ACVIM American College of Veterinary Internal Medicine | **ACVIM FORUM** Annual Conference

LOGO NAME SLOGAN HERE

Small Animal Internal Medicine

Sucralfate protects against acid-induced gastric mucosal barrier dysfunction

Tracy Hill[1], Karen Young[2], Duncan Lascelles[2], Anthony Blikslager[2]

[1]The Royal (Dick) School of Veterinary Sciences, University of Edinburgh, Scotland, UK, [2]Department 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

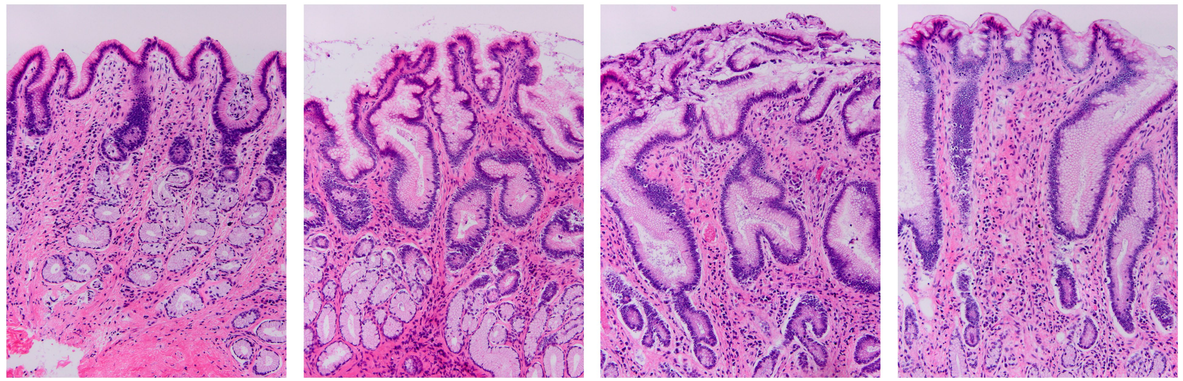
Results

- 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.

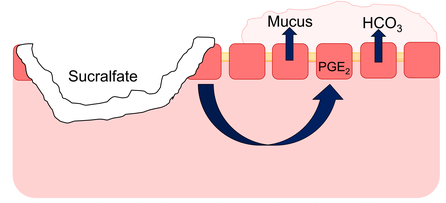


Scan to **Download all abstract details**

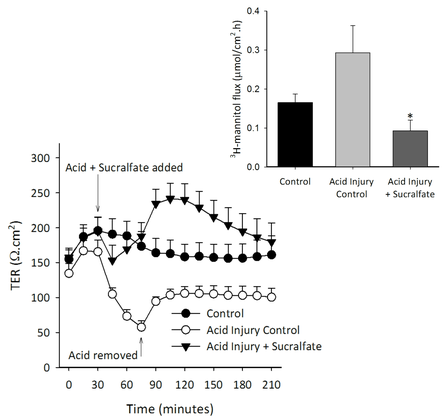
FINANCIAL DISCLOSURE/S:
No relevant financial conflicts exist.

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

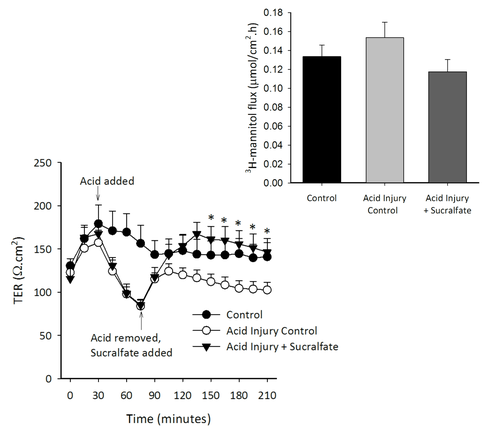
Extra Figures & Tables



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 ^{3}H-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 ^{3}H-mannitol flux (p=0.214). N=10, values represent means±SE.