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Effects of sevoflurane pretreatment on renal Src and FAK expression in diabetic rats after renal ischemia/reperfusion injury

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Abstract The diabetic kidney is sensitive to ischemia–reperfusion (I/R) injury due to microvascular complications, such as cellular apoptosis and necrosis. The aim of this study was to determine if sevoflurane pretreatment could help preserve renal function in rats with diabetes mellitus (DM) by altering non-receptor tyrosine kinases steroid receptor coactivator (Src) and focal adhesion kinase (FAK) expression (Src and FAK are mediators of cellular apoptosis and necrosis). Male rats ($N = 40$) were randomly assigned to one of five groups: Group A, sham operation; Group B, renal I/R injury; Group C, DM + sham operation; Group D, DM + renal I/R injury; and Group E, DM + sevoflurane pretreatment + renal I/R injury. Sevoflurane pretreatment comprised exposure to 2.5 % sevoflurane for 30 min, followed by exposure to air for 10 min. After 24 h, serum creatinine (Cr) and blood urea nitrogen (BUN) levels, and renal Src and FAK expression (immunohistochemistry) were assessed. Compared with rats in C, rats in D had significantly higher Cr and BUN levels, but significantly lower renal Src and FAK expression. Rats in E had significantly lower serum Cr and BUN levels and significantly higher renal Src and FAK expression levels than rats in D. Our findings suggest that sevoflurane pretreatment in rats with DM protects the kidneys from ischemia/reperfusion injury in part due to increased renal Src and FAK expression.

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Introduction

Ischemia/reperfusion (I/R) injury is a major cause of acute organ dysfunction and that often occurs in association with pathophysiological conditions, including major surgery, trauma, large area burns, and acute hypovolemia due to heavy fluid and blood loss. Although reperfusion after ischemia generally facilitates the recovery of organ dysfunction caused by ischemia, in some instances, reperfusion may exacerbate the damage associated with ischemia [1].

Diabetes mellitus (DM) is a metabolic disorder, the major complications of which include microvascular lesions and lesions in major vessels. As the kidney has a rich blood supply with abundant microvascular networks, it is particularly susceptible to DM-induced vessel lesions. With the occurrence of DM-induced vessel lesions, renal tolerance to I/R is significantly compromised and the kidney is more likely to develop acute renal failure [2]. Thus, investigating the mechanisms underlying I/R-induced renal injury in DM is crucial for improving clinical outcomes.

Renal I/R injury is mediated by multiple pathophysiological mechanisms of which cellular apoptosis and necrosis are two key events [3]. During renal I/R injury, apoptosis pathways are activated, resulting in extensive apoptosis of tubular epithelial cells [4]. Among these factors, non-receptor tyrosine kinases steroid receptor coactivator (Src) and focal adhesion kinase (FAK) are involved in the pathophysiology of apoptosis through multiple signaling pathways [5].

Src protein kinases play key roles in cell adhesion and movement, cell proliferation and survival, vasoconstriction,