Autophagy: Chapter 18. Blockage of Lysosomal Degradation is Detrimental to Cancer Cell Survival: Role of Autophagy Activation

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Autophagy is a major catabolic process used by cells to remove superfluous or damaged proteins and organelles. In the final stages of autophagy, acidic organelles (lysosomes) act to degrade autophagic cargo and to facilitate their recycling. Little is known about how cancer cells undergoing autophagy, often as a consequence of stress, respond when lysosomal function is blocked. To elucidate this mechanism, several recent studies report that lysosomes and their hydrolytic proteases (cathepsins) play a critical role in autophagy and subsequent cancer progression. Our studies in breast cancer suggest that inhibition of cathepsins D and L using the BH3-mimetic, obatoclax, is effective in reducing the cell density of anti-estrogen sensitive and resistant breast cancer cells. Furthermore, blockage of cathepsin protein expression with obatoclax leads to the accumulation of autophagic vacuoles and impairs the ability of cells to use degraded material to restore homeostasis. While cancer cells are dependent on effective lysosomal function, neoplastic transformation induces changes in lysosomal volume, number, and protease activity. Recent reports suggest that pro-oncogenic changes render cancer cells more susceptible to lysosomal-associated death pathways. A number of distinct stimuli have been shown to permeabilize the lysosomal membrane, leading to the release of hydrolases into the cytosol and, ultimately, cell death. Thus, changes in cathepsin and lysosomal membrane permeabilization (LMP) regulation during cancer cell progression suggest that strategies targeting this cellular compartment may be exploited to improve outcomes for cancer patients.

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