Arylsulfatases

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Editorial

The three arylsulfatases, A, B, and C are isoenzymes, which are differentiated from one another in terms of substrate specificity, response to inorganic inhibitors, pH optimum, cellular location and solubility [1]. Arylsulfatase A (ASA, EC 3.1.6.8) is a lysosomal enzyme known as cerebroside-3-sulfohydrolase. It desulfates the galactose-3-sulfate residues in cerebroside sulfate and other sulfated galactolipids [2]. Cerebroside sulfate has an important role during developmental period including myelination and it is one of the myelin sheath constituents [3]. Metachromatic leukodystrophy is a disease in which there is a deficiency in ASA leading to neurological disorders [4]. An increase in this enzyme concentration was found in the body fluids of the lung, central nervous system, colon cancer and malignant lymphoma patients [5]. The pH optimum for activity is 5.2, when hydrolyzing p-nitrocatechol sulfate and its isoelectric point is 4.3. ASA is inhibited by sulfate, sulfite, silver, magnesium, manganese and calcium ions [6]. On the other hand, Arylsulfatase B (ASB, EC 3.1.6.12) is a lysosomal hydrolase that desulfates the non-reducing terminal N-acetylgalactosamine-4-sulfate residues that are present in Glycosaminoglycan (GAG) [7]. The Mucopolysaccharidoses (MPS) type VI (Maroteaux-Lamy disease) is a lysosomal storage disease that occurs due to a deficiency of ASB activity results in the accumulation of dermatan sulfate. MPS type VI is characterized by short stature, distress, dysostosis multiplex, cranial and tracheal abnormalities, cornea and cardiovascular defects, and degenerative joint disease [8]. In addition, ASB activity is increased in bladder tumors [2] and colorectal carcinoma [9]. In visceral neoplasm, the ASB activity is either elevated or depressed in a tissue specific manner [10]. ASB displayed a pH optimum of activity at 5.8 using p-nitrocatechol sulfate as a substrate and isoelectric point range from 8.6 to 8.9. ASB is inhibited by chloride, sulfate, sulfite and silver ions [6]. It was surprising that ASB was found to regulate the interaction of chondroitin-4-sulfate and kinosin in renal epithelial cells [11].

Arylsulfatase C (ASC, EC 3.1.6.2) is a steroid sulfatase which is available in a wide variety of organisms and tissues. In mammals, the enzyme is resistant to inhibition by phosphate and being able to catalyze a variety of synthetic substrates including p-acetylphenyl sulfate, p-nitrophenyl sulfate and 4-methylumbelliferyl sulfate. The enzyme was found in the microsomes of rat liver, kidney and spleen cells [12].

In fact, the activity of arylsulfatases increases in biological materials under physiological and pathological conditions such as pregnancy, wound healing, inflammation, intoxication and malignancy [13]. It was previously reported that ASA and ASB are modified by sialylation and phosphorylation in some tumors [14-16] and ASB activity was changed due to its phosphorylated by cAMP-dependent protein kinase in lung cancer [17,18]. Moreover, it was found previously that hepatic ASB displayed a significant change of its specific activity in Schistosomiasis, whereas ASA showed a non-significant change [19].

In addition to the above mentioned types, there are other types, whose their function is still subject to investigation by many researchers to elucidate a clear chart of the various types of arylsulfatases.

References


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