# Metformin ameliorates testicular damage in diabetes and prostate cancer model

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Diabetes is a chronic metabolic disease which lasts for the whole life. Cancer is the second cause of death in the world, according to World Health Organization data. Association of diabetes with cancer is a major health concern. Diabetes and cancer is a serious metabolic disorder with many functional and structural complications as well as having a significant impact both directly and indirectly on all systems (1). Prostate cancer has a great importance for male morbidity and mortality observed both in our country and also in the globe. It is at the second rank among cancer-related mortality cases. Prostate cancer can be determined as the alteration of the balance between cell proliferation and cell death in the prostate which causes a malign increase of the organ volume. Dunning prostate cancer model is formed by subcutaneous injection of strongly metastatic MAT-Lylu cells in a Copenhagen rats (2). Experimental diabetes model is widely induced by streptozotocin (STZ). Metformin is a drug that used for the treatment of type 2 diabetes. Besides, the studies related to reduce the risk of cancer of the metformin have recently drawn attention (3). The aim of this study is to investigate the role of metformin on testicular damage in diabetic+prostate cancer model.

Male Copenhagen rats were divided into three groups: 1) Control group: % 0.9 physiological saline was received during 14 days, 2) Diabetic+ cancer group: $2\times10^4$ Mat-LyLu cells were received after injection of 65 mg/kg STZ. 3) Diabetic+ cancer+metformin group: metformin was received 250 mg/kg during experimental period, following injection of STZ and inocculation of Mat-LyLu cells. At the end of the experimental period (day 14) testes tissues were taken. Tissues were stained with hematoxylin and eosin and periodic acid-Schiff reaction and determined the degree of histopathological damage. The degree of histopathological damage in the seminiferous tubules

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were evaluated as: normal, regressive, degenerative and atrophic (4). Apoptotic cells in testes tissue were detected with TUNEL reaction. Biochemically, serum glucose, glutathione, malondialdehyde, prostate specific antigen levels and testis protein carbonyl levels and myeloperoxidase, xanthine oxidase activities were determined.

Testes tissue of the control group presented a normal testicular morphology and regular seminiferous tubules. The histopathological damage score of testicular tissue was significantly increased in diabetic+cancer group compared to control group. The number of regressive and degenerative tubules in diabetic+cancer+metformin group was decreased by metformin treatment. TUNEL positive cells were observed in all groups. The total number of TUNEL positive cells throughout the testes was increased in diabetic+cancer+metformin group compared to diabetic+cancer group. According to biochemical data, serum glutathione levels were decreased in diabetic+cancer group compared to control group. Serum glucose, malondialdehyde, prostate specific antigen levels, and testis protein carbonyl levels and myeloperoxidase and xanthine oxidase activities were increased in diabetic+cancer group. Treatment with metformin reversed these effects.

It was indicated that metformin has been shown to be protective against testicular damage in diabetic male rats (5). It was has been reported that metformin was used as protective agent to prevent high-fat diet induced testicular damage. Metformin inhibits the growth of cancer cell lines which suggests that it also has an inhibitory effect on cancer progression (6). Our results suggest that administration of metformin prevents the testicular damage by ameliorating the oxidative stress parameters and tissue damage. In conclusion, we can say that metformin has a potential protective effect on the testes tissue in diabetes and Dunning prostate cancer model.

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