

Glycogenolysis

Glycogen to Glucose

Glider to Glue

Glycogenolysis is the conversion of glycogen into free glucose. When glucose is required in the body, whether it be to maintain adequate blood glucose levels or to fuel working muscles, it often comes from glycogen stores. A series of reactions are required to liberate glucose from glycogen molecules for use in glycolysis.

Glycogen Phosphorylase + Vitamin B6 Cofactor

Glider Phosphor-P-lace and Viking (B) Bee (6) Sax Crow-flagger

Glycogen phosphorylase is the first enzyme involved in glycogenolysis, and acts on the branches of glycogen. This enzyme adds a phosphate group to the glycogen branch resulting in the liberation of one glucose-1-phosphate. This enzyme requires vitamin B6 as a cofactor. This enzyme is only capable of releasing glucose from the ends of glycogen branches. Once glucose-1-phosphate is produced, phosphoglucomutase will transfer the phosphate group from the 1' position to the 6' position, setting it up to enter glycolysis.

Limit Dextrin

Limit-Desks

Once a branch of glycogen has been shortened enough so only two to four residues remain, the shortened branch is called a "limit dextrin". Glycogen phosphorylase is not capable of acting on glycogen residues in these short branches. Debranching enzyme is instead needed to break these bonds.

Debranching Enzyme

Enzyme Cutting-branches

The shortened four residue "limit dextrin" branches of glycogen are acted upon by debranching enzyme, a special enzyme with two different activities. The first debranching enzyme activity (α -1,4-D-glucanotransferase) moves three of the glucose subunits from the branch onto the main linkage chain of the glycogen complex. This leaves just one glucose subunit behind as a tiny 'branch', attached to the main linkage by a α -1,6 bond. The second debranching enzyme activity (α -1,6-glucosidase) then liberates this remaining residue as free glucose. Deficiency of debranching enzyme is inherited in an autosomal recessive manner known as glycogen storage disease type III (Cori disease, or limit dextrinosis). Limit dextrans accumulate in tissues, especially the liver and muscles, causing damage. A small amount of glycogen is broken down within lysosomes by the enzyme α -1,4-glucosidase (acid maltase); a deficiency of this enzyme causes glycogen storage disease type II (Pompe disease).