

Resolution of the anti-coagulant property of sulfated glycosaminoglycan from the cephalothorax of white leg shrimp (*Penaeus vannamei*) - Vincent S Arnold - Centro Escolar University

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In this study, the anticoagulant property of Sulfated Glycosaminoglycan was evaluated using Plasma Recalcification Test. Extraction of the Sulfated Glycosaminoglycan from the White Leg Shrimp was performed by defatting the sample with acetone. The defatted sample was treated with 0.4M Sodium Sulfate and Aluminum disulfate crystal to collect the supernatant. The supernatant was treated with 90% ethanol. The mixture was centrifuged using a refrigerated centrifuge at 8000 rpm for 3 minutes and the collected precipitate was washed using absolute ethanol. The Sulfated Glycosaminoglycan was tested using Plasma Recalcification Test. The results in the said test showed that at 30ug/mL was significant and at 60ug/mL and 90ug/mL were very significant. The Sulfated Glycosaminoglycan from the White Leg Shrimp exhibited an anticoagulant property. Dermatan sulfates and heparin, almost like the mammalian glycosaminoglycans, but with differences within the degree and position of sulfation were previously isolated from the body of the ascidian *Styela plicata* and *Ascidia nigra*. These differences produce profound effects on their anticoagulant properties. *S. plicata* dermatan sulfate composed by 2-O-sulfated alpha-L-iduronic acid and 4-O-sulfated N-acetyl-beta-D-galactosamine residues may be a potent anticoagulant thanks to a high heparin cofactor II activity. Surprisingly, it's a lower potency to stop thrombus formation on an experimental model and a lower bleeding effect in rats than the mammalian dermatan sulfate. In contrast, *A. nigra* dermatan sulfate, also enriched in 2-O-sulfated alpha-L-iduronic acid, but during this case sulfated at O-6 of the N-acetyl-beta-D-galactosamine units, has no in vitro or in vivo anticoagulant activity, does not prevent thrombus formation but shows a bleeding effect almost like the mammalian glycosaminoglycan. Ascidian heparin, composed by 2-O-sulfated alpha-L-iduronic acid, N- and 6-O-sulfated glucosamine (75%) and alpha-L-iduronic acid, N- and 6-O-sulfated glucosamine (25%) disaccharide units has an anticoagulant activity 10 times lower than the mammalian heparin, is about 20 times less potent in the inhibition of thrombin by antithrombin, but has the same heparin cofactor II activity as mammalian heparin

Cardiovascular diseases are the number one cause of death in the world according to the World Health Organization. In 2012, 7.4 million deaths were reported due to coronary heart diseases and 6.7 million were due to stroke (WHO 2016). In the Philippines, the leading cause of mortality is heart disease. In 2009, 100,908 thousand deaths reported with the rate of 109% (DOH 2013). The most important behavioral risk factors of heart condition and stroke are unhealthy diet, physical inactivity, tobacco use and harmful use of alcohol. The effects of behavioral risk factors may show up in individuals as raised vital sign, raised blood sugar, raised blood lipids, and overweight and obesity. These "intermediate risks factors" are often measured in medical care facilities and indicate an increased risk of developing an attack, stroke, coronary failure and other complications. Cessation of tobacco use, reduction of salt within the diet, consuming fruits and vegetables, regular physical activity and avoiding harmful use of alcohol are shown to scale back the danger of cardiovascular disease. In addition, drug treatment of diabetes, hypertension and high blood lipids could also be necessary to scale back cardiovascular risk and stop heart attacks and strokes. Health policies that make conducive environments for creating healthy choices affordable and available are essential for motivating people to adopt and sustain healthy behavior. There also is variety of underlying determinants of CVDs or "the causes of the causes". These are a mirrored image of the main forces driving social, economic and cultural change – globalization, urbanization and population ageing. Other determinants of CVDs include poverty, stress and hereditary factors.