



Autophagy: Chapter 11. The Presence of Dioxin in Kidney Cells Induces Cell Death with Autophagy

Filomena Fiorito, Luisa De Martino

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Autophagy is a cellular process of “self-eating,” which involves the digestion of cytoplasmic components via the lysosomal pathway as resources during starvation or other limiting conditions. However, many studies have indicated that autophagy may represent a cellular response to stress conditions. As a consequence, autophagy can directly induce cell death or act as a mechanism of cell survival. It is well established that 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) may promote a multiplicity of toxic effects in mammalian cells. In particular, kidney anomalies are correlated with dioxin exposure by inducing augmented renal immune complex deposition, glomerulonephritis, and hydronephrosis. In Madin–Darby Bovine Kidney (MDBK), a kidney epithelial cell line, our group demonstrated that TCDD provokes cell proliferation and impairs cellular iron homeostasis, leading to changes in the extent of the labile iron pool. These processes could be associated with neoplastic transformation of the bovine kidney cell. Analysis of MDBK cell morphology revealed some death alterations in a large number of exposed cells where signs of neither apoptosis nor necrosis were detected, but we found that dioxin activated cell death with autophagy. Our data establish the requirement for autophagy in the maintenance of MDBK cells exposed to dioxin, as well as providing evidence suggesting that autophagy protects against proliferative effects induced by non-genotoxic compounds such as TCDD. Indeed, multiple evidences indicate that autophagy plays a critical role in kidney maintenance, diseases, and aging. Generally, autophagy serves as a protective mechanism, but persistent activation of autophagy can result in cell death, mainly after exposure to toxic agents.

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