Original Article

INHIBITION OF GROWTH FACTOR SIGNALING PATHWAYS BY IMATINIB MESYLATE IN MOUSE NORMAL LEYDIG CELLS

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Abstract

***Background & Aims*:** Cancer cells proliferation may be mediated by abnormal phosphorylation of signaling pathways downstream of tyrosine kinase receptors such as Platelet derived growth factor receptor α (PDGFR-α) and β (PDGFR-β). We aimed to study the phosphorylation level of PDGFR-α and PDGFR-β and apoptosis in mouse normal leydig cells being exposed to Imatinib.

***Materials & Methods*:** The mouse TM3 leydig cells were treated with 0, 2.5,5,10 and 20 μM Imatinib for 2, 4, and 6 days. The apoptosis and phosphorylation level of PDGFRs were assessed by caspase-3 activities colorimetric and fluorescence immunoassay methods, respectively. For statistical analysis, one-way ANOVA and T-test were performed.

***Results*:** Phosphorylation level of PDGFR-α in the treated (0.21±0.001) and control cells (0.35±0.13) was significantly different ($P<0.05$), and its level decreased with increasing drug dosage ($P<0.05$). PDGFR-β level and apoptosis had no significant differences between groups, although PDGFR-β level decreased significantly with increasing exposure duration ($P<0.05$).

***Conclusion*:** By inhibition of signaling pathways downstream of growth factors specifically PDGFR-α phosphorylation blockage in normal leydig cells, Imatinib may interfere with cellular growth. It seems that this drug has no effect on apoptotic pathways.

***Keywords*:** Apoptosis, Imatinib mesylate, Leydig cells, Platelet derived growth factor receptor

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