Suppl. Fig. 2

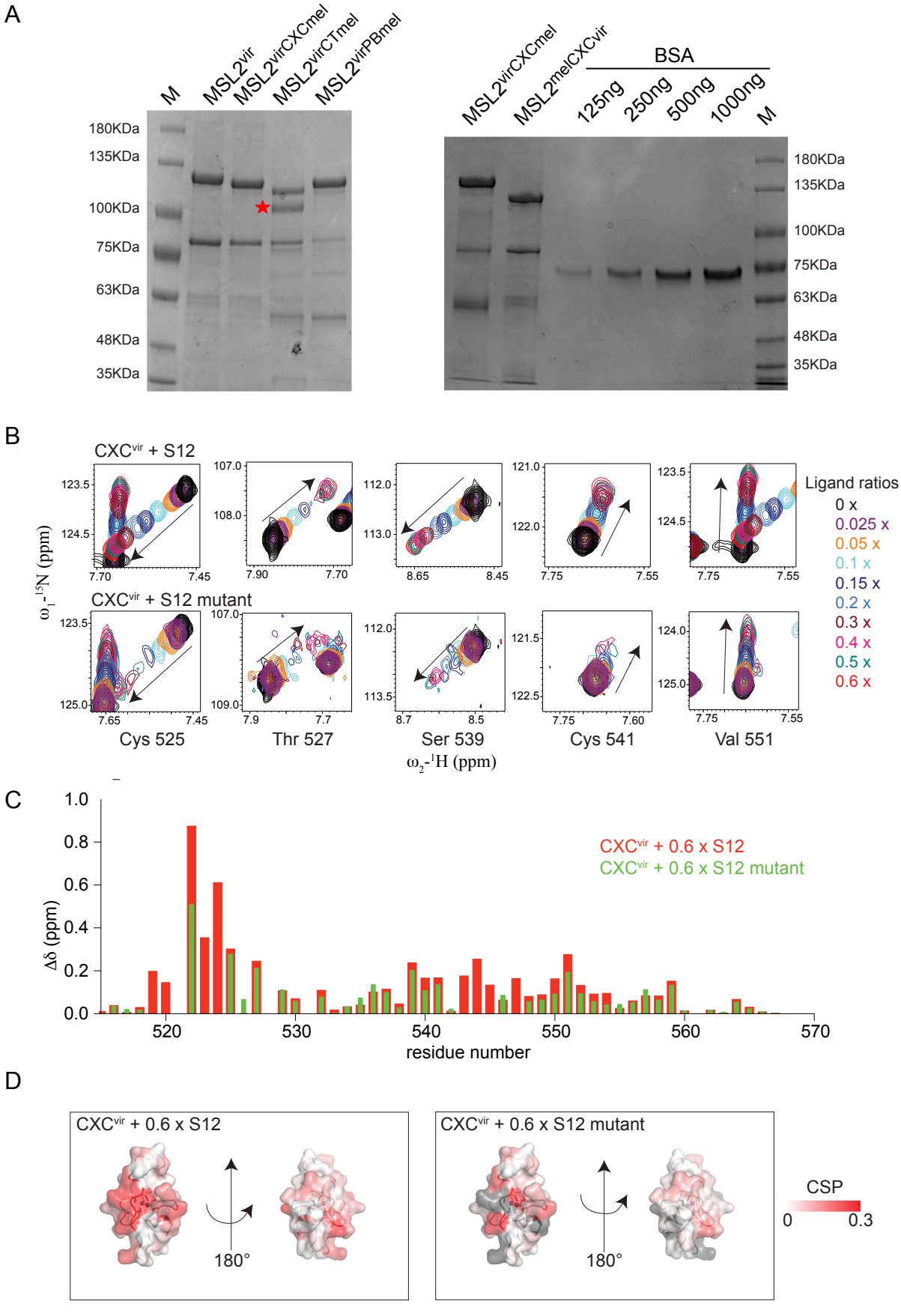
A
M
MSL2vir
MSL2virCXCmel
MSL2virCTmel
MSL2virPBmel
180KDa
135KDa
100KDa
75KDa
63KDa
48KDa
35KDa
MSL2virCXCmel
MSL2melCXCvir
BSA
125ng
250ng
500ng
1000ng
M
180KDa
135KDa
100KDa
75KDa
63KDa
48KDa
35KDa
B
CXCvir + S12
CXCvir + S12 mutant
ω1-15N (ppm)
Ligand ratios
0 x
0.025 x
0.05 x
0.1 x
0.15 x
0.2 x
0.3 x
0.4 x
0.5 x
0.6 x
Cys 525
Thr 527
Ser 539
Cys 541
Val 551
ω2-1H (ppm)
C
Δδ (ppm)
CXCvir + 0.6 x S12
CXCvir + 0.6 x S12 mutant
residue number
D
CXCvir + 0.6 x S12
180°
CXCvir + 0.6 x S12 mutant
180°
CSP
0
0.3

Figure S2. Characterization of the DNA binding specificity of the CXC^{vir} domain.

(A) Coomassie staining of proteins used in DIP assays. The MSL2^{virCTmel} runs as a double band due to a deletion at the N-terminus of the protein (star). Deletion at the N-terminus of MSL2 do not affect the DNA binding properties of the protein (Villa et al. 2016).

(B) Fitted NMR titration curves for few of the residues are shown with calculated affinity. Errors represent standard error of the group fitting of all the residues showing CSPs above mean.

(C) Residue wise chemical shift perturbation (CSP) plot upon titration with saturating concentrations of S12 (red) and S12 mutant (green) DNA in *D. virilis* CXC. Residue 526 in S12 DNA titration and residues 519, 520, 523, 524 and 543-545 during S12 mutant titration could not be tracked due to severe line broadening.

(D) CSPs plotted on the homology model of *D. virilis* CXC domain showing that the S12 and S12 mutant DNA binding surface is similar. The homology model was made SWISS-MODEL web server using crystal structure of *D. melanogaster* MSL2 CXC domain as template (Schwede et al. 2003). Resides which showed severe line broadening are shown in grey.