

Reading: an article

Prof.Dr.Nuray ARI, 2018

- [Oxid Med Cell Longev](#). 2015:534873. doi: 10.1155/2015/534873. Epub 2015 Mar 23.
- Oxidative stress and adipocyte biology: focus on the role of AGEs.
- [Boyer F](#), [Vidot JB](#), [Dubourg AG](#), [Rondeau P](#), [Essop MF](#), [Bourdon E](#).

- Abstract

- Diabetes is a major health problem that is usually associated with obesity, together with hyperglycemia and increased advanced glycation endproducts (AGEs) formation. Elevated AGEs elicit severe downstream consequences via their binding to receptors of AGEs (RAGE). This includes oxidative stress and oxidative modifications of biological compounds together with heightened inflammation. For example, albumin (major circulating protein) undergoes increased glycoxidation with diabetes and may represent an important biomarker for monitoring diabetic pathophysiology. Despite the central role of adipose tissue in many physiologic/pathologic processes, recognition of the effects of greater AGEs formation in this tissue is quite recent within the obesity/diabetes context. This review provides a brief background of AGEs formation and adipose tissue biology and thereafter discusses the impact of AGEs-adipocyte interactions in pathology progression. Novel data are included showing how AGEs (especially glycated albumin) may be involved in hyperglycemia-induced oxidative damage in adipocytes and its potential links to diabetes progression.