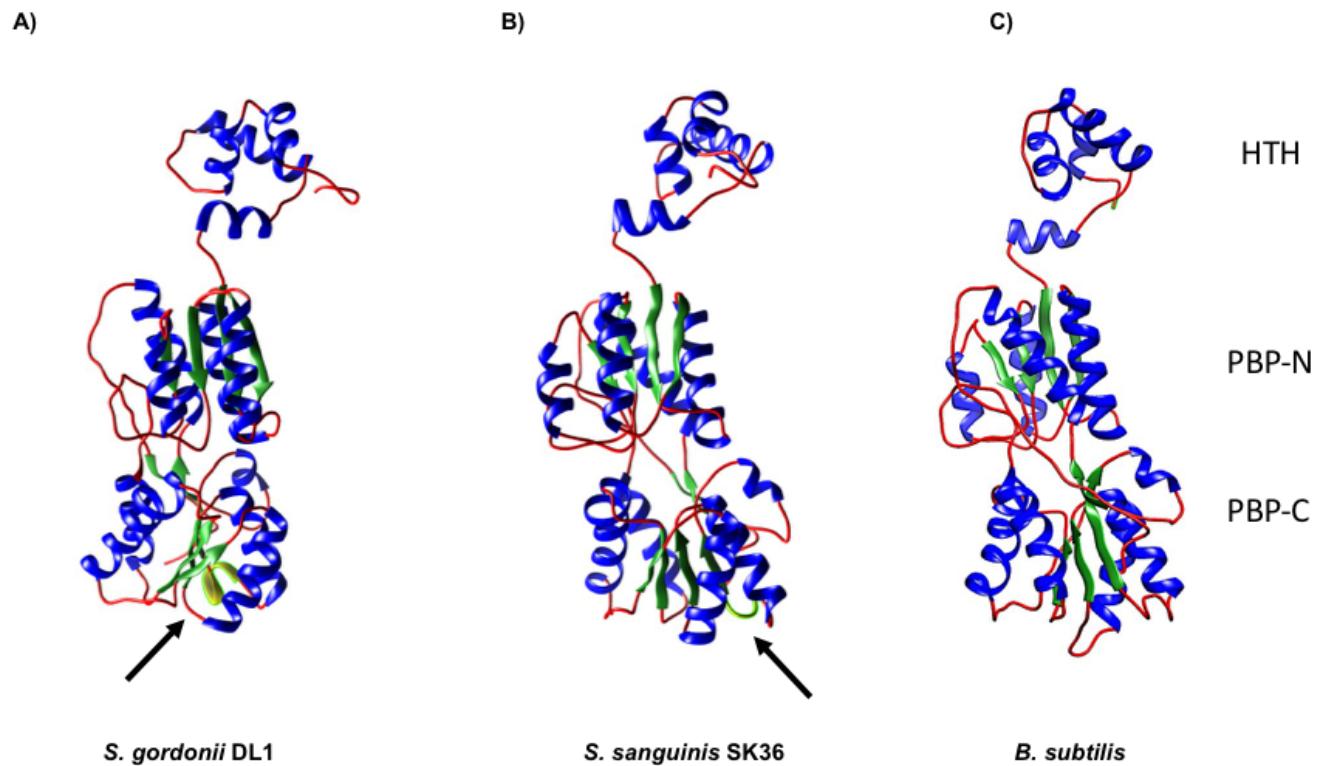
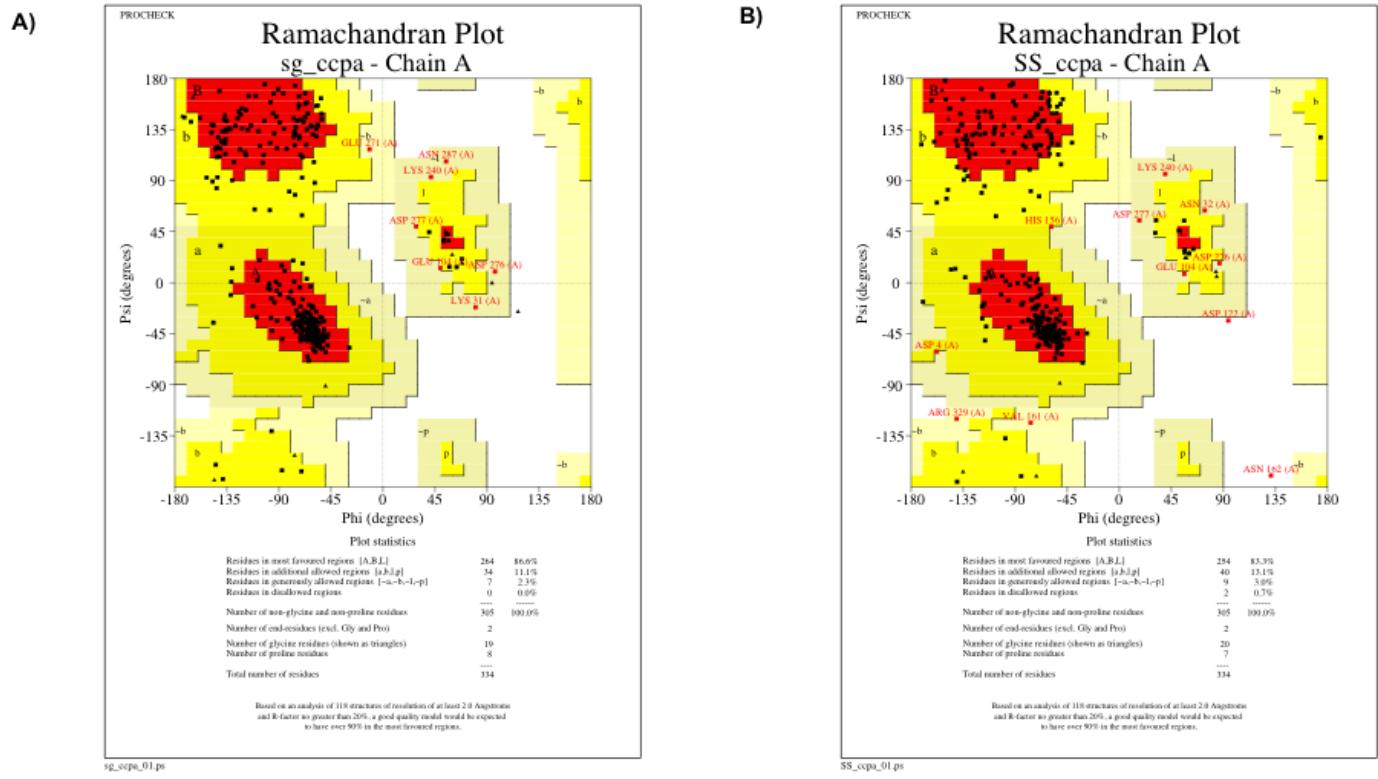


**Fig. S1:** EMSA analysis of CcpA binding to *spxB<sub>p</sub>* in the presence of unlabeled competitor *spxB<sub>p</sub>* DNA. Increasing concentrations of unlabeled competitor *spxB<sub>p</sub>* DNA was added to the reaction mixture containing fixed amounts of CcpA (20 pmol) and biotin-labeled *spxB<sub>p</sub>* probe (1 fmol). 1 = negative control; 2 = plus 1 fmol unlabeled probe; 3 = plus 10 fmol unlabeled probe; 4 = plus 50 fmol unlabeled probe; 5 = plus 100 fmol unlabeled probe; 6 = plus 200 fmol unlabeled probe; 7 = plus 300 fmol unlabeled probe; 8 = plus 400 fmol unlabeled probe; 9 = plus 500 fmol unlabeled probe. A representative picture is shown (n = 3).



**Fig. S2:** I-TASSER proposed CcpA structural models. A) *S. gordonii* and B) *S. sanguinis*. Predicted  $\beta$ -sheet regions are depicted in green and  $\alpha$ -helical regions are depicted in blue. The arrows point to the major difference between both CcpA. The difference is highlighted in yellow in the *S. gordonii* structural model. C) CcpA from *Bacillus subtilis* (PDB ID: 1ZVV) which served as template.



**Fig. S3:** Ramachandran plots for structure validation of proposed CcpA structural models. A) *S. gordonii* and B) *S. sanguinis*.

**Group I**

*S. oralis* J22 **TAA**AAGCGAGCTTGCCTTTATTATAAACAGTCAC**AT AAAACGT TT TCAA**ACTGTACTGTAATAGAGAATAAAAACCTCTGAGCAGATTCTAGTAAAGTCAAAATAATTGTGATAAAATTAGTAGAGTAAAATTG**TG AAACGT TT CCAA**ATAATAT**ATGAAACGGATGAAT**CAAGTTTAAAATATTGA~~AGGAGAGTTATT~~**ATG**  
*S. infantis* **TAA**- (N)<sub>125</sub>-TAACCGCTTTCTATTCTAAAACAGATT**TTCAAAACCGCTTCAA**ACTGTACTGTAATAGAGAATGAAAGTTCTGAGCATACTTAGAAATCTAAATGTGATAAAATTAGGATTGATAATT**CTG AATCGTTTCAA**ATAACAT**ATAAAGCGTTTTG**TAATATGAAATTATTG**AGGAGAGTTATCATT****ATG**  
*S. dentisanii* 2-1B **CAT**- (N)<sub>139</sub>-TTTTTGAAATCTATTATAAAATCACATTCAAATTCAGAAAAATCTCATATTGTTATGTAATAGTTCTAACAGAGTTGAAATTGAGTTAAGTT**TTG AAACGT TT TCAT**TAACCT**ATCTGAAACGT TT CCAA**TAATAAATTAAATTG**AGGAGAGTTATCATT****ATG**  
*S. mitis* 3-1 **TAA**AAGCGGGCTTGCCTTTATTATAAACAGAAAG**CTAAACACGT TT CCAA**ACTGTACTGTAATAGAGAATAAAAACCTCTGAGCAGATTCTAGTAAAGTCAAAATAATGTGATAAAATTAGT**TG AAACGT TT CCAA**AAACTATATGAAACCTTTAGTAGATTAAAATTG**AGGAGAGTTATCATT****ATG**  
*S. parasanguinis* **TAA**- (N)<sub>299</sub>-AATAGGGATTGAGCATTCTGAGCAGATTCTTACATGTGAATCTTTGTGATAAAATTAAAT**TGTAATCGTTTGTAATCGTTTCAT**GAGAAATGTCAGAAAAACTCATTTCAGCAAGATGAAG**TAG AAGCGCTATGCT**ACATTAAAGGATCAAATAGATTAA~~AGGAGAGTTAGA~~**ATG**  
*S. parasanguinis* 1-9 **stop**- (N)<sub><208</sub>-AATAGGGATTGAGCATTCTGAGCAGATTCTTACATGTGCACTCTTGTGATAAAATTAAAT**TGTAATCGTTTGTAATCGTTTCAT**GAGAAATGTCAGAAAAACTCATTTCAGCAAGGAGATGATCGCAAAGATGAAG**TAG AAGCGCTATGCT**GCATTAAAGGATCAAATAGATTAA~~AGGAGAGTTAA~~**ATG**

**Group II**

*S. parasanguinis* 1-7 **TAA**- (N)<sub>319</sub>-GAATAGGGATTGAGCATTGAGCAGATCTTACATGTGAATCTTTGTGATAAAATTAAAT**TGTAATCGTTTGTAATCGTTTCAT**TAGAAAAGTCTGAAAAACTCATTTCAGACGAGATGATCGCAAAGATGAAG**TAG AAGCGCTATGCT**GCATTAAAGGATCAAATAGATTAA~~AGGAGAGTTAA~~**ATG**  
*S. sanguinis* SK36 **TAG**- (N)<sub>108</sub>-AATTATTATAAACACATTGGACTTTGTGAAAAAAATCAGGGCTGGTGTATGTAATAGTCTATATCATCTTGGCATATTCTTACAGAGCGAAAAACATGATAAATTGATAGACTAAAGTT**TG AAGCGTTTCAT**TCACAA**ATGGAATATTTC**CAA~~CTGAAACCTTTATTG~~**AGGAGAGTTATT****ATG**

**Group III**

*S. gordonii* DL1 **TAA**- (N)<sub>245</sub>-TATCCTATTCTACTATAGTTGTGAAACTTCTAAAAAGAGCATCTGTTATGTAATAGGGATTGAGCATCTGAGCATATTCTTACAGTGAGAAAAACATGGTAAATTGATAGTAAAGTT**TG AAGCGTTTCAT**TCACAA**ATGGAATGT**~~TTTCAA~~TAAGTAAATTATTG**AGGAGAGTTATT****ATG**  
*S. mitis* 2-7 **TAA**AAGCGGGCTTGCCTTTATTATAAACAGTT**CATAAACACGT TT CCAA**ACTGTACTGTAATAGATAATAAAAACCTCTGAGCAGATTCTTAGTAAACTAAATTAAATGTGGTAAATTAGAAATGTAATAAATTG**TAAACACGT TT TCAT**GGTTATCTGAATGCTCCTTAGTAAATTAAAATATTG**AGGAGAGTTATA****ATG**  
*S. mitis* 2-8 **TAA**- (N)<sub>107</sub>-TTAATTTCATTGAGTATTATAAACAGAAAT**CTAAACACGT TT CCAA**ACTGTACTGTAATAGAGAATAAAAACCTCTGAGCAGATTCTTAGTAAACTAAATTAAATGTGGTAAATTAGAATTGTAATAATTG**CTAAACACGT TT TCAT**AATTATCTGAATGTTCTTAGTAAATTAAAATATTG**AGGAGAGTTATCATT****ATG**

**Group I**

*S. oralis* J22 **S. oralis** J22  
*S. infantis* **S. infantis**  
*S. dentisanii* 2-1B **S. dentisanii** 2-1B  
*S. mitis* 3-1 **S. mitis** 3-1  
*S. parasanguinis* **S. parasanguinis**  
*S. parasanguinis* 1-9 **S. parasanguinis** 1-9

**Group II**

*S. parasanguinis* 1-7 **S. parasanguinis** 1-7  
*S. sanguinis* SK36 **S. sanguinis** SK36

**Group III**

*S. gordonii* DL1 **S. gordonii** DL1  
*S. mitis* 2-7 **S. mitis** 2-7  
*S. mitis* 2-8 **S. mitis** 2-8

**Fig. S4:** Sequence comparison of the *spxB* promoter region of several oral streptococci classified in three groups, representing their H<sub>2</sub>O<sub>2</sub> release regulation type. Start codons of *spxB* and upstream genes as well as stop codons of upstream genes are underlined and displayed in bold. Shine-Dalgarno sequences and predicted *cre* sites (and known *cre* sites of SK36 and DL1) are boxed. In addition matching nucleotides are displayed in bold, in case of the core palindromic sequence, nucleotides are displayed in the same color as in the consensus sequence (5'-WWGW**AARCGYTTWCWW**-3'). Nucleotides not matching the consensus sequence and gaps are highlighted in grey. Please note the overlapping *cre* sites of *S. parasanguinis*. In group one only one representative of *S. parasanguinis* is shown, because there were no differences in the displayed sequence. The *cre* prediction is based on similarities to the given consensus sequence and followed the rule of allowing no more than 4 variations (mismatches or gaps). Sequence prediction was manually performed.