b_2GPI PLASMA LEVEL INCREASE IN RESPONSE TO BACTERIAL TRANSLOCATION THROUGH INTESTINAL MUCOSA IS HIGHER THAN AFTER INTRAVENOUS INJECTION

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β_2GPI is a circulating protein produced mainly in liver and intestines. We have previously shown that both intravenous sepsis and bacterial translocation (BT) associated intestinal infection induce hepatic β_2GPI expression. Local damage associated production lead us to hypothesize a mucosal contribution for β_2GPI plasma levels in BT. In order to prove this point, we determined β_2GPI in plasma of Wistar rats submitted to controlled experimental sepsis or BT. Briefly, sepsis was induced by endovenous inoculation of E. coli R-6, 10^7 or 10^9 CFU/mL/100g. BT was produced by oroduodenal catheterization and confinment of E. coli R-6 (10^{10} CFU/10mL/100g) in the ligated small intestine. Control groups received vehicle. Blood samples were obtained after 3h. Plasma β_2GPI levels were determined by indirect ELISA. Tissues were collected under sterile conditions for quantitative microbiology. Increased β_2GPI plasma levels were proportional to infection intensity and clinical prognosis in experimental sepsis. However, an infection route dependence for β_2GPI *in vivo* expression was also unequivocally demonstrated. Accordingly, low intensity BT infection promotes higher plasma β_2GPI levels than intravenous infection. This apparent paradox highlights the intestinal synthesis contribution to β_2GPI plasma levels and suggests a dynamic association with intestine immune system physiology. (CNPq)