

Sucralfate protects against acid-induced gastric mucosal barrier dysfunction

Tracy Hill¹, Karen Young², Duncan Lascelles², Anthony Blikslager²

¹The Royal (Dick) School of Veterinary Sciences, University of Edinburgh, Scotland, UK, ²Department of Clinical Sciences, North Carolina State University, Raleigh, NC, USA

Introduction

- Stress-related gastric mucosal disease (SRMD) occurs when critical illness disrupts gastric protective mechanisms.
- Acid suppressants have adverse events in critically ill people.
- Sucralfate is a gastroprotectant with fewer adverse events.

Hypothesis

Sucralfate would preserve barrier function in a canine *ex vivo* acid injury model.

Methods

- Canine gastric mucosa was kept alive *ex vivo* on a Ussing chamber and injured with acidic solution.
- Sucralfate was administered both with injury and immediately post-injury.
- Barrier function was assessed with: mannitol flux, transepithelial resistance, and histologic examination

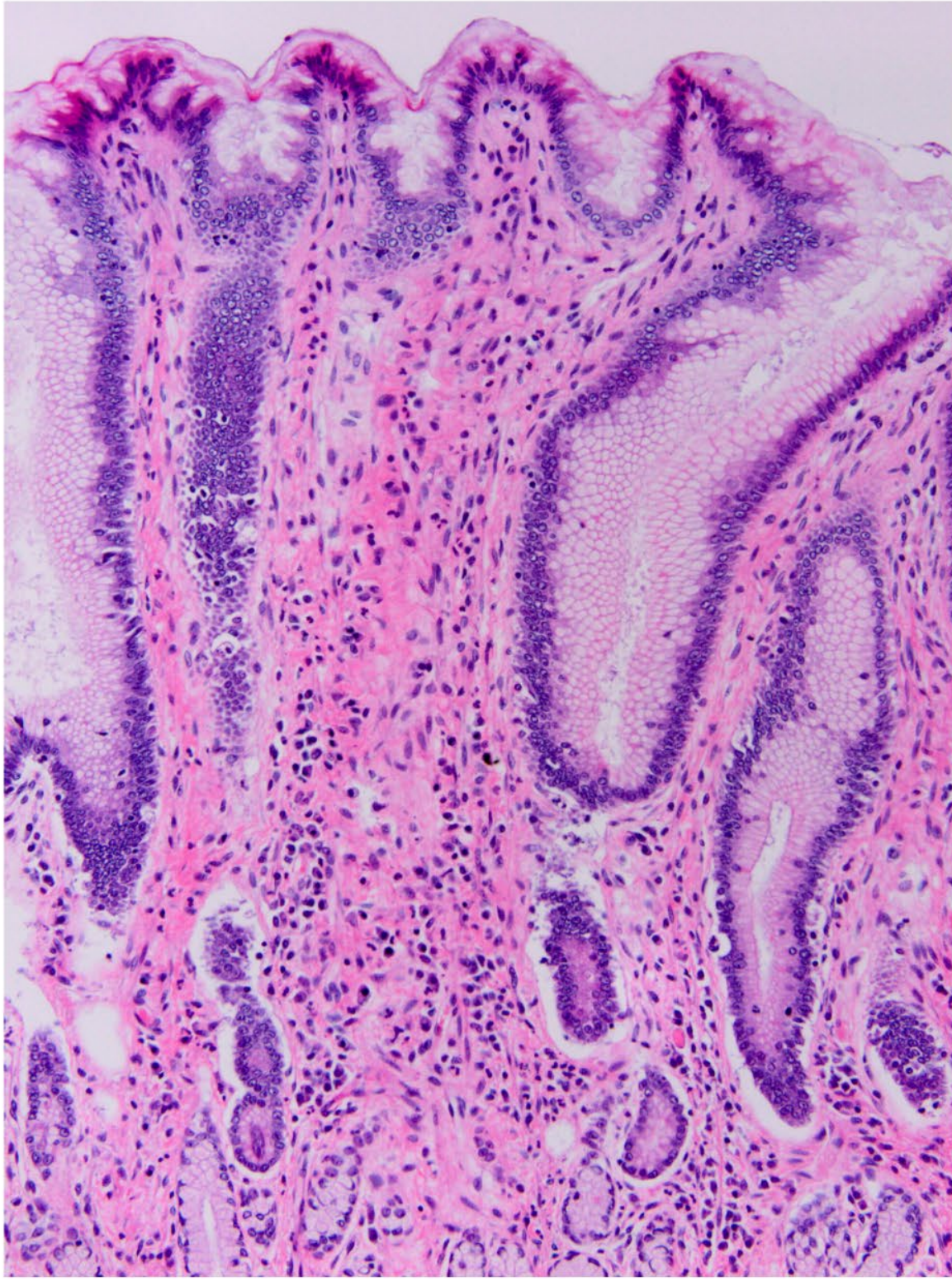
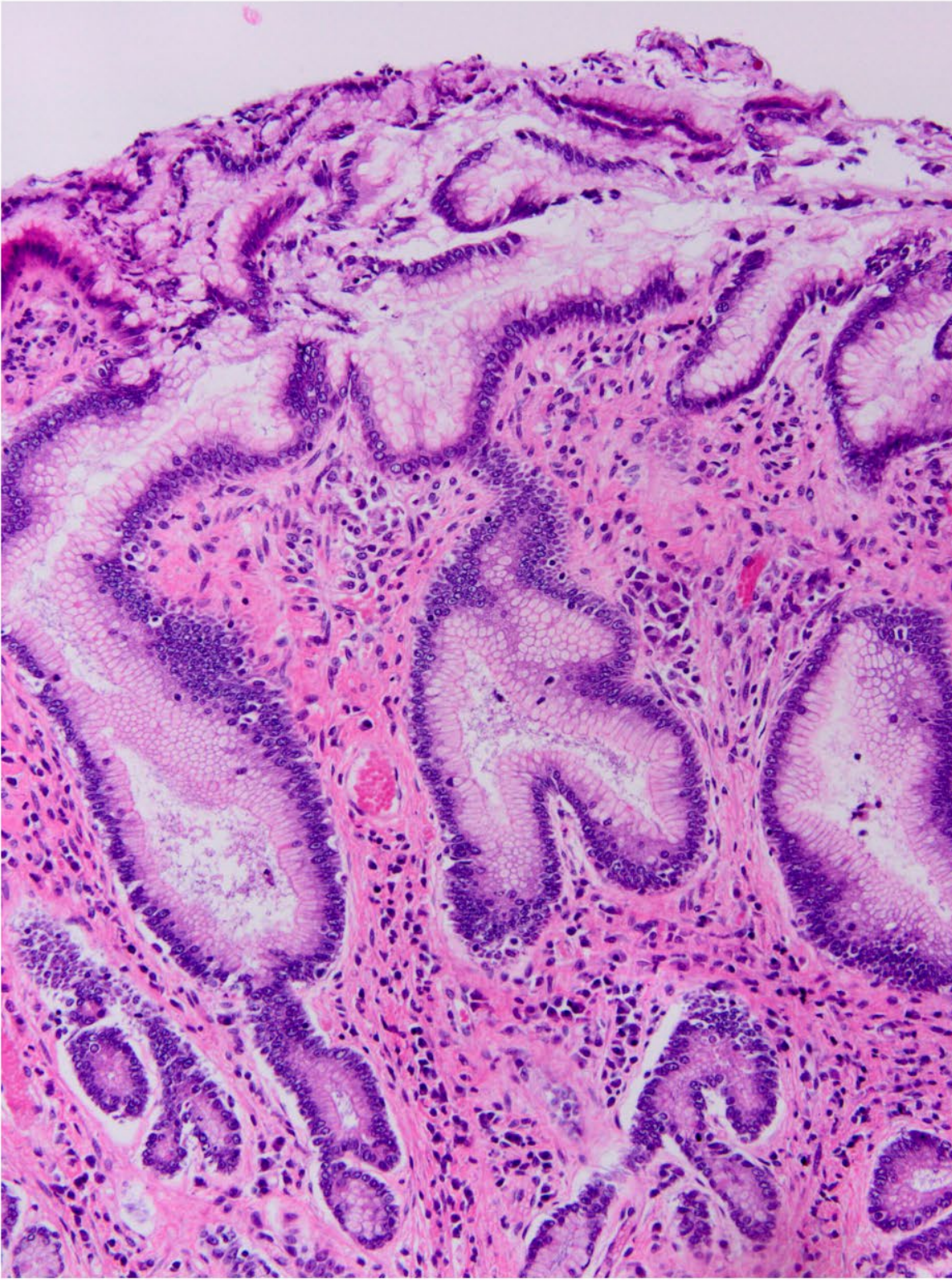
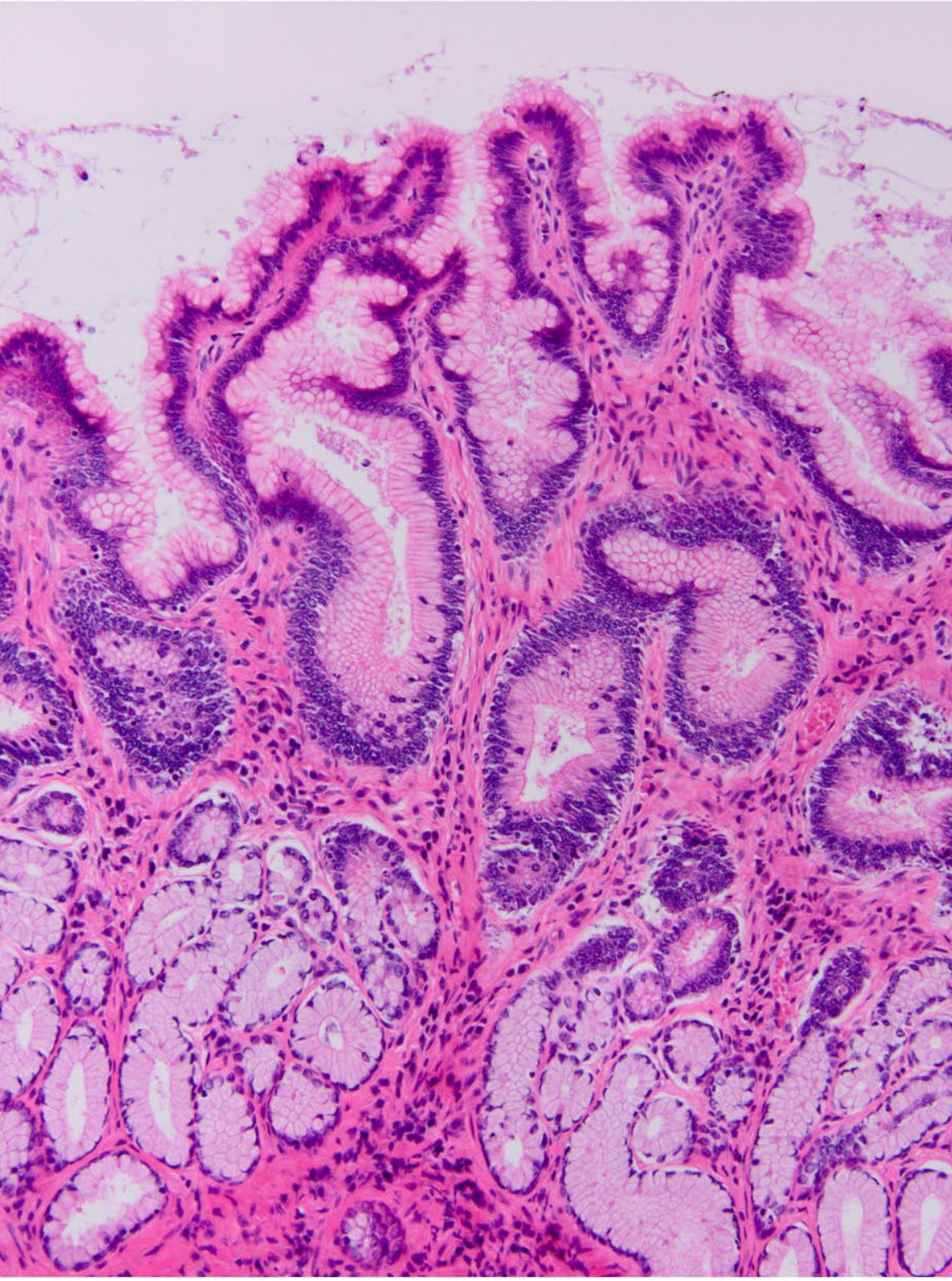
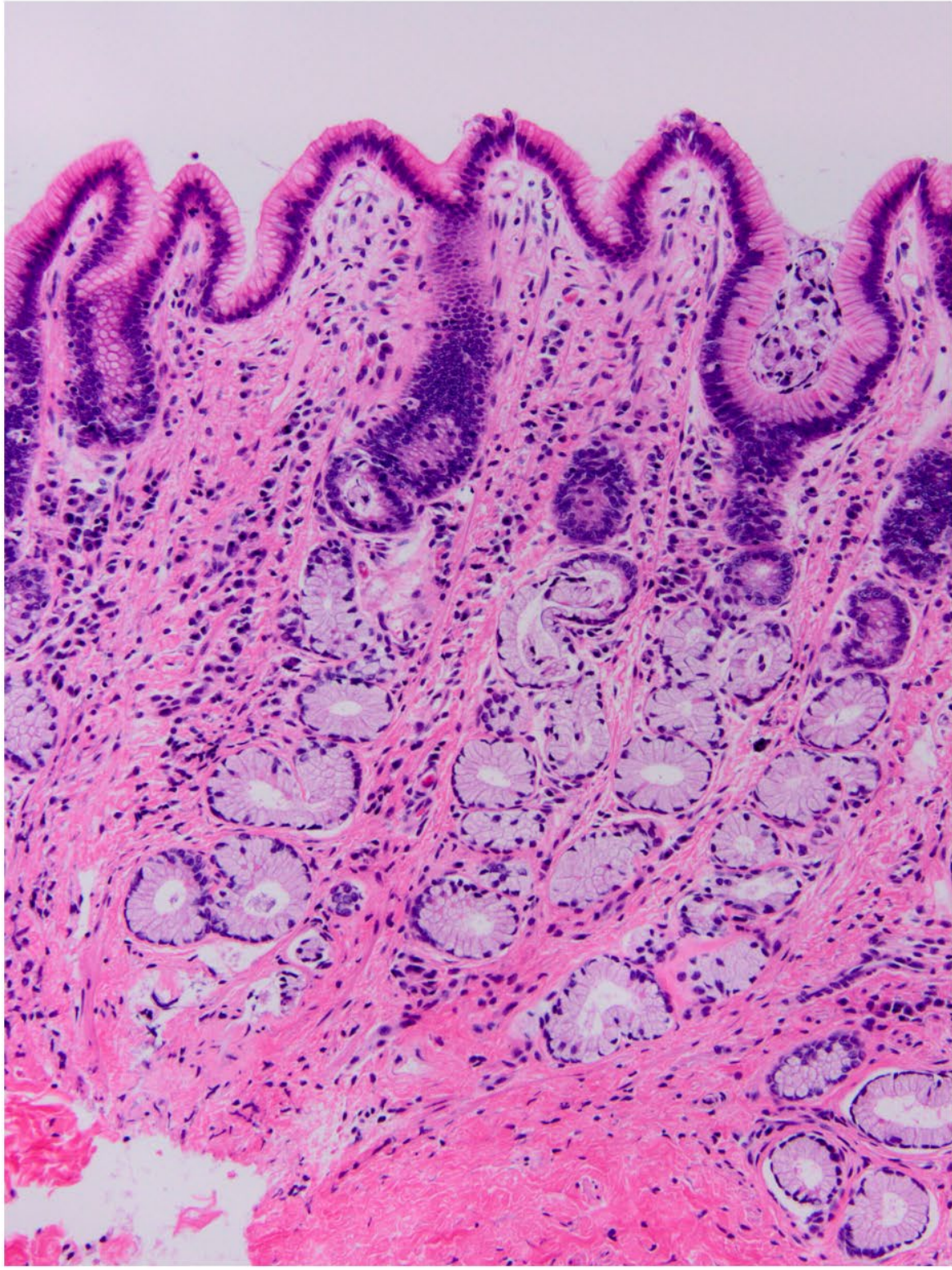
Results

- Sucralfate prevented acid-induced mucosal barrier dysfunction.
- It also increased recovery of barrier function after injury.

Discussion

- Sucralfate protects against and speeds recovery of mucosal barrier function after acid injury
- With its lower adverse event profile, sucralfate might be useful to prevent SRMD in people and dogs.

Sucralfate, an oral gastroprotectant, helped prevent and speed recovery from gastric injury.



BaselineControlAcid InjuredAcid + Sucralfate

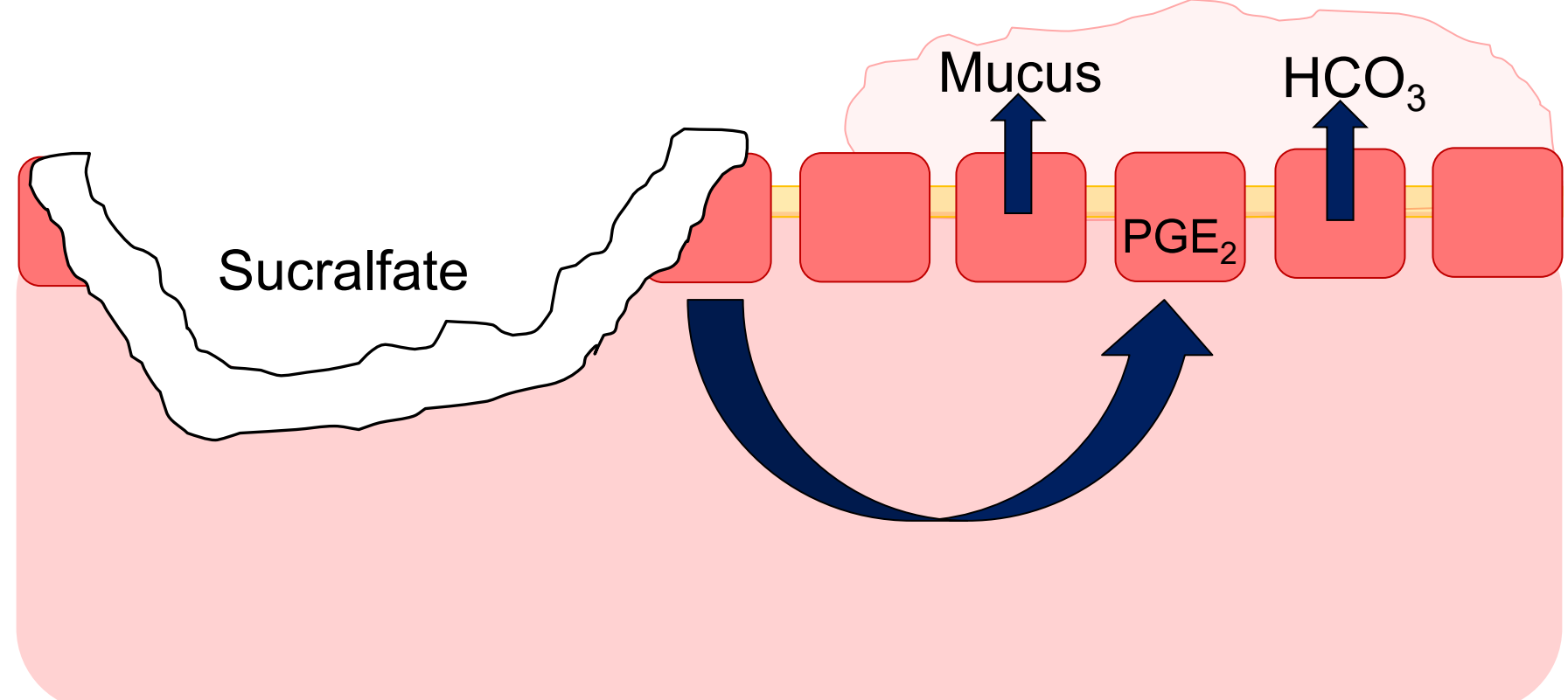


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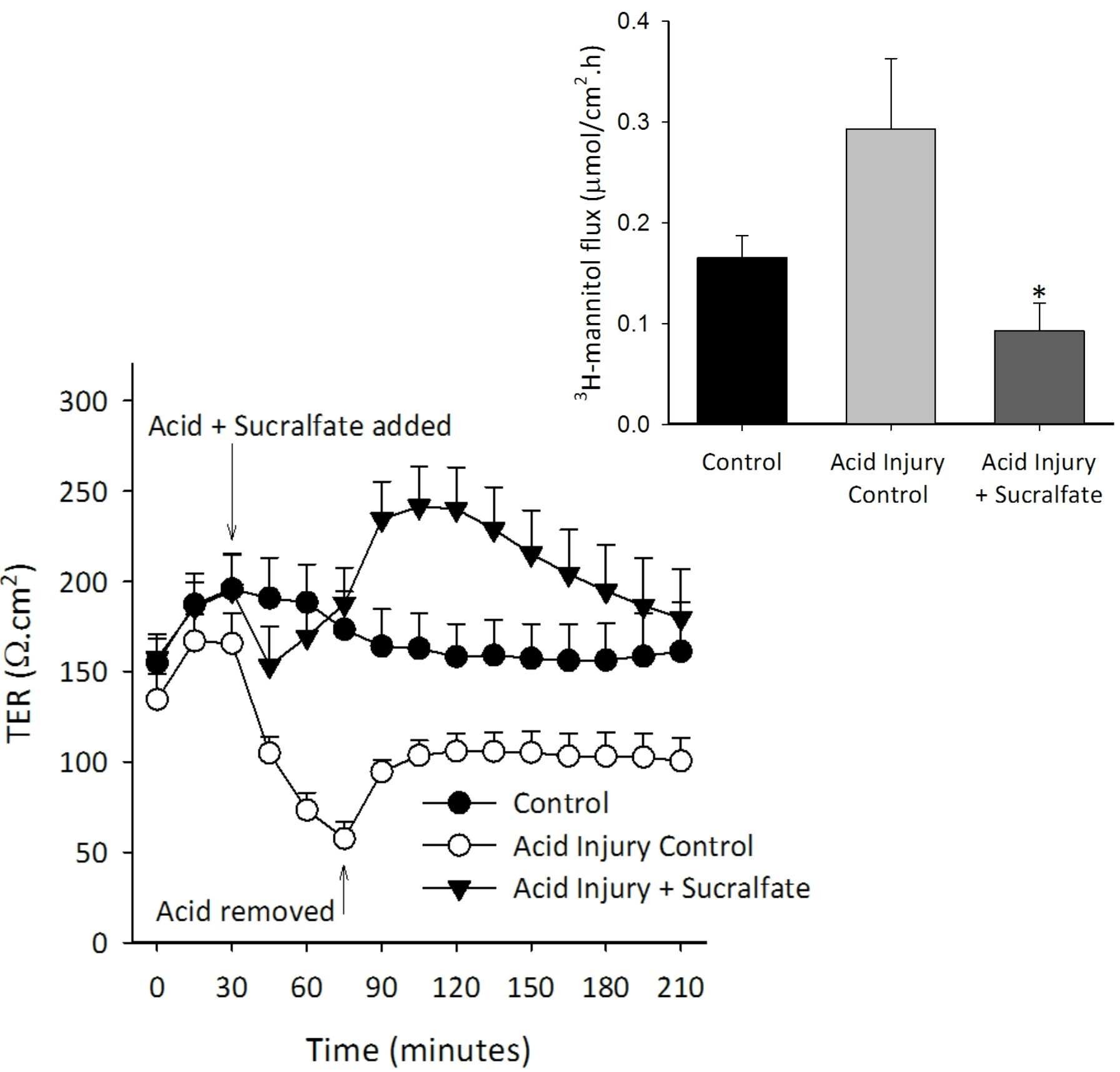
FINANCIAL DISCLOSURE/S:
No relevant financial conflicts exist.

UNLABELED/UNAPPROVED USES DISCLOSURE:
Sucralfate is not labeled for use in dogs.

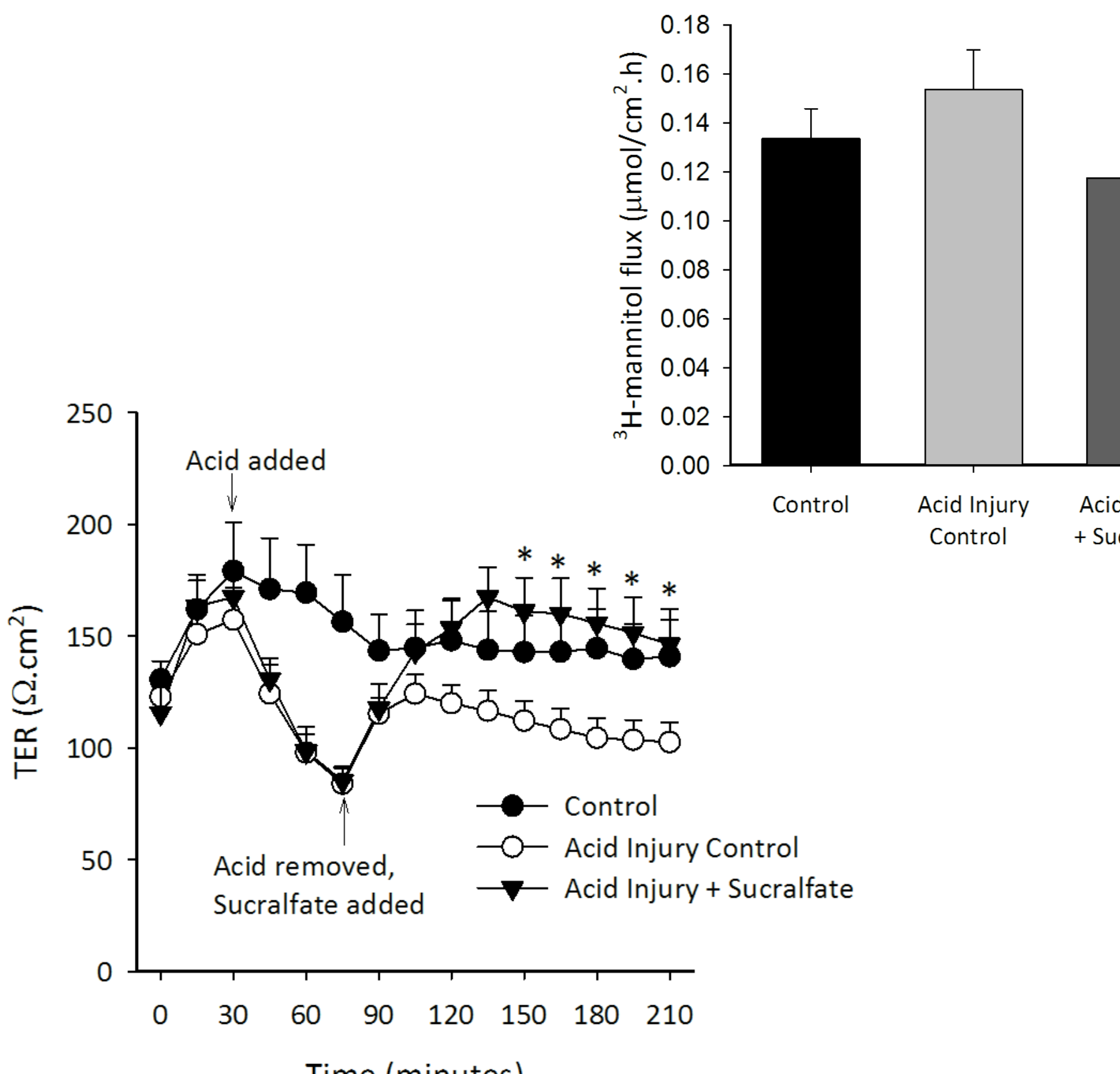
Extra Figures & Tables



Proposed mechanisms of protection of sucralfate.^{6,7}



Sucralfate prevents acid-induced mucosal barrier dysfunction. Figure 1. Transepithelial resistance decreases during acid injury and subsequently recovers but remains significantly lower than control (p=0.029). Sucralfate, applied at the time of injury, increases TER as compared to acid injury control (p<0.001) and is significantly higher than control non-injured tissue at 90-120 minutes (p=0.010). **Figure 2.** Sucralfate administration commensurate with acid injury leads to decreased paracellular flux of ³H-mannitol (p=0.008). N=10, values represent means±SE.



Sucralfate increases recovery of barrier function after acid injury. Figure 3. Sucralfate treatment subsequent to acid injury resulted in TER recovery to a greater degree than in acid-injured mucosa without treatment (* p<0.035). **Figure 4.** There was no significant effect of sucralfate applied subsequent to acid injury on ³H-mannitol flux (p=0.214). N=10, values represent means±SE.