Obesity has increased at an alarming rate over the past three decades. It is likely to be caused by increased caloric intake combined with genetic predisposing factors and is a major risk factor for type 2 diabetes (T2D) and cardiovascular disorders. Obesity is linked to chronic inflammation, which was proposed as a cause for insulin resistance and T2D. However, the molecular mechanism of obesity driven T2D still lacks knowledge and thus is inaccessible to therapeutic intervention. In our research, we are exploring the molecular mechanism underpinning this association and aims to identify novel therapeutic targets for the rapidly growing population of obese individuals living with diabetes. The obese state creates a microenvironment within the adipose tissue that is susceptible to the accumulation of reactive oxygen species (ROS). Accumulated ROS causes oxidation of macromolecules such as DNA. Oxidative DNA damage encompasses a variety of DNA lesions such as DNA single-strand breaks (SSBs) and DNA double-strand breaks (DSBs) including non-telomeric and telomeric DSBs. Broken or dysfunctional telomeres have been hypothesized as a cause of cellular senescence, a state of irreversible cell cycle arrest observed in aging illnesses and even in obesity with accumulation of senescent adipocytes. A major player in detecting and defending against accumulation of oxidative DNA damage is PARP-1. PARP-1 activation has been shown to be associated to diabetes and obesity. We are exploring the role of PARP-1 in ROS-induced senescent and inflamed adipocytes by using a combination of biochemical, molecular and cell biological assays in primary human and mice adipocytes treated with hydrogen peroxide (H₂O₂) as a source of ROS.