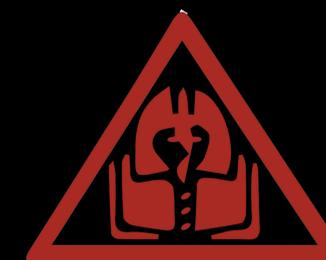


# Remote ischemic preconditioning regulates post-resuscitation microcirculation via a $K_{ATP}$ channel-dependent mechanism in a rat model of CPR

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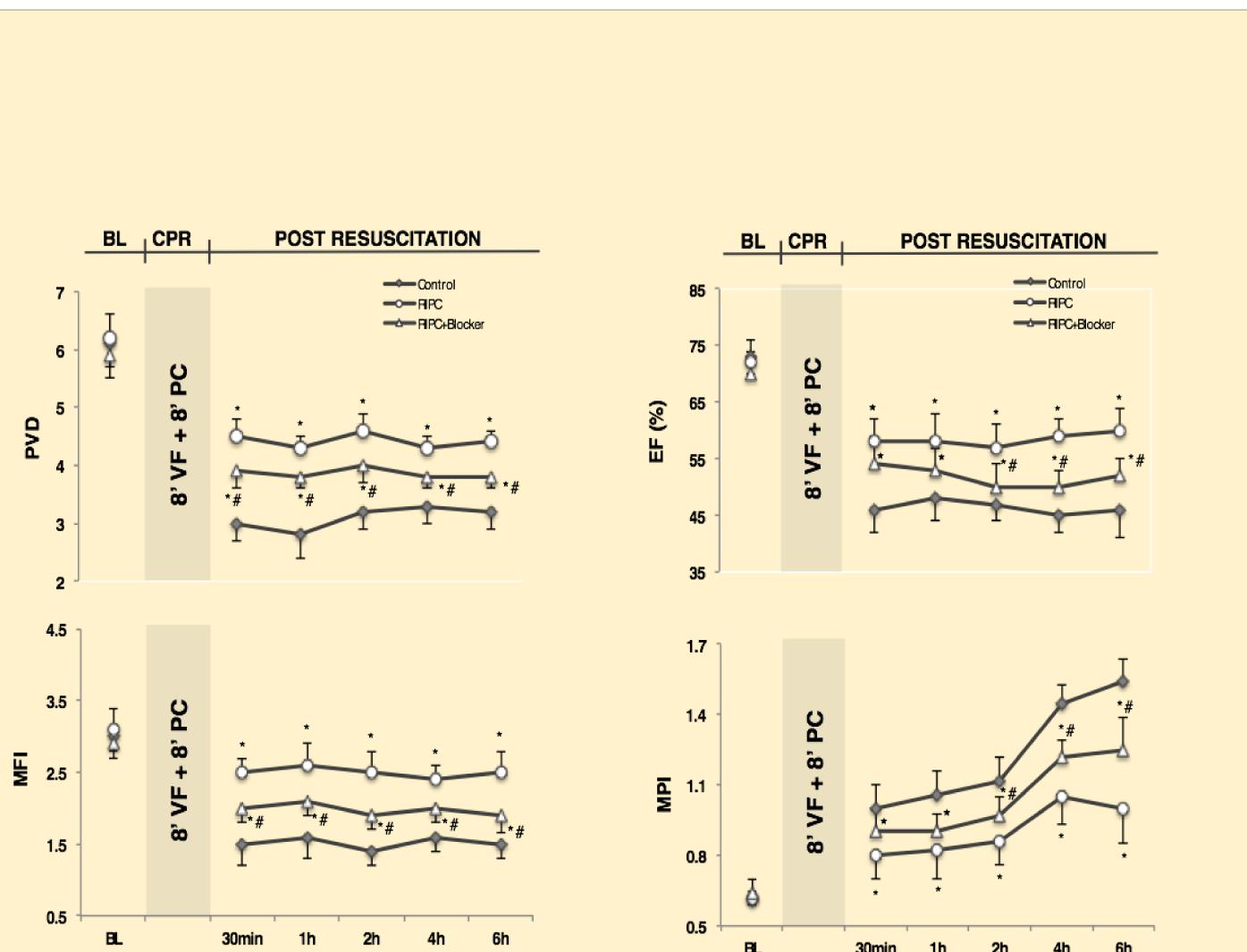
## Background

Remote ischemic preconditioning (RIPC) provides potent protection on tissue microcirculation against ischemic-reperfusion injury. However, the underlying microvascular mechanism of RIPC is not fully understood. This study investigated whether RIPC would reduce the impairment of sublingual microcirculation and improve the outcome of cardiopulmonary resuscitation (CPR) via  $K_{ATP}$  channel activation. We hypothesized that the activation of the  $K_{ATP}$  channel induced by RIPC would result in improved microcirculation and cardioprotection following resuscitation.

## Methods

Twenty-four male Sprague-Dawley rats were randomized into three groups (n=8 for each group): RIPC, RIPC with  $K_{ATP}$  channel blocker, and control. Remote ischemic preconditioning was induced by four cycles of 5 minutes of limb ischemia followed by reperfusion for 5 minutes. Ventricular fibrillation was induced and untreated for 8 minutes followed by 8 minutes of CPR. The animals were monitored for 6 hours and observed for an additional 66 hours. Sublingual microcirculation was assessed by a side stream dark-field imaging device, and myocardial function was measured by echocardiography at baseline, 30 minutes, 1 hour, 2 hours, 4 hours, and 6 hours post-resuscitation.

## Results



**Fig. 1. Changes of parameters of sublingual microcirculation.** PVD, perfused vessel density; MFI, microcirculatory flow index. BL, baseline; CPR, cardiopulmonary resuscitation; VF, ventricular fibrillation; PC, precordial compression. \* $P < 0.05$  versus Control group, # $P < 0.05$  versus RIPC group.

**Fig. 2. Changes of myocardial function.** EF, ejection fraction; MPI, myocardial performance index. BL, baseline; CPR, cardiopulmonary resuscitation; VF, ventricular fibrillation; PC, precordial compression. \* $P < 0.05$  versus Control group, # $P < 0.05$  versus RIPC group.

At 30 minutes and subsequent intervals post-resuscitation, both perfused vessel density and microcirculatory flow index were greater in the RIPC group than in the control group ( $P < 0.05$ ). Compared with the control group, better ejection fraction, myocardial performance index, and the duration of survival were observed in the RIPC group ( $P < 0.05$ ). Pretreatment with the  $K_{ATP}$  channel blocker (glibenclamide) reversed the microvascular changes and myocardial protective effects of RIPC ( $P < 0.05$ ) (Figure 1 and Figure 2).

## Conclusions

In a rat model of CPR, the beneficial effect of RIPC on sublingual microcirculation and cardiac function with improved survival was partly due to the activation of the  $K_{ATP}$  channel.

## References

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## Disclosure

None