

Role of Infiltrated Tissue Macrophages in Delayed Ischemic Preconditioning

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Thirty min of bilateral renal ischemia prior to ischemia/reperfusion (I/R), known as ischemic preconditioning (IP), protects the kidneys from I/R injury induced several weeks later in mice. However a detailed description of the protective mechanisms of IP remains to be defined. We hypothesized that macrophages infiltrated after IP contribute to the long-term protection. To test our hypothesis, IP mice underwent either IP or non-IP on day 0, were administered dichloromethylene bisphosphonate (Cl2MBP; clodronate, a remover of tissue macrophages) encapsulated by liposome intravenously on day 6, and were then exposed to ischemia on day 8. IP increased the accumulation of macrophages, and this increase was sustained for at least 8 days. Clodronate depleted macrophages in the ischemic preconditioned kidney, however, the protection afforded by IP was not abolished by clodronate. These results suggest that infiltrated tissue macrophages after ischemic preconditioning is not a critical factor for protection afforded by ischemic preconditioning.