



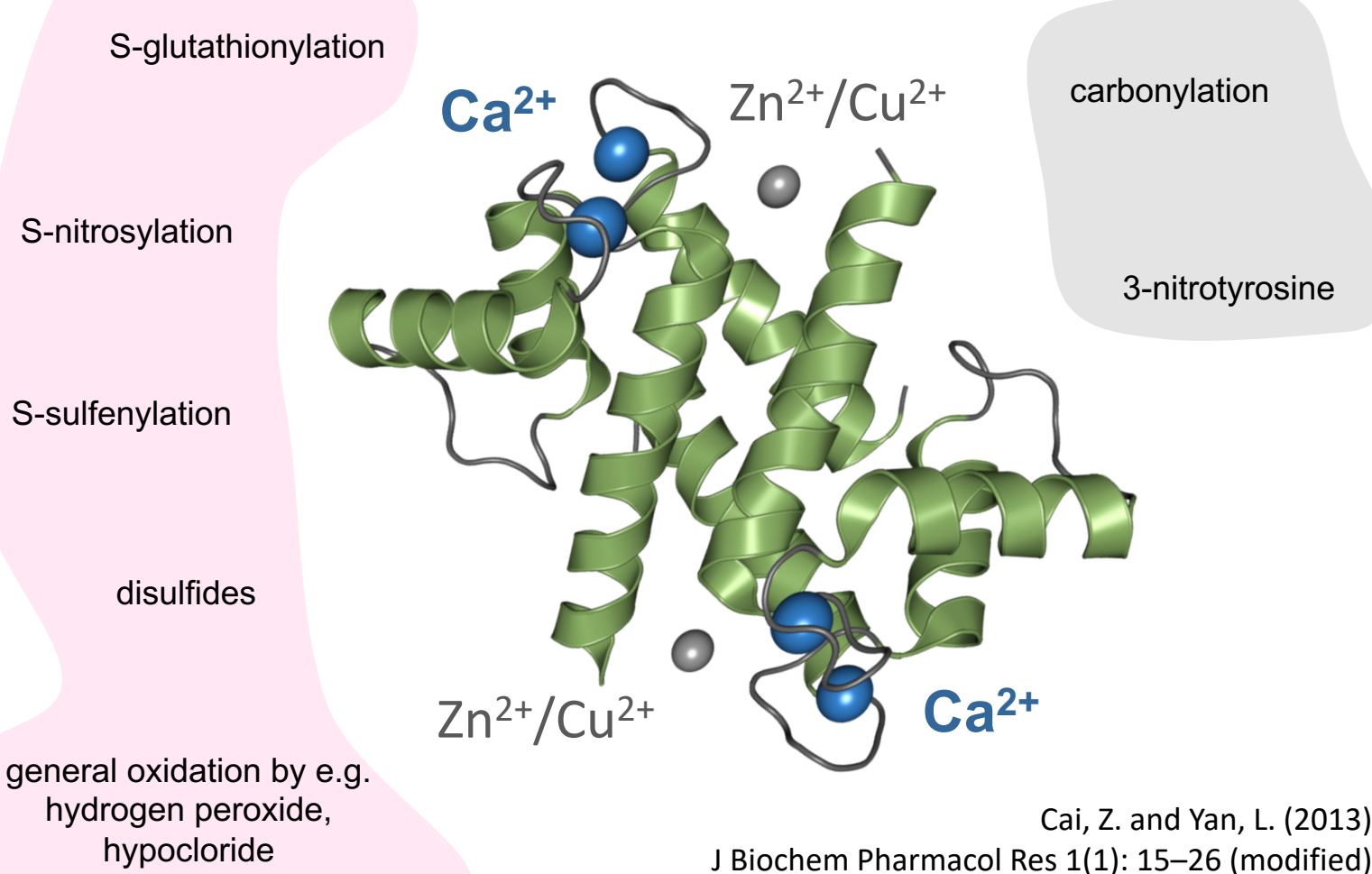
Oxidized S100B modulates its chaperone activity against A β aggregation

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Here we combine biochemical and biological experiments to report a role of oxidized S100B protein as possibly a key modulator of A β_{42} aggregation in Alzheimer's disease.

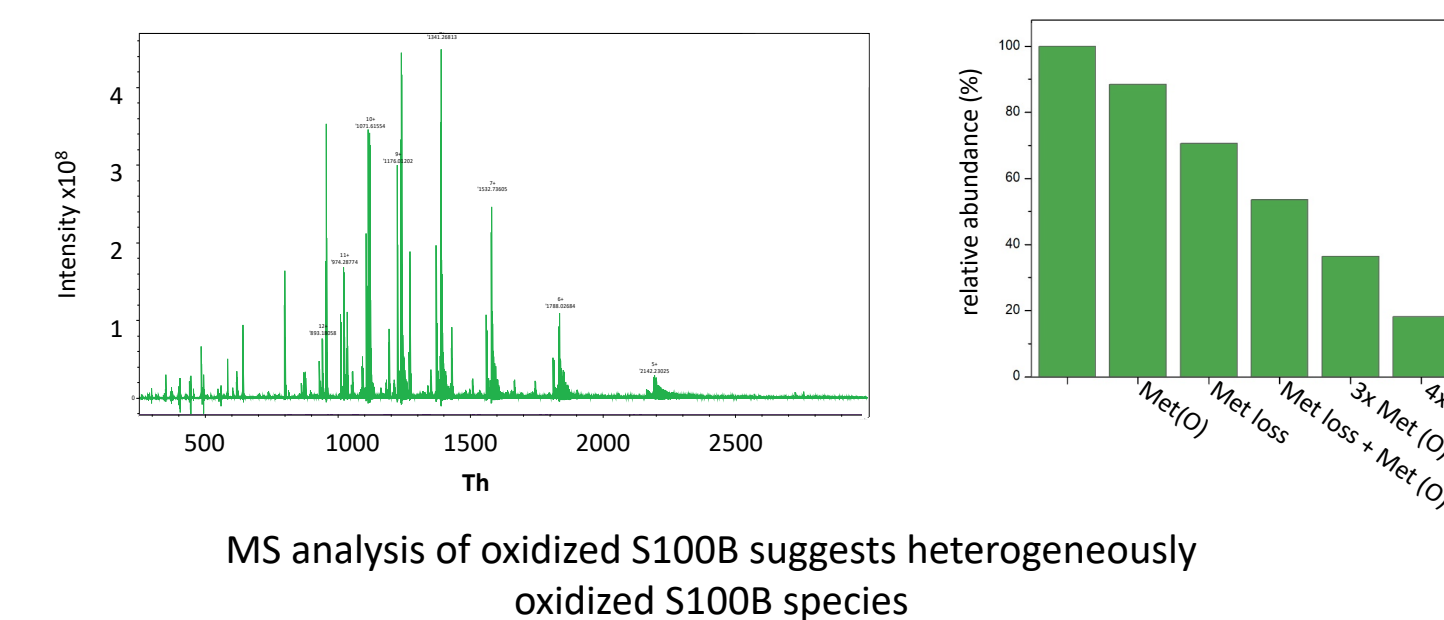


Oxidation protocol

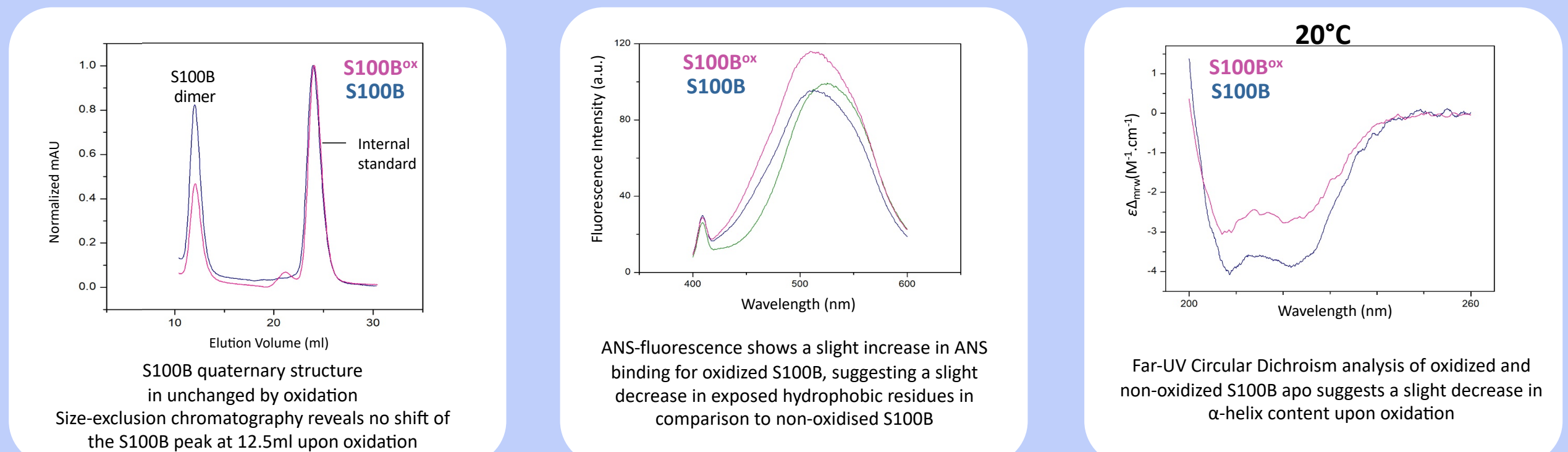
50 μ M S100B + 476nM hypochlorite
↓ 10 min, RT
centrifuge 5min, 12400rpm
↓
Size-exclusion chromatography
↓
concentrate protein
↓
store at -20°C

Rafferty, M. et al. 2001, J. Biol. Chem. 276: 33393-33401.

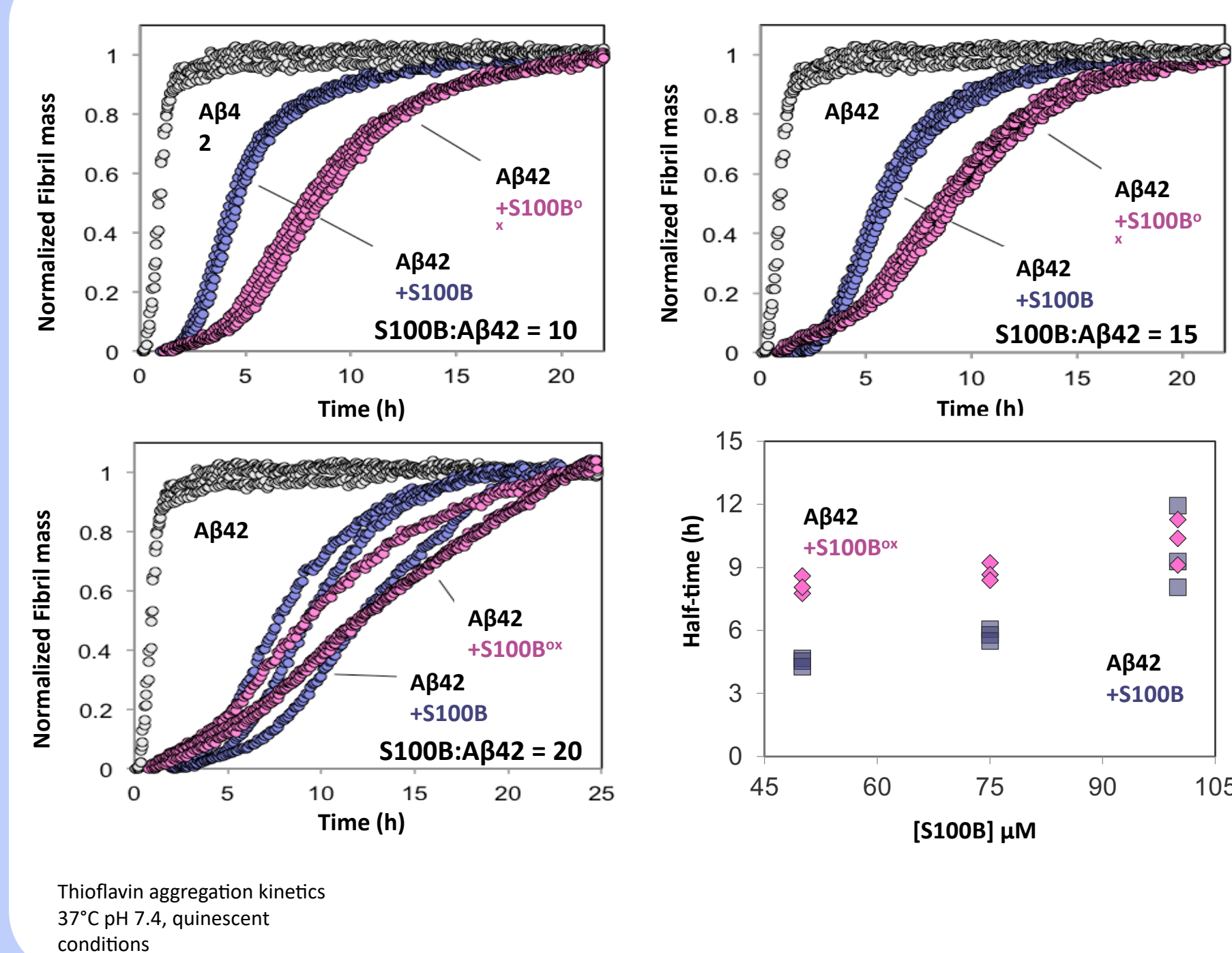
Mass spectrometry analysis of S100B^{ox}



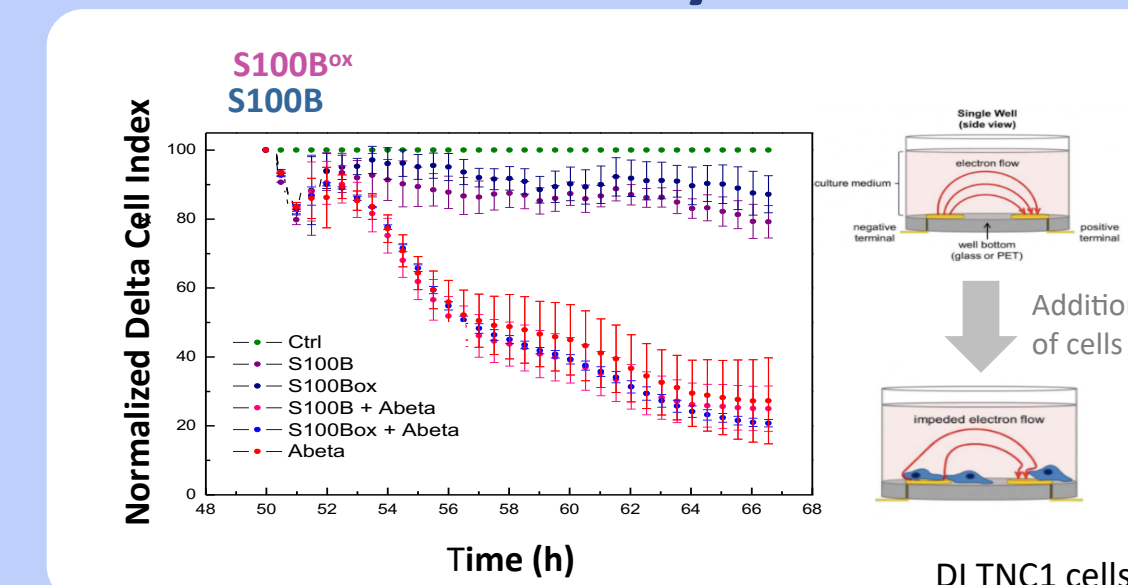
S100B structure and folding is not substantially affected by oxidation



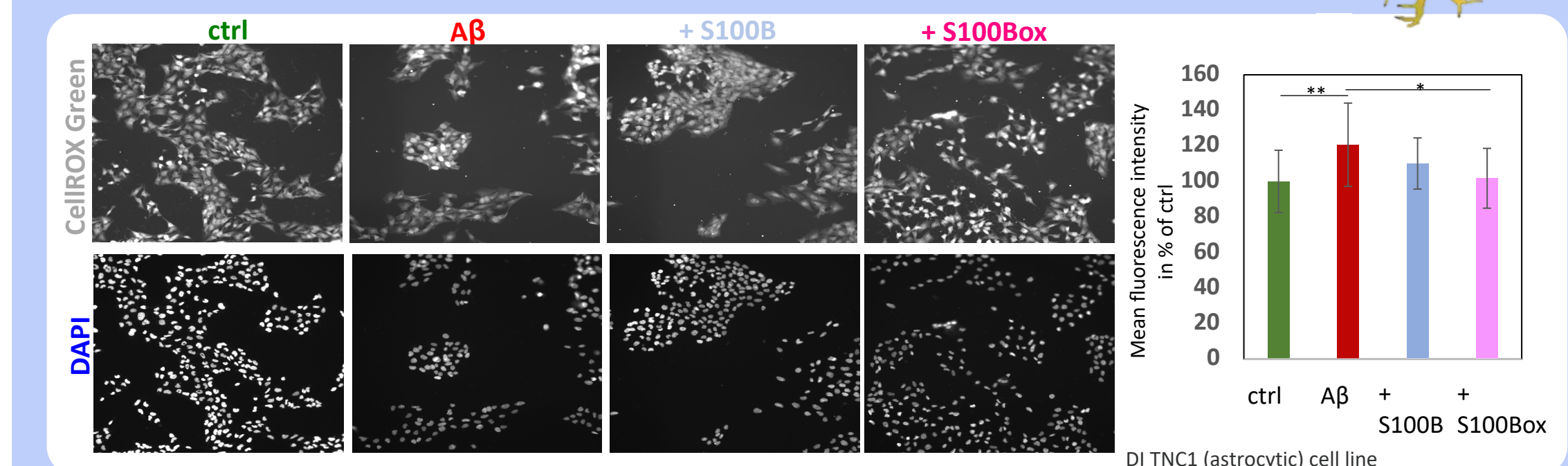
Oxidized S100B-Ca²⁺ more efficiently delays amyloid β aggregation



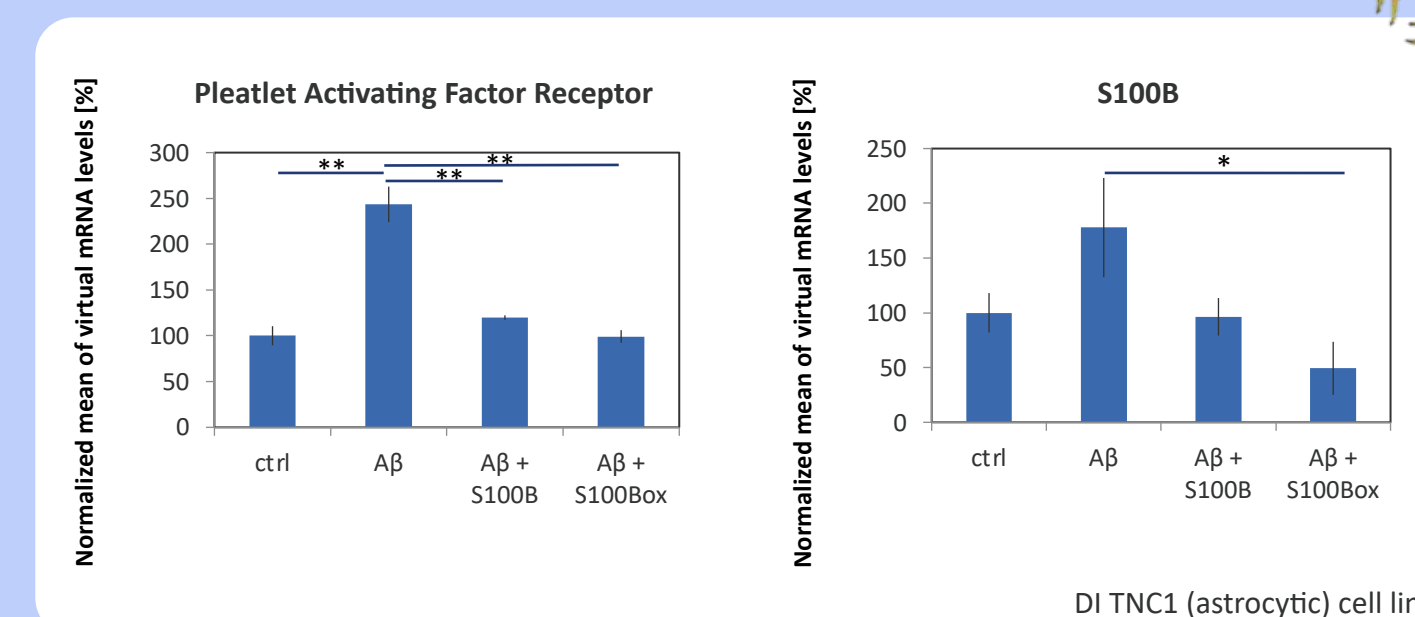
No toxicity rescue by S100B^{ox} in astrocytes



Effects of A β induced oxidative stress



Lower inflammatory marker expression with S100B^{ox}



The results that have emerged from this study show so far that oxidative modification of the calcium-activated S100B cytokine may potentiate its newly discovered chaperone activity against amyloid β species by interfering with fundamental processes of A β_{42} aggregation. While not interfering significantly with protein folding i.e. preserving its structural characteristics. Further, oxidized S100B shows a tendency of decreasing inflammation.

