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Neuroinflammation is regulated by angiotensin-related drugs: Possible implications for neurodegenerative disease

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Neuroinflammation is associated with the pathology of Alzheimer's disease (AD). Brain glial cells are responsible for the release of cytokines and reactive oxygen species that eventually leads to neuronal damage. Renin-Angiotensin System (RAS) is a hormonal system and its major effector is angiotensin II (Ang II). Lately this system has been discovered also in the brain. Ang II is formed from angiotensin I by angiotensin converting enzyme (ACE). There is increasing evidence that RAS may contribute to neuroinflammation associated with AD. Thereby, specific inhibition of brain RAS has been suggested as a potential therapeutic strategy for AD. In this study we examined the role of Ang II AT1 Receptor Blockers (ARBS) and angiotensin converting enzyme inhibitors (ACEI) in regulation of neuroinflammation. The *in vivo* study was conducted on transgenic mice that express five familial AD mutations (5 FAD). These mice exhibit amyloid plaque pathology similar to that found in AD. Intranasal administration of 1 mg/kg/day of Telmisartan (ARB) to 5FAD mice for 3-8 weeks resulted in a significant decrease of amyloid plaques, an important feature of AD, and in microglial activation in the hippocampuses and the cortex. Similar changes were observed in the cortex of mice treated with the same dose of Perindopril (ACEI). Our results indicate that intervention in the brain renin angiotensin system with ARBs or ACEI may serve as a new approach for the treatment of brain disorders, such as AD, where inflammation plays a significant role.

Biography

Sigal Fleisher-Berkovich has completed her PhD at the age of 29 years from Ben-Gurion University of the Negev. She is an Associate Professor in the department of clinical Biochemistry and Pharmacology. She has published papers and book chapters in reputed journals and serves as a reviewer of repute journals. She also received highly competitive national and international grants.

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