Inflammatory Effects of Hypothermia and Inhaled H2S During Resuscitated, Hyperdynamic Murine Septic Shock

Titre	Inflammatory Effects of Hypothermia and Inhaled H2S During Resuscitated, Hyperdynamic Murine Septic Shock
Type de publication	Article de revue
Auteur	Wagner, Florian [1], Wagner, Katja [2], Weber, Sandra [3], Stahl, Bettina [4], Knöferl, Markus-W. [5], Huber-Lang, Markus [6], Seitz, Daniel-H. [7], Asfar, Pierre [8], Calzia, Enrico [9], Senftleben, Uwe [10], Gebhard, Florian [11], Georgieff, Michael [12], Radermacher, Peter [13], Hysa, Vladislava [14]
Editeur	Lippincott, Williams & Wilkins
Type	Article scientifique dans une revue à comité de lecture
Année	2011
Langue	Anglais
Date	2011/04
Numéro	4
Pagination	396 - 402
Volume	35
Titre de la revue	Shock
ISSN	1073-2322

Résumé en anglais

Inhaling hydrogen sulfide (H2S) reduced energy expenditure resulting in hypothermia. Because the inflammatory effects of either hypothermia alone or H2S per se still are a matter of debate, we tested the hypothesis whether inhaled H2S amplifies the hypothermia-related modulation of the inflammatory response. Fifteen hours after cecal ligation and puncture or sham laparotomy, anesthetized and mechanically ventilated normothermic and hypothermic mice (core temperature kept at 38°C and 27°C, respectively) received either 100 ppm H2S or vehicle. In the sham-operated animals, inhaled H2S and hypothermia alone comparably reduced the plasma chemokine and IL-6 levels, but combining hypothermia and inhaled H2S had no additional effect. The lung tissue cytokine and chemokine patterns revealed a similar response. During sepsis, inhaled H2S reduced the blood cytokine concentrations only, without effects on the plasma chemokine or the lung tissue levels. Again, inhaled H2S had no major additional effect during hypothermia. With or without sepsis, inhaled H2S and hypothermia alone comparably reduced the lung tissue heme oxygenase 1 expression, whereas inhaled H2S had no additional effect during hypothermia. Lung tissue nuclear transcription factor κB activation was reduced by combining H2S with hypothermia in the sham-operated animals, whereas it was increased by inhaled H2S during sepsis. Hypothermia amplified this response. Hence, during anesthesia and mechanical ventilation, inhaled H2S exerted anti-inflammatory effects, which were, however, not amplified by adding deliberate hypothermia. Sepsis attenuated these anti-inflammatory effects of inhaled H2S, which were at least in part independent of the nuclear transcription factor κB pathway.

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DOI	10.1097/SHK.0b013e3181ffff0e [16]
Lien vers le document	http://dx.doi.org/10.1097/SHK.0b013e3181ffff0e [16]

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