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Cerebral hypoperfusion and cerebrovascular protein alterations induced by diabetes

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Morbidity associated with uncontrolled diabetes includes cardiovascular disease, loss of sight and renal dysfunctions. Despite recent advances in diabetes treatment, an estimated 40% or more of people currently on available treatment options are not in optimal control of their disease. Therefore, an increased risk of experiencing diabetes complications exists. My research interests amongst others focus on the molecular pathology of diabetic complications, in particular cerebrovascular complications characterized by hypoperfusion/cerebral insufficiency, predisposition to stroke and worst stroke outcome in diabetes. Experimental studies have revealed that chronic dysregulation of glycemic control leads to deficits in cerebrovascular structure and function which may explain some of the clinical complications observed. Such observations as edema, inflammations, neovascularization, protease expression, cerebrovascular remodeling accompanied by altered vascular functions suggesting potential therapeutic targets. We have investigated the effects of diabetes on cerebral blood flow, signaling molecules and essential protein expressions in cerebral microvessles. The identification of potential proteins that can mediate altered cerebrovascular structure and function will serve as therapeutic targets. These protein targets can provide cerebrovascular protection which would not be limited to prevention of diabetes-induced deleterious changes in cerebrovascular structure/function before the occurrence of cerebrovascular incidence but can also be targeted acutely to preventing vascular dysfunction and loss of vascular integrity. The attending consequences and complications of diabetes induce-stroke can be abated with targeted intervention. Therefore, the results from this study would provide an insight into the cerebrovascular changes that is induced by chronic hyperglycemia; identify profiles of proteins affected which contribute to the remodeling and the cerebrovascular complications as therapeutic targets.

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