Hypoxia is Involved in Deep Tissue Injury Formation in a Rat Model

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Abstract	Pressure ulcers that develop from deep tissue are known as deep tissue injuries (DTI). Although several mechanisms, including ischemic hypoxia, are attributed to pressure ulcer formation, the mechanisms involved in DTI formation are still unclear. Previous studies have suggested that hypoxia is involved in DTI in vitro, but it has yet to be determined whether hypoxia is also involved in DTI in vivo. Therefore, this study aimed to investigate whether hypoxia is involved in DTI using a newly established DTI model. Rats were divided into control, low pressure DTI, and high pressure DTI groups. Results of wound healing tests indicated that more severe DTI resulted in prolonged healing time, more severe inflammation and muscle damage, higher levels of exudate creatine phosphokinase, and greater muscle edema. Increased hypoxia was observed in severe DTI-nuclear localization of hypoxia-inducible factor-1 alpha was markedly increased in the high pressure DTI group, while the low pressure group showed more increased cytoplasm localization compared to the control group on day 3. Study results revealed that hypoxia is involved in DTI in vivo.
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