INFLUENCE OF EPIGALLOCATECHIN-3-GALLATE ON INSULIN FIBRIL FORMATION

Sakalauskas Andrius[1], Smirnovas Vytautas[1]

[1]Institute of Biotechnology, Life Sciences Center, Vilnius University, Vilnius, Lithuania
sakalauskas.and@gmail.com

Amyloid protein aggregation into fibrils has been known to cause neurodegenerative diseases, such as Alzheimer‘s, Parkinson‘s and prion diseases [1]. For a long time there has been a search to find compounds that could inhibit such aggregation into amyloid fibrils and prevent the onset or progression of these diseases. A polyphenol from green tea-known as epigallocatechin-3-gallate (EGCG) was shown on multiple occasions to inhibit fibril formation [2]. A model protein used to study amyloid aggregation is insulin and it has been documented that EGCG is capable of inhibiting it‘s fibrilization [3].

In this work we examine the inhibition effect of insulin aggregation dependence on the oxidation time of EGCG. We found that the EGCG autooxidation process time is one of the key parameters to increase the inhibition of insulin aggregation. The inhibition mainly affect the nucleation stage, suggesting that the inhibition pathway is towards biding the native state of insulin and/or primary nucleation complexes.

[1] Choi M.L., Gandhi S. 2018. Crucial role of protein oligomerization in the pathogenesis of Alzheimer ’ s and Parkinson ’ s diseases, 285: 3631–3644.
[2] Doig A.J., Derreumaux P. 2015. ScienceDirect Inhibition of protein aggregation and amyloid formation by small molecules, Current Opinion in Structural Biology 30: 50–56.
[3] Wang S., Dong X., Sun Y. 2012. Effect of - (-) epigallocatechin-3-gallate on human insulin fibrillation / aggregation kinetics, Biochemical Engineering Journal 63: 38–49.