Glycosylation is enzymatic modification of proteins or lipids with saccharides. Glycation is a non-enzymatic chemical reaction that results in the addition of sugar. Both sugar modification processes are related to aging and diseases. This talk will give examples and discuss each modification and the relationship to aging.

Glycation can form advanced glycation end products (AGEs) and contributes to aging-related diseases. Insulin resistance can result in an excess of aldehydes and the aldehydes such as glyoxal can non-enzymatically react with free amino acid to form AGEs (Vasdev et al. 2007). The changes can result in endothelial dysfunction and inflammation which are characteristics of hypertension and atherosclerosis. Attenuating insulin resistance and lowering AGEs together with a healthy lifestyle and diet might be promising therapies for these diseases.

There are two types of glycosylation: N-linked and O-linked. A large proportion of the animal proteins are glycosylated and the sugar modification can affect protein functions. The prion linked diseases are believed to be associated with the conversion of two forms of prion protein: α-helical cellular prion protein (PrP^C) and the β-sheet rich scrapie isoform (PrP^Sc). Prion protein is a glycoprotein with two N-linked glycosylation domains. One dimensional protein gel and immunoblots of PrP^C show three major bands: 35kDa, 32kDa and 28kDa, representing di-glycosylated, mono-glycosylated and un-glycosylated isoforms, respectively. The expression pattern changes of the three isoforms among different age groups of mice indicate that the prion protein glycosylation changes may contribute to the normal aging process (Goh et al. 2007). The interaction of PrP^C and lectin detected by enzyme-linked immunosorbent assay (ELISA) suggests that the lectin binding profile changes in aging.

References: