A Novel Approach to Alzheimer's Disease

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To date all approaches to developing an effective treatment for Alzheimer's disease have failed, and there is an increasing lack of confidence in the efficacy of combating the well-established targets amyloid or tau. Moreover, existing marketed therapies for Alzheimer's disease target symptoms in the late stages, but don't arrest progression. We have focused instead on identifying the pivotal basic mechanism and have subsequently suggested neurodegeneration is an inappropriate reactivation of a developmental mechanism that becomes toxic in the context of the mature brain, selectively in the subpopulation of primarily vulnerable cells. The key signalling molecule underlying this process is a 14mer peptide, 'T14'. T14 is (a) is doubled in the Alzheimer brain and CSF; (b) can potentially be monitored in plasma, as a blood biomarker, during the 10-20 years before cognitive impairment is apparent; (c) can drive the subsequent, secondary production of amyloid and p-tau; (d) can be intercepted pharmaceutically to stabilise any further cell loss by means of a first-in-class type of drug, i.e. a cyclised variant (NBP14) of the linear toxin T14 itself. NBP14 has significantly beneficial effects on the memory and brain histochemistry of Alzheimer model mice. The eventual goal will be to develop a treatment package whereby the biomarker monitored in a routine blood test could detect presymptomatic degeneration already underway, such that a variant of the cyclised T14 drug could be given immediately that stabilised any further cell loss. The symptoms of cognitive impairment would this be prevented from ever appearing, an effective 'cu.

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