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 asanO 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.