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# Redox state of human serum albumin in serum and cerebrospinal fluid of multiple sclerosis patients

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Background/Aim: Like in various neurodegenerative diseases, oxidative processes are involved in the pathophysiology of multiple sclerosis (MS). Human serum albumin (HSA) is the most abundant protein in blood and other body fluids and can serve as an indicator for oxidative stress. We aimed to analyze the redox state of HSA in serum and cerebrospinal fluid (CSF) of MS patients and controls, and to further find potential associations with disease activity and severity.

Methods/Results: We performed HPLC on serum and CSF of 20 MS patients and 21 controls to determine the redox state of HSA regarding cysteine-34 (Cys-34) in allocation to the fractions human mercaptalbumin (HMA), human nonmercaptalbumin 1 (HNA1) and human nonmercaptalbumin 2 (HNA2). HMA is the reduced form of the protein, with a free thiol group on Cys-34. In HNA1, HSA is reversibly oxidized with Cys-34 as disulfide together with another thiol, like another cysteine, homocysteine, or glutathione, whereas HNA2 is irreversibly oxidized with Cys-34 as sulfinic or sulfonic acid. In CSF, we found significantly higher fractions of HMA than in serum, and lower fractions of HNA1 and HNA2. There was no significant difference between patients and controls, although CSF of patients showed a trend to higher HNA2 fractions. However, we found an association between albumin redox state in serum and physical disability in remission, and between albumin redox state in CSF and disease activity.

Conclusion: In conclusion, our data affirm the involvement of oxidative stress in MS pathophysiology. This study should serve as a basis for further investigation of HSA in MS, particularly in larger cohorts and patients with more advanced disease stages. Additionally, HSA appears to be an interesting molecule to explore still poorly understood redox processes in CSF.