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The Chemistry of Oxidative Stress and Glycoxidation As Risk Factors for Developing Degenerative Disease

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Abstract

Oxidative stress conceived as an abnormal production of highly reactive oxygen species has been strongly implicated in degenerative and chronic diseases such as diabetes, cancer, coronary artery disease, vitiligo, arteriosclerosis, lupus, rheumatoid arthritis, and neurodegenerative pathologies. Additionally, glycoxidation is intimately associated with diabetes, coronary heart disease, retinopathy, Parkinson, Lewy body disease and aging. These oxidative processes are strongly interconnected and related to common alterations and therefore it is primordial to understand their chemistry and what is the impact of the oxidative stress and glycoxidation on the modulation and homeostasis of the body, with the aim of establishing strategies that could lead to the prevention or control of chronic and degenerative disease.

Keywords: Reactive oxygen species (ROS); Glycosylation; Diabetes; Amadori adducts; Advanced glycosylation end products; Autoimmune; Degenerative disease

Introduction

Oxidative stress has been defined as an imbalance between oxidants and antioxidants in favor of the oxidants, potentially leading to damage [1] The reactive species known as reactive oxygen species (ROS), are the superoxide anion ($O_2^{\cdot-}$), the hydroxyl radical (•OH), hydrogen peroxide (H_2O_2), hydroperoxyl radical (HOO•) and singlet oxygen (1O2) (Figure 1).

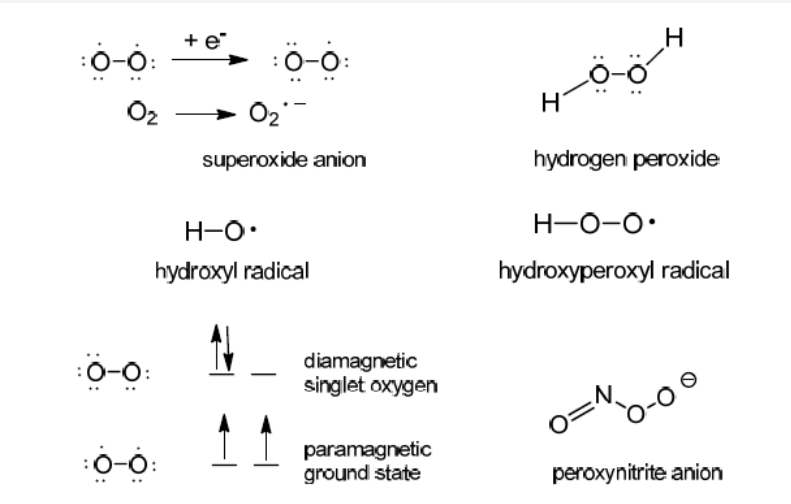

Figure 1: last 10 years' Brent crude oil price.

On the other hand, reactive nitrogen species (RNS) also participate in the form of nitric oxide (NO•) and peroxynitrite anion ($ONOO^-$) [2]. ROS are mainly expressed in mitochondria, cell membrane and nucleus and combines extracellular components such as superoxide dismutase and glutathione, with abnormal redox processes. Different studies pointed out that the increase of ROS is associated with anomalous production of hydrogen peroxide (H_2O_2) and peroxynitrite ($ONOO^-$) in the mitochondria [3,4]. The generation of superoxide anion ($O_2^{\cdot-}$) as a central player is attributed to the combination of electrons coming from the NOX2/

p22phox complex and the subunits p47, p67, and p40 with free oxygen. This highly reactive specie is quickly transformed to hydrogen peroxide (H_2O_2) by the action of superoxide dismutase or to hydroxyl radical in combination with Fe^{2+}. The superoxide anion can also react with nitric oxide (NO) to form the potent oxidant peroxynitrite ($ONOO^-$) associated with oxidative damage of biomolecules [5] (Figure 2).

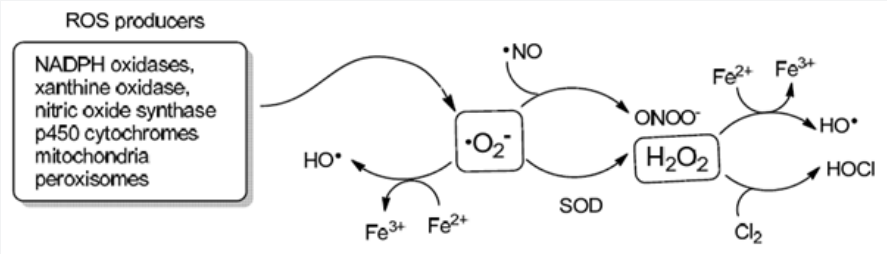

Figure 2: Simplified scheme representing the main ROS producers.

On the other hand, the degradation of oxidative species is mediated by reduced glutathione (2GSH) which in turn is trans formed to the oxidized glutathione (GSSG) and reactivated to the reduced form by the system NADPH/H+ → NAP^+.

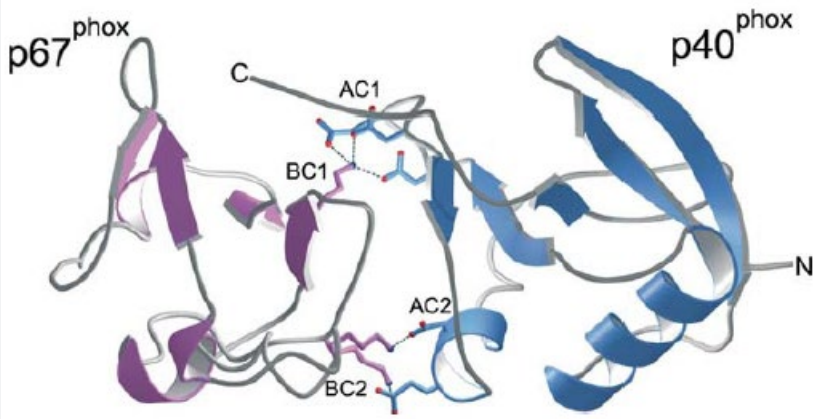

Figure 3: Enzyme NADPH oxidase (PDB: 1OEY).

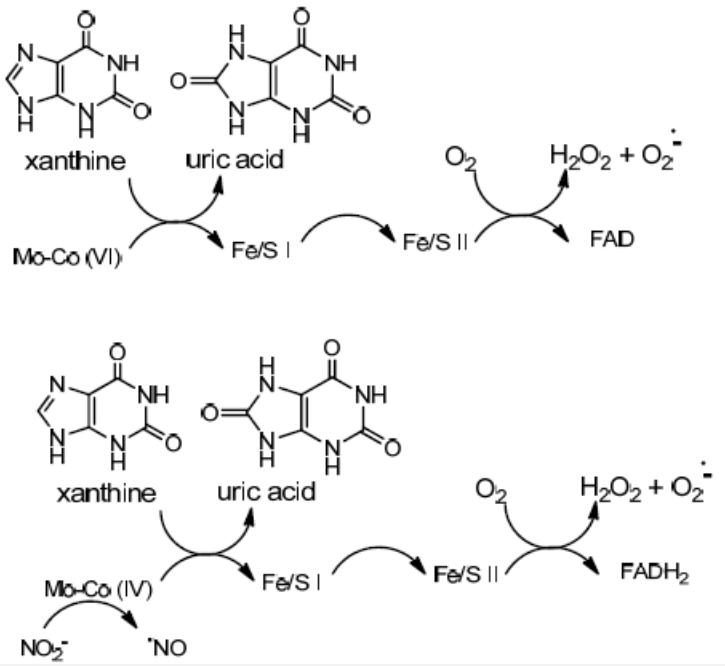

Figure 4: Proposed pathways for producing superoxide anion and hydrogen peroxide involving xanthine oxidase.

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Enzymes involved in the production of ROS Some of the enzymes identified as ROS producers are NADPH oxidases, xanthine oxidase, nitric oxide synthetase, p450 cytochromes and organelles (mitochondria, peroxisomes). A well identified source of ROS in humans is the NADPH oxidase consisting in a multi subunit enzyme complex of flavocytochrome b558 formed by two membrane subunits $p22^{phox}$ and $gpp1^{phox}$, activated by cytosolic $p40^{phox}$, $p47^{phox}$, $p67^{phox}$ and the GTPase Rac [6]. The normal production of ROS is necessary for autoimmune response, cellular signaling, and the regulation of gene expression, however the overproduction give place to serious alterations including cancer and autoimmune diseases [7]. The maximal activation of NADPH oxidase requires a complex formation between p40phox and p67phox subunits via association of their PB1 domains [8] (Figure 3).

Xanthine oxidase is a molybdenum hydroxylase family of proteins having one FAD site and two Fe/S clusters which catalyses the hydroxylation of hypoxanthine to xanthine as well as xanthine to uric acid [9]. During inflammatory conditions xanthine dehydrogenase is converted to xanthine oxidase, giving as result a NAD+ decrease while oxygen affinity increase, with the resulting generation of superoxide anion and hydrogen peroxide. The proposed mechanism for the generation of oxidative species such as superoxide anion ($O_2^{\cdot-}$) and hydrogen peroxide (H_2O_2) involves xanthine oxidation to uric acid where electrons are transferred to FAD resulting in O_2 reduction to H_2O_2 and ($O_2^{\cdot-}$).

Alternatively, superoxide anion and hydrogen peroxide can be generated by NO_2^- reduction to $^{\cdot}NO$ at the Mo cofactor with electrons donated by xanthine [10] (Figure 4).

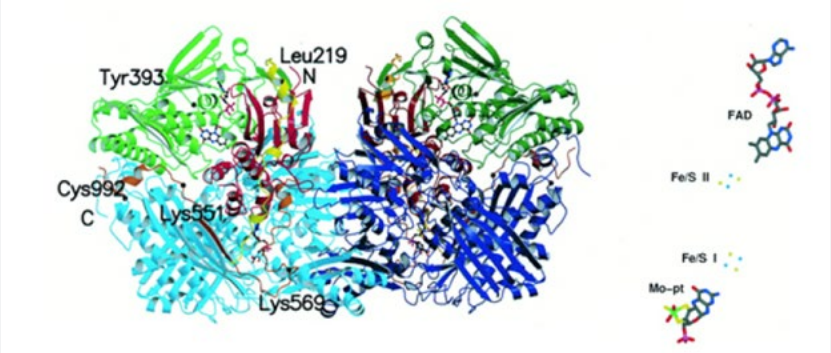

Figure 5: Bovine xanthine oxidase structure (PDB: 1FIQ).

The crystal structure of bovine xanthine oxidase shows a molybdenum-containing enzyme conformed by two monomers with domains iron/sulphur-center domains (residues 3-165), FAD domain (residues 226-531), and Mo-pt domain (residues 590-1,331) [11]. This butterfly shaped dimer catalyses the hydroxylation of a wide variety of aromatic heterocycles, including the physiological purine substrates, hypoxanthine and xanthine, and aldehydes [12] (Figure 5).

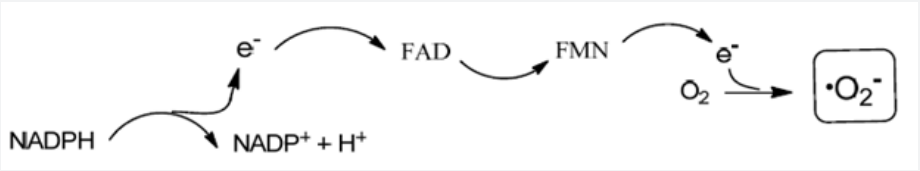

Figure 6: Simplified pathway to produce superoxide involving nitric oxide synthase.

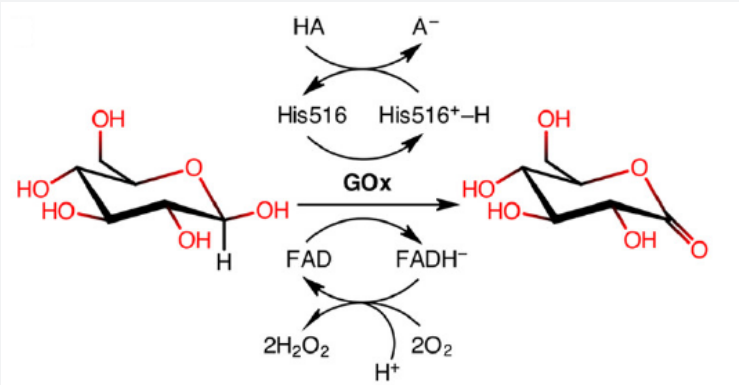

Figure 7: Conversion of β-D-glucose to D-gluconolactone and hydrogen peroxide, catalyzed by glucose oxidase.

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Nitric oxide synthase (NOS) isoforms uses L-arginine as substrate and in combination with O_2 and NADPH produce superoxide anion ($O_2^{\cdot-}$). The whole process involves an electron transfer from reduced nicotinamide-adenine-dinucleotide phosphate (NADPH), to flavin-adenine-dinucleotide (FAD) and flavin-mononucleotide (FMN), which ultimately will reduce molecular oxygen to superoxide ($O_2^{\cdot-}$), although with limited capacity [13] (Figure 6).

Glucose oxidase is a homodimer consisting in two identical subunits and two noncovalently bound flavin adenine dinucleotides (FAD), and its main function is to produce H_2O_2 as antibacterial and anti-fungal repulsive agent [14]. Glucose oxidase is responsible of the conversion of β-D-glucose to D-gluconolactone and hydrogen peroxide. A detailed reductive half-reaction shows the oxidation reaction of β-D-glucose involving a hydride migration of C1 to His516 and FAD (Figure 7).

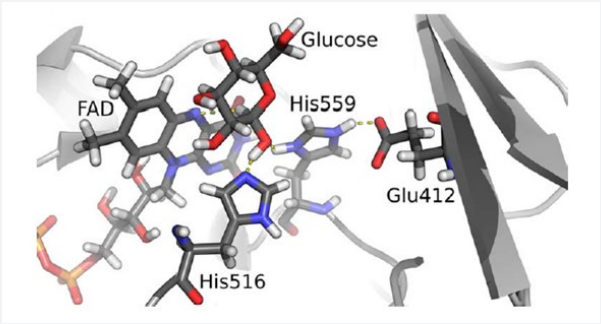

Figure 8: Glucose oxidase with FAD inside the catalytic site.

The catalytic site involving FAD, His516, His559, Glu412 and glucose as substrate, indicates that the protonation of His516 and hydride migration to the N5 of the flavine adenine dinucleotide result critical in the transition state [15] (Figure 8).

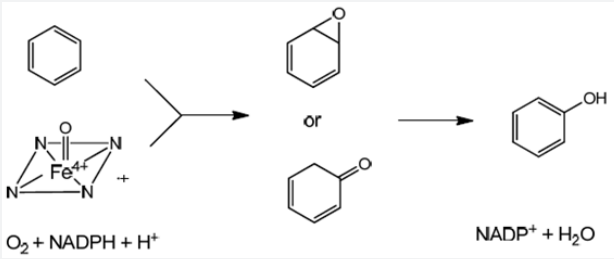

Figure 9: Oxidation of benzene by cytochrome p450.

Cytochrome p450 is an enzyme complex that plays a central role in detoxification processes mainly of drugs during the phase I and carries out several oxidative transformations on alkenes, heteroatoms, carbonyl groups, aromatic and other alpha activated positions. The oxidation products include mainly hydroxylation, however epoxidation, N-oxides, S-oxides, oximes, O- and N-dealkylations are usually observed. The p450 enzyme family utilize in the catalytic site the heme group, the iron atom, and the NADPH as a redox system as it can be visualized in the hydroxylation reaction of benzene (Figure 9).

The catalytic cycle of cytochrome p450 postulates different intermediates some of them isolated and studied extensively by different spectroscopies [16,17] (Figure 10).

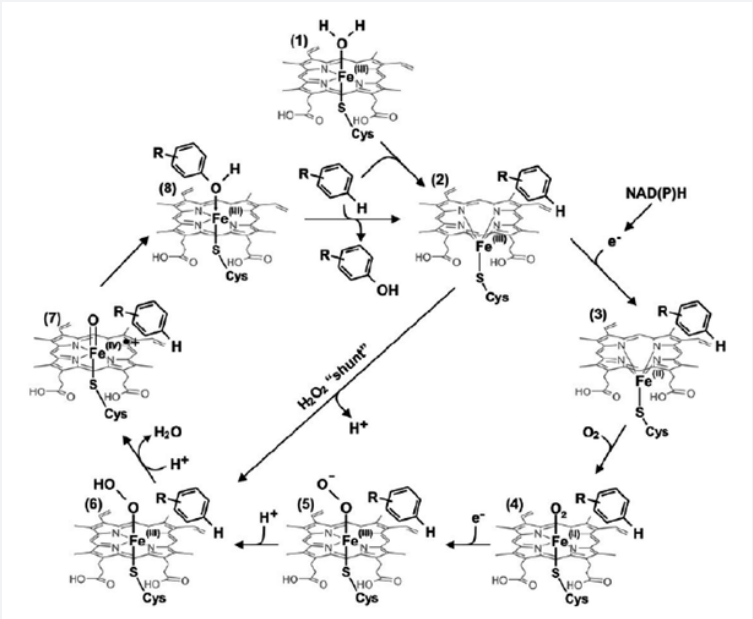

Figure 10: Intermediates identified during the oxidative transformation of benzene by cytochrome p450.

The vast majority of P450s requires NAD(P)H as a redox partner *in vivo*, although an alternative route involves hydrogen peroxide (H_2O_2) to generate iron-oxo species [18]. The crystal structure of P450 3A4 (CYP3A4) which metabolize more drugs than any other isoform was determined, showing the heme grupo as a ball-and-stick model in the center of the molecule, flanked by helix [19] (Figure 11).

Mitochondria organelle is another important source for the generation of reactive oxidative species (ROS) besides his primor-

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dial activity regarding the ATP production within the cell. The increase in the production of ROS superoxide anion ($O_2^{\cdot-}$) is observed under two circumstances, a diminished ATP levels and when there is a high NADH/NAD+ ratio in the matrix [20]. The generation of superoxide anion also depends on the proton motive force Dp determined by the redox systems NADH/NAD+, CoQH2/CoQ ratios and the O_2 concentrations. However, according to literature small molecule electron carriers such as NADH, NADPH, $CoQH_2$ (reduced coenzyme Q) and glutathione (GSH) do not react with O_2 to generate $O_2^{\cdot-}$, so this reduced form is mainly generated at low ATP production levels and high NADH/NAD^+, or $CoQH_2$/CoQ ratios during the reverse electron transport (RET) consisting in the transfer of two electrons to the CoQ pool where in the presences of Dp force the electrons moves back from $CoQH_2$ into the complex I of the respiratory chain and reduce NAD^+ to NADH at the FMN site [21-24] (Figure 12).

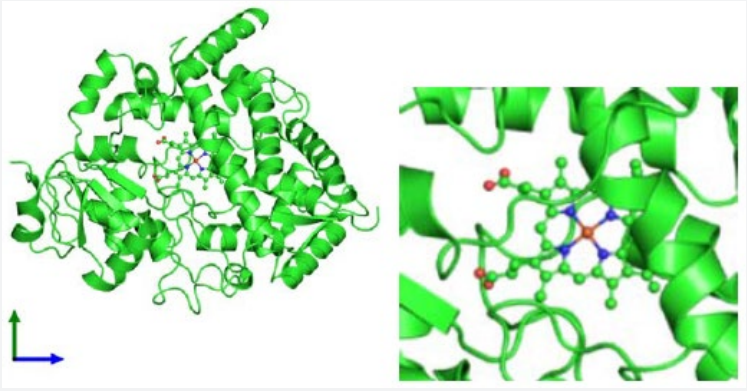

Figure 11: Ball and stick model of cytochrome p450 (PDB: 1W0E)

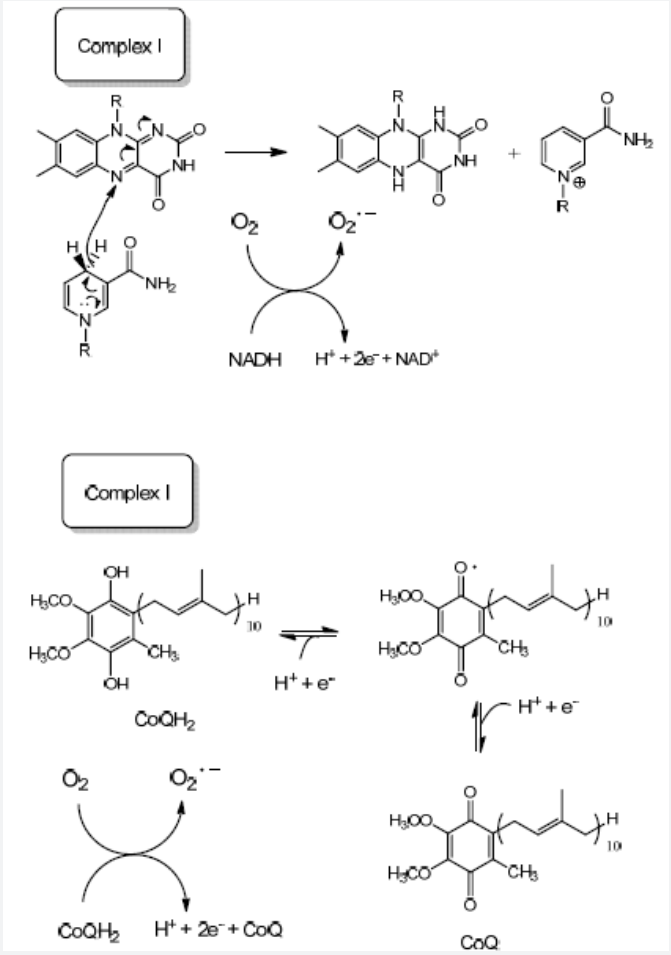

Figure 12: Formation of superoxide anion in the mitochondria involving and NADH $CoQH_2$ cofactors.

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Peroxisomes are membrane-bound organelles involved primarily in the lipid metabolism and not less important in the productions of reactive oxygen species (ROS). With the discovery of an alternative respiratory pathway in peroxisomes, which is characterized by the generation of electrons from different metabolites, which convert the O_2 into H_2O_2 and finally to H_2O, several studies have been directed with the aim of establishing a potential connexion between the oxidative processes with human malignancies. It is well known that the main metabolic processes associated with the H_2O_2 production in peroxisomes are the β-oxidation of fatty acids. The enzymes presents in peroxisomes that generate ROS are: Acyl-CoA oxidase (Palmitoyl-CoA oxidase, Pristanoyl-CoA oxidase, Trihydroxycoprostanoyl-CoA oxidase), Urate oxidase, Xanthine oxidase, D-amino acid oxidase, pipecolic acid oxidase, D-aspartate oxidase, sarosine oxidase, L-alpha hydroxyl acid oxidase, poly amine oxidase, nitric oxide synthase and plant sulphite oxidase [25].

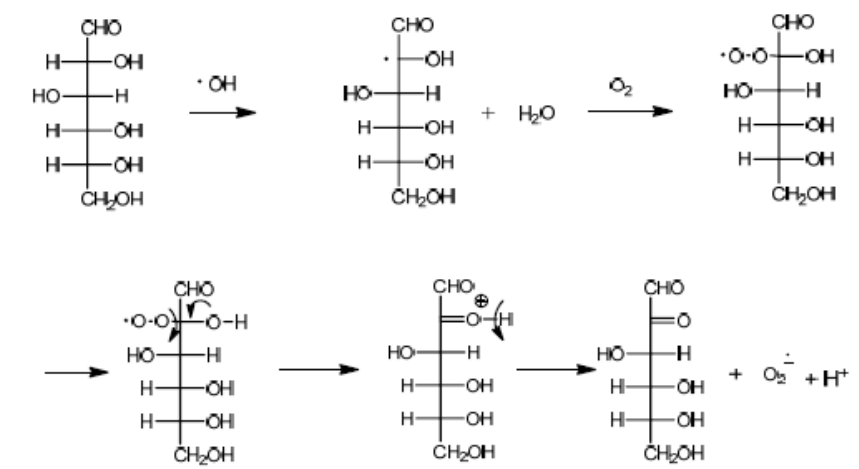

Figure 13: Formation of α-keto aldoses by hydroxyl radicals.

Glycoxidation

Carbohydrates are biological units with unquestionable importance for the well-functioning of the cell, however the intake of high amounts of some sugars specially sucrose, fructose and starch which ultimately will deliver glucose, had modified dramatically the conception about the good reputation of the carbohydrates, and how to deal with this primordial substance considering that glucose is the first alternative as a fuel by the cell. Numerous studies have been undertaken to understand the implications of the carbohydrates in alterations or dysfunctions associated with the oxidative stress and glycoxidation which has been identified with accuracy through intensive analysis. Monosaccharaides such as glucose, fructose, and disaccharides (sucrose and maltose) may react under non enzymatic and enzymatic conditions with ROS. Example of the former case is the reaction of monosaccharaides and disaccharides with hydroxyl radicals ($HO^{\bullet}$) to produce initially a secondary hydroxyl alcohol radical which is further oxidized with O_2 to hydroxyalkylperoxy radical. Final decomposition will furnish the corresponding ketone and superoxide anion $O_2^{\cdot-}$ [26] (Figure 13).

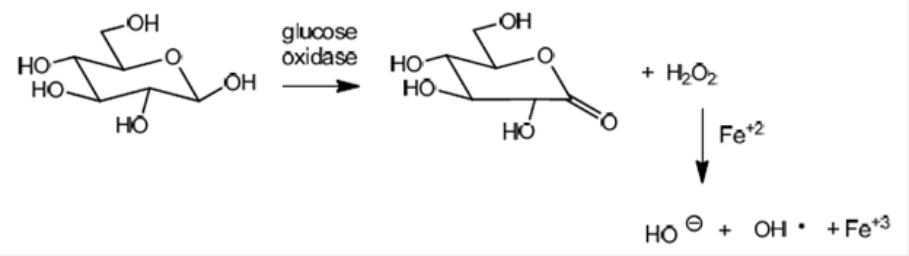

Figure 14: The Fenton reaction.

On the other hand, the well-known Fenton reaction is an example of enzymatic reaction, consisting in the oxidation mediated by glucose oxidase of the anomeric hydroxyl group of β-D-glucose to form D-gluconolactone and hydrogen peroxide which in the presence of Fe^{+2} decompose to hydroxyl anion and hydroxyl radical [27] (Figure 14).

Another important transformation involving saccharides is the Amadori reaction for glucose (Figure 15) or Heyns for fructose [28] (Figure 16) also known in food chemistry as Millard reaction when the amines belong to amino acid residues present in proteins. This process has been largely considered a non-enzymatic reaction although recent evidence suggests the participation of

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enzymes especially for the attachment of some amino acids [29]. This important transformation with high implications in food chemistry as well as in degenerative processes mainly diabetes mellitus and autoimmune disease is associated with the formation of α-ceto aldehydes considered oxidation products implicated in the formation of advanced glycation end products (AGES). Such Amadori adducts have been identified in hemoglobin through the attachment with lysine residues [30].

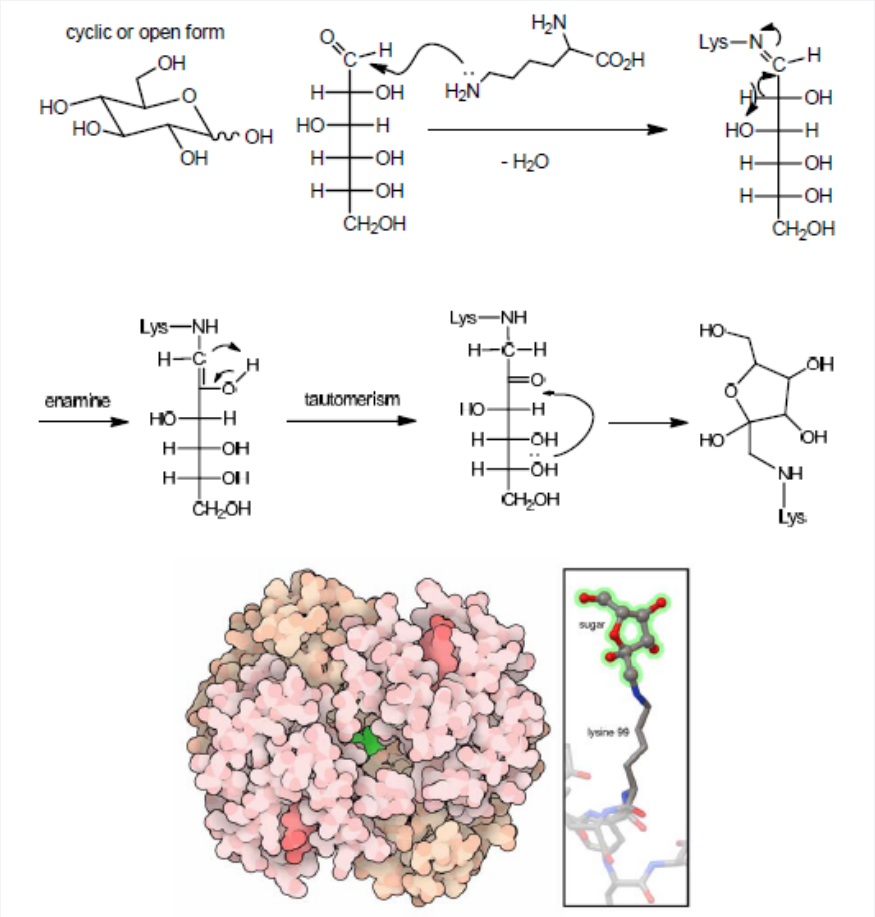

Figure 15: Amadori reaction between glucose with L-Lysine, and haemoglobin attached to Amadori adducts (PDB: 3B75).

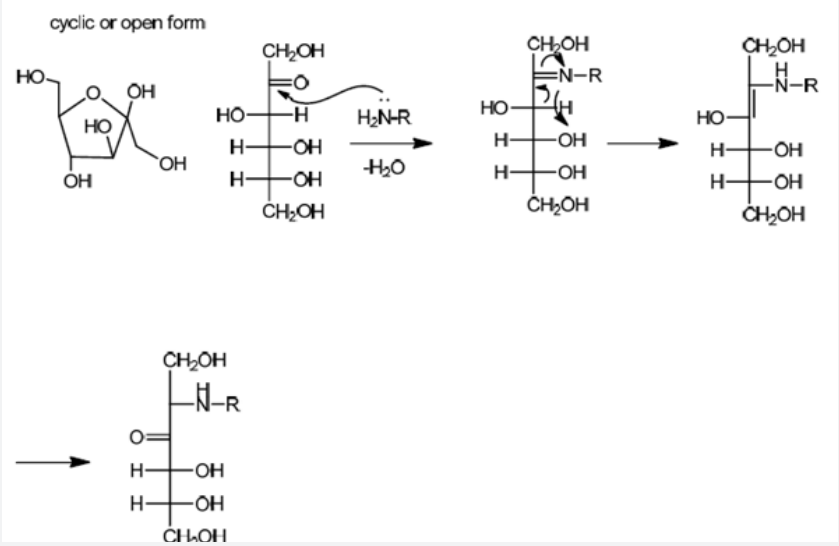

Figure 16: The Heyns reaction.

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Glucuronic acid is another monosaccharide with importance in phase II of metabolism implicated in the detoxification of drugs that has been described to generate Amadori adducts under non-enzymatic conditions, and it has been conjugated to lysine containing pentapeptides under mild conditions [31] (Figure 17).

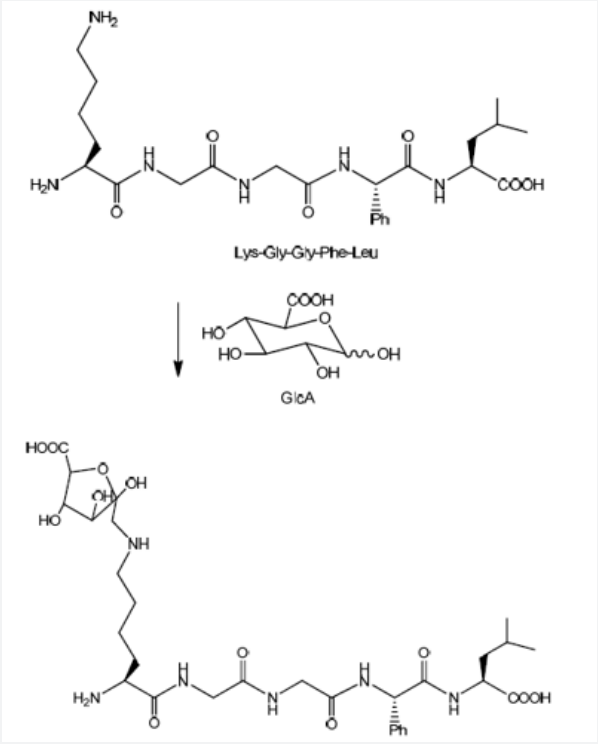

Figure 17: Amadori glucuronide-lysine peptides.

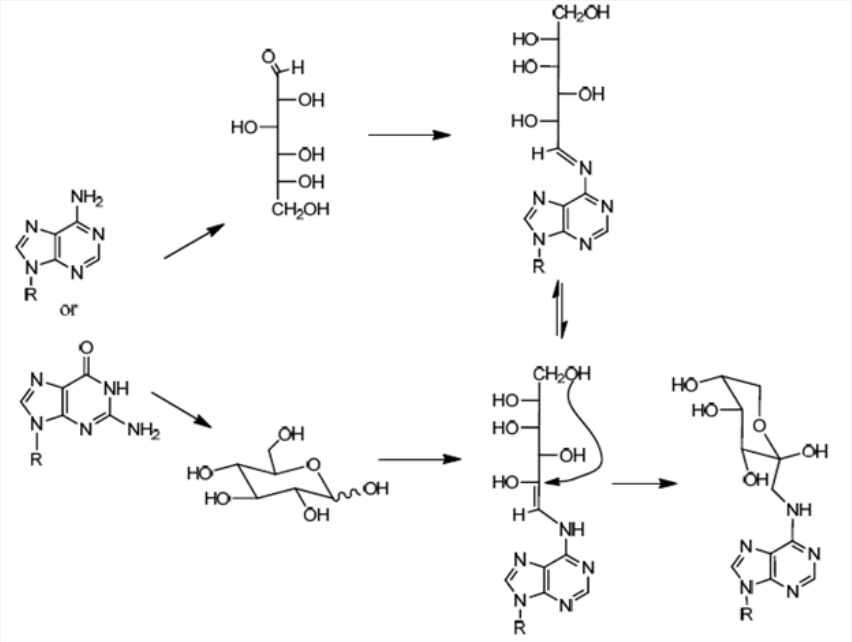

Figure 18: Amadori-purine adducts.

As predicted, amino purines also react with glucose either in the open or cyclic forms leading through the imine of Schiff product which establish an equilibrium with the enaminol product, and then addition to the enamine to form the cyclic Amadori-purine, adducts [31-33] (Figure 18).

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Also, purines can be modified by oxygen radicals that react with polyunsaturated fatty acid residues in phospholipids producing a cocktail of products being Malondialdehyde (MDA) a major product of lipid peroxidation [34] (Figure 19).

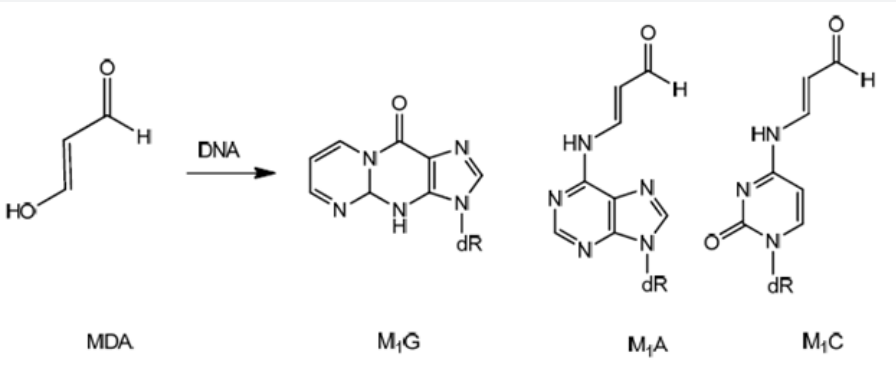

Figure 19: Adducts of MDA with deoxyguanosine, deoxyadenosine, and deoxycytidine.

High glucose concentration brings as a pathologic consequence the abnormal attachment with proteins to generate Amadori adducts, advanced glycosylation end products (AGES) and reactive carbonyl species such as glyoxal (GO) and methyl glyoxal (MGO) 3-deoxyglucosone (3-DG). The reactive carbonyl species may react with amino acid lysine and arginine to produce imidazole derivatives and other AGES adducts [35] (Figure 20).

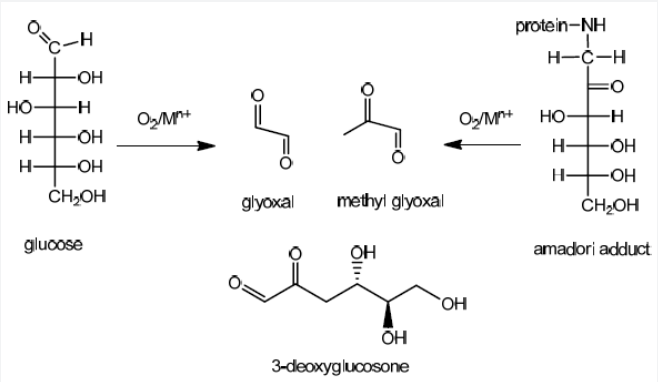

Figure 20: General scheme for the formation of reactive carbonyl species glyoxal (GO), methyl glyoxal (MGO) and 3-deoxyglucosone (3-DG).

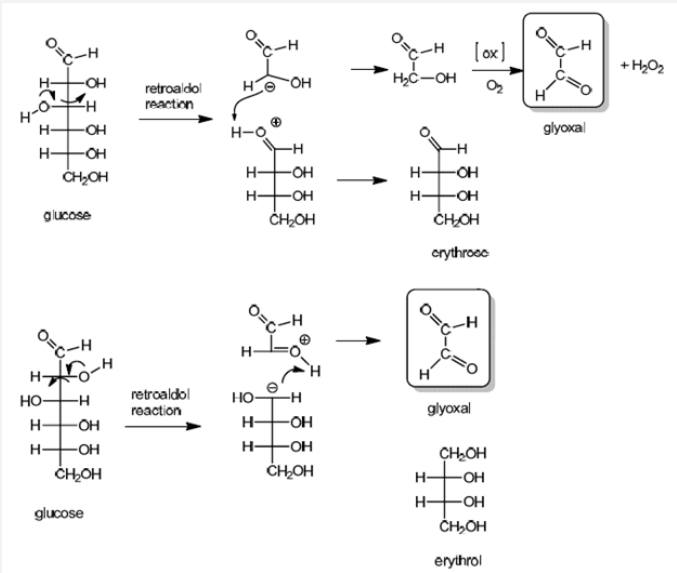

Figure 21: Retroaldol reaction for the formation of glyoxal and methyl glyoxal.

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Non enzymatic glyoxal (GO) and methyl glyoxal (MGO) formation from glucose is a process with high implications since these α-oxoaldehydes are responsible of most of the advanced glycosylation product (AGES) especially after attachment with amino acids arginine and lysine. A proposed mechanism for converting glucose into glyoxal considers a retroaldol reaction followed by autooxidation, or direct glyoxal formation and erythrol [36,37] (Figure 21).

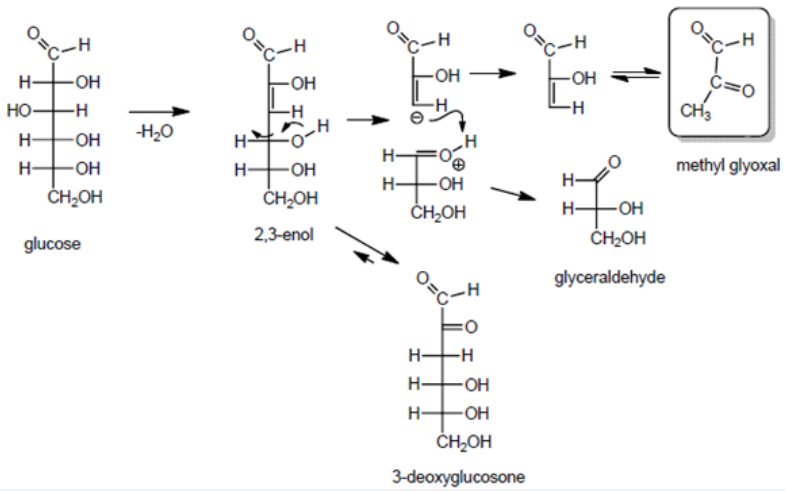

Figure 22: Alternative pathway for the formation of glyoxal and methyl glyoxal via 2,3, -enol intermediate.

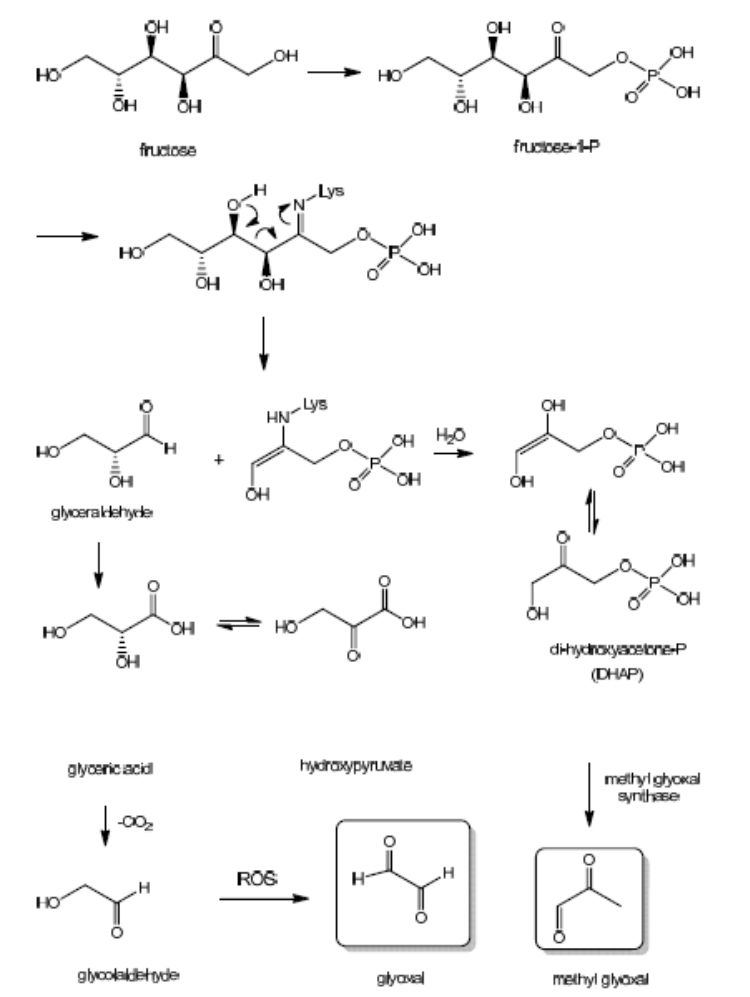

Figure 23: Formation of reactive α-keto aldehydes from fructose.

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Alternatively, methyl glyoxal can be generated from glucose through the formation of 2,3-enol, which is converted by conjugated retroaldol reaction to methyl glyoxal or by tautomerism to 3-deoxyglucosone (Figure 22).

Fructose is undisputedly very important monosaccharide for his extended use as sweetener present in soft drinks and corn syrup, not to mention his presence in sucrose disaccharide. Fructose metabolism differs from glucose for his non dependence from insulin, and instead is phosphorylated to fructose-1-phosphate by fructokinase and ATP [38,39] (Figure 23).

On the other hand, glucose and fructose are interconnected trough the polyol pathway which has been highly associated with the generation of reactive oxygen species leading to tissue damage and apoptosis. An increase flux of glucose through this pathway is related with diabetes mellitus complications due the increase of NADH/NAD+ ratio and decrease of cytosolic NADPH which is required for regenerating glutathione (GSH) and important oxygen radical scavenger [40] (Figure 24).

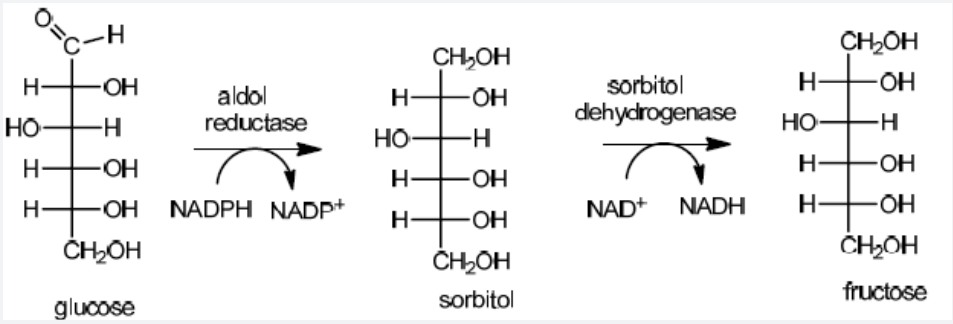

Figure 24: The polyol pathway.

Advanced glycation end products (AGES)

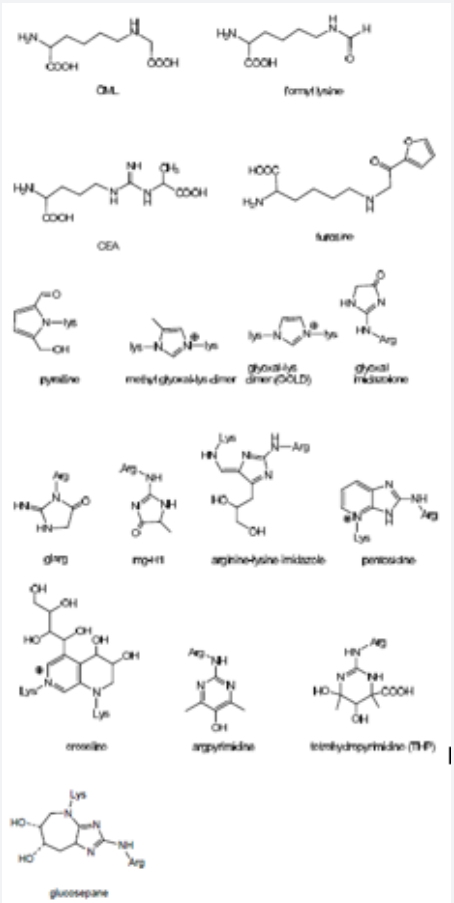

Figure 25: Structure of advanced glycation end products (AGES).

This term is referred to an array of compounds resulting from the condensation of amino acids mainly lysine and arginine with α-dicarbonyl products (glyoxal, methyl glyoxal), and carbohydrates. The conjugates that have been identified by high resolution mass spectrometry and rasonance spectroscopy includes open structures such as Nε- (carboxymethyl)lysine (CML), formyl lysine, Nδ-(carboxyethyl)arginine (CEA), or 5 and 6 membered heterocyclic rings including pyrrole, imidazole, imidazolone, imidazo [4,5- b] pyridine, and tetrahydropyrimidin [41] (Figure 25).

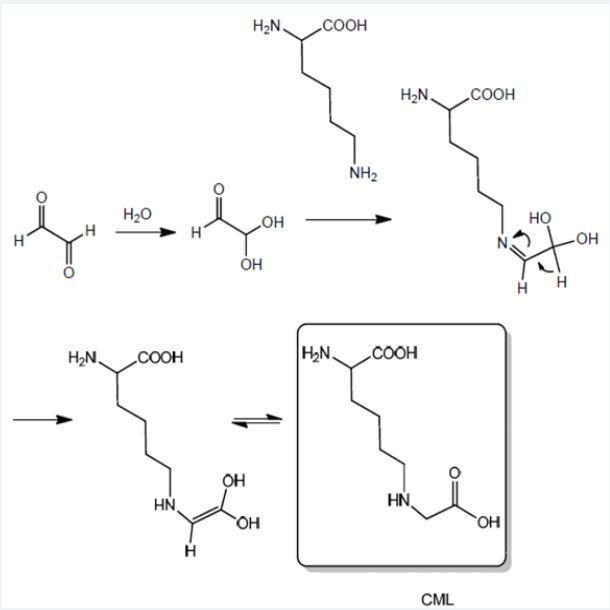

Figure 26: Formation of Nε-(carboxymethyl)lysine (CML).

Nε-(carboxymethyl) lysine (CML) is a commo advanced glycation product found in foods described for over 30 years. It can be

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prepared from casein and glucose incubated at 95 °C for 8h [42]. According to the literature CML can be prepared *in vivo* from the Schiff base, the Amadori product, and from glyoxal with lysine [43] (Figure 26).

Pentosidine is a fluorescent imidazo [4,5-b] pyridine AGE adduct, [44] associated with diabetic complications, aging, kidney damage, and can be prepared from fructose, ascorbate, glucose, and ribose with lysine and arginine residues [45]. The structural assignment has been described by using mono and heteronuclear two dimensional 1H and 13CNMR spectroscopy [46] (Figure 27).

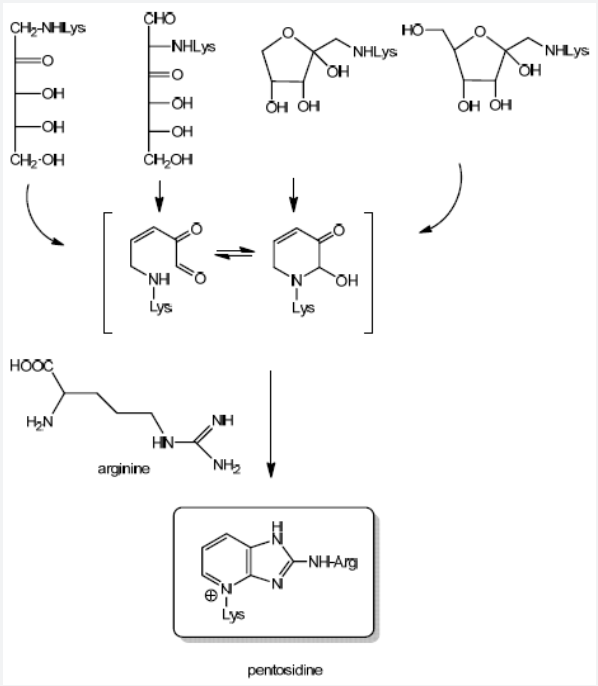

Figure 27: Formation of Pentosidine.

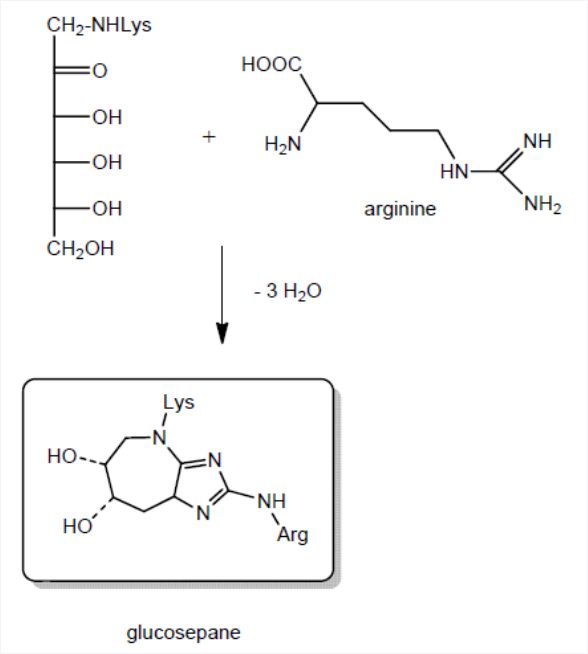

Figure 28: Formation of glucosepane.

Glucosepane is an advanced gycation product less investigated with implications in retinopathy, neuropathy and nephropathy which is *in vivo* prepared by conjugation of arginine with Amadori adduct [46,47] (Figure 28).

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Furosine is a ε-N-(furoylmethyl)-L-lysine formed by hydrolysis of the Amadori adducts fructosyl-lysisne, lactulosyl-lysisne and maltulosyl-lysisne under acid conditions, and it is produced by lysine conjugation with glucose, lactose and maltose [48] (Figure 29).

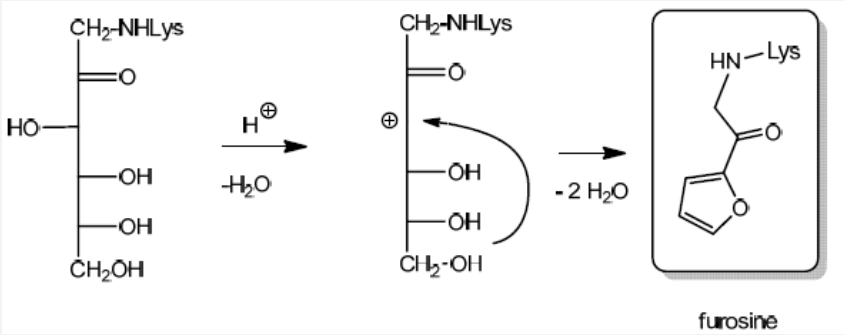

Figure 29: Formation of furosine.

Conclusion

The oxidative stress is design by the living systems for the regulation of important processes such as proliferation-differentiation, apoptosis, tissue homeostasis and self defense against pathogens, however when there is an imbalance between ROS production an anti-oxidative mechanism, a oxidative stress scenario occurs with severe consequences in the wellness of the living system deriving in pathologies that compromise the survival. Likewise glycoxidation is a process conceived to provide essential small molecules such as pyruvate from monosaccharide's mainly glucose and fructose which are the main source of energy by the cell, however under hyperglycaemia the oxidative processes stimulate the production of highly reactive molecules such as glyoxal and methyl glyoxal which combined with proteins produce advanced glycation end-products responsible for expression of numerous degenerative disease.

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