Supporting information S2

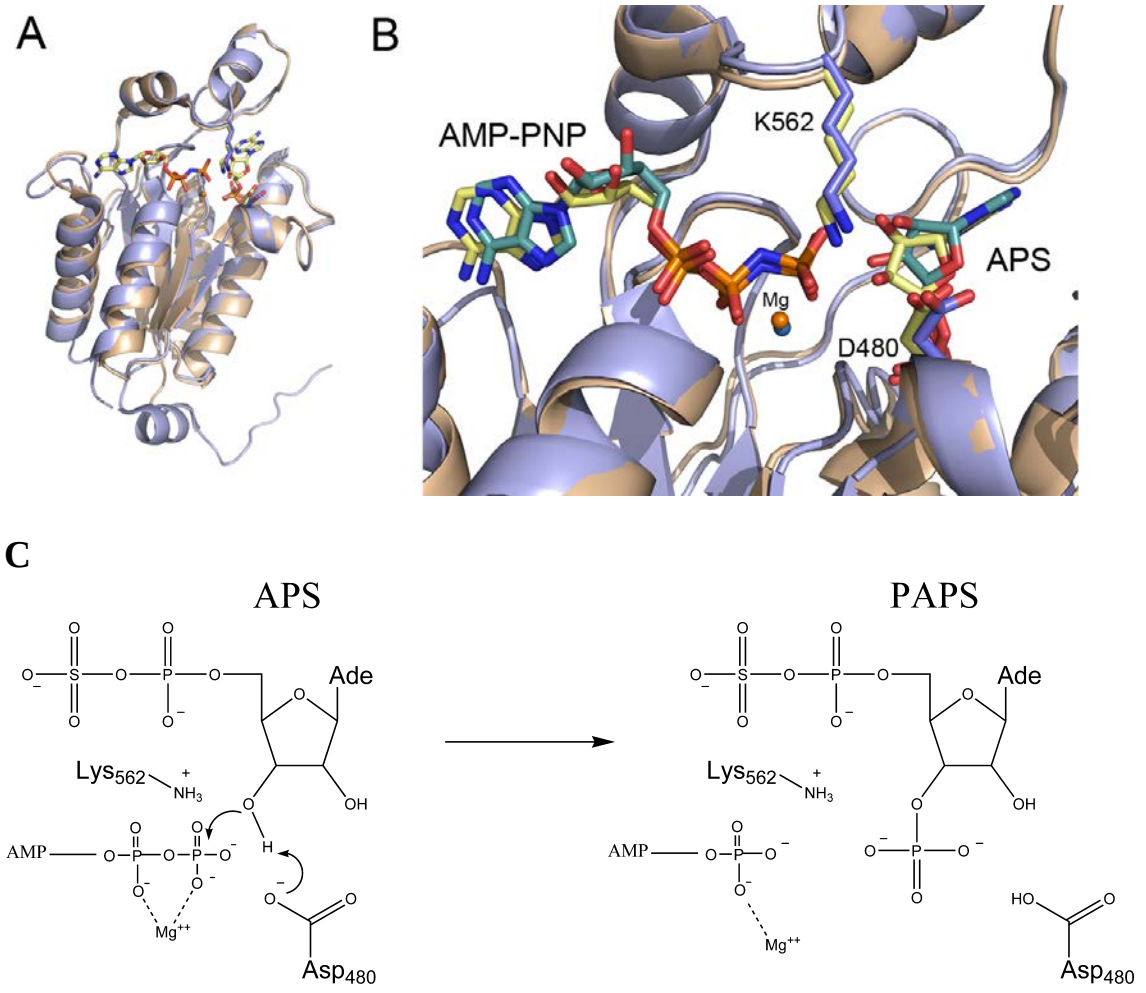
Evidence of redox regulation by a single cysteine residue in the kinase domain of the sulfate-activating complex in *Mycobacterium tuberculosis*

Ömer Poyraz[a,$], Katharina Brunner[a], Bernhard Lohkamp[a], Hanna Axelsson[b], Lars G.J. Hammarström[b], Robert Schnell[a] and Gunter Schneider[a]*

Department of Medical Biochemistry and Biophysics, Karolinska Institutet, S-171 77 Stockholm, Sweden

Chemical Biology Consortium Sweden, Science for Life Laboratory Stockholm, Department of Medical Biochemistry and Biophysics, Karolinska Institutet, Solna, Sweden.

[$]Present address: Department of Clinical Neuroscience, Therapeutic Immune Design Unit, Karolinska Institutet, Stockholm, Sweden



S2 Figure. Comparison of *M. tuberculosis* and *A. thaliana* APS kinase. The active site cavity of CysC (A) and catalytic residues (B) are shown in comparison with *Arabidopsis thaliana* APS-kinase (PDB:3uie). *M. tuberculosis* CysC and *Arabidopsis thaliana* APSK are shown as cartoon in beige and light blue, respectively. The non-cleavable ATP analogue (AMP-PNP), APS and residues Asp480 and Lys562 (CysC numbering) are shown as sticks in yellow and blue for *M. tuberculosis* CysC and *A. thaliana* APSK, respectively. Mg^{2+}-ions present in both structures are indicated by a blue or orange sphere. (C). Mechanistic proposal for phosphoryl transfer by *M. tuberculosis* CysC. Asp480 acts as a base abstracting the proton from the 3' OH of APS that subsequently attacks the γ phosphate of ATP. The side chain of Lys562 stabilizes the additional negative charge developing in the transition state.