Spring 3-15-2011

Leptin Protects Dendritic Cells from Chemically-Induced Cell Death

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Leptin is a pleiotropic adipokine that exerts its physiological effects by binding to its receptor, which is expressed in many immune cells including T cells, macrophages, and dendritic cells. Multidrug resistance-associated protein (MRP) is a transmembrane transporter commonly associated with cancer cells conferring resistance to chemotherapy. The purpose of this study is to determine if leptin supersedes the cytotoxic death signal in dendritic cells. We further hypothesize that the mechanism underlying leptin protection is a result of over expression of MRPs. To address this question, we examined the effect of leptin on monocytes treated with cytotoxic drugs *in vitro.* Bone marrow-derived dendritic cells (BM-DCs) and JAWSII were treated with camptothecin (CPT), plumbagin (PB8) or doxorubicin (Doxo) in the presence or absence of leptin. CPT, Doxo and PB8 are anticancer drugs that inhibit topoisomerase I, topoisomerase II and induce free radical formation respectively. Cell line viability was assessed via a colorimetric assay. Induction of cell death was measured by annexin V/propodium iodine staining. BMDCs generated from DB mice with a nonfunctional leptin receptor were used in response to camptothecin, leptin or a combination. The data demonstrates that leptin inhibits cell death upon treatment with cytotoxic agents.