Inverse relationship between body mass index and mitochondrial oxidative phosphorylation capacity in human subcutaneous adipocytes.

Abstract:

Obesity is characterized by a substantial increase in adipose tissue that may contribute to energy balance. Recently, obesity was suggested to be associated with impaired mitochondrial function in adipocytes. In this study, we investigated the following: 1) the respiratory capacities of mitochondria isolated from mature adipocytes of female subjects whose body mass index (BMI) values were distributed over a wide range and 2) the amounts of electron transport chain complexes in these mitochondria. Fat cells were isolated from adipose tissue specimens by collagenase digestion. Mitochondria were isolated from these fat cells, and their respiratory capacity was determined using a Clark-type electrode. Fat cells were also sorted on the basis of their size into large and small fractions to assess their respiration. Western blot analyses were performed to quantify respiratory chain complex components. We also examined mitochondrial activity development during differentiation using human Simpson-Golabi-Behmel syndrome cells. Our results showed that mitochondrial respiratory capacities in adipocytes were inversely associated with BMI values but were independent of cell size. Western blot analyses revealed significantly fewer complex I and IV components in adipose tissues from obese compared with nonobese.
women. These results suggest that differences at the level of respiratory chain complexes might be responsible for the deterioration of respiratory capacity in obese individuals. In particular, electron transport at the level of complexes I and IV seems to be most affected.