

Evaluation of miR-146a, miR-155, NF- κ B, TRAF6 and IRAK1 mRNA expression in the aorta of diabetic male rats

Dr. Mohammad Reza Alipour, Dr. Farhad Ghadiri Soufi, Shadi Sadat Emadi

Department of Physiology, Faculty of Medicine, Tabriz University of medical sciences

Abstract

Introduction: Diabetic angiopathy mechanism is complicated. Many parts of this mechanism has remained unknown. NF- κ B presence as a factor involved in diabetic inflammation has been illustrated in majority of studies. In addition to, some of miRNAs including miR-146a and miR-155 as regulating elements are noted in NF- κ B signaling pathway, recently. Therefore, the present study designed to evaluation miR-146a and miR-155 expression in aorta in probable signaling alters result from diabetes. Also this investigation evaluated IRAK1 and TRAF6 mRNA expression, ingredient of feedback loop between NF- κ B, miR-146a and miR-155.

Methods and Materials: Fourteen male wistar rats were randomly divided into control and diabetic groups (n=7 in each). Diabetes was induced by a single injection of streptozotocin (55 mg/kg; i.p.). The gene expression of miR-146a, miR-155, NF- κ B, IRAK1, and TRAF6 were determined by real time PCR.

Results: The expression of miR-146a was down-regulated in diabetic aorta when compared with the control group ($p < 0.05$). The mRNA expression levels of miR-155, NF- κ B, TRAF6 and IRAK1 also increased in the diabetic rat aorta when compared with their control counterparts ($p < 0.01$ for all comparisons).

Conclusion: These results suggest that it is possible miR-146a down-regulation result from hyperglycemic products lead to IRAK1 and TRAF6 activity and flowing diabetic aorta inflammation, by miR-146a-NF- κ B likely negative feedback loop. Furthermore miR-155 up-regulation can be inflammatory process provoker, because of miR-155-NF- κ B probable positive feedback loop.

Key words: Diabetes, miR-146a, miR-155, NF- κ B, Aorta