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Oral presentation

Immunity and neuron: new evidence relating immunoglobulins to cytosceletal damage

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Previous studies have suggested an increased activation of humoral immunity in neurodegenerative diseases, it remains unclear whether this phenomenon is secondary to lesion formation or contributes directly to their development. Using stereotaxic injections in macaque monkey cerebral cortex, we studied the effects of human immunoglobulins on the neuronal cytoskeleton. Under these conditions, several MC-1-immunoreactive axons were observed in the vicinity of injection site. No MC-1 or TG-3 staining was detected in neuronal soma. Ultrastructurally, several axons in the same area displayed curly formations and accumulation of twisted tubules but not paired helical filaments. These data suggest that Fc fragment induce conformational changes of tau and subtle structural alterations in axons in this model. Immunocytochemical analyses in human autopsy materials revealed the presence of human Fc fragments as well as Fc receptors only in large pyramidal neurons known to be vulnerable in brain aging and Alzheimer's disease, further supporting a possible role of immunoglobulins in neurodegeneration. Moreover, the influence of human immunoglobulins Ig in neuronal cytoskeleton stability was studied in vitro. Here we show that human Ig and Fc fragments stimulate animal and human microtubule assembly by binding to microtubules via tau isoforms. In presence of Ig microtubules show increased aggregation twisting and rigidity. Non-immune Ig and Fc fragments promote microtubule assembly in temperature-dependent manner and stabilize microtubules at a molecular ratio of Ig per tubulin dimers. These in vitro data provide an experimental support for an immuno-mediated modulation of the cytoskeleton. These observations should be interpreted in conjunction with the consistent development of cytoskeletal pathology in the aged human brain, raising the possibility that nonspecific immune reactions may influence the neuronal cytoskeleton and participate in structural changes in the early phases of neurodegeneration