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Structural characterization and biological implications of sulfated N-glycans in a serine protease from the neotropical moth Hylesia metabus (Cramer [1775]) (Lepidoptera: Saturniidae)

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Contact with the urticating setae from the abdomen of adult females of the neo-tropical moth Hylesia metabus gives rise to an urticating dermatitis, characterized by intense pruritus, generalized malaise and occasionally ocular lesions (lepidopterism). The setae contain a pro-inflammatory glycosylated protease homologous to other S1A serine proteases of insects. Deglycosylation with PNGase F in the presence of a buffer prepared with 40% H_2 18O allowed the assignment of an N-glycosylation site. Five main paucimannosidic N-glycans were identified, three of which were exclusively α(1-6)-fucosylated at the proximal GlcNAc. A considerable portion of these N-glycans are anionic species sulfated on either the 4- or the 6-position of the α(1-6)-mannose residue of the core. The application of chemically and enzymatically modified variants of the toxin in an animal model in guinea pigs showed that the pro-inflammatory and immunological reactions, e.g., disseminated fibrin deposition and activation of neutrophils are due to the presence of sulfate-linked groups and not on disulfide bonds as demonstrated by the reduction and S-alkylation of the toxin. On the other hand, the hemorrhagic vascular lesions observed are attributed to the proteolytic activity of the toxin. Thus, N-glycan sulfation may constitute a defense mechanism against predators.

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Hyaluronan reverses Imatinib dependent-senescence in chronic myeloid leukemia cell lines

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Chronic myeloid leukemia (CML) is a myeloproliferative syndrome characterized by the presence of the Philadelphia chromosome which encodes a constitutively activated tyrosine kinase *(BCR-ABL)*. The first line treatment for CML consists on *BCR-ABL* inhibitors such as Imatinib. Nevertheless, such treatment may lead to the selection of resistant cells. Hyaluronan (HA) is the main glycosaminoglycan of the extracellular matrix which is involved in tumor progression and multidrug resistance. We have previously demonstrated that HA induce cell proliferation in CML cells. However, the effect of HA on Imatinib therapy remains unknown. The aim of this work was to determine whether HA is able to reverse the anti-proliferative, pro-apoptotic and pro-senescent effect of Imatinib in human CML cell lines. For this purpose, K562 (ATCC) and Kv562 (multidrug resistant derived) were exposed to HA, Imatinib or a combination of both. Cell proliferation was evaluated by ^{3}H-T uptake; apoptosis was determined by annexin-V-PE-7AAD kit, SubG1 peak and DNA fragmentation; senescence was evaluated by senescence-associated β-galactosidase activity (SA-β-gal) and senescence-associated heterochromatin foci (SAHF) presence. We showed that HA reversed the anti-proliferative effect of Imatinib without modifying Imatinib-dependent apoptosis in both cell lines. However, the combination of HA and Imatinib decreased SA-β-gal activity and SAHF presence when compared to Imatinib alone. Moreover, Imatinib treatment decreased pAkt/Akt ratio in both cell lines and reduced the pERK/ERK ratio only in K562 cells, while the addition of HA reversed such effects. We conclude that HA reverses de anti-proliferative and pro-senescent effect of Imatinib without modifying apoptosis.

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