

makale

- [Format: Abstract](#)
- [Send to](#)
- [J Diabetes](#). 2017 Feb;9(2):141-148. doi: 10.1111/1753-0407.12475. Epub 2016 Sep 25.
- **Glycation and cardiovascular disease in diabetes: A perspective on the concept of metabolic memory.**
- [Yamagishi SI](#)¹, [Nakamura N](#)¹, [Matsui T](#)¹.
- [Author information](#)
- **Abstract**
- Epidemiological studies have suggested that cumulative diabetic exposure, namely prolonged exposure to chronic hyperglycemia, contributes to the increased risk of cardiovascular disease (CVD) in diabetes. The formation and accumulation of advanced glycation end-products (AGEs) have been known to progress under hyperglycemic conditions. Because AGEs-modified collagens are hardly degraded and remain in diabetic vessels, kidneys and the heart for a long time, even after glycemic control has been achieved, AGEs could become a marker reflecting cumulative diabetic exposure. Furthermore, there is a growing body of evidence that an interaction between AGEs and the receptor for AGEs (RAGE) plays a role in the pathogenesis of CVD. In addition, AGEs induce the expression of RAGE, thus leading to sustained activation of the AGEs-RAGE axis in diabetes. Herein we review the pathological role of the AGEs-RAGE axis in CVD, focusing particularly on the phenomenon of metabolic memory, and discuss the potential clinical usefulness of measuring circulating and tissue levels of AGEs accumulation to evaluate diabetic macrovascular complications.