO cells which do not express human DDC. Our results provide evidence for involvement of DDC regulation in cytotoxicity and in Androgen–Receptor Independent prostate cancer cell survival. The mechanism of the observed carbidopa-induced cell death, in addition to the study of the involvement of DDC in AR-independent prostate cancer cell viability, are currently under investigation.

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