Summary

Excessive accumulation of adipose tissue is causally related to the development of insulin resistance and glucose intolerance with subsequent development of type 2. diabetes and increased cardiovascular mortality. Although there is relatively convincing epidemiological evidence, mechanisms mediating adverse effects of obesity and associated metabolic disorders still remain only partially elucidated. Among the factors that may play a causal role were determined increased lipolytic activity and related increased release of free fatty acids from adipose tissue. Excessive accumulation of adipose tissue, including all related comorbidities is associated with the development of sleep disorders including obstructive sleep apnea syndrome (OSA). OSA is a relatively common disease that is characterized by the partial obturation of the upper airway during sleep, the so-called Intermittent hypoxia. IH according to the severity of the resulting decrease in hemoglobin saturation is also associated with adipose tissue hypoxia. As confirmed in this study, both mild and severe hypoxia in adipose tissue, even upon exposure to cell cultures resulted in increased lipolytic activity and increased accumulation of triglycerides compared to control conditions. Furthermore, it was confirmed that pharmacological intervention of this diabetic phenotype may completely prevent its development. This may be a crucial step in the search for some new treatments of metabolic comorbidities which are associated with the development of OSA and obesity. Exposures of cell culture and tissues to intermittent and continuous hypoxia was due to present artifacts and limitations very difficult to interpret. In this study, we have managed to design a system that allowed us to bypass these limitations and it can become an important tool in the context of future metabolic research, not only in our laboratory.