



Fig. S1. Proposed model for the metabolism of Fuc- α -1,3-GlcNAc and the regulation of the *alf* operon. Fuc- α -1,3-GlcNAc is transported by the PTS encoded by *alfEFG* cluster and it is intracellularly split by AlfB into fucose and GlcNAc. This is metabolized through glycolysis and the fucose is expelled from the cells by an unknown mechanism. The *alfBR* and *alfEFG* clusters are induced by Fuc- α -1,3-GlcNAc mediated by the transcriptional repressor AlfR and repressed by glucose via CcpA. Fuc- α -1,3-GlcNAc is probably the effector of AlfR and mediates its release from the operator, allowing the expression of *alfBR* and *alfEFG*. In the presence of glucose both CcpA and AlfR are bound to *cre* motif and operator, respectively, blocking the transcription. In the presence of a mix of glucose and Fuc- α -1,3-GlcNAc there is some expression of the *alf* operon.