Erratum

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In Eur. Surg. Volume 39, Supplement No. 218, Abstract 02, one author was missing. The correct abstract is shown below. The publishers do apologize for this error.

02 Lipocalin-2 – a novel inflammatory regulator during ischemia and reperfusion and acute allograft rejection

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Background: The main focus of this work was to analyze the possible implication of Lipocalin-2 (Lcn-2) upregulation for the course of ischemia/reperfusion (IR) during heart transplantation as well as its role in acute allograft rejection. Lcn-2 expression was also analyzed in terms of acute rejection following liver transplantation in a clinical setting.

Methods: Male inbred C3H and C57BL/6 mice as well as the Lcn-2−/− mouse were used in our transplantation experiments. Western blot, RT-PCR and immunohistochemistry and TUNEL assay were performed to determine Lcn-2 expression and apoptosis in the graft. Cardiac rejection was classified following the ISHLT score (0–3). Additionally, Lcn-2 protein expression was analyzed in the serum of recipients at day 0–15 following liver transplantation using ELISA technology.

Results: Infiltrating polymorphonuclear cells (PMN) were the major contributors to Lcn-2 expression during IR peaking 24 h after reperfusion. The number of infiltrating PMN was significantly reduced in Lcn-2−/− recipients (by 79% at 12 h [$p < 0.01$]). No difference was observed in the apoptotic rate between wildtype and Lcn-2−/− donors in our murine heart transplantation model. Concomitant upregulation of Lcn-2 in the serum of liver transplant recipients could be observed during acute rejection episodes in a timely dependent course (3 to 7-fold).

Conclusions: Our data suggest a chemoattractant function of increased Lcn-2 expression in the transplanted heart due to infiltrating PMN. Lcn-2 is a novel inflammatory regulator upregulated during IR and is proposed an early biomarker for acute graft rejection. Our observations shed light on a possible function of Lcn-2 in the recruitment of PMN to the site of IR and identify possible targets for therapeutic intervention.

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