3, 4-METHYLENEDIOXY-BETA-NITROSTYRENE AMELIORATES EXPERIMENTAL BURN WOUND PROGRESSION BY INHIBITING THE NLRP3 INFLAMMASOME ACTIVATION (182)

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**Question:** Burn wound progression remains a challenging problem in clinic. Secondary tissue damage caused by unlimited inflammatory response is considered to be one of the most important contributing factors to this clinical problem. Nucleotide-binding oligomerization domain like receptor family, pyrin domain containing 3 (NLRP3) inflammasome is recently found to play important roles in immune activation and inflammatory response after burn/trauma. This experimental study aims 1) to observe the expression and distribution of NLRP3 inflammasome in burn wounds of a rat burn model; 2) to study whether inhibiting the NLRP3 inflammasome activation would ameliorate burn wound progression.

**Methods:** A deep second degree burn was inflicted on the back of Wistar rats. The expression of NLRP3 inflammasome components and IL-1β were determined by western blot and co-immunoprecipitation. The distribution of NLRP3 inflammasome was assessed by immunohistochemical staining and double-labelling immunofluorescence. Neutrophil infiltration, wound perfusion, burn depth and wound healing time were assessed.

**Results:** Burn induced remarkable NLRP3 inflammasome activation and cleavage of IL-1β. The NLRP3 inflammasome was mainly observed in macrophages of the zone of stasis. 3, 4-Methylenedioxy-β-nitrostyrene (MNS) significantly inhibited the NLRP3 inflammasome activation and inflammatory cytokines production in burn wounds. Consequently, neutrophil infiltration was reduced, wound perfusion was restored, burn wound progression was ameliorated and wound healing was accelerated.

**Conclusion:** In this study, we demonstrated that burn induced NLRP3 inflammasome activation and inflammatory response in wounds, which may be associated with burn wound progression. Treatment with MNS inhibited NLRP3 inflammasome activation, ameliorated burn wound progression and promoted wound healing.