

P005 Lack of hemopexin dampens mercury-induced autoimmune response in mice
Fagoonee S.¹, Tolosano E.^{1,2}, Giovarelli M.^{2,3}, Stoltenberg M.⁴, Silengo L.^{1,2}, Altruda F.^{1,2}, Camussi G.⁵, Bussolati B.⁵

¹Department of Genetics, Biology, and Biochemistry, ³Department of Medicine and Experimental Oncology, Section of Pathology, University of Turin, Italy. ²Experimental Medicine Research Center, ⁵Department of Internal Medicine, San Giovanni Battista Hospital, Turin, Italy. ⁴Department of Neurobiology, Institute of Anatomy, University of Aarhus, Denmark

Mercury-induced autoimmunity in mice is characterised by immunostimulation, anti-nuclear antibodies (ANA) and systemic immune-complex (IC) deposits. We explored the role of the heme-binding protein, hemopexin, in regulating mercury-induced autoimmunity. Wild-type and hemopexin-null 129Sv mice were injected subcutaneously with 1.6 mg/kg of mercuric-chloride (HgCl_2) every 3 days for 4 weeks. After treatment, Hemopexin-null mice had a lower titer of serum ANA and significantly reduced IC deposits in kidney, skin, heart and muscle compared to wildtype mice. No significant differences in T_H1/T_H2 responses were seen between the two genotypes but Hemopexin-null mice had significantly increased spleen T_H17 versus non-treated ones. Moreover, CD11b-expressing splenocytes increased in wildtype and decreased in hemopexin-null mice. Hemopexin did not control the distribution of mercury to the different tissues, as observed by similar autometallographic silver-enhancement staining patterns in both genotypes. We here report that the absence of hemopexin dampens the HgCl_2 -triggered production of autoimmune antibodies and renal immune deposits in this model. Lack of anti-inflammatory molecule, hemopexin, can down-regulate the autoimmune component in 129Sv mice.