CO2 laser therapy accelerates the healing of ulcers in the oral mucosa by inducing the expressions of heat shock protein-70 and tenasin C. (CO2 レーザー療法は、heat shock protein-70 および tenasin C 発現を誘導し、口腔粘膜潰瘍の治癒を促進する)

Background
The treatment of ulceration or stomatitis by laser irradiation is known to accelerate healing and relieve pain, but the underlying biological mechanism is not fully understood.

Purpose
The present study used a mouse model of ulceration to investigate the molecular mechanisms by which CO2 laser therapy accelerated the wound healing process.

Materials and Methods
The ulcer was artificially created in mouse palatal mucosa and irradiated with a CO2 laser. The wound healing process was analyzed using histology and immunohistochemistry. Fibroblast cells and HaCaT epithelial cells were cultured to assess the effects of thermal or laser stimulation.

Results
Laser irradiation induced the proliferation of epithelial cells and faster re-epithelialization of the wound area. Immunohistochemistry experiments showed that heat shock protein-70 (HSP70) was expressed mainly in the palatal epithelium under normal conditions. Laser irradiation induced HSP70 mRNA and protein expression in the lamina propria. In contrast, there was little tenasin C (TnC) expression in both the epithelium and mesenchyme of normal palatal tissue. However, laser irradiation induced TnC expression in the mesenchyme underlying the renewing epithelium. To examine whether hyperthermia mimicked the effect of laser irradiation, epithelial cells and fibroblasts were exposed to heated culture medium or laser irradiation. Culture of fibroblasts in heated medium increased the expressions of both TnC and TGF-β1, whereas laser irradiation induced only TnC expression.

Conclusion
The present study indicates that photobiogenic factor of CO2 laser irradiation caused by laser irradiation up-regulates TnC expression without TGF-β1 induction. We suggest that CO2 laser irradiation has an advantage over thermal stimulation.