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$\alpha-$synuclein aggregation and inhibition: the role of β-synuclein

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$\alpha-$synuclein (αS) is a small neuronal intrinsically disordered protein (IDP) that self-associates to form oligomers and fibrils in the brains of patients with Parkinson's disease. The highly homologous protein β-synuclein (βS) co-localizes with αS and can act as a neuro-protector of αS toxicity *in vivo* to inhibit pathological αS aggregation. Using NMR and biophysical approaches, we will discuss the molecular mechanisms of αS inhibition by βS and demonstrate the presence of an environmentally sensitive pH switch for βS that serves as an on/off fibrillation switch at mildly acidic physiological pH. These results have several implications for the role of βS in disease and highlight the complex interplay of αS and βS in the cell.

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