* Do not include theses or articles with unknown journal

## Proposed EC/sub‐subclass

B4GALNT1 is classified under the Enzyme Commission number EC 2.4.1.92, which places it in the subclass of glycosyltransferases that transfer hexosamine residues via a β‐linkage (kellokumpu2016glycosyltransferasecomplexesin pages 3-4, petit2021aphylogeneticview pages 10-11). This EC designation reflects its role in transferring an N-acetylgalactosamine moiety from UDP-GalNAc to specific glycolipid acceptors (taniguchi2014handbookofglycosyltransferases pages 183-187, taniguchi2014handbookofglycosyltransferases pages 45-49). The enzyme’s classification under EC 2.4.1.92 is based on rigorous biochemical characterization that demonstrates its specificity for glycolipid substrates rather than glycoproteins (taniguchi2014handbookofglycosyltransferases pages 183-187, taniguchi2014handbookofglycosyltransferases pages 187-190). EC 2.4.1.92 distinguishes B4GALNT1 from other glycosyltransferases that transfer different sugars or catalyze glycosidic bonds with alternate anomeric configurations (hennet2002thegalactosyltransferasefamily pages 12-13, li2018congenitaldisordersof pages 1-4). This value is supported by kinetic assays and substrate reactions that define the enzyme’s functional role in ganglioside biosynthesis (taniguchi2014handbookofglycosyltransferases pages 183-187, taniguchi2014handbookofglycosyltransferases pages 187-190).

## Accepted name

The recommended and accepted name for the enzyme is Beta-1,4 N-acetylgalactosaminyltransferase 1. This designation is accompanied by alternative names such as GM2 synthase, GM2/GD2 synthase, and GalNAc-T, reflecting its activity in ganglioside biosynthesis (taniguchi2014handbookofglycosyltransferases pages 183-187, taniguchi2014handbookofglycosyltransferases pages 187-190). Additional synonyms including (N-acetylneuraminyl)-galactosylglucosylceramide transferase emphasize its substrate context and catalytic function (taniguchi2014handbookofglycosyltransferases pages 45-49, unknownauthors2003molecularcloninggene pages 9-10). These designations are consistent across multiple publications and databases, with the accepted name being used in biochemical and genetic studies (hennet2002thegalactosyltransferasefamily pages 12-13, alecu2022functionalvalidationof pages 5-7).

## Phylogeny

B4GALNT1 is evolutionarily conserved across vertebrates, and orthologs have been identified in species such as human, mouse, and rat (taniguchi2014handbookofglycosyltransferases pages 183-187, alecu2022functionalvalidationof pages 5-7). Phylogenetic analyses indicate that this enzyme shares significant sequence conservation with other glycosyltransferases involved in the synthesis of gangliosides, underscoring its essential role in the formation of complex glycolipids in the nervous system (petit2021aphylogeneticview pages 6-7, petit2021aphylogeneticview pages 11-12). Within the animal kingdom, B4GALNT1 clusters in a distinct subgroup of glycosyltransferases that specialize in glycolipid modification, and its sequence conservation suggests a common ancestral gene from which other GT31 family members have diverged (petit2021aphylogeneticview pages 4-5, petit2021aphylogeneticview pages 11-12). In evolutionary context, the conservation of catalytic motifs such as the DXD motif and key amino acid residues in the catalytic domain reflects its preserved functional role across divergent metazoan lineages (breton1999structurefunctionstudiesof pages 8-8, breton1999structurefunctionstudiesof pages 5-7).

## Glycosyltransferase family

B4GALNT1 belongs to the CAZy glycosyltransferase family GT31, a group of enzymes that are involved predominantly in glycolipid and glycoprotein biosynthesis (petit2021aphylogeneticview pages 4-5, petit2021aphylogeneticview pages 11-12). Members of the GT31 family are characterized by conserved domain structures, including a catalytic domain that adopts a GT-A fold, and a specific set of conserved sequence motifs such as the DXD motif critical for metal ion coordination and donor substrate binding (breton2001structuralandfunctional pages 2-4, breton1999structurefunctionstudiesof pages 3-4). The enzyme is grouped with other β1,4-galactosyltransferases that share similar substrate specificities and domain organizations, distinguishing its activity from enzymes in other CAZy families (petit2021aphylogeneticview pages 15-16). This family assignment is supported by phylogenetic studies that consistently place B4GALNT1 in the context of ganglioside synthesizing enzymes (petit2021aphylogeneticview pages 11-12).

## Reaction Catalyzed

The enzyme catalyzes the transfer of N-acetylgalactosamine (GalNAc) from the nucleotide sugar donor UDP-GalNAc to specific glycolipid acceptors. In this reaction, B4GALNT1 converts ganglioside precursors such as GM3, GD3, GT3, and GA3 into the more complex species GM2, GD2, GT2, and GA2, respectively (taniguchi2014handbookofglycosyltransferases pages 183-187, taniguchi2014handbookofglycosyltransferases pages 45-49). The reaction proceeds via the formation of a β1,4-glycosidic linkage, and it is central to the biosynthetic pathways that generate the gangliosides enriched in neural tissues (cogez2023nglycanonthe pages 3-4, taniguchi2014handbookofglycosyltransferases pages 183-187). The enzyme exhibits strict donor specificity for UDP-GalNAc, and its acceptor specificity for glycolipids underlines its role in the ganglio-series biosynthetic cascade (unknownauthors2016investigatingthebiological pages 16-20, taniguchi2014handbookofglycosyltransferases pages 187-190).

## Cofactor Requirements

B4GALNT1 requires divalent metal ions as cofactors for its catalytic activity. In particular, its activity is dependent on the presence of manganese ions (Mn²⁺), which are essential for coordinating the UDP-GalNAc donor substrate via conserved amino acid residues in the catalytic domain (taniguchi2014handbookofglycosyltransferases pages 183-187, taniguchi2014handbookofglycosyltransferases pages 45-49). This requirement for Mn²⁺ is a common feature among glycosyltransferases, and it plays a critical role in stabilizing the transition state during the transfer reaction (breton1999structurefunctionstudiesof pages 3-4, kapitonov1999conserveddomainsof pages 1-2). The ionic cofactor binds in conjunction with the conserved DXD motif, thus facilitating the proper orientation of the donor substrate for efficient catalysis (breton2001structuralandfunctional pages 2-4, taniguchi2014handbookofglycosyltransferases pages 183-187).

## Substrate Specificity

The substrate specificity of B4GALNT1 is marked by its exclusive action on glycolipid substrates, particularly those that are precursors to complex gangliosides. The enzyme recognizes and acts on gangliosides such as GM3, GD3, GT3, and GA3, incorporating an N-acetylgalactosamine residue to generate GM2, GD2, GT2, and GA2, respectively (taniguchi2014handbookofglycosyltransferases pages 183-187, taniguchi2014handbookofglycosyltransferases pages 187-190). B4GALNT1 does not exhibit activity toward glycoprotein substrates, and its substrate specificity is determined by the configuration of the acceptor glycolipid, which includes a terminal sialylated galactose residue (taniguchi2014handbookofglycosyltransferases pages 183-187, taniguchi2014handbookofglycosyltransferases pages 45-49). Kinetic studies indicate that the enzyme demonstrates distinct affinity parameters for different glycolipid acceptors, with reported differences in Km and Vmax values when using substrates such as GM3 versus lactosylceramide (taniguchi2014handbookofglycosyltransferases pages 183-187). The strict specificity for glycolipid substrates plays a crucial role in directing the biosynthetic flow toward the formation of gangliosides that are pivotal in neural function (taniguchi2014handbookofglycosyltransferases pages 183-187, taniguchi2014handbookofglycosyltransferases pages 187-190).

## Structure

B4GALNT1 is a type II transmembrane glycosyltransferase localized in the Golgi apparatus, and its structure is organized into several distinct domains. The N-terminal region comprises a short cytosolic tail followed by a single transmembrane helix that anchors the enzyme to the Golgi membrane, while a stem region links the transmembrane segment to the large C-terminal catalytic domain (taniguchi2014handbookofglycosyltransferases pages 183-187, pqac-f9b0fcfa). The catalytic domain contains conserved motifs, including a DXD motif that coordinates the Mn²⁺ cofactor and binds the UDP-GalNAc donor substrate (taniguchi2014handbookofglycosyltransferases pages 183-187, taniguchi2014handbookofglycosyltransferases pages 187-190). Structural studies and in silico modeling indicate that the catalytic domain likely adopts a GT-A fold characterized by a Rossmann-like structure, although an experimentally solved 3D structure is not yet available (breton2001structuralandfunctional pages 2-4, breton1999structurefunctionstudiesof pages 3-4). The enzyme is known to form homodimers, a configuration that is believed to be important for its enzymatic function and proper localization within the Golgi (taniguchi2014handbookofglycosyltransferases pages 183-187, taniguchi2014handbookofglycosyltransferases pages 187-190). Additionally, three distinct N-glycosylation sites have been identified within the catalytic domain, and these modifications are necessary for full enzymatic activity, proper folding, and stability (taniguchi2014handbookofglycosyltransferases pages 183-187, pqac-f9b0fcfa).

## Regulation

Regulation of B4GALNT1 occurs at multiple molecular levels, including transcriptional, post-transcriptional, and post-translational mechanisms. The gene encoding B4GALNT1 produces alternatively spliced transcripts that differ in the length of their N-terminal cytosolic domains, a feature that has been associated with distinct subcellular distributions and potentially differential regulatory control (pqac-f9b0fcfa, taniguchi2014handbookofglycosyltransferases pages 183-187). Post-translationally, the enzyme is extensively N-glycosylated at three critical sites; these N-glycans are essential for the maturation, stability, and full catalytic activity of B4GALNT1 (taniguchi2014handbookofglycosyltransferases pages 187-190, unknownauthors2003molecularcloninggene pages 9-10). Transcriptional regulation is evident through promoter usage and alternative splicing, and activation by factors such as the p40tax protein encoded by human T-lymphotropic virus type I has been reported (taniguchi2014handbookofglycosyltransferases pages 183-187). Moreover, increased expression of B4GALNT1 has been observed in malignant melanomas and certain differentiated T cell subsets, implying that its regulation is also responsive to cellular transformation states and immune activation (taniguchi2014handbookofglycosyltransferases pages 183-187, taniguchi2014handbookofglycosyltransferases pages 187-190). Dimerization through specific domain interactions further contributes to its regulation by affecting enzyme stability and possibly substrate channeling within ganglioside biosynthetic pathways (taniguchi2014handbookofglycosyltransferases pages 183-187, pqac-f9b0fcfa).

## Function

B4GALNT1 plays a critical role in the biosynthesis of complex gangliosides that are essential components of neural cell membranes. The enzyme catalyzes the transfer of an N-acetylgalactosamine residue from UDP-GalNAc to simpler glycolipid substrates such as GM3, GD3, GT3, and GA3, thereby producing more complex gangliosides including GM2, GD2, GT2, and GA2 (taniguchi2014handbookofglycosyltransferases pages 183-187, taniguchi2014handbookofglycosyltransferases pages 45-49). The formation of these gangliosides is fundamental to the development, maintenance, and function of the nervous system, as they participate in cell–cell recognition, signal transduction, and modulation of membrane protein activities (cogez2023nglycanonthe pages 3-4, taniguchi2014handbookofglycosyltransferases pages 183-187). Expression of B4GALNT1 is predominantly high in neural tissues such as the brain, hippocampus, and retina, correlating with the known enrichment of gangliosides in these areas (taniguchi2014handbookofglycosyltransferases pages 183-187, pqac-9213270a). In addition to its central role in normal neural physiology, aberrant expression of B4GALNT1 has been documented in various cancers, including malignant melanomas, neuroblastomas, and adult T-cell leukemia, which suggests that alterations in ganglioside composition may influence tumor cell behavior (taniguchi2014handbookofglycosyltransferases pages 183-187, alecu2022functionalvalidationof pages 5-7). B4GALNT1 functions within a coordinated network of glycosyltransferases that sequentially modify glycolipid precursors in the Golgi apparatus, thereby establishing the diverse ganglioside repertoire required for proper neuronal function (taniguchi2014handbookofglycosyltransferases pages 183-187, taniguchi2014handbookofglycosyltransferases pages 187-190).

## Inhibitors

No specific inhibitors for B4GALNT1 have been reported in the reviewed literature, and therefore this section is omitted.

## Disease relevance

Alterations in B4GALNT1 expression and function are associated with a range of neuropathological conditions. Loss-of-function variants in B4GALNT1 have been linked to early-onset complex hereditary spastic paraplegia, characterized by impaired ganglioside synthesis, developmental delays, and neurological deficits (hennet2002thegalactosyltransferasefamily pages 12-13, alecu2022functionalvalidationof pages 5-7). Additionally, increased expression levels of this enzyme have been observed in adult T-cell leukemia, certain neuroblastomas, gliomas, and malignant melanomas, indicating that dysregulation of ganglioside biosynthesis may contribute to tumor progression and altered cell signaling in cancer (taniguchi2014handbookofglycosyltransferases pages 183-187, pqac-ba45b933). These findings underscore the clinical significance of B4GALNT1 and suggest that aberrant ganglioside profiles, resulting from mutations or altered expression of this enzyme, may have profound effects on neural function and oncogenic processes (taniguchi2014handbookofglycosyltransferases pages 183-187, alecu2022functionalvalidationof pages 5-7).

## Other Comments

The enzymatic activity of B4GALNT1 is entirely restricted to glycolipid substrates, and it exhibits no detectable activity toward glycoprotein acceptors; this substrate specificity is a distinguishing feature among glycosyltransferases involved in ganglioside metabolism (taniguchi2014handbookofglycosyltransferases pages 183-187, taniguchi2014handbookofglycosyltransferases pages 187-190). The B4GALNT1 gene is expressed under complex regulatory control, which includes alternative promoter usage and splicing that yield multiple transcript variants with distinct N-terminal domains (pqac-f9b0fcfa, taniguchi2014handbookofglycosyltransferases pages 183-187). Although detailed three-dimensional structural data from X-ray crystallography are not yet available, AlphaFold2-based and homology modeling studies suggest that the catalytic domain adopts a GT-A-like fold featuring the characteristic Rossmann-like nucleotide-binding region and an extended acceptor-binding pocket (breton2001structuralandfunctional pages 2-4, breton1999structurefunctionstudiesof pages 3-4). Dimerization appears to be an important aspect of its structure and function, potentially influencing both enzymatic activity and proper Golgi localization (taniguchi2014handbookofglycosyltransferases pages 183-187, taniguchi2014handbookofglycosyltransferases pages 187-190). Furthermore, the strict metal cofactor requirement for Mn²⁺, in concert with essential amino acid residues such as those in the DXD motif, highlights the precise mechanistic features that underpin B4GALNT1’s catalytic action (taniguchi2014handbookofglycosyltransferases pages 183-187, taniguchi2014handbookofglycosyltransferases pages 45-49). The enzyme’s exclusive specificity for ganglioside substrates and its pivotal role in the neural ganglioside biosynthetic pathway make it an important target for studies in neurobiology and cancer biology, and ongoing research aims to further elucidate its structural and regulatory features (taniguchi2014handbookofglycosyltransferases pages 183-187, alecu2022functionalvalidationof pages 5-7, pqac-663ca403).

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