# IMPACT OF SEVERE BURNS ON PANCREATIC ISLETS: AN EXPERIMENTAL MODEL IN RATS

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## INTRODUCTION

The underlying mechanisms of beta-cell failure in burn patients with acquired insulin resistance after a burn trauma is not well understood. Our aim in this study was to describe the histopathological changes in the pancreatic islets secondary to severe burns in an experimental animal model.

## MATERIAL AND METHODS

**Groups:**

Fourteen Wistar albino rats weighing 350 to 400 g were randomly divided into 2 groups:

1. Sham group: Seven days after sham procedure (shaving + wound dressing), pancreatectomy was performed.
2. Burn group: Seven days after burn induction + wound dressing pancreatectomy was performed.

**Full-thickness burn model:**

- Animals were anesthetized with a mixed intraperitoneal injection of 100 mg/kg ketamine hydrochloride and 10 mg/kg xylazine hydrochloride
- A brass plate of 4 cm × 4 cm was heated under the flame of a Bunsen burner, monitoring the temperature using a thermocouple device of a multimeter (Fluke 116 HVAC)
- The brass plate was heated to 255 °C and placed onto the shaved area for 10 seconds once reached 250 °C (Figure 1).

**Histopathology and cell count:**

- Pancreatic tissues were examined under light microscopy, and islet size and cellularity were calculated. (Figure 2).
  - Light microscopic examination: Hematoxylin-eosin
  - Digitizing the slides: 3Dhistech Panoramic P250 Flash III scanner
  - Imaging processing: ViraPath application (ViraSoft Software Trade Inc, Istanbul, Turkey).

**Statistics**:

We performed statistical analyses using IBM SPSS Statistics for Windows (Statistical Package for the Social Sciences, version 25.0, Armonk, NY, IBM Corp).

- Means ± SD
- One-way analysis of variance and the Bonferroni correction for normal distributions
- *P* < .05

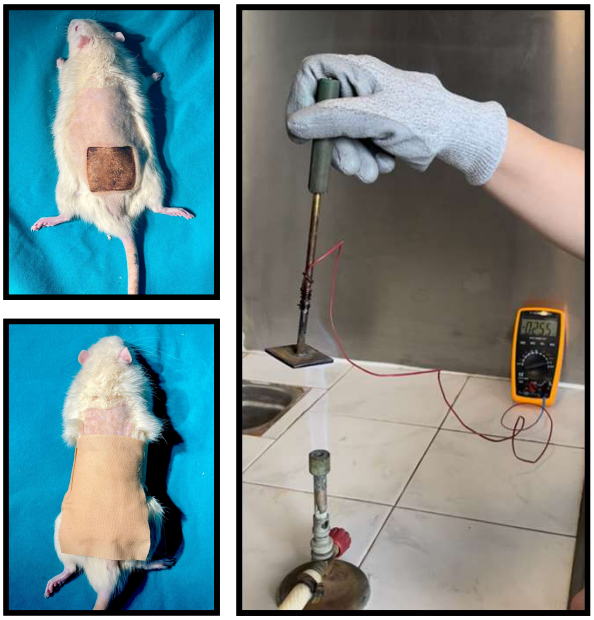

**FIGURE 1.** Materials Used and Macroscopic Evolution Observed in the Rat Experimental Burn Model

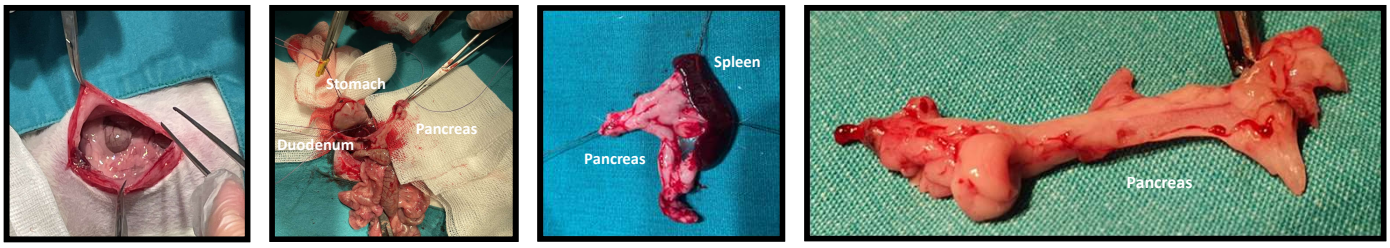


**FIGURE 2.** Intraoperative Findings During Pancreatectomy

## RESULTS

- The histopathologic examination was unremarkable.
- The mean number of islets per pancreatic tissue was lower in the burn group than in the sham.
- Significant difference was found in the mean number of cells per one islet with the cell count higher in the burn group (P < .05).
- The total volume of islets did not differ after burn trauma, and the residual islets in the Burn group were hypercellular in comparison to the Sham group.

| | Group | | P Value |
|---|---|---|---|
| | Sham | Burn | |
| Mean No. of islets/pancreatic tissue, cm² | 3864 ± 0.94 | 2394 ± 0.98 | .014* |
| No. of islet cells/islet | 122 420 ± 27.20 | 167 714 ± 3411 | .017* |
| Mean calculated areas of islets, cm² | 0.0168 ± 0.0046 | 0.0184 ± 0.0078 | .668 |

**TABLE 1.** Quantitative Assessment of Pancreatic Islets: Quantity of Islets, Density of Beta Cells in Islets, and Islet Size

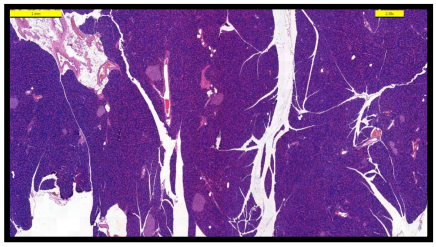

**FIGURE 3.** Pancreatic tissue with acinar structures and islets.

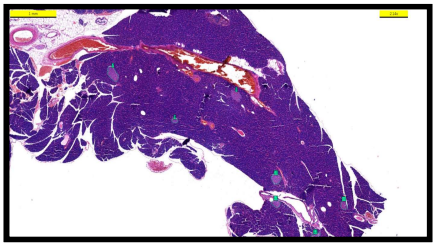

**FIGURE 4.** Pancreatic tissue from the sham group. Islets marked with green lines.

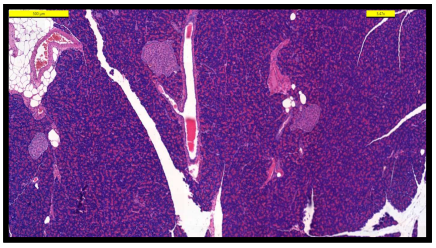

**FIGURE 5.** Analysis of pancreatic islets and cells.

## CONCLUSIONS

- Major burn injury causes significant morphological changes in the pancreatic islets in rats during the acute phase of burn.
- The decrease in the number of islets in the rats at 1 week postburn may be explained by the inadequate blood supply in some islets, leading to ischemic cell damage.
- The hypercellularity might have increased the total volume in the burn group, closing the gap with the sham group with regard to volume.
- Although it is not clear which cell type in the islets increased in number, we suggest that they most probably were beta cells as these cells constitute the most active cells in islets during the metabolic changes after burn trauma.

## APPLICABILITY TO CLINICAL PRACTICE

Identifying the adaptive mechanisms of the pancreas during the acute phase of burns allow us to understand the alterations in glucose metabolism and the insulin-signaling cascade associated with impaired wound healing, increased skin graft loss, increased muscle protein catabolism, increased incidence of infections, and mortality.