Australian and New Zealand Burn Association
Annual Scientific Meeting

BURNS ACROSS THE AGES

15 – 18 October 2019
Hotel Grand Chancellor Hobart

BURN WOUND PROGRESSION: A HISTOLOGICAL ANALYSIS OF THE MECHANISMS RESPONSIBLE

Raymond Quan[1,2,3], Damien Harkin[3], Christine Andrews[4], Leila Cuttle[1,2,3]

[1]Centre for Children's Burns & Trauma Research, Centre for Children's Health Research; [2]Tissue Repair and Translational Physiology Program, Institute of Health and Biomedical Innovation; [3]School of Biomedical Sciences, Queensland University of Technology; [4]Mater Research Institute - The University of Queensland

Centre For Children's Burns & Trauma Research

BACKGROUND

- Burn wound progression is the phenomenon in which further tissue damage occurs in burn wounds following the initial damage at the time of injury.
- Histological staining can comprehensively visualise burned skin structures to identify burn wound pathologies responsible for burn wound progression.
- Previously, mechanisms identified to promote burn wound progression in the wound include:
  - Collagen denaturation
  - Vascular pathologies
  - Cellular necrosis
  - Cellular apoptosis

AIMS

1. To develop and optimise a catalogue of stains to accurately identify burn wound progression and measure burn depth.
2. Identify the significance of mechanisms contributing to burn wound progression at different time points post burn.

METHODS

Burn model selection: Porcine scald burn models were previously created and biopsies were collected at 1, 24 and 72 hours post burn.

Optimising histological stains: Haematoxylin and eosin, Verhoeff's van Gieson and Gomori's Trichrome are specialised stains to identify mechanisms of progression.

Burn damage markers: Markers of damage include: blood vessels, endothelial cell injury, infiltration of inflammatory cells and cell death.

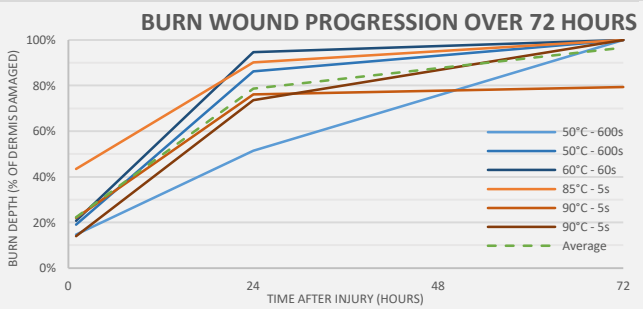

Figure 1: Burn conditions distinguished by line colour. Blue: Low temperature – long durations. Orange: High temperature – short durations. Wound progression occurs to cause deep dermal partial thickness burns in the initial 24 hours.

RESULTS

Cellular or structural markers of damage and wound progression were identified through different histochemical stains performed on tissue 1, 24 and 72 hours post burn.

Cellular markers

Haematoxylin and Eosin

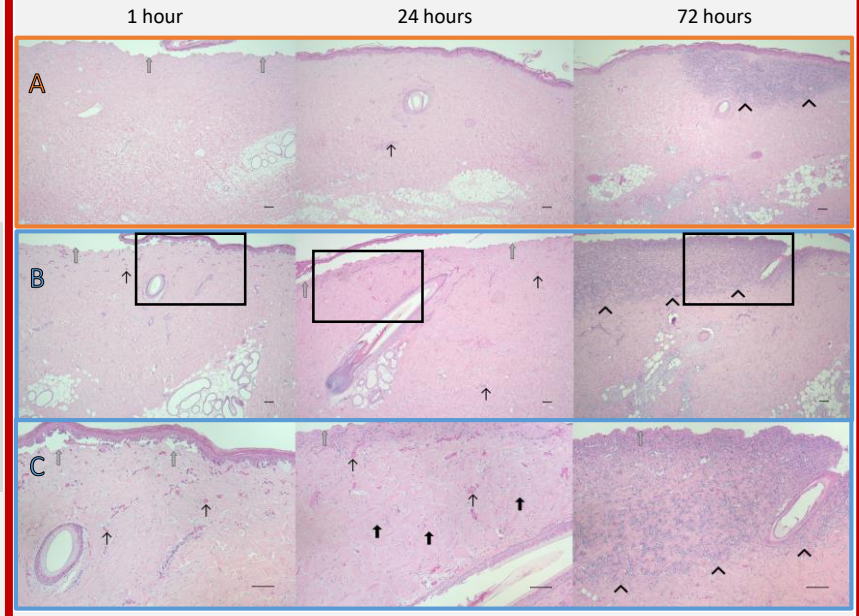

Figure 2: (A) Low temperature – long duration burn condition; magnification 4x. (B) High temperature – short duration burn condition; magnification 4x; boxes displayed at 10x in C. (C) 10x magnification shows markers of damage. Thick arrows indicate dead cells, thin arrows indicate red blood cell aggregation, shaded arrows indicate epidermis loss of adherence or absence, vector arrows indicate band of inflammatory cells. Scale = 100µm.

Structural markers

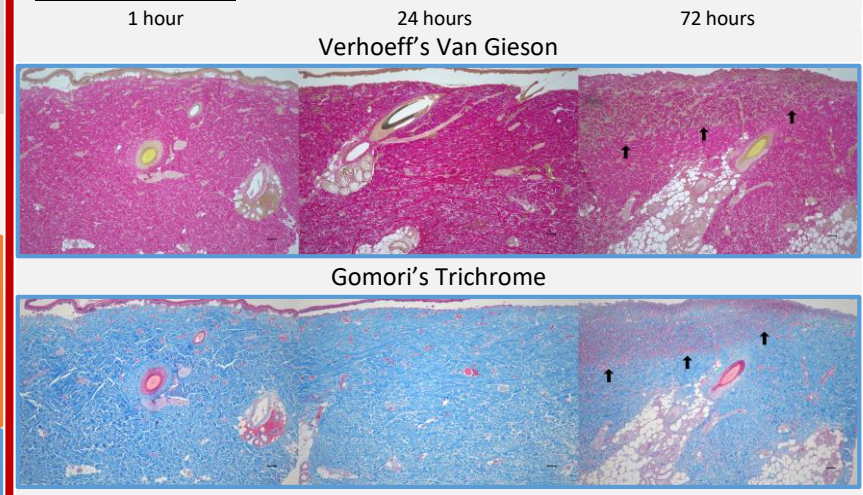

Figure 3: Low temperature – long duration burns stained with Verhoeff's van Gieson and Gomori's Trichrome to observe collagen changes due to thermal injury. Thick arrows show a band of contrasting stain appearing at 72 hours, consistent between both stains. No collagen changes were observed within the first 24 hours post burn. Images at 4x magnification. Scale = 100µm

DISCUSSION

- Burn wound progression can be histologically detected up to 72 hours post injury with H&E, VVG and MT. Effectively staining cellular and structural damage.
- The thick band observed at 72 hours may not represent denatured collagen, due to its time of presentation and similarity to figure 2B at 72 hours. This may represent inflammatory cells or an alternate pathology.

FUTURE DIRECTIONS

To further detect mechanisms of damage, other stains should be considered such as Martius Scarlet Blue to detect possible fibrin in thick band and cleaved Caspase 3 for apoptotic cell death