

A glutamine-amidotransferase-like protein modulates FixT anti-kinase activity in *Sinorhizobium meliloti*

ABSTRACT

The isolation of the *asnO* mutant demonstrates that *fixT* has a physiological regulation, which makes it unlikely that *FixT* serves as merely 'homeostatic' in *S. meliloti* (species hypotheses state that *asnO* may regulate the activity of its own protein, but our data suggest that this remains to be determined. An alternative function of *asnO* might be to pair nitrogen fixation gene expression in

INTRODUCTION

The glutamine-amidotransferase (GAT) is a member of the glutamine-amino acid transferase family, and is present in many foodstuffs. It is also known as glutamine-amidotransferase-like protein (GATL-AMP). GATL-AMP is a protein that is present in many proteins.

Figure 1. View largeDownload slide A glutamine-amidotransferase (GAT) is a member of the glutamine-amino acid transferase family, and is present in many foodstuffs. It is also known as glutamine-amidotransferase-like protein (GATL-AMP). The development of N₂-fixing nodules on the roots of alfalfa (*Medicago sativa*) and closely related plants is regulated by a regulatory cascade. FixLJ, whose two-component regulatory system activates nitrogen fixation genes in response to microoxic conditions inside the nodula, acts as an intermediary regulator for *nifA* and *fixK*. We report the isolation of a mutant strain of *S. meliloti* that does not respond to *FixT*'s repressor activity. The mutation is located in *pnO*, coding for glucidation protein and homologous to glutamine-amidotranferases; we explain how this observation may shed light on nitrogen fixation in bacteria.

CONCLUSION

Remarkable conclusions *FixT* is an intriguing protein because it has not been described in any other bacterium except for *S. meliloti*. Furthermore, its mechanism of action is unique since it can block the phosphorylation and hence activity of the *FixL* sensor histidine kinase. There are only a few examples of such anti-kinastic proteins in the literature, and its primary sequence did not provide clues to its function. Therefore, there is great interest in determining what its biological role is in *S.* Alternatively, By modifying the *asnO* gene, the authors demonstrate that *fixT* may have a physiological function. They conclude that this discovery supports previous hypotheses by suggesting that *Fixt* may facilitate integration of an additional signal by the *FixLJ* two-component regulatory system, whose activity is mainly controlled by oxygen (Figure 6). The relationship between *fixT*, *fixL*, and *asnO* needs to be further investigated. We propose a working model that suggests the absence of *Asno* may result in an imbalance in the pool of metabolites associated with *FixT* or other interactions.