

Active neutrophil responses counteract *Candida albicans* burn wound infection of *ex vivo* human skin explants.

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Abstract

Burn wounds are highly susceptible sites for colonization and infection by bacteria and fungi. Large wound surface, impaired local immunity, and broad-spectrum antibiotic therapy support growth of opportunistic fungi such as *Candida albicans*, which may lead to invasive candidiasis. Currently, it remains unknown whether depressed host defenses or fungal virulence drive the progression of burn wound candidiasis. Here we established an *ex vivo* burn wound model, where wounds were inflicted by applying preheated soldering iron to human skin explants, resulting in highly reproducible deep second-degree burn wounds. Eschar removal by debridement allowed for deeper *C. albicans* penetration into the burned tissue associated with prominent filamentation. Active migration of resident tissue neutrophils towards the damaged tissue and release of pro-inflammatory cytokine IL-1 β accompanied the burn. The neutrophil recruitment was further

increased upon supplementation of the model with fresh immune cells. Wound area and depth decreased over time, indicating healing of the damaged tissue. Importantly, prominent neutrophil presence at the infected site correlated to the limited penetration of *C. albicans* into the burned tissue. Altogether, we established a reproducible burn wound model of candidiasis using ex vivo human skin explants, where immune responses actively control the progression of infection and promote tissue healing.

Involved units

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Leibniz-HKI-Authors



Fionnuala Bulman

[Details](#)



Petra Eißmann

[Details](#)



Ilse Denise Jacobsen

[Details](#)



Alessandra Marolda

[Details](#)



Daniel Rosenberger

[Details](#)



Christin von Müller

[Details](#)



Slavena Vylkova

[Details](#)



Lysett Wagner

[Details](#)

Identifier

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