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Autophagy in regulation of aquaporins

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Aquaporins (AQPs) are water channel proteins – widely expressed among living organisms –facilitating water transport across cell membranes. At least 13 AQPs (AQP0-12) are found expressed in mammals, which transport not only water but also some small neutral solutes (e.g. glycerol, H_2O_2, gas). The modulation and transport properties of AQPs are intensively investigated in the body and a number of important functions have been already described both in health and diseases. Autophagy is a highly conserved biological process that involves removal of protein aggregates and damaged organelles and the subsequent transport of these components to the lysosome for degradation. Emerging data suggest that autophagy influence renal epithelial transport. Autophagic degradation of AQP2, a key aquaporin located in the renal collecting ducts, is found to contribute to urine concentration defect in several animal models of nephrogenic diabetes insipidus (hypokalemia, hypercalcemia, lithium-treatment, BUO). Our recent data shows that hypotonic stress induces downregulation of AQP1 and activation of autophagy in mouse IMCD3 cells, which is improved when autophagy is suppressed by 3-MA, CQ, or ATG5 knockdown, a key autophagy factor. The significance of autophagy regulation of AQPs in epithelial cells is still unknown, but may be in favor of saving and producing energy in times of stress. On the other hands, evidence indicates that AQPs may be involved in autophagy activation in some conditions, for examples, aquaglyceroporins AQP3 and AQP9 are shown to regulate autophagy activity in cancer or aging cells. More studies on the interaction between aquaporin and autophagy are of significance in better understanding molecular mechanism of certain relevant diseases.