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## PAR-2-induced colonic inflammation and does not depend upon paracellular permeability and lymphocyte infiltration

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Activation of colonic PAR-2 receptor provokes colonic inflammation and increases mucosal paracellular permeability in mice. The mechanism of inflammation is under debate and could be a consequence of tight junction opening with passage of exogenous pathogens into the lamina propria

**Methods** : The present study aimed to further characterise PAR-2-induced effects. To investigate: (i) a dose-effect relationship between the peptide agonist (SLIGRL) concentration and paracellular permeability, four groups received an intracolonic administration of SLIGRL at the dose of 5, 10 or 100 µg/mouse; (ii) a possible relationship between paracellular permeability and mucosal inflammation, four groups received the MLCK inhibitor ML-7 (2mg/kg, i.p.) or vehicle and SLIGRL (100 µg/mice) or vehicle; (iii) a possible cause and effect relationship between TCD4<sup>+</sup> cells and paracellular permeability, two groups of SCID mice received SLIGRL (100 µg/mice) or vehicle. For all groups, colonic permeability was assessed with an intracolonic infusion of 51Cr-EDTA for 4h after SLIGRL or vehicle administration, followed by the determination of 51Cr-EDTA radioactivity in blood (cpm/ml). Pieces of colon were taken for myeloperoxidase assay (MPO; U/g of protein) and histologic examination (microscopic damage score, MDS). Values are expressed as mean ± SEM for each group.

**Results** : (i) At 10 and 100 µg SLIGRL increased paracellular permeability ( $903 \pm 102$  and  $1721 \pm 241$  vs.  $307 \pm 41$ ,  $p < 0.01$ ), MPO activity ( $239 \pm 14$  and  $638 \pm 198$  vs.  $49 \pm 5$ ,  $p < 0.05$ ) and MDS ( $4.8 \pm 0.1$  and  $5.8 \pm 0.60$  vs.  $0.8 \pm 0.06$ ,  $p < 0.01$ ). In contrast, at 5 µg SLIGRL increased paracellular permeability ( $307 \pm 41$  vs.  $754 \pm 78$ ,  $p < 0.05$ ) but did not affect MPO nor MDS. (ii) ML-7 prevented SLIGRL-induced increase in permeability ( $119.3 \pm 56.39$  vs.  $675.6 \pm 94.5$ ,  $p < 0.01$ ) but did not prevent MPO increase nor MDS increase. (iii) In SCID mice SLIGRL increased paracellular permeability ( $1685 \pm 257$  vs.  $939 \pm 173$ ,  $p < 0.05$ ), MPO activity ( $300 \pm 78$  vs.  $44 \pm 9$ ,  $p < 0.01$ ) and MDS ( $5.8 \pm 0.60$  vs.  $0.8 \pm 0.06$ ,  $p < 0.001$ ).

**Conclusion** : The SLIGRL-induced inflammation does not result from an increase of paracellular permeability linked to activation of MLCK and does not require the presence of mature lymphocytes. In addition, distinct pathways account for SLIGRL-induced inflammation and alterations of permeability observed for a non-inflammatory dose.

10<sup>th</sup> United European Gastroenterology week.

"UEGW 2002"

Geneva, Switzerland, 19-23 October 2002

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June 2002

10th United European Gastroenterology Week "UEGW 2002"  
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Richard Torriani  
Project Manager