

## Evaluation of the minimal core in Galactofuranose structure capable to stimulate the immune response

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Carbohydrate-containing molecules are distributed at external layers of all organisms, being involved in many functions related to specific interactions of the exocellular environment. Recently was observed that a human lectin that binds to galactofuranose stimulated the response of innate immune. In order to understand the role of the galactofuranosyl epitopes in the macrophages phagocytic activation we produced some galactofuranosyl derivatives to study *in vitro* their immunostimulant activity. Commercial galactose was converted in Me- $\beta$ -Gal/Me- $\alpha$ -Gal 7:3 ratio (0.5% w/w MeOH-H<sub>2</sub>SO<sub>4</sub>) and these anomers were purified by column chromatography packed with silica-gel 60 G and monitored on TLC by orcinol-H<sub>2</sub>SO<sub>4</sub>. Pure  $\beta$ - and  $\alpha$ -galactofuranosides were obtained (GFB-Me and GFA-Me, respectively). Octyl-galactofuranosides were prepared from the Me-Gal mixture and the separation was performed as described above, giving pure  $\beta$ - and  $\alpha$ -Octyl-galactofuranosides (GFB-O and GFA-O, respectively). Pure GFB-Me and GFA-Me were converted to isopropylidene derivatives, resulting in specific 5,6 hydroxyl blocked isopropylidenes (GFB-5,6-IPP and GFA-5,6-IPP). The purity and authenticity of all galactofuranosides were confirmed by NMR and ESI-MS analysis. Phagocytic capacity was performed with resident macrophages by zymosan incorporation. GFB-Me and GFB-O had a stimulant effect at 5ug /mL with an enhancement of 35.12 % $\pm$  0.06 SD and 11.90%  $\pm$  0.11 SD, respectively, GFA-Me and GFA-O had low response. The hydroxyl blocked galactofuranosides GFA-5,6-IPP and GFB-5,6-IPP had negative values relative to the control group -4.17% $\pm$  0.10 SD and -34.22% $\pm$  0.07 SD, respectively. These results suggest that the freely 5 and 6 hydroxyls and the  $\beta$  configuration are essential characteristics for the galactofuranose unit to macrophage phagocytic activation.

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