

### 牡丹柱枝孢叶斑病 (*Cylindrocladium canadense*) 对叶片光合系统功能的影响

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#### Effect of Infection by *Cylindrocladium canadense* on Behaviors of Photosystems in Tree Peony Leaves

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摘要 以牡丹 (*Paeonia suffruticosa*) ‘藏枝红’为试验材料, 通过测定叶绿素荧光快速诱导动力学曲线、820 nm 光吸收曲线以及气孔交换参数和H<sub>2</sub>O<sub>2</sub> 含量, 研究了柱枝孢叶斑病 (*Cylindrocladium canadense*) 对牡丹叶片光合性能和光系统功能的影响。结果显示: 在病原菌侵染后, 牡丹叶片的净光合速率 (P<sub>n</sub>)、气孔导度和叶绿素含量均下降, 而细胞间隙CO<sub>2</sub> 浓度升高, 表明P<sub>n</sub> 的降低是受非气孔因素的影响。*Cylindrocladium canadense* 的侵染, 使H<sub>2</sub>O<sub>2</sub> 含量显著升高, 快速叶绿素荧光诱导动力学曲线形状发生明显改变, F<sub>v</sub>/F<sub>m</sub> 和PIABS 均显著下降。PS II 供体侧、反应中心和受体侧活性均受到抑制, 但PS II 受体侧所受抑制比供体侧大。同时, 病原菌侵染也使PS I (ΔI/I<sub>0</sub>) 活性快速下降。由上述研究结果推测: *Cylindrocladium canadense* 的侵染, 使H<sub>2</sub>O<sub>2</sub> 积累增加, 对PS I 和PS II 的功能造成伤害。PS I 活性的下降阻碍了PS II 向PS I 的电子传递, 过剩激发能增加, 导致ROS 增加, 抑制D1 蛋白的合成, 加剧了对PS II 伤害, 这是其侵染抑制牡丹光合系统的主要原因。

关键词: 牡丹 柱枝孢叶斑病 光系统 I 光系统 II 活性氧

Abstract: In the present study, Effect of infection by *Cylindrocladium canadense* on behaviors of photosystem I (PS I) and photosystem II (PS II) in tree peony (*Paeonia suffruticosa* ‘Zangzhihong’) leaves was estimated by simultaneously measuring their Chlorophyll fluorescence transient, light absorbance at 820 nm, gas exchange and hydrogen peroxide (H<sub>2</sub>O<sub>2</sub>) content. The data showed that the net photosynthetic rate (P<sub>n</sub>), stomatal conductance and chlorophyll content were significantly decreased compared with that of the control after infection by *C. canadense*, whereas intercellular CO<sub>2</sub> concentration was significantly increased. These results indicated that the decreased photosynthesis in infected leaves was resulted from non-stomatal factor. Furthermore, significantly enhanced H<sub>2</sub>O<sub>2</sub> content, reduced F<sub>v</sub>/F<sub>m</sub> and PIABS, as well as the changed chlorophyll fluorescence transient was also observed in leaves of tree peony infected by *C. canadense*. The donor sides (Oxygen evolution complex), reaction centers and acceptor sides of PS II were significantly inhibited by *C. canadense* infection. But the acceptor sides were more suffered than the donor sides. At the same time, the activity of PS I was also significantly reduced. Taken together, the data presented here indicated that *C. canadense* infection resulted in the accumulation of H<sub>2</sub>O<sub>2</sub>, which in turn damaged the functions of PS I and PS II. The decline of PS I activity inhibited transportation of electrons from PS II to PS I, which then led to the accumulation of excessive excitation energy and reactive oxygen species (ROS). The increased ROS inhibited synthesis of protein D1, accelerating damage to PS II. It was the main reason for inhibition of photosystems in tree peony leaves after infected by *C. canadense*.

Keywords: tree peony, *Cylindrocladium canadense*, PS I, PS II, reactive oxygen species

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