modulated in the monocyte/macrophage lineage, and their littermate controls by injecting 1ml 4% thioglycollate to the peritoneal cavity. The composition of the cells infiltrating the peritoneum was analysed at 0, 6 and 24 hours post-injection by multi-colour flow cytometry. We also measured the cytokine content of the lavage fluids via the BDTM Cytometric Bead Array (CBA).

Results At 6 and 24 hours post-injection we found a significant increase in the infiltration of monocytes to the peritoneal cavity in *trb-1* KO animals compared to littermate controls, while the *trb-1* over-expressing monocytes refrained from entering the peritoneum. We quantified the amount of IL-1a/b, TNFa, MIP1a/b and KC in the samples and found no significant difference in the production of any of these cytokines.

Conclusions *Trb-1* is involved in the regulation of monocyte migration under inflammatory conditions and therefore may play a significant role in the early steps of monocyte recruitment. Via this mechanism, *trb-1* may contribute to the development of chronic inflammation and atherosclerosis. Further investigations will determine whether differences in the expression of leukocyte cell adhesion molecules (CAM) or other signalling abnormalities are responsible for the altered migration pattern of *trb-1* KO and *trb-1* transgenic monocytes.

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TRIBBLES-1 CONTRIBUTES TO MONOCYTE MIGRATION IN EXPERIMENTAL PERITONITIS

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doi:10.1136/heartjnl-2013-304019.235

Introduction Mitogen activated protein kinase (MAPK) cascades are involved in the regulation of several cellular responses including migration or proliferation. We have shown previously that *tribbles-1* (*trb-1*), a member of the *tribbles* family of adaptor/scaffold proteins controls vascular smooth muscle proliferation and chemotaxis via a direct interaction with the JNK activator kinase, MKK4. However, a regulatory role in monocyte/macrophage function for members of the *tribbles* family has also been reported. With the current work, we aimed to gain a better understanding of the early steps of monocyte recruitment under inflammatory conditions, and in particular, the contribution of *tribbles-1* to these processes.

Methods We induced experimental peritonitis in *trb-1* KO and *trb-1* transgenic mice, where the expression of this gene was specifically