

IDENTIFICATION OF POTENTIAL SKIN-DEGRADING CONSTITUENTS IN ILEOSTOMY DEJECTA

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Background

About 1 million people live with an ostomy in the United States – with 1000,000 to 300,000 ostomies performed annually¹. An **ostomy** is a surgically created opening in the abdominal wall (**stoma**), that attaches the distal small bowel (**ileostomy**) or large bowel (**colostomy**) to the skin surface.

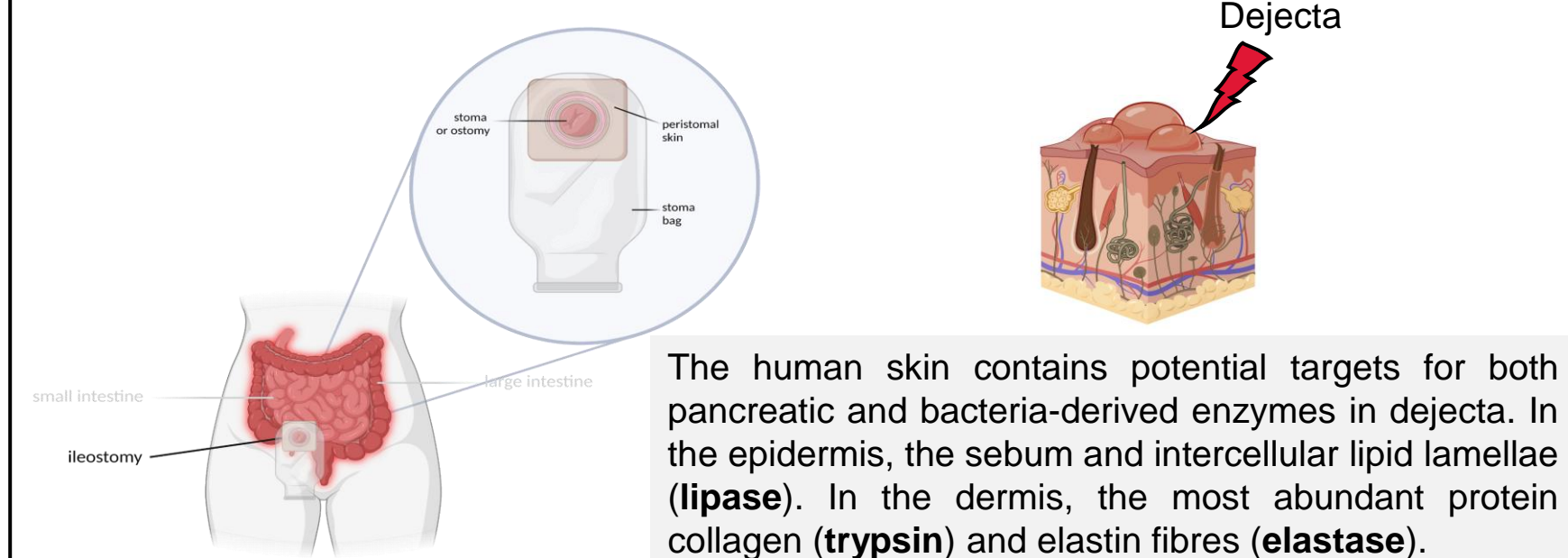
Peristomal skin complications (PSC), damage to skin around a stoma, commonly occur following an ileostomy and have been attributed to peristomal skin being exposed to the constituents in the semi-solid stool (**dejecta**)². It has been suggested that the skin-degrading effect of dejecta could be due to the interplay of dejecta constituents, especially pancreatic enzymes, gut bacteria and bile. However, the contribution of dejecta constituents to PSC formation has not been fully established³. Hence, there is a need to characterize the properties, constituents and cellular impact of ileostomy dejecta as a step towards developing more effective solutions to PSC. This knowledge will eventually improve the ileostomate's quality of life via informed development of improved stoma care products, practices and interventions.

Project Aim

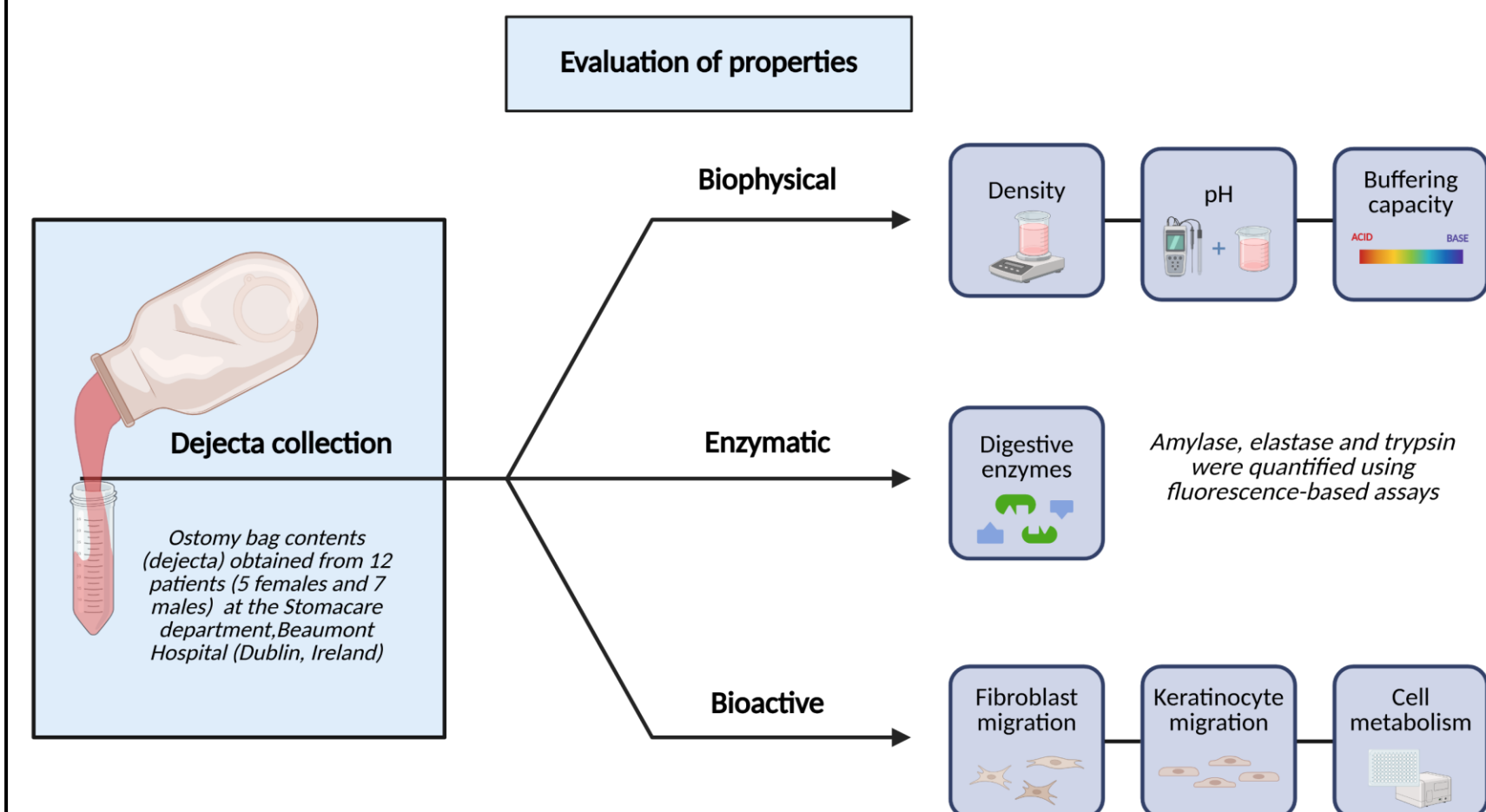
To characterize the biophysical and biological properties of ileostomy dejecta related to its possible contribution to PSC formation

Repeated exposure of peristomal skin to dejecta (in the event of leakage or spillage) is often accompanied by irritation, inflammation or infection around the stoma.

Dejecta consists of masticated food, bile, gut bacteria, digestive enzymes (e.g. amylase, elastase, lipase, trypsin), and additional enzymes. Dejecta could prove potentially deteriorating towards peristomal skin.



Methodology



Results

Heterogeneity observed in ileostomy dejecta profile amongst individuals

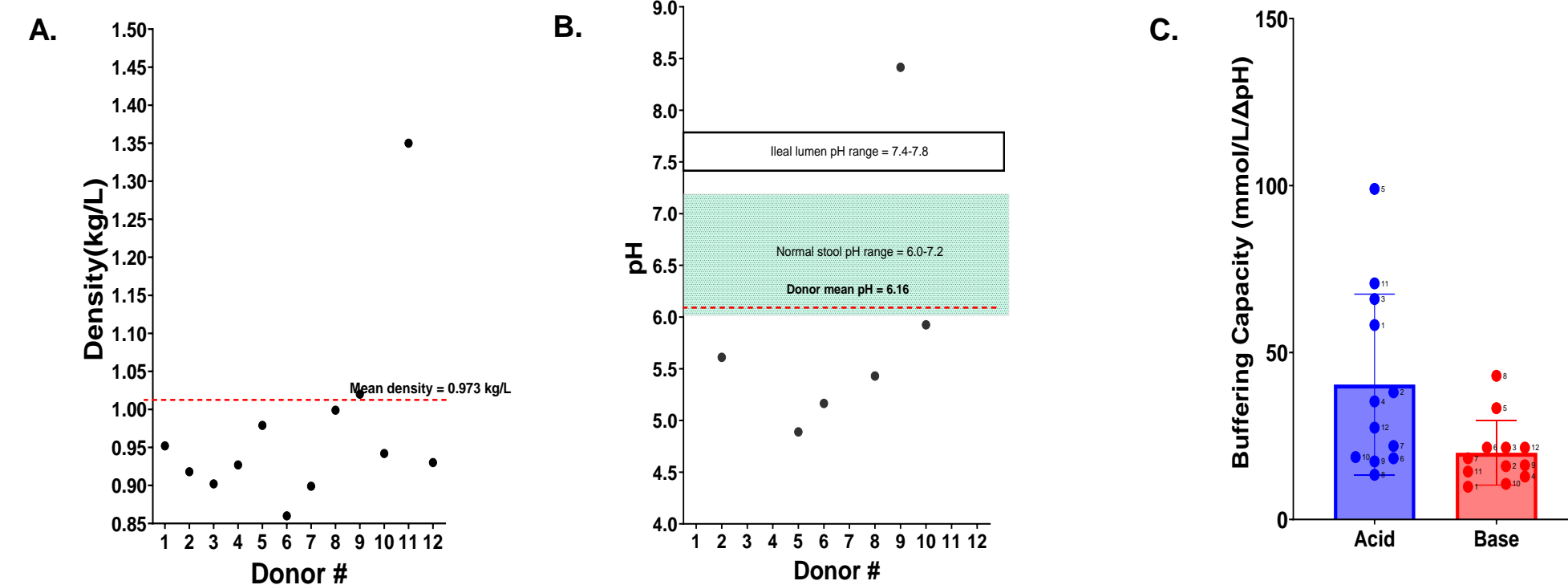


Figure 1 Biophysical properties of ileostomy dejecta. (A) Density, (B) pH and (C) buffering capacity (i.e., the ability to buffer controlled volumes of an acid or a base) of ileostomy dejecta from 12 individuals.

Significant protease activity was detected in ileostomy dejecta

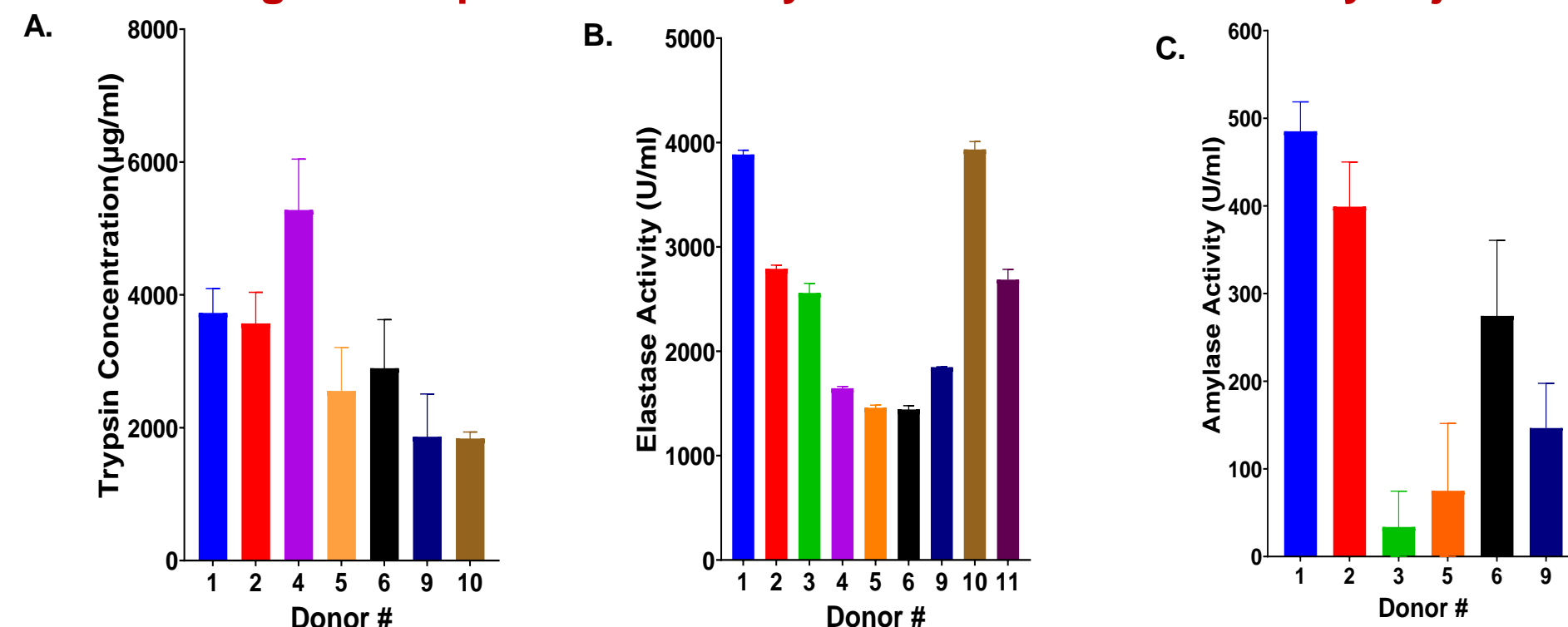


Figure 2 Digestive enzymes are active constituents in ileostomy dejecta. (A) Trypsin concentration, (B) elastase concentration, and (C) amylase activity of ileostomy dejecta supernatant from 12 individuals. Included donors are represented by solid bars with means \pm SD. Donors with values outside the standard curve range (e.g., Donor 3 from the trypsin concentration graph) were excluded.

Ileostomy dejecta significantly reduced cell migration *in vitro*

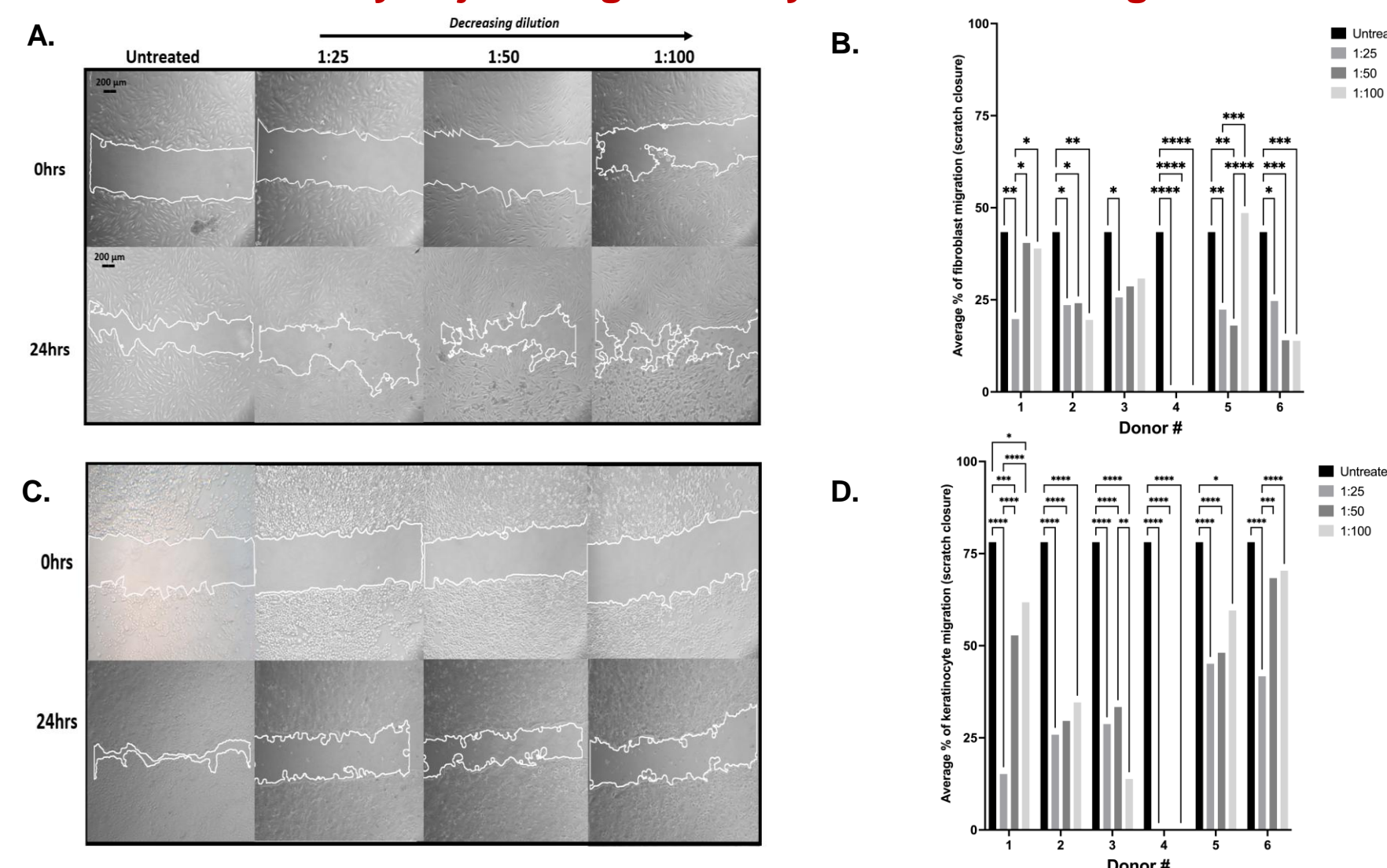


Figure 3 The effect of different ileostomy dejecta supernatant concentrations on cell migration over 24 hours. (A) Representative images of the migratory ability of human fibroblasts and (B) the corresponding scratch closure (%) after 24 hours; (C) representative images of the migratory ability of human keratinocytes and (D) the corresponding scratch closure (%) after 24 hours - upon exposure to different ileostomy dejecta supernatant concentrations compared to the untreated group. Reduced migratory ability observed following exposure to the 1:25 ileostomy dejecta supernatant dilution. Additionally, 1:25 diluted dejecta supernatant significantly reduced both fibroblast and keratinocyte viability *in vitro* (data not shown).

Conclusions and Future Directions

Key Takeaways:

- ❑ The difference in pH between ileostomy dejecta (pH 4.89-8.41) and the skin mantle pH range (4.1-5.8) could negatively affect peristomal skin flora and barrier function, eventually contributing to PSC formation.
- ❑ In this study, the ileostomy dejecta samples were resistant to pH changes in the presence of a strong acid – this could support the likelihood of metabolically active enzymes in ileostomy dejecta when in direct contact with peristomal skin.
- ❑ Proteolytic activity in ileostomy dejecta was confirmed and could partly explain the peristomal skin damage in ileostomates.
- ❑ Exposure to ileostomy dejecta supernatant significantly reduced the migration of keratinocytes and fibroblasts.

Conclusively, this study identifies possible mechanisms involved in the skin-damaging effect of ileostomy dejecta that contribute to PSC formation in ileostomates.

Future work will apply ileostomy dejecta to *ex vivo* murine skin models to study its resulting effect on skin integrity. This approach is targeted at understanding how PSC formation occurs. Additionally, mechanical damage will be incorporated to account for mechanical stripping of the peristomal skin during stoma bag changes.

References

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Acknowledgements

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