Ammonia channel (Amt) proteins are ubiquitous in archaea, eubacteria, fungi and plants. They allow these organisms to take up ammonium from their environment and the flux through these channels is regulated in response to the intracellular nitrogen status. In bacteria, the $P_{II}$ signal transduction protein, GlnK, can form a complex with AmtB and can thereby regulate ammonium uptake. Complex formation is reversible and is controlled by intracellular metabolite pools.

The regulatory properties of $P_{II}$ proteins, like GlnK, derive from their ability to bind three small molecule effectors: ATP, ADP, and 2-oxoglutarate (2-OG). To investigate how these effectors influence ammonium uptake we have analysed the fluctuations in intracellular effector pools in *E. coli* during association and dissociation of the AmtB-GlnK complex. Using the effector combinations that we observed *in vivo* we were then able to replicate the observed AmtB-GlnK interactions *in vitro*. Using our recently determined X-ray structure of the *Azospirillum brasilense* GlnK homologue, GlnZ, with bound 2-OG and ATP we are now able to propose a model that explains the way in which interactions between GlnK and its effectors mediate control of ammonium influx via AmtB.