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The Impact of Intestinal Ischaemia-Reperfusion Injury upon the Biological Function of Mesenteric Lymph

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Abstract : Intestinal ischaemia-reperfusion injury drives systemic inflammation and organ failure following trauma/haemorrhagic shock (T/HS), through the release of pro-inflammatory mediators into the mesenteric lymph (ML). However, changes in the biological function of ML are not fully understood, and therefore, a specific model of intestinal ischaemia-reperfusion injury is required to obtain ML for the study of its biological function upon inflammatory cells. ML obtained from a model of intestinal ischaemia-reperfusion injury was used to assess biological function upon inflammatory cells and investigate changes in the biological function of individual ML components. An additional model was used to determine the effect of vagal nerve stimulation (VNS) upon biological function. Rat ML was obtained by mesenteric lymphatic duct cannulation before and after occlusion of the superior mesenteric artery (SMAO). ML was incubated with human polymorphonuclear neutrophils (PMNs), monocytes and lymphocytes, and the biological function of these cells was assessed. ML was then separated into supernatant, exosome and micro-vesicle components, and biological activity was compared in monocytes. A model with an additional VNS phase was developed, in which the right cervical vagal nerve was exposed and stimulated, and ML collected for comparison of biological function with the conventional model. The biological function of ML was altered by intestinal ischaemia-reperfusion injury, increasing PMN activation, monocyte activation, and lymphocyte apoptosis. Increased monocyte activation was only induced by the exosome component of ML, with no significant changes induced by the supernatant or micro-vesicle components. VNS partially attenuated monocyte activation, but no attenuation of PMN activation was observed. Intestinal ischaemia-reperfusion injury induces changes in the biological function of ML upon both innate and adaptive inflammatory cells, supporting the role of intestinal ischaemia-reperfusion injury in driving systemic inflammation following T/HS. The exosome component of ML appears to be critical to the transport of pro-inflammatory mediators in ML. VNS partially attenuates changes in innate inflammatory cell biological activity observed, presenting possibilities for future novel treatment development in multiple organ failure patients.

Keywords : exosomes, inflammation, intestinal ischaemia, mesenteric lymph, vagal stimulation
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