研究报告

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一氧化氮合酶途径参与SO_2胁迫下蚕豆气孔运动调节

Involvement of nitric oxide synthase in stomatal movement regulation in the response of *Vicia faba* L. to sulfur dioxide stress

关键词：蚕豆 SO_2 一氧化氮合酶 气孔运动

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摘要：以蚕豆为材料,研究一氧化氮合酶(NOS)途径在SO_2诱发气孔运动中的作用.研究发现:浓度7.5~200 μmol·L^{-1}的SO_2衍生物处理后,蚕豆叶面气孔开度减小,气孔开度与SO_2衍生物浓度呈负相关;SO_2衍生物处理组叶组织中NOS活性增强;加入NO清除剂c-PTIO或NOS抑制剂L-NAME可抑制SO_2衍生物诱发的气孔关闭;SO_2衍生物处理组保卫细胞内NO和Ca^{2+}水平升高,用c-PTIO降低胞内NO水平后Ca^{2+}水平随之下降.结果表明,SO_2衍生物胁迫可诱发保卫细胞内NO合成增加,NO通过调节胞内Ca^{2+}水平升高,激活下游信号转导途径,调节气孔运动;NOS途径介导的NO合成参与了SO_2胁迫下蚕豆气孔运动的调节.

Abstract: The potential role of nitric oxide synthase (NOS) in stomatal movement regulation in the response of plants to sulfur dioxide (SO_2) was investigated in *Vicia faba* L. The results showed that SO_2 hydrates significantly decreased stomatal aperture in the concentration range of 7.5 to 200 μmol·L^{-1}. SO_2 hydrates induced stomatal closure in a concentration-dependent manner. SO_2 hydrates-evoked stomatal closure was correlated with increasing activities of NOS in the whole leaves from four-week-old *V. faba* plants. Both NO scavenger c-PTIO and NOS inhibitor L-NAME can block SO_2 hydrates-evoked stomatal closure. Moreover, exposure to SO_2 hydrates enhanced the intracellular levels of NO and Ca^{2+} in guard cell. Application of exogenous NO scavenger c-PTIO blocked the increases of intracellular NO and Ca^{2+} levels in SO_2-treated guard cells. These results indicated that SO_2 hydrates-triggered NO production mediated stomatal movement regulation via Ca^{2+} signaling, and that nitric oxide synthase-dependent NO production played a crucial role in SO_2 hydrates-evoked stomatal movement in *V. faba*.

Key words: *Vicia faba* L. SO_2 nitric oxide synthase (NOS) stomatal movement