BOTH ACUTE AND CHRONIC CELL VOLUME CHANGES ARE ENGAGED IN REGULATION OF ADIPOCYTE FUNCTIONS

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Recent article of Edwin Mariman published in this volume of Adipobiology (1) brings new interesting view on the mechanism involved in weight loss and regain: changes of the cell volume and content of adipocytes and their impact on secretion of adipokines. While acute changes of cell volume are well known to be integrated into a signal transduction network affecting various functions including secretion, macromolecule biosynthesis and degradation, cell migration, proliferation and apoptosis (reviewed in 2,3), impact of chronic changes of cell volume on function have been rarely considered.

As recently reviewed, acute cell volume changes induced in adipocytes by an application of a hypertonic or a hypotonic solution alter one of their fundamental physiological functions: glucose uptake and metabolism (4). Osmotically shrunken adipocytes show insulin resistance, which is a characteristic of type 2 diabetes mellitus, whereas glucose uptake is facilitated in osmotically swollen adipocytes (4).

As discussed in the model by Mariman, during weight loss adipocytes build up resistance against releasing more fat, which is demonstrated by the differential expression of markers for cellular stress. This stress may be based on mechanical forces that arise between the shrinking cell and the surrounding rigid basal lamina, raising the importance of cell-extracellular matrix interactions in the regulation of adipocyte functions. For adipocytes the best way to alleviate this stress would be returning to their original volume. Secretion profile is related to adipocytes size isolated even from the same individual (5); during weight loss and shrinkage of cells, adipocytes will automatically change their adipokine secretion profile. Model is based on the fact that chronic change of cell volume results in long lasting stimulation or inhibition of secretion (secretion of leptin and other proinflammatory adipokines is positively related to adipocyte volume) (5). Changed secretion profile of adipokines is a strong regulatory factor promoting regain of body weight. Moreover, lipid droplets have recently been identified as a substrate for macroautophagy (6,7); whether this latter function may be altered in response to changes of adipocyte volume, remains to be evaluated.

In brief, Mariman’s adipobiological model for weight regain after weight loss (1) – important role of adipocyte volume and cell-matrix interactions in handling body weight – offers possibility of a novel therapeutic approach in obesity and related diseases.

References


