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Histidine residues in human phenol sulfotransferases

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Abstract

Sulfotransferases are phase II drug-metabolizing enzymes that catalyze the sulfation of hydroxyl-containing compounds, leading to detoxification of xenobiotic toxicants. The universal sulfuryl donor is adenosine 3′-phosphate-5′-phosphosulfate. Human simple phenol sulfotransferase (P-PST) is one of the major human sulfotransferases that catalyze the sulfation of most phenols. Human monoamine phenol sulfotransferase (M-PST) has high affinity for monoamines and also catalyzes the sulfation of simple phenols at high substrate concentrations. In this report, the amino acid modification method was used for studies of His residues in the active site of P-PST and M-PST. The His specific modification reagent diethylpyrocarbonate was used for the modification of His residues in P-PST and M-PST. Diethylpyrocarbonate inactivation kinetic data suggest that there is one His residue in the active site that is critical for catalytic activity of both P-PST and M-PST. The modification has no effect on phenol or monoamine substrate binding for M-PST, but it does have an effect on adenosine 3′-phosphate-5′-phosphosulfate binding with M-PST. The experimental results agree with amino acid sequence alignment, mutation, and the crystal structures of P-PST and M-PST and suggest that His108 is the only critical His residue in both P-PST and M-PST. The differing roles His108 plays in P-PST and M-PST may explain the substrate specificity of the two isoforms.

Keywords: Sulfotransferases; Amino acid modification; Structure–function studies; Histidine; Enzyme active site; Diethylpyrocarbonate

1. Introduction

Sulfotransferases (STs) catalyze the sulfation of many structurally diverse drugs, carcinogens, and other xenobiotics as well as endogenous molecules, such as steroid hormones, bile acids, thyroxin, glycosaminoglycans and glycoproteins, proteins and peptides (tyrosine residue), and neurotransmitters [1,2]. The co-substrate for sulfation of all STs is adenosine 3′-phosphate-5′-phosphosulfate (PAPS). The substrate specificities of some STs are very broad. Most hydroxyl groups in phenols, alcohols, and *N*-substituted hydroxylamines are substrates for one of the STs [3]. Sulfation of drugs and xenobiotics is primarily associated with detoxification by which a relatively hydrophobic xenobiotic is biotransformed into a more water-soluble sulfuric ester that is readily excreted. However, there are numerous important exceptions wherein the formation of chemically reactive sulfuric esters is an essential step in metabolic pathways leading to toxic or carcinogenic bioactivation [4–7]. Detoxification or bioactivation is highly dependent upon the electrophilic reactivity of the individual sulfuric ester products formed.

Structure–activity relationship studies of STs began in the 1980s. Certain peptide sequences and active site amino acid residues have been identified as important for the catalytic activities of different STs. Protein sequence alignments of the different STs have revealed two highly conserved regions, one (PKSGTTW) in the N-terminal region and one (RKGXXGDWK) in the C-terminal region [8]. Since all STs use the same sulfuryl donor, it was speculated that these two regions were involved in PAPS binding [9,10]. Site-directed mutagenesis and [^{35}S]PAPS affinity labeling studies of plant flavonol 3-ST have supported the conclusion that the two regions mentioned above are involved in PAPS binding [11]. Point mutations and [^{35}S]PAPS affinity labeling studies of guinea pig estrogen sulfotransferase (EST) have also strongly supported the C-terminal conserved region mentioned above as being part of the PAPS binding site [12]. The X-ray crystal structure of mouse EST supports

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Abbreviations: DEPC, diethylpyrocarbonate; P-PST, human simple phenol sulfotransferase (SULT1A1); MBP-PST, maltose-binding protein P-PST; M-PST, human monoamine phenol sulfotransferase (SULT1A3); MBM-PST, maltose-binding protein M-PST; PAPS, adenosine 3′-phosphate-5′-phosphosulfate; PAP, adenosine 3′-phosphate-5′-phosphate; PNPS, *p*-nitrophenyl sulfate; PNP, *p*-nitrophenol; STs, sulfotransferases; EST, estrogen sulfotransferase; PST, phenol sulfotransferase; SA, specific activity; 4PP, 4-phenylphenol.

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the idea that these two regions, residues 259–265 and 45–51 in mouse EST, are directly involved in PAPS binding [13]. In addition to these two regions, Thr227, Trp53, and Phe229 of mouse EST have also been shown to be involved in PAPS binding.

Limited information is available on the structural determinants of the ST substrate-binding site. Sakakibara *et al.* reported localization and a functional analysis of the substrate specificity and catalytic domains of the human M-PST and P-PST [10]. By comparing the kinetic parameters of a series of expressed chimeric PSTs, where the M-form and P-form coding regions were exchanged, it was concluded that amino acid residues 84–148 contain the structural determinants for substrate specificity in both forms. They also demonstrated the differential roles of the two highly variable regions (amino acid residues 84–89 and 143–148) in substrate binding, catalysis, and sensitivity to inhibition by 2,6-dichloro-4-nitrophenol. A mutation study of P-PST and M-PST demonstrated the importance of Ala146 (P-PST) and Glu146 (M-PST) in the determination of the substrate specificity of these two enzymes [14]. Similar studies on human aryl STs (HAST1 (SULT1A1, P-PST) and HAST3 (SULT1A3, M-PST)) suggested that the two highly divergent regions, region A (amino acids 44–107) and region B (amino acids 132–164), determine the substrate specificity of human aryl STs [15,16]. A single amino acid change in HAST1 (A146E) changed its activity for *p*-nitrophenol (PNP) to that of HAST3. Studies on guinea pig 3-hydroxysteroid ST isoforms revealed that the amino acid residue at position 51 plays a fundamental role in determining the stereospecificity exhibited by the α- and β-isoforms. Mutational studies on mouse EST demonstrated that Tyr81 determines the substrate specificity of this enzyme [17]. The X-ray crystal structure of mouse EST showed that residues Phe142, Ile146, and Tyr149 contribute to binding of steroid 17β-estradiol. Asn86 is believed to be in a position to form a hydrogen bond with the 17β-hydroxyl group, while Lys106 and His108 are within hydrogen bonding distance of the steroid's 3α-phenol group [13].

To our knowledge, studies using amino acid modification reagents to identify specific residues in the active site of STs are limited. Borchardt and Schasteen used 2,3-butanedione and phenylglyoxal to identify the arginyl residues in the active site of rat liver PST [18,19]. *N*-Ethylmaleimide has also been used for the identification of essential sulfhydryl residues in rat PST [19]. Ribonucleotide dialdehydes (ATPDA, ADPDA, AMPDA, APSDA) have been characterized as affinity labeling reagents for rat liver PST and it was speculated that the dialdehydes inactivated the ST by possible formation of a Schiff's base adduct with an active site lysine residue [19,20]. ATPDA has been used as an affinity labeling reagent for the identification of a peptide sequence in the PAPS-binding site of rat liver aryl ST IV (AST-IV) [21]. We have studied the carboxyl amino acid residues in the active site of human P-PST [22]. Our results indicated that the conserved residue Asp134 is critical for the binding of PAPS. Glu83 may be important for the binding of substrate and may be involved in the catalytic reaction.

Since the crystallization of the mouse EST [13], a number of human STs have also been crystallized and their crystal structures partially solved. These include M-PST (SULT1A3) [23,24], human EST (SULT1E1) [25] and hydroxysteroid ST (DHEA-ST, SULT2A1) [26], and P-PST (SULT1A1) [27].

Crystal structure determination, computer modeling, and site-directed mutagenesis are all established methods for protein structure–activity relationship studies. Each of these methods has its limitations though. Even with the known crystal structure, the roles of amino acid residues in the active site still need to be determined using experimental methods when the protein is in solution or during catalytic reaction. An amino acid mutation (site-directed mutagenesis) may or may not exactly reflect the importance of that residue with regard to the catalytic activity of the enzyme. Protein folding and packing may inactivate a mutant even though the amino acid residue is not important for the enzyme's catalytic activity. Experimental methods such as amino acid modification and affinity labeling are complimentary methods to those mentioned above for the study of active site amino acids.

In this report, we studied His residues in the active site of human PSTs (P-PST and M-PST) using amino acid modification, amino acid sequence alignment, and computer modeling. His residues have been known to be essential amino acids among many enzymes. Our results, combined with those previously published, demonstrate that His108 is a critical residue for human PSTs and other STs. Other His residues in human PSTs are not likely to be critical.

2. Materials and methods

2.1. Material

Diethylpyrocarbonate (DEPC), 4-phenylphenol (4PP), 2-naphthol, *p*-nitrophenyl sulfate (PNPS), adenosine 3′-phosphate-5′-phosphate (PAP), and PAPS were purchased from Sigma Chemical Co. Amylose affinity resin and maltose were purchased from New England Biolabs. All other chemicals and solvents were of the highest grade available.

2.2. Purification of maltose-binding protein M-PST (MBP-PST) and maltose-binding protein M-PST (MBM-PST)

Human MBP-PST cDNA and MBM-PST cDNA were expressed using the pMAL-c2 expression system as previously described [29,31]. Briefly, the maltose-binding fusion protein (MBP-PST and MBM-PST) was expressed in XL1-Blue cells containing the pMAL-P-PST vector. The cytosol of fusion protein was loaded onto an amylose

affinity column (New England Biolabs) prewashed with 5 mM phosphate buffer, pH 7.4. After washing out other proteins, the fusion protein was eluted using a 0–1 mM maltose gradient in the same buffer. The final purified MBP-PST and MBM-PST appeared homogenous according to SDS–PAGE analysis. These fusion proteins are constructed in Dr. Charles Falany's laboratory. Kinetic data from Dr. Falany's laboratory [29,31] and our laboratory suggest that the maltose-binding protein does not affect the kinetic behavior of these enzymes.

2.3. Enzymatic assay

2.3.1. PNPS assay

This method utilizes PNPS to regenerate PAPS from product PAP. At the same time, the color reagent PNP is generated for colorimetric measurement. The PNPS assay was used for the determination of both MBP-PST and MBM-PST sulfation activity. The detailed procedure was described in a previous publication [28–30]. In summary, the reaction mixture, 250 μL total volume, contained 50 mM sodium phosphate, pH 6.2, 5 mM PNPS, 0.02 mM PAPS, and 2–5 μg MBP-PST or MBM-PST. After incubation at 37.0° for 2 min, the reaction was started by addition of either 5 μL of 5 mM 2-naphthol or 5 μL ethanol as a control. After a 30-min incubation at 37.0°, the reaction was stopped by addition of 250 μL of 0.25 M Tris buffer, pH 8.7. The absorbance at 401 nm was measured within 30 min. Assays were done in triplicate and the average of the measurements minus the controls was used to calculate the enzymatic activity.

2.4. MBP-PST and MBM-PST inactivation by DEPC

MBP-PST (or MBM-PST) (0.1 mg/mL) was incubated in 0.1 M phosphate buffer, pH 6.0, at room temperature. Different concentrations of DEPC (in ethanol) were added to start the inactivation reaction. Aliquots (10 μL) were taken at different times for standard assay of MBP-PST (or MBM-PST) activity. Imidazole (0.1 M) was included in the enzyme assay reaction mixture (for time- and concentration-dependent inactivation) or used to stop the reaction (for partial inactivation experiments). For substrate or PAPS protection experiments, phenol substrates (2-naphthol or dopamine) or PAPS was added to the enzyme solution before the addition of DEPC.

3. Results

3.1. Time- and concentration-dependent inactivation of MBP-PST (SULT1A1) and MBM-PST (SULT1A3) by DEPC

DEPC is a reagent specifically reactive toward the His residues of a protein (Fig. 1). It has been used for the characterization of His residues in various enzymes since the 1960s [32–35].

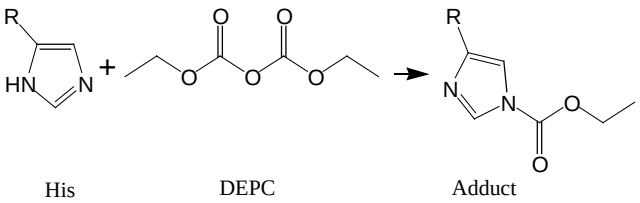

Fig. 1. Chemical reaction for DEPC inactivation of His residues. See Ref. [38] for detailed information of this reaction.

DEPC inactivated both human PSTs, MBP-PST (SULT1A1) and MBM-PST (SULT1A3), in an efficient, time- and concentration-dependent manner (Figs. 2A and 3A). At all DEPC concentrations, log(% activity) is linear vs. time. These facts indicate that the inactivation occurs through His residues in the active site. According to the equation $d[E]/dt = k_{app}[E]$ or $\log[E] = 2.3k_{app}t$ (where E represents active enzyme, t is time, k_{app} is apparent first order rate constant), k_{app} can be calculated from the slopes of Figs. 2A and 3A. According to equation $k_{app} = k[DEPC]^n$ or $\log k_{app} = \log k + n \log[DEPC]$ (where k is the rate constant, n is the reaction order for DEPC), when $\log(k_{app})$ is plotted against the log[DEPC], a straight line

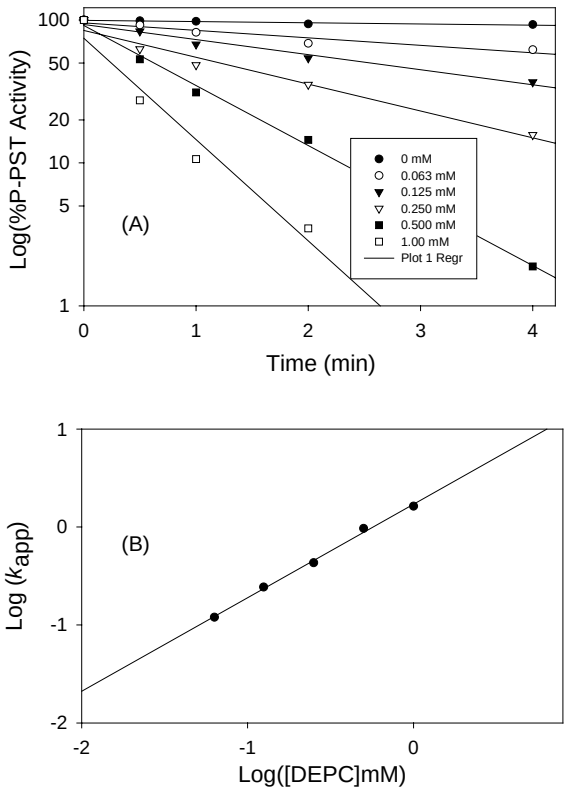

Fig. 2. Time- and concentration-dependent inactivation of MBP-PST by DEPC. (A) log(% activity) vs. time. MBP-PST (0.1 mg/mL) was incubated with different concentrations of DEPC at room temperature in phosphate buffer, pH 6.0. Aliquots were taken at different time points. Remaining MBP-PST activity was assayed using the PNPS assay method in a mixture containing 0.1 M imidazole. (B) $\log(k_{app})$ vs. log(DEPC).

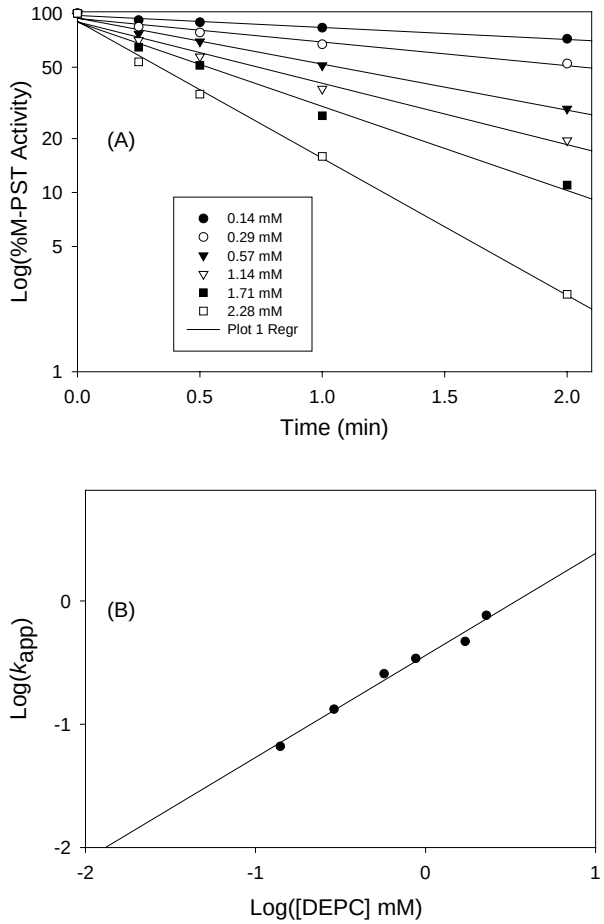

Fig. 3. Time- and concentration-dependent inactivation of MBM-PST by DEPC. (A) log(% activity) vs. time. MBM-PST (0.1 mg/mL) was incubated with different concentrations of DEPC at room temperature in phosphate buffer, pH 6.0. Aliquots were taken at different time points. Remaining MBM-PST activity was assayed using the PNPS assay method in a mixture containing 0.1 M imidazole. (B) log(k_{app}) vs. log(DEPC).

results (Figs. 2B and 3B). From the data in Fig. 2B, $n = 0.95$ and $k = 1.7$ min/mM. This further demonstrates that DEPC is a specific reagent for the inactivation of a single critical His residue in MBP-PST. The DEPC reaction order n is approximately 1. This suggests that there may be only one His residue in the active site whose modification could lead to inactivation of MBP-PST. From the data in Fig. 3B, $n = 0.86$ and $k = 0.36$ min/mM. This also suggests that for MBM-PST, there should be one His residue in the active site that is critical for enzyme catalytic activity. These indications cannot exclude the possibilities that there may be crucial His residues in the active sites of MBP-PST and/or MBM-PST which cannot react with DEPC because of some special structural reasons.

3.2. Substrate and PAPS protection for DEPC inactivation of MBP-PST and MBM-PST

Fig. 4 suggests that PAPS or 2-naphthol protect MBP-PST from inactivation by DEPC, but this protection is very weak. This suggests that the His108 in MBP-PST is not located within the PAPS- or substrate-binding site. The limited protection of both 2-naphthol and PAPS suggests that the His108 in P-PST is close to both the substrate- and PAPS-binding site. The His108 residue in the active site does not significantly contribute to the binding of either PAPS or substrate but does contribute to the enzyme's catalytic activity. For MBM-PST, neither 2-naphthol nor dopamine protected the enzyme from DEPC inactivation (Fig. 5). In contrast, PAPS provided significant protection. This suggests that the modified His108 is not in close proximity to the substrate-binding site but rather is close to the PAPS-binding site. Further support is provided by the changes seen with the kinetic parameter data for DEPC

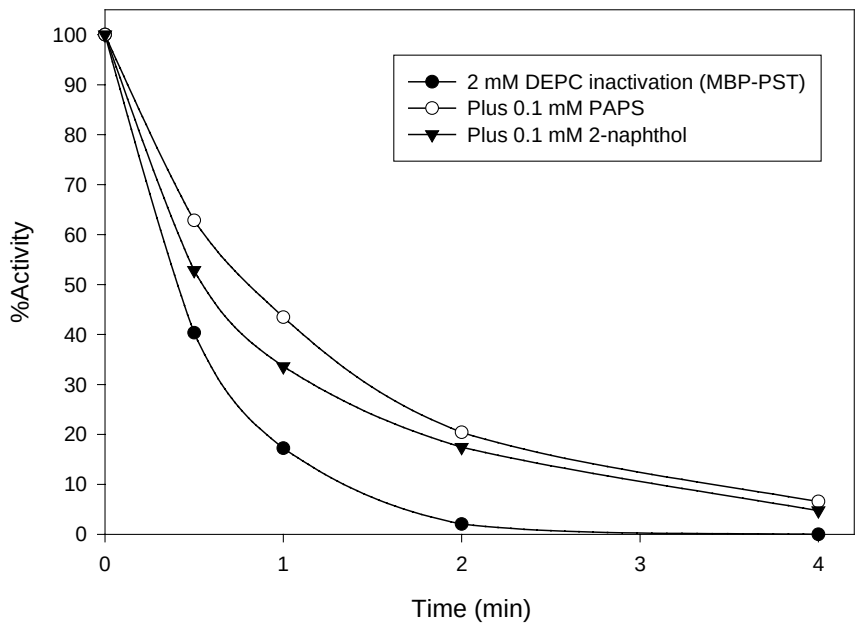

Fig. 4. PAPS and substrate protection of P-PST from DEPC inactivation. The PNPS assay was used to determine remaining enzyme activity.

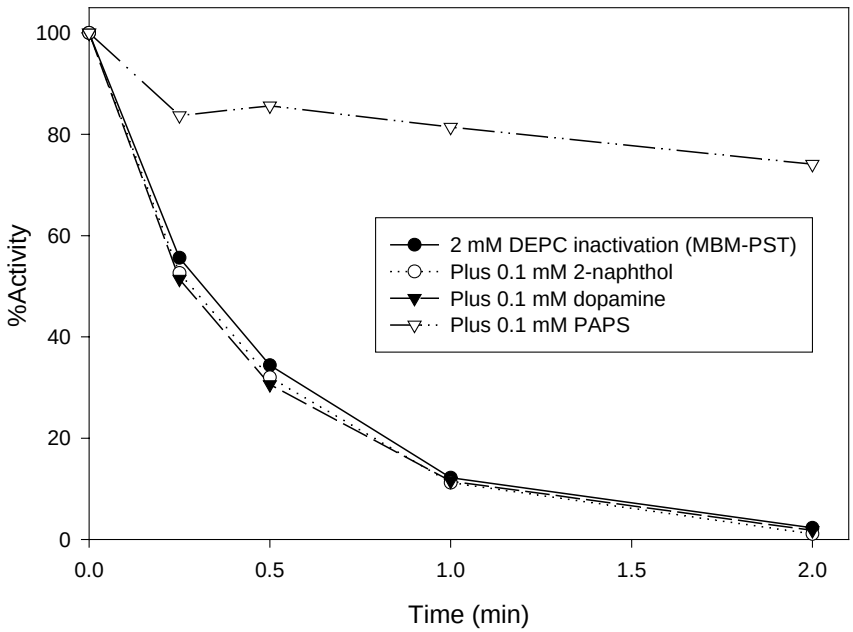

Fig. 5. PAPS and substrate protection of MBM-PST from DEPC inactivation. The PNPS assay was used to determine remaining enzyme activity.

partial inactivation of MBP-PST and MBM-PST (data not shown).

3.3. Protein sequence alignment of mammalian STs

Both P-PST and M-PST have 295 amino acids in their sequences. The two isoforms share 93% homology. Both isoforms can catalyze simple phenol sulfation, P-PST having higher activity. M-PST has high affinity toward positively charged monoamines, such as dopamine, while P-PST has very low affinity toward these substrates. Amino acid sequences of P-PST (Accession #: L19999) and M-PST (U08032) have been used for the multiple sequence alignment with three PSTs (dog (D29807), cow (U35253), and mouse (U08032)), four ESTs (human (X84816), guinea pig (U35115), rat (M33329), and mouse (AF026072)), and four hydroxysteroid STs (human (U20521), mouse (S78182), guinea pig (U09552), and rat (M86758)), each from mammalian species (data not shown). Sequence alignment demonstrates that only His108 in both P-PST and M-PST is completely conserved in all mammalian STs. Other His residues in P-PST and M-PST are not significantly conserved.

3.4. Crystal structure of P-PST

Crystal structures of P-PST [27] and M-PST [23,24] (not shown) are available. Fig. 6 shows the P-PST crystal structure (1LS6) [27] (displayed as strands). The structure shows His residues (displayed as sticks), including His108 (displayed as spacefill), PAP (displayed as spacefill), and two molecules of PNP substrate (displayed as spacefill). His108 is in close proximity to the site of sulfuryl group transfer. It is in direct contact with both the sulfate group on PAPS and the hydroxyl group on substrate PNP (within 2–3 Å). Combining both the DEPC inactivation and amino acid sequence alignment results, His108 is further shown to be a critical amino acid for P-PST, M-PST, and other mammalian STs activity.

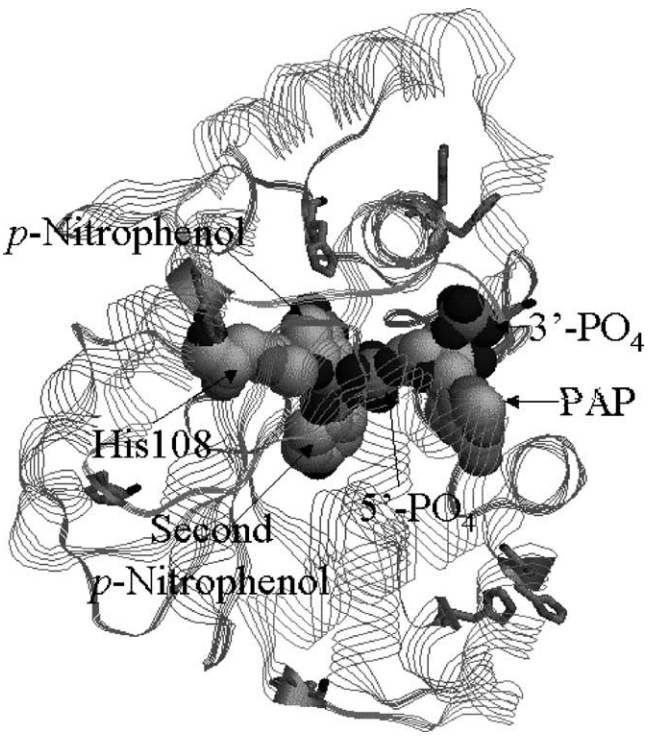

Fig. 6. Crystal structure of P-PST showing His residues and ligands. The crystal structure of P-PST ((1LS6) [27]) was depicted by RasMol 2.6. His108, PAP, and PNP molecules were displayed as ''spacefill''. Other His residues were displayed as ''sticks''. Other parts of the structure were displayed as ''ribbons''.

4. Conclusion

In summary, our amino acid modification results suggested experimentally that His108 is a critical residue for both human P-PST and M-PST. Our kinetic data suggest that there is only one His residue in the active site of P-PST and M-PST which contributes to catalytic activity. Protection experiments suggest that the modification of the His residue does not affect the binding of phenol substrate and PAPS for MBP-PST and does not affect the binding of monoamine for MBM-PST. The modification does, however, affect PAPS binding to a certain extent for MBM-PST. These results agree with amino acid sequence alignment and crystal structure results. Sequence alignment and crystal structure suggest that His108 is the only critical His residue. The imidazole group in His108 may act as a base catalyst for the sulfuryl transfer.

Site-directed mutagenesis studies on His residues in STs have been reported. Mutation of His108 (H108A) in both P-PST and M-PST resulted in complete loss of sulfation activity [36]. They concluded that His108 is important in the catalytic mechanism for both P-PST and M-PST. Mutation results alone do not indicate if His108 is important for sulfuryl group transfer, PAPS binding, or substrate binding. Crystal structure and mutation studies on mouse EST demonstrated that His108 (at the same conserved position as human P-PST and M-PST) is critical for catalytic activity [37]. They concluded that His108 is in position to both stabilize the transition state and/or to function as the catalytic base for mouse EST. Our experimental results further prove these conclusions for human MBP-PST and MBM-PST in buffer solution.

Our experimental results suggest that although His108 is critical for both MBP-PST and MBM-PST activity, its function within each enzyme differs. PAPS protected MBM-PST from being inactivated by DEPC, whereas it did not protect MBP-PST from being inactivated. His108 in MBM-PST is in closer proximity to the PAPS-binding site compared to its position in MBP-PST. This may be related to the substrate specificity differences of the two isoforms.

Acknowledgments

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