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비만 유발 생쥐에서 AICAR의 투여가 신장 합병증에 미치는 효과

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The Effect of AICAR on Renal Injury in Diet-induced Obesity Mice

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Background: Obesity itself can cause renal injury and chronic kidney disease. Although the inflammation in adipose tissue is suggested as the cause of various end organ damage in obesity, the exact mechanism of obesity induced renal injury is not clear yet. AMPK is a major regulator of energy metabolism found in various human organs. When activated, it induces weight reduction, improvement of lipid metabolism and decrease of insulin resistance. In this study, we analyzed the effect of AMPK activation on renal injury by administrating AICAR in diet-induced obesity mice model.

Material and Method: Thirty two 6-week-old C57/BL6 mice were devided into 3 groups. Group 1 was fed with normal diet. Group 2 and 3 were fed with high fat diet containing 60% of lipid for three months. Group 1 and 2 were injected with saline as control, and group 3 was injected with 500mg/kg of AICAR intraperitoneally 3 times a week. We measured body weight, the amount of proteinuria, serum creatinine, fasting blood glucose and Hba1c every month. After 3 months of experiments we sacrificed them and analyzed lipid metabolism, insulin resistance and structural change in kidney and adipose tissue.

Result: Twenty nine mice survived and were analyzed after 3 months. Obesity was induced by high fat diet in group 2. AICAR administration showed weight reducing effect in group 3. Although there was no difference in serum creatinine level among groups, the amount of proteinuria was significantly decreased in group 3 compared to group 2. However, there was no difference in the level of fasting blood glucose and Hba1c, so the anti-proteinuric effect of AICAR was not mediated by hypoglycemic effect. Moreover, total cholesterol, triglyceride and LDL levels were decreased in AICAR-treated group. However, there was no difference in HOMA-IR or profibrotic molecule expression in the kidney.

Conclusion: AICAR treatment showed a beneficial effect on kidney function in diet-induced obesity mice model and the effect was not mediated by hypoglycemic effect. Further studies will be needed to clarify the mechanism of obesity-induced renal injury and to find the new therapeutic target of obesity.

Key Words: 비만, 신손상, AICAR
Obesity, Renal injury, AICAR