Control of the Mineralocorticoid Receptor Enhances Wound Healing and Mitigate Fibrosis After Thermal Injury

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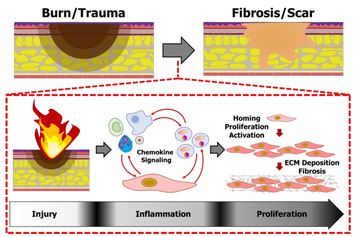
INTRODUCTION

Scars remain a major problem in care of the burned patient.

The central pathology driving fibrosis is known to be the imbalance of extracellular matrix.

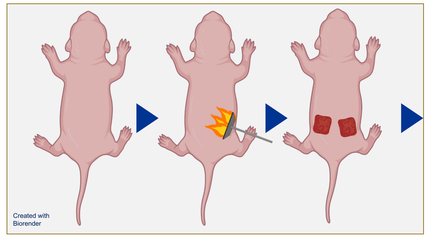
Mineralocorticoid (MR) antagonists have been shown to inhibit inflammation and fibrosis.



Aim: To enhance healing while mitigating hypertrophic collagen deposition after burns in mice with MR antagonists.

METHODS

Female athymic mice sustained bilateral 1 cm full-thickness thermal injury and were stratified.

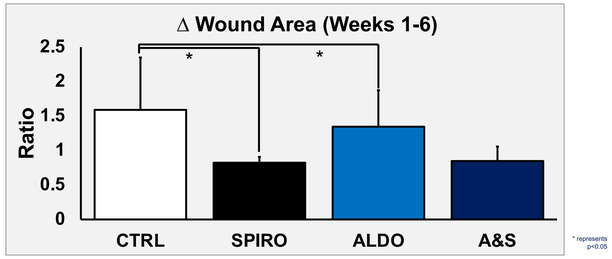


- Control
- Spironolactone IP injections
- Aldosterone Alzet pump
- Spironolactone and aldosterone

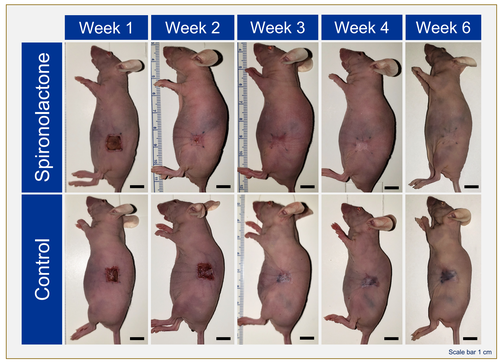
Mice were followed with weekly photography for 6 weeks.

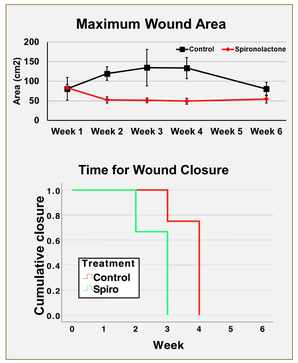
At the time of sacrifice, samples were collected for H&E, Trichrome, VVG and Pentachrome staining.

RESULTS - 1



Spironolactone only and Spironolactone and Aldosterone treatment decreased scar area after thermal injury compared with the controls.



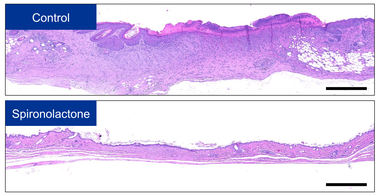


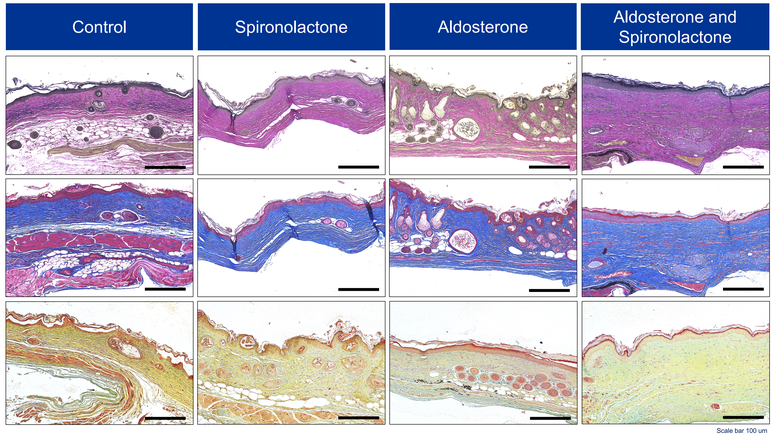
Burns kept growing and expanding in control but not in spironolactone treated mice.

Time to closure after injury improved in murine burns with spironolactone.

RESULTS - 2

Gross scar morphology showed hyperproliferative response in untreated but not spironolactone-treated mice.





Trichrome, VVG and Pentachrome demonstrated decreased extracellular matrix deposition with spironolactone therapy.

CONCLUSION

We found **early improvement** in wound epithelialization and overall decrease in scar area with spironolactone therapy.

These findings support that **MR-inhibition** in the treatment of burn scars.

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