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The potential role of red blood cells in amyloid toxicity Alzheimer's disease and vascular dementia

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Oxidative stress is a primary cause for neuronal dysfunction and is considered a causative factor contributing to impaired cognitive function. Red blood cells (RBC) in intimate contact with the vasculature in the cerebral circulation are constantly undergoing autoxidation that is a source for oxidative stress. This process is enhanced by the uptake of amyloids, readily transferred from the vasculature to the circulating RBCs. Although the in vivo levels of RBC amyloids are very low, this is attributed to the transfer of amyloids back to the vasculature and the relatively rapid removal from circulation of RBCs containing amyloids. The transfer of amyloids from RBCs back to the vasculature contributes to the spread of cerebral amyloid angiopathy found in Alzheimer's disease (AD). The oxidative stress triggered while the amyloids are still associated with RBCs, is thought to be responsible for the impaired RBC morphology observed in subjects with AD, even after the amyloids are removed. These changes are responsible for the impaired deformability and adherence of RBCs to the endothelium, which contribute to cerebral hypoperfusion. These same modifications are also expected to result in the transfer or reactive oxygen species to the vasculature, which can trigger an inflammatory response and neuronal dysfunction. These studies suggest that although RBCs do not accumulate a significant fraction of the amloid generated during AD, the transient effects of the interaction of RBCs with amyloids can contribute to cerebral amyloid angiopathy, cerebral hypoperfusion and neuronal dysfunction associated with AD and other forms of vascular induced dementia.

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