



Title	Endothelium-specific activation of AMP-activated protein kinase alleviates diabetes-induced impairment in endothelial repair in mice
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The use of acupuncture in relieving dizziness, nausea, and vomiting after cerebellar stroke: a case report

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Introduction: Acupuncture is found to be effective in relieving chemotherapy-induced and postoperative nausea and vomiting. We sought to explore whether similar acupuncture technique is helpful in reducing dizziness, nausea, and vomiting after acute cerebellar stroke.

Methods: We report on a 66-year-old man who presented with sudden collapse in mid-August 2009 and was found to have acute bilateral cerebellar infarcts. He ran a very stormy acute course and was complicated by obstructive hydrocephalus and respiratory failure due to aspiration pneumonia. He was transferred to our rehabilitation unit at week 5 after stroke eventually. Patient remained bed- and chair-fast because of severe dizziness and nausea whenever he changed his positions. Acupuncture was applied on Neiguang (PC6) of both forearms. Even reinforcing-reducing needling technique was employed to achieve soreness, numbness and distension in the local areas. Needles were retained for 30 minutes and manipulated once every 10 minutes to intensify the needling sensation. The treatment was given once every other day, 3 times a week, for 2 weeks. Dizziness, nausea and vomiting were measured by global self-rating (nil or minimal, mild, moderate, and severe) and visual analogue scale (VAS) before and after treatment.

Results: The patient reported remarkable improvements with dizziness and nausea reduced from severe grade to mild grade and VAS from 8 to 3. No complication was observed.

Conclusion: Acupuncture by needling the Neiguang points is potentially beneficial. Further large-scale, randomised and controlled studies are warranted.

Endothelium-specific activation of AMP-activated protein kinase alleviates diabetes-induced impairment in endothelial repair in mice

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Introduction: Endothelial injury, an initiating step in various cardiovascular diseases, can be repaired in part by endothelial progenitor cell (EPC). Impaired functionality and reduced number of EPC are commonly observed in diabetic patients. AMP-activated kinase (AMPK) is a well-known target of several anti-diabetic and cardiovascular drugs. The objective of this study was to test whether or not activation of AMPK in endothelium alone is sufficient to prevent diabetes-induced impairment in EPC function and endothelial repair using a tissue-specific transgenic mouse model.

Methods: The transgenic mice with endothelium-selective expression of a constitutively active AMPK (AMPK-Tg) were generated using the T-cadherin gene promoter. Diabetes was induced by injection with streptozotocin (STZ). Bone marrow-derived EPCs (BM-EPCs) were assessed for (i) adhesion function and (ii) tubular formation capacity in vitro. Wire-mediated injury was introduced to the right common carotid artery of the mice which were allowed to recover for 3 days, and vascular repair was assessed using Evans blue staining. Post-injury circulating EPC numbers were quantified by flow cytometry analysis.

Results: In healthy mice, adhesion and tube formation were enhanced in BM-EPCs isolated from the AMPK-Tg mice when compared to wild type (WT) mice. These were, however, diminished in response to high glucose (25 mM) treatment in WT mice but not in AMPK-Tg EPCs. Re-endothelialisation after wire-mediated carotid injury was accelerated in AMPK-Tg mice and this was associated with a reduced number of circulating EPCs during the recovery process. In mice rendered diabetic with STZ, adhesion and tube formation of BM-EPCs were further impaired in wild type but not the AMPK-Tg mice in response to glucose (25 mM) treatment. Re-endothelialisation after wire injury was much less in WT mice compared to the AMPK-Tg mice, concomitant with a higher number of circulating EPCs after carotid injury.

Conclusion: These findings collectively suggest that endothelial activation of AMPK can prevent diabetes-induced endothelial injury through improving the EPC function. AMPK may represent an appealing therapeutic target for prevention of cardiovascular disease in diabetes.

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