The sulfation pattern of heparan sulfate (HS) chains influences signaling events mediated by heparan sulfate proteoglycans (HSPGs) located on cell surface. These specific domains are critical for the interactions with growth factors and their receptors at cell surface. Human sulfatase-1 (SULF1) is a heparin-degrading extracellular endosulfatase that desulfates cell surface HSPGs. Endosulfatases removes 6-O sulfate from HS chains, thereby modulating growth factor activity. SULF1 protein is widely expressed in normal tissue, but has been associated with tumor suppressor effects in various human cancers, e.g., the ovarian, breast, pancreatic, hepatic, head and neck squamous cell carcinomas. However, SULF1 gene expression is increased in leukemia, in lung adenocarcinoma and in renal carcinoma compared to corresponding normal tissues. Therefore, SULF1 function in cancer is still complex, and its molecular mechanism has not been well understood. To further investigate the functions of SULF1 gene in regulating prostatic and colorectal tumorigenesis, we overexpressed this gene in DU-145 and HCT-116 cell lines, respectively. Transfection of DU-145 and HCT-116 cancer cells with a full-length SULF1 expression vector resulted in increased viability and proliferation. Furthermore, forced expression of SULF1 augmented cell migration in both cancer cell lines. Our results showed that SULF1 has an oncogenic effect in DU-145 and HCT-116 cancer cells, suggesting an important role of this enzyme in prostatic cancer and colorectal cancer progression.

Key-words: heparan sulfate, endosulfatase-1, cancer progression