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Proliferation and Differentiation of Human Adipocyte Precursor Cells: Differences Between the Preperitoneal and Subcutaneous Compartments

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ABSTRACT

Human adipocyte precursor cells (APC) have been characterized in their proliferation and differentiation potential from subcutaneous, omental, and mesenteric depots, mostly from morbidly obese patients. Cells from the preperitoneal adipose compartment have not been characterized yet, least of all when obtained from normal weight subjects. The aim was to compare proliferation and differentiation of subcutaneous (SC) and preperitoneal (PP) APC derived from adipose tissue in healthy subjects with different body mass. SC and PP adipose tissue was obtained during surgery of inguinal hernias in five healthy non-obese subjects and three obese otherwise healthy men. APC, obtained by collagenase digestion, were cultured. Proliferation was assayed by cell counting and differentiation by oil red O staining and flow cytometry using Nile Red staining. Proliferation of SC was higher than PP APC. Such differences between both compartments were even higher in APC obtained from obese patients. Conversely PP APC differentiated earlier in vitro compared with SC cells. These results agree with published data on fat cell proliferation. However regarding differentiation, our data show that APC from deeper depots (in this case PP) differentiate earlier than subcutaneous APC. This is different to previous studies performed in mesenteric or omental adipose tissue. J. Cell. Biochem. 111: 659–664, 2010.

KEY WORDS: ADIPOSE; PROLIFERATION; DIFFERENTIATION; FAT; HUMAN; PRECURSOR; PREADIPOCYTE

One of the main roles of adipose tissue is storage of triglycerides, according to energy balance. This is accomplished by hypertrophy of mature adipocytes as well as proliferation and differentiation of adipocyte precursor cells (APC) towards lipid-laden adipocytes. However, there is scarce information on human adipocyte expansion, and most studies have been performed in morbidly obese subjects. Limited information derived from normal or slightly overweight subjects is available; furthermore, tissue has been obtained from cancer patients requiring laparotomy. Obviously, adipose tissue samples obtained during hernia surgery and from non-obese subjects are smaller and thus, it is harder to accomplish primary cultures of APC.

The main cellular components of adipose tissue are mature adipocytes of different sizes and stromal-vascular cells, which include macrophages and preadipocytes or APC. Adipose tissue mass is determined by both adipocyte number and size. Therefore, obesity is caused both by hyperplastic growth through mitotic activity in precursor cells and further differentiation, and by hypertrophic growth, this is an increase in the size of adipocytes, due to lipid accumulation within the cell. Hyperplastic growth predominates during the third trimester of gestation and during prepuberty and puberty, but it also occurs in adulthood; long-term overfeeding and weight regain could induce adipocyte hypertrophy and hyperplasia [Löfgren et al., 2005; Jackman et al., 2008; Spalding et al., 2008]. It has been shown that adipose cells are able to re-enter the cell cycle during early stages of adipogenesis, resulting in increase of cell number [Fajas et al., 2001].

In adult human beings, adipose tissue depots show specific body distribution, in relation to degree of overweight, gender and genetic background, among other factors. Abundant research data relate abdominal fat with cardiovascular risk and metabolic diseases. Abdominal fat depots vary in size, function, and potential contribution to disease. Most studies focus on two main compartments: subcutaneous (SC) and visceral or intraperitoneal. However, recent studies have even detected differing capacities for replication, adipogenesis, and apoptosis between APC obtained from two distinct intraperitoneal visceral fat depots, mesenteric, and omental [Tchkonia et al., 2007]. Another less studied abdominal fat depot is the preperitoneal (PP), which widens in obese patients [Bortolotto et al., 2005], can be estimated adequately by ultrasonography [Liu

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