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Review Article

Tyrosine *O* Sulfation: An Overview

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Abstract

Tyrosine *O* sulfation is a post translational modification (PTM) originally discovered by Bettelheim in 1954 in the bovine protein fibrinogen. Currently, this PTM is found only in secreted and transmembrane proteins of higher eukaryotes. This article gives an overview of experimental tools to study tyrosine *O* sulfation and also describes the biological function of this PTM.

INTRODUCTION

The Tyrosine *O* sulfation reaction is catalyzed by two Type II transmembrane enzymes, tyrosylprotein sulfotransferases 1 & 2 (TPST 1 & 2). Huttner identified the trans-golgi as the compartment in which this PTM occurred [1]. The sulfate donor for the reaction is 3′-phosphoadenosine 5′-phosphosulfate (PAPS). The major feature of the sulfated tyrosines is the presence of acidic amino acids within ± 5 residues of the sulfated residues [2,3]. Figure 1 shows the schematic representation of the tyrosine *O* sulfation reaction in the trans-golgi compartment. Currently, only secreted and transmembrane proteins of higher eukaryotes are subject to this PTM.

To study the role of tyrosine sulfation *in-vivo*, knockout animals were generated that had targeted gene disruptions in the genes that code for TPST 1 [4] or 2 [5]. Studies on these animals showed a distinct phenotype for each animal, suggesting no functional redundancy between TPST 1 and TPST 2. *Tpst1* $^{-/-}$ animals had a 5% lower average body weight compared to wild-type animals and the *Tpst1* $^{-/-}$ females had smaller litter sizes due to increased post implantation fetal death [4]. In addition, retinal function of these animals is compromised as assessed by reduced rod ERG function in early development, but these retinas become electrophysiologically normal by postnatal day 90 [6].

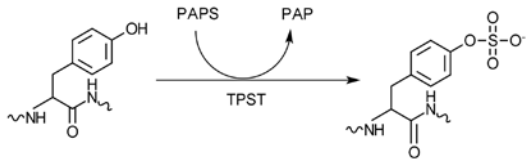

Figure 1 Tyrosine *O* sulfation reaction. The enzyme tyrosylprotein sulfotransferase (TPST) in the trans-golgi compartment, transfers a sulfate group from the universal sulfate donor 3′-phosphoadenosine-5′-phosphosulfate (PAPS) to the tyrosine residue in the protein, resulting in the formation of a tyrosine O sulfate ester and 3′,5′-ADP (PAP).

Tpst2 $^{-/-}$ animals had reduced body weight compared to age matched wild-type animals, and the males are sterile. *In-vitro* fertilization assays in these animals showed fewer eggs were able to be fertilized by sperm from *Tpst2* $^{-/-}$ males. Further analysis of *Tpst2* $^{-/-}$ sperm showed a decreased motility in viscous media and an inability to penetrate zona pellucida of intact eggs [5]. Retinal function of these animals is also compromised as assessed by reduced rod ERG and cone ERG values, that do not become electrophysiologically normal during the entire age of the animal [6].

Double knockout animals (DKO) generated by selective matings between the two individual knockout animals [7] had 95% mortality by postnatal day 5 and no animal survived beyond 2 months of age [7]. Autopsy studies on these pups indicated poor aeration of the lungs due to improper expansion of the alveoli. The hearts of these animals were also abnormal due to the enlargement of the atrium and vena cava. And, in addition to these effects, the follicles of the thyroid gland were devoid of colloid suggesting that these animals are hypothyroid [7]. These animals also had the most drastically reduced visual function as assessed by the rod and cone ERG values becoming 25% and 15% of normal wild-type levels, respectively [8]. The rod and cone synaptic terminals were disorganized and defects were seen in their ultrastructure at the EM level [8].

DETECTION METHODS

Tyrosine *O* sulfation in proteins can be detected by radioactive and non-radioactive methods.

Non-radioactive methods

Two antibodies are widely used to identify tyrosine-sulfated proteins [9,10]. These antibodies were developed using phage display technology. The epitope for both antibodies was the tyrosine-sulfated N-terminal region of PSGL-1 [9,10]. Therefore, a limitation to using these antibodies to identify tyrosine-sulfated protein is that both antibodies identify tyrosine-sulfate residue

in proteins only if the amino acids flanking the tyrosine-sulfate are similar to PSGL-1 (acidic amino acids within ± 2 residues) [10]. Any tyrosine-sulfated protein that diverges from the PSGL-1 consensus sequence by having a basic residue ±1 to the tyrosine-sulfate will be revealed as a non tyrosine-sulfated protein. Therefore, multiple detection methods need to be used to positively identify a tyrosine-sulfated protein.

Mass spectrometric analysis is commonly used to identify post translational modifications such as phosphorylation, glycosylation, methylation and ubiquitination. However, since tyrosine *O* sulfation is a modification that is labile in positive ion MS/MS, this PTM is detected by comparing spectra using a combination of positive and negative ion mode analyses [11-13]. Another mass spectrometric analysis method used to identify tyrosine-sulfated residues in peptides involves acetylating the unmodified tyrosine residues by sulfosuccinimidyl acetate and then subjecting the protein to positive ion MS/MS analysis. In this method by Yu et. al [13], peptide fragmentation analysis then identifies a tyrosine-sulfate as an unmodified tyrosine.

Radioactive methods

This method involves metabolic labeling of the protein with radioactive S^{35}sodium sulfate, which gets incorporated into the sulfate donor PAPS, then into sulfated carbohydrates and tyrosine residues in proteins. To distinguish between the carbohydrate sulfates and tyrosine-sulfates the radiolabelled protein is then immunoprecipitated from the cell and subjected to digestion into individual amino acids by pronase enzyme digestion [14] or barium hydroxide hydrolysis [7,15-18] followed by thin layer electrophoresis of the hydrolysates in the presence of non-radioactive tyrosine-sulfate standards. Co-localization of the radioactive tyrosine-sulfate from the protein of interest with the non-radioactive tyrosine-sulfate standard positively identifies the tyrosine-sulfated status of the protein.

FUNCTION

Multiple studies on tyrosine-sulfated proteins reveal a major role for this PTM in enhancing protein-protein interaction. Some of the major functions in the body that require tyrosine *O* sulfation are:

Blood coagulation

Tyrosine *O* sulfation has been detected in multiple proteins involved in the blood coagulation process. Some of these proteins are fibrinogen [19], Factor V [17,20], Factor VIII [21,22], Factor IX [23] and hirudin [24]. Mutation of the tyrosine-sulfated 1680 residue to phenylalanine in Factor VIII, results in non-optimum binding to von Willebrand factor and moderate hemophilia in patients [21,22]. In addition, tyrosine-sulfated residues in fibrinogen and hirudin are needed for optimum binding to thrombin [24-26].

Optimum rolling of leukocytes on endothelial cells

Host response to tissue damage, is to recruit leukocytes to local sites of inflammation. This is achieved by the rolling of leukocytes on endothelial cells in blood vessels. The rolling of leukocytes on endothelial cells is mediated by P-selectin glycoprotein ligand-1 (PSGL-1), which is expressed on leukocytes interacting with P-selectins expressed on endothelial cells. Studies have shown that Tyr 46, 48 and 51 are sulfated in PSGL-1 and that elimination of tyrosine-sulfation reduces binding and rolling on endothelial cells in *in-vitro* flow assays [27,28].

Chemokine receptor ligand binding

Chemokine receptors are 7 transmembrane G-protein coupled receptors found on the surface of leukocytes, with 19 members identified thus far. Of these, nearly 13 are predicted to be tyrosine-sulfated according to the position specific scoring matrix algorithm [29]. Tyrosine-sulfation has been experimentally confirmed in the N-terminus of five receptors, CCR5 [30], CCR2b [31], CXCR3 [32], CXCR4 [33] and CX3CR1 [34], in a region that is critical for chemokine binding. Studies done on these receptors have proven the role of tyrosine *O* sulfation in ligand binding specificity and downstream signaling [35].

Chemokine CCR5 binding to HIV-1 gp120 and entry of virus into cells

It has been shown that chemokine receptor CCR5 is necessary for binding of the HIV-1 gp120/CD4 complexes and entry of the virus into cells [30]. Experiments using tyrosine sulfation inhibitor chlorate or using peptides that contained the tyrosine-sulfated N terminal region of CCR5 or had them eliminated, showed that tyrosine sulfation of CCR5 is important for the HIV gp 120/CD4 complexes from binding to CCR5 receptor and entry of the virus into CCR5 expressing cells [30,36,37].

Hormone binding to receptors

Glycoprotein hormone receptors such as Thyrotropin receptor (TSHR), Luteinizing/choriogonadotropin (LH/CG) receptor and follicle stimulating hormone (FSH) receptors recognize TSH, LH and FSH hormones as their ligands and are responsible for sexual development and reproduction. These three receptors are highly conserved with a 70% sequence identity. A highly conserved Y-D/EY motif in the extracellular domain of TSHR and LH/CG, and an FDY motif in FSHR domain of these proteins contain the tyrosine *O* sulfation sites. Site-directed mutagenesis studies on these receptors have established the role of tyrosine-sulfation of these motifs in the high affinity binding of these hormones to the receptors [38].

Protein interactions in the ECM

Multiple proteins of the ECM such as fibronectin, fibromodulin, lumican and osteoadherin were found to be tyrosine-sulfated [12,39]. These proteins are rich in cartilage tissue and implicated in binding collagens, which are also predicted to be tyrosine-sulfated according to the tyrosine-sulfation predicting program Sulfosite [40] and by experimental detection by the barium hydroxide hydrolysis radioactive method [39]. Therefore a possible role for the presence of these highly tyrosine-sulfated proteins in the cartilage may be to stabilize the fibrillar network by collagen binding.

Growth promoting effects in plants

Three tyrosine-sulfated peptides, PSK, PSY1 and RGF have been identified in Arabidopsis plants [41]. These peptides are involved in a multitude of growth promoting functions such

as germination of pollen [42], root growth [43], growth of hypocotyls [44] and host defense against bacterial pathogens [45]. Elimination of these peptide ligands or their receptors can lead to deleterious effects in the plants such as dwarfism, reduced ability to repair wounds [46], small leaves and early senescence [46,47].

REGULATION

Enzyme regulation in biological systems is usually done by controlling the transcription, translation, or controlling the activity of the enzymes. While there is no transcription/translation regulation of TPST activity reported, the activity of TPST enzymes can be modulated by numerous factors. Prostaglandin PGE2, enhances the activity of TPSTs by decreasing the Km from 1.4 pM to ~0.12 pM, compared to the absence of prostaglandin [48]. Two other members of the prostaglandin family, $PGF_{2\alpha}$ and 6-Keto-$PGF_{1\alpha}$, also increase the activity to a lesser extent by 22% and 23% respectively, when added at 100 μM concentration [48]. Numerous factors have been shown to decrease the activity of the TPST enzymes. Manganese ion (Mn^{2+}) suppressed the activity of TPST by ~ 50% when added at 0.3 mM concentration for 24 hours [49]. Sphingosine and 2-choloroadenosine inhibited the activity by 50% when added at 150 μM and 50 μM respectively [50,51]. Tyrosine sulfation has been shown to be completely abolished in the presence of 1 mM sodium chlorate [52]. The mechanism of action of this chemical is by inhibition of action of ATP-sulfurylase, the first enzyme responsible for the synthesis of the sulfate donor PAPS. The addition of this drug causes the inhibition of all sulfation (both tyrosine sulfation and carbohydrate sulfation) by > 95%. Therefore, this drug can be used to study the effects of sulfation in general.

RECENT ADVANCES

New tools have been recently developed that have led to the advancement of the field of tyrosine *O* sulfation. A brief description of these tools is given below.

Flourescent assay for detection of tyrosine-sulfation

A novel method has been developed to study protein tyrosine *O* sulfation *in-vitro* [53]. This method involves incubation of a substrate protein with two enzymes, phenol sulfotransferase (PST) and tyrosylprotein sulfotransferase (TPST) along with two sulfate donors, 4-methylumbelliferyl sulfate (MUS), which is a fluorophore, and adenosine 3′, 5′-diphosphate (PAP). In this reaction, the PST enzyme catalyzes the transfer of a sulfate group from the sulfate donor MUS to PAP to generate 3′-phosphate 5′-phosphosulfate (PAPS). Then, the tyrosine sulfating enzyme, TPST utilizes PAPS as the sulfate donor and catalyzes the transfer of the sulfate to the substrate protein. This entire reaction is monitored by the fluorescent generation of 4-methylumbelliferone (MU). This assay is especially important if one wants to study the enzyme kinetics of TPST 1 or 2 reactions.

Expression of tyrosine-sulfated proteins in *E. coli*

A novel method has been developed to produce tyrosine-sulfated proteins in *E. coli*, which normally does not have this post translational modification. This method involves mutation of the codon that incorporates tyrosine-sulfate to the amber nonsense codon, TAG. The protein containing the mutated codon is then co-expressed along with the tRNA/aminoacyl-tRNA synthetase pair that recognizes tyrosine-sulfate, which leads to a tyrosine-sulfate residue being incorporated into the nonsense codon TAG site [54,55]. This method can potentially lead to the synthesis of large amounts of tyrosine-sulfated proteins as recombinant protein synthesis in bacteria is an established method.

CONCLUSION

A major drawback in the field of tyrosine *O* sulfation is the absence of an antibody that identifies the tyrosine-sulfate residue independent of its neighboring residues. Other methods such as mass spectrometric analysis and radioactive methods require expensive instruments. Therefore, more tools need to be developed to study this post translational modification. As more tools are developed, more novel tyrosine-sulfated proteins will be identified and the sites of tyrosine-sulfates determined. Finally, the true function of tyrosine *O* sulfation in specific proteins will be revealed from studies of *in-vivo* 'mutant knock-in' mice that have their tyrosine-sulfated residues mutated to phenylalanines. A study of this mouse model in comparison to the wild-type tyrosine-sulfated protein will reveal the true function of this post translational modification *in-vivo*.

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