

Histopathology of the obese adipose organ

Saverio Cinti

Dpt Experimental and Clinical Medicine, University of Ancona, Italy. cinti@univpm.it

Most of white and brown adipocytes, in spite of their well known different functions: i.e. storing energy (white) and thermogenesis (brown), are contained together in visceral and subcutaneous depots (adipose organ) in all mammals including humans. A growing body of evidence suggests that the reason for this anatomical mixture could reside in the fact that adipocytes have peculiar plastic properties allowing them to convert directly each other under appropriate stimuli (1). Under chronic cold exposure white convert into brown to support the need for thermogenesis and under obesogenic diet brown convert into white to satisfy the need of energy storing. Adipocyte population in the mammary gland offers another striking example of adipocyte plasticity: during pregnancy and lactation adipocytes transdifferentiate into milk-producing epithelial glands and vice versa in the post-lactation period. The white into brown transdifferentiation is of great medical interest because the brown phenotype of the adipose organ is associated with obesity resistance and drugs inducing the brown phenotype curb obesity and related disorders.

Type 2 (T2) diabetes is the most common disorder associated to visceral obesity. Macrophages infiltrating the adipose organ are responsible for the low-grade chronic inflammation dealing to insulin resistance and T2 diabetes. This inflammation is caused by the need of removal debris deriving from the death of adipocytes (2). Death of adipocytes is tightly related to their hypertrophy up to the critical death size. Visceral adipocytes have a critical death size smaller than subcutaneous adipocytes, thus explaining the higher inflammation and higher morbidity of visceral fat (3). The smaller critical death size of visceral adipocytes could be explained by their origin from brown adipocytes transformed into small white adipocytes less expansible than the constitutive white adipocytes (4).

1) [Frontini A](#) and [Cinti S](#). *Distribution and development of brown adipocytes in the murine and human adipose organ*. *Cell Metab*. 2010 Apr 7;11(4):253-6.

2) [Cinti S](#), [Mitchell G](#), [Barbatelli G](#), [Murano I](#), [Ceresi E](#), [Faloia E](#), [Wang S](#), [Fortier M](#), [Greenberg AS](#), [Obin MS](#). *Adipocyte death defines macrophage localization and function in adipose tissue of obese mice and humans*. *J Lipid Res*. 2005 Nov;46(11):2347-55.

3) [Murano I](#), [Barbatelli G](#), [Parisani V](#), [Latini C](#), [Muzzonigro G](#), [Castellucci M](#), [Cinti S](#). *Dead adipocytes, detected as crown-like structures, are prevalent in visceral fat depots of genetically obese mice*. *J Lipid Res*. 2008 Jul;49(7):1562-8.

4) [Cinti S](#). *Reversible physiological transdifferentiation in the adipose organ*. *Proc Nutr Soc*. 2009 Nov;68(4):340-9. Epub 2009 Aug 24.