Biochemistry & Physiology: Open Access

Balbaa, Biochem Physiol 2014, 3:4
DOI: 10.4172/2168-9652.1000e125

Editorial Open Access

Arylsufatases

Mahmoud Balbaa*

Department of Biochemistry, Faculty of Science, Alexandria University, Alexandria, Egypt

***Corresponding author:** Mahmoud Balbaa, Department of Biochemistry, Faculty of Science, Alexandria University, Alexandria, Egypt, Tel: +96179858583; E-mail: mahmoud.balbaa@alexu.edu.eg

Rec date: Aug 11, 2014, **Acc date:** Aug 12, 2014, **Pub date:** Aug 19, 2014

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, 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*-nitrocatecholsulfate 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 kininogen 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

1. Gasa S, Makita A, Kameya T, Kodama T, Araki E, et al. (1980) Elevated activities and properties of arylsulfatases A and B and B-variant in human lung tumors. Cancer Res 40: 3804-3809.
2. Boyland E, Wallace DM, Williams DC (1955) The activity of the enzymes sulphatase and beta-glucuronidase in the urine, serum and bladder tissue. Br J Cancer 9: 62-79.
3. Kolodny EH, Moser HW (1983) Sulfatide lipidosis: metachromatic leukodystrophy. In The Metabolic Basis of Inherited Disease. Stanbury JB, Wyngaarden JB, Fredrickson DS, Goldstein JL, Brown MS, editors. 5th ed., McGraw-Hill, NewYork 881-905.
4. Kolodny EH, Fluharty AL (1995) Metachromatic leukodystrophy and multiple sulfatase deficiency: sulfatide lipidosis. In Scriver CR, Beaudet AL, Sly WS, and Valle D. (eds.), The Metabolic and Molecular Bases of Inherited Disease, Vol. 2, McGraw-Hill, New York 2693–2739.
5. Laidler P, Kowalski D, Silberring J (1991) Arylsulfatase A in serum from patients with cancer of various organs. Clin Chim Acta 204: 69-77.
6. Wojczyk B (1986) Lysosomal arylsulfatases A and B from horse blood leukocytes: purification and physico-chemical properties. Biol Cell 57: 147-152.
7. Draper RK, Fiskum GM, Edmond J (1976) Purification, molecular weight, amino acid, and subunit composition of arylsulfatase A from human liver. Arch Biochem Biophys 177: 525-538.
8. Valayannopoulos V, Nicely H, Harmatz P, Turbeville S (2010) Mucopolysaccharidosis VI. Orphanet J Rare Dis 5: 5.
9. Morgan LR Jr, Samuels MS, Thomas W, Krementz ET, Meeker W (1975) Arylsulfatse B in colorectal cancer. Cancer 36: 2337-2345.
10. Morgan LR, Reehlmann NJ, Maddux B, Rodriguez L, Gidman G, et al. (1971) Arylsulphatase activity of human lung, liver and kidney neoplasms. Clin Chim Acta 32: 316-320.
11. Bhattacharyya S, Kotlo K, Danziger R, Tobacman JK (2010) Arylsulfatase B regulates interaction of chondroitin-4-sulfate and kininogen in renal epithelial cells. Biochim Biophys Acta 1802: 472-477.
12. Thomas SB, Rose FA (1976) The intracellular distribution of arylsulphatase C and oestrogen sulphatase activities in rat kidney and spleen. Biochem J 158: 631-633.
13. Dzialoszynski LM, Gniot-Szulzycka J (1967) The clinical aspects of arylsulphatase activity. Clin Chim Acta 15: 381-386.
14. Gasa S, Makita A, Kameya T, Kodama T, Koide T, et al. (1981) Arylsulfatases of human-lung tumors transplanted into athymic mice. Cancer-associated modification of arylsulfatase B variant. Eur J Biochem 116: 497-503.

15. Gasa S, Makita A (1983) Phosphorylation on protein and carbohydrate moieties of a lysosomal arylsulfatase B variant in human lung cancer transplanted into athymic mice. J Biol Chem 258: 5034-5039.
16. Nakamura M, Gasa S, Makita A (1984) Arylsulfatase a from normal human lung and lung tumors showed different patterns of microheterogeneity. J Biochem 96: 207-213.
17. Gasa S, Balbaa M, Nakamura M, Yonemori H, Makita A (1987) Phosphorylation of human lysosomal arylsulfatase B by cAMP-dependent protein kinase. Different sites of phosphorylation between normal and cancer tissues. J Biol Chem 262: 1230-1238.
18. Balbaa M, Gasa S, Makita A (1988) Alterations of protein kinase isozymes in transplantable human lung cancer with special reference to the phosphorylation of arylsulfatase B. Biochem Biophys Res Commun 150: 163-169.
19. Balbaa M, El-Kersh M, Mansour H, Yacout G, Ismail M, et al. (2004) Activity of some hepatic enzymes in schistosomiasis and concomitant alteration of arylsulfatase B. J Biochem Mol Biol 37: 223-228.