

## Letter to the Editor

### Mature adipocytes: Are there still novel things that we can learn from them?

The popular press, clinicians, and scientists are all warning us that consuming excess energy may be health-hazardous or even pathologic. Despite the caveat, people (especially in developed countries) are getting fatter (Wang and Lobstein, 2006) and fast foods sales are reaching new heights. Fewer people are sitting down for a family meal, which is time consuming. Instead, fast foods are popular—even for the home. Mixed into the health equation are other (rather unknown) dysfunctions related to body composition. For example, we know that there are instances such as the case involving metabolic syndrome whereby consuming excess energy is not the entire story as to the pathology observed (Batsis et al., 2007; Tchernof, 2007).

A key to the understanding of energy flow, storage or (otherwise) involvement in body composition is the adipocyte. Once considered as a part of a mechanism by which excess energy is stored, the adipocyte is emerging as a complex endocrine organ that is very important to our understanding of body composition. Depot-specific adipocytes produce a variety of systemic factors which regulate a myriad of physiologies—including the amount of body fat (Fonseca-Alaniz et al., 2007). Terms such as leptin, adipokines, and resistin have been coined to identify a few of the adipocyte-secreted compounds that influence/regulate distribution of fat, adipose depot sensitivity to other compounds, or immune functions (Lago et al., 2007). Studying adipocytes is in vogue and advances are being made in rapidly with respect to identifying the extent of adipocyte involvement in human health.

Are there still novel things that we can learn from adipocytes? Our present idea of the cellularity of adipocytes involves the term adipogenesis. Adipogenesis, a component of morphogenesis, may be compartmentalized/defined in general terms as the proliferation and subsequent differentiation of a cell within the fat cell lineage that is then capable of the assimilation of lipid to form a lipid-containing adipocyte. Mature adipocytes function in lipid storage, energy flow, and the production of regulatory compounds. However, adipocytes also appear capable of proliferating (at least *in vitro*) with loss of cytosolic lipid in an asymmetric or symmetric manner with each subsequent cell division (Dodson et al., 2005; Fernyhough et al., 2005b). This process has

been termed dedifferentiation (reviewed in Fernyhough et al., 2005a).

While the proliferation of an otherwise “differentiated” adipocyte may only occur *in vitro*, what valuable insight might we gain from this process? We have proposed numerous possibilities for the reversion of an apparently mature adipocyte undergoing proliferation *in vitro* (Fernyhough et al., 2005a,b). Moreover, we now hypothesize that deciphering the regulation of this process (perhaps at the single cell level) may reveal new insights into the regulation of conventional adipogenesis. This may result in identifying potential new lipostatic compounds with which to combat obesity-related issues involving fat cells. Furthermore, as the proliferative-competent progeny cells appear fibroblastic in phenotype, there is a potential to identify new (stem-like) cells for use in tissue engineering/replacement therapy (Fernyhough et al., 2008). Whatever the immediate future may bring with respect to new knowledge being obtained from mature adipocytes, it seems reasonable to suggest that these cells, and the process of dedifferentiation, will become a new avenue for research for many years to come.

### References

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