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N SIGNALING A MAJOR DRIVER OF *RHIZOBIUM*-LEGUME SYMBIOSIS

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A major hallmark of legumes is their adaptation to mineral N limitation by developing root symbiotic nodules with N₂-fixing rhizobia. Systemic N signaling mechanisms are tightly regulating nodule activity and development as a function of the N status of the whole plant and adjust the symbiotic N fixation capacity to the N demand of the plant. These mechanisms have been evidenced by using split-root experimental systems in symbiotic plants, particularly in the model legume *Medicago truncatula*/Sinorhizobium. Addition of mineral nitrogen to a half root system results in systemic repression of nitrogen fixation and nodulation as well as systemic induction of nodule senescence on the whole root system. Alternatively, suppression of the fixation activity of a half root system (by removing N₂ from the root atmosphere by Ar/O₂ treatment or by using Fix⁻ bacteria) results in systemic N signaling that stimulates both de novo nodule formation and mature nodule expansion in the roots not directly exposed to the N limitation treatment. Molecular identification of the mechanisms behind these regulations begins to be elucidated and reveals an unexpected complexity as several pathways are likely to operate in the control of nodule development and function. Cross-talk of systemic N signaling with the autoregulation of nodule number, the nodule organogenesis pathways, nodules C-N metabolites allocations will be discussed. Additional evidence indicating that the response of symbiosis to N signaling may vary according to the bacterial partner will be presented. Symbiotic organs are highly sensitive to environmental constraints that inhibit symbiotic nitrogen fixation (water deficit, inefficient bacterial partner etc...). Evidences indicate that N signaling mechanisms have major role in the adaptation of the symbiotic plant to various stresses by promoting a compensatory response of the plant. These mechanisms may be important targets for breeding symbiotic traits in legumes.