# Article information:

AGEs, rather than hyperglycemia, are responsible for microvascular complications in diabetes: a "glycoxidation-centric" point of view - PubMed
<https://pubmed.ncbi.nlm.nih.gov/23786818/>

# Article summary:

1. Advanced glycation end products (AGEs) are responsible for microvascular complications in diabetes, rather than hyperglycemia.

2. AGEs have several intra- and extracellular targets, making them a "bridge" between intracellular and extracellular damage.

3. An effective approach to prevention and treatment of diabetic complications should focus on reducing factors related to oxidative stress, including dietary intake of exogenous AGEs.

# Article rating:

Appears moderately imbalanced: The article provides some useful information, but is missing several important points or pieces of evidence that would be required to present the discussed topics in a balanced and reliable way. You are encouraged to seek a more balanced perspective on the presented issues by exploring the provided research topics and looking at different information sources.

# Article analysis:

The article "AGEs, rather than hyperglycemia, are responsible for microvascular complications in diabetes: a 'glycoxidation-centric' point of view" by N.C. Chilelli et al. presents a review of the role of advanced glycation end products (AGEs) in the pathophysiology of chronic diabetic complications. The authors argue that AGE excess is one of the most important mechanisms involved in the development of microvascular complications such as diabetic retinopathy, nephropathy, and neuropathy.

The article provides a comprehensive overview of recent studies that demonstrate the central role of AGEs in the progression of these complications. The authors argue that AGEs are not just "actors" but "directors" of processes leading to these complications due to their ability to target both intra- and extracellular structures. Furthermore, they suggest that AGE-related intracellular glycation triggers a vicious cycle that amplifies AGE formation and contributes to metabolic memory.

While the article provides valuable insights into the role of AGEs in diabetic complications, it has some potential biases and limitations. Firstly, it focuses solely on the role of AGEs and does not consider other factors such as inflammation or oxidative stress that may also contribute to microvascular complications. Secondly, it presents an overly simplistic view by suggesting that hyperglycemia is not a significant factor in diabetic complications when evidence suggests otherwise.

Additionally, while the article highlights the importance of reducing factors related to oxidative stress and dietary intake of exogenous AGEs for prevention and treatment, it does not provide specific recommendations or guidelines for achieving this goal. Moreover, it does not explore potential risks associated with reducing dietary intake of exogenous AGEs or provide evidence for claims made regarding their impact on diabetic complications.

Overall, while this article provides valuable insights into the role of AGEs in diabetic microvascular complications, its narrow focus and potential biases limit its usefulness as a comprehensive guide for prevention and treatment. Further research is needed to fully understand the complex mechanisms involved in diabetic complications and develop effective strategies for prevention and treatment.

# Topics for further research:

* Role of inflammation in diabetic microvascular complications
* Contribution of oxidative stress to diabetic retinopathy
* nephropathy
* and neuropathy
* Importance of hyperglycemia in the development of diabetic complications
* Strategies for reducing dietary intake of exogenous AGEs
* Risks associated with reducing dietary intake of exogenous AGEs
* Evidence-based recommendations for prevention and treatment of diabetic complications

# Report location:

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