



4th International Congress of Turkish Neuroendocrinology Society | ISTANBUL

26 | 28
November
2020



Melatonin may regulate apoptotic pathway via affecting Bax, Bcl2l1 and XIAP levels in myocardial ischemia-reperfusion injury

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Poster # PC36



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AIM: Restoring blood flow after ischemia is very important for maintaining the viability of ischemic tissue. The reperfusion of ischemic tissue causes enzyme degradation, an excessive increase in reactive oxygen species and secondary injuries such as apoptosis. Melatonin released from the pineal gland is one of the important endogenous antioxidants. This study aimed to investigate the effects of melatonin on pro-apoptotic B-cell lymphoma 2 associated X (Bax) and anti-apoptotic B-cell lymphoma 211 (Bcl211) and X-linked inhibitor of apoptosis (XIAP) levels in ischemia/reperfusion (I/R) injury in rat heart.

METHODS: Rats were randomly divided into 3 groups as control, I/R, and I/R+melatonin. The left main coronary artery was occluded for 30 min followed by 120 min reperfusion. Melatonin was administrated by intraperitoneal injection during the last 10 days. Bax, Bcl211 and XIAP levels were analyzed by real time-PCR. MDA levels were measured by spectrophotometry.

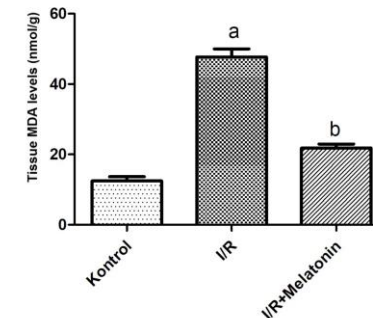
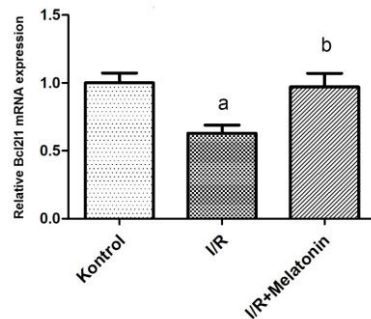
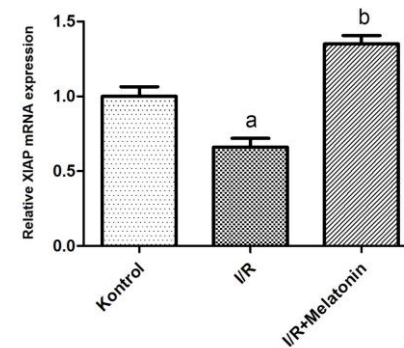
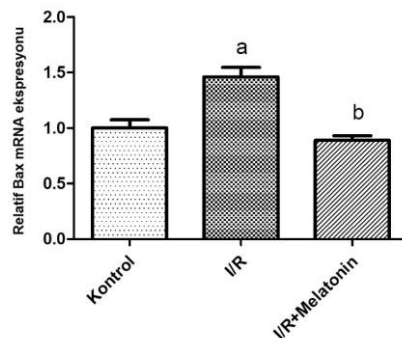


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RESULTS: The tissue Bax and MDA levels increased (1,47 and 3,82 fold, respectively) while Bcl211 and XIAP levels decreased with I/R injury. Melatonin administration showed protection against I/R induced myocardial injury by inhibiting all these changes.



a: Significant difference compared to control, b: Significant difference compared to I / R due to melatonin administration (p < 0.05).



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CONCLUSION:

Antiapoptotic XIAP and Bcl2l1, and proapoptotic Bax may be involved in signaling pathways in the pathology of myocardial I/R.

The protective effects of melatonin may be due to its activity in apoptotic pathways.