

Glycation

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Glycation (sometimes called non-enzymatic glycosylation) is the result of typically covalent bonding of a protein or lipid molecule with a sugar molecule, such as fructose or glucose, without the controlling action of an enzyme. All blood sugars are reducing molecules. Glycation may occur either inside the body (endogenous glycation) or outside the body (exogenous glycation). Enzyme-controlled addition of sugars to protein or lipid molecules is termed glycosylation; glycation is a haphazard process that impairs the functioning of biomolecules, whereas glycosylation occurs at defined sites on the target molecule and is required in order for the molecule to function. Much of the early laboratory research work on fructose glycations used inaccurate assay techniques that led to drastic underestimation of the importance of fructose in glycation.^[1]

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Exogenous

Exogenous, meaning *outside the body*, may also be referred to as *dietary* or *pre-formed*. Exogenous glycations and Advanced Glycation Endproducts (AGEs) are formed when sugars are cooked with proteins or fats. Temperatures over 120°C (~248°F) greatly accelerate the reactions, but lower temperatures with longer cooking times also promote their formation.^[*citation needed*]

These compounds are absorbed by the body during digestion with about 30% efficiency. Browning reactions (usually Maillard type reactions) are evidence of pre-formed glycations. Indeed, sugar is often added to products such as french fries and baked goods to enhance browning. Glycation may also contribute to the formation of acrylamide,^[2] a potential carcinogen, during cooking. Until recently, it was thought that exogenous glycations and AGEs were negligible contributors to inflammation and disease states, but recent work has shown that they are important.^[3] Although most of the research on this topic has been done with reference to diabetes, these results are likely to be important for all people, as exogenous AGEs are implicated in the initiation of retinal dysfunction, cardiovascular diseases, type II diabetes, and many other age-related chronic diseases.

Food manufacturers have added AGEs to foods, especially in the last 50 years, as flavor enhancers and colorants to improve appearance.^[4] Foods with significant browning, caramelization, or directly added preformed AGEs can be exceptionally high in these proinflammatory and disease-initiating compounds. A very partial listing of foods with very high exogenous AGEs includes donuts, barbecued meats, cake, and dark colored soda pop.^[5]

Endogenous

Endogenous glycations occur mainly in the bloodstream to a small proportion of the absorbed simple sugars: glucose, fructose, and galactose. It appears that fructose and galactose have approximately ten times the

glycation activity of glucose, the primary body fuel.^[6] Glycation is the first step in the evolution of these molecules through a complex series of very slow reactions in the body known as Amadori reactions, Schiff base reactions, and Maillard reactions; which lead to advanced glycation endproducts (AGEs). Some AGEs are benign, but others are more reactive than the sugars they are derived from, and are implicated in many age-related chronic diseases such as cardiovascular diseases (the endothelium, fibrinogen, and collagen are damaged), Alzheimer's disease (amyloid proteins are side-products of the reactions progressing to AGEs),^{[7][8]} cancer (acrylamide and other side-products are released), peripheral neuropathy (the myelin is attacked), and other sensory losses such as deafness (due to demyelination). This range of diseases is the result of the very basic level at which glycations interfere with molecular and cellular functioning throughout the body and the release of highly oxidizing side-products such as hydrogen peroxide.

Red blood cells have a consistent lifespan of 120 days and are easily accessible for measurement of recent increased presence of glycating product. This fact is used in monitoring blood sugar control in diabetes by monitoring the glycated hemoglobin level, also known as HbA1c. As a consequence, long-lived cells (such as nerves and different types of brain cell), long-lasting proteins (such as crystallins of the lens and cornea), and DNA may accumulate substantial damage over time. Cells such as the retina cells in the eyes, and beta cells (insulin-producing) in the pancreas are also at high risk of damage^[citation needed]. Damage by glycation results in stiffening of the collagen in the blood vessel walls, leading to high blood pressure, especially in diabetes.^[9] Glycations also cause weakening of the collagen in the blood vessel walls^[citation needed], which may lead to micro- or macro-aneurisms; this may cause strokes if in the brain.

See also

- Advanced glycation end-product
- Alagebrium
- Fructose
- Galactose
- Glucose
- Glycosylation
- List of aging processes

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