

学会/受賞報告書

第75回日本循環器学会総会 学術集会 国際留学生YIA(Young Investigator's Award) 受賞

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今回、第75回日本循環器学会総会学術集会において、国際留学生 YIA (Young Investigator's Award) という大変素晴らしい賞をいただき、大変光栄に思います。また、ご指導いただいた下川宏明先生、加賀谷豊先生に心から感謝申し上げます。

今回、私は、遺伝子組み換えヒトエリスロポイエチン (EPO) が圧負荷心不全のマウスモデルにおいて、心筋保護効果を持つことを報告いたしました。本研究は、遺伝子組み換えヒトEPOが、圧負荷による左室収縮不全モデルにおいて、心筋保護効果を持ち、生命予後を改善することを示した初めての報告であり、臨床的にも意義の高い研究であると思っております。

このような名誉ある賞を頂いたことを喜ばしく思うと同時に、本賞に恥じぬよう一層の努力をしなければならぬと身の引き締まる思いでございます。将来も研究を続けていくことを強く希望しており、引き続き、母国中国において循環器領域の研究を継続していきたいと思っております。

受賞研究：

Cardioprotective Effects of Recombinant Human Erythropoietin in Pressure-Overloaded Hearts in Mice in Vivo

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抄録：

Background: We have demonstrated that endogenous erythropoietin (EPO)-EPO receptor system in non-hematopoietic cells plays a protective role against pressure overload-induced left ventricular (LV) dysfunction in mice. In the present study, we investigated that recombinant human EPO also exerts a protective effect against pressure-overload induced LV remodeling from a more clinical point of view.

Methods and Results: Mice subjected to transverse aortic constriction (TAC) (n=63) were randomly assigned to the treatment with PBS (TAC-PBS) or EPO (2000 U/kg twice a week) (TAC-EPO). At 8 weeks after TAC, LV weight and cardiomyocyte size were comparably increased in both TAC groups compared with sham-operated mice (Sham) (both $P<0.001$). EPO improved the survival of TAC mice compared with PBS (80 vs. 47%, $P<0.01$), which was associated with amelioration of myocardial fibrosis and cardiomyocyte apoptosis (both $P<0.05$). Echocardiography revealed that TAC increased LV chamber diameter and decreased LV fractional shortening compared with Sham ($P<0.05$), which was ameliorated by EPO ($P<0.05$). In TAC-EPO as compared to TAC-PBS, phosphorylations of STAT3, Akt and eNOS were increased, while that of p38 was decreased (all $P<0.05$). VEGF expression and capillary density in LV were similar among the 3 groups.

Conclusion: These results suggest that recombinant human EPO exerts cardioprotective effects against the development of cardiac remodeling and premature death induced by LV pressure overload by mechanisms independent of angiogenesis.