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Commentary
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Novel Evidence on hCdc14A as an *in vitro* Regulator in Cell Cycle Events in Diabetic Cerebrovascular Insults

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Short-Communication

Cell division cycle protein 14 (Cdc14) is an important cell cycle regulatory phosphatase. In budding and fission yeasts, Cdc14 can inactivate the activity of mitotic cyclin-dependent kinases (Cdks), which is required for mitotic exit and cytokinesis [1,2]. However, there are few published studies on the exact function of human Cdc14 (hCdc14) in cell cycle regulation. HCdc14 has two homologues, hCdc14A and hCdc14B. The report by Su et al. adds to the comprehensive knowledge on the *in vitro* regulatory effects of hCdc14A in diabetic cerebrovascular damage [3]. This study demonstrated an *in vitro* cerebrovascular damage model of diabetic stroke characterized by the combined addition of high glucose, free fatty acids (FFA), and hypoxia to cultured human brain vascular endothelial cells (HBVECs). One important issue is that the combined addition of the three stimuli largely blocks cell cycle progression and down-regulates hCdc14A expression. The preliminary results may have something to tell us that hCdc14A might be involved in the cell cycle regulation during the stimulus-mediated insults.

Another more important issue is that hCdc14A can accelerate and block cell cycle progression by conditional overexpression and small interfering RNA of hCdc14A, respectively, after exposure to three stimuli in cultured HBVECs. A recent study found that hCdc14A may directly contribute to cell migration and adhesion in tumor cells [4]. Despite the metastatic potential of tumors of hCdc14A, the role of hCdc14A in cerebrovascular endothelial damage during diabetic stroke remains unclear. This study [3] presents novel evidence to underscore the important role of hCdc14A in cell cycle progression under the combined stimulation of high glucose, FFA and hypoxia in HBVECs. However, there are still some limitations about this study. It should be pinpointed the effects of individual component from three stimuli on cell cycle progression and hCdc14A expression. Furthermore, the role of hCdc14A in cell cycle progression under individual stimuli of high glucose, FFA and hypoxia should be mentioned because these results will be useful and informative.

To summarize, this study is an important contribution to our understanding of pathological mechanism in cerebrovascular endothelial damage during diabetic stroke. Furthermore, it may also provide us with the clinical therapeutic targets for diabetic stroke in future studies.

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