PRINT: ISSN 0972-3757 ONLINE: ISSN 2456-6330
International Journal of
HUMAN GENETICS

Full text open access online (Since 2001)
Int J Hum Genet, 22(3): 233-241 (2022)
DOI: 10.31901/24566330.2022/22.03.836

PRINT: ISSN 0972-3757 ONLINE: ISSN 2456-6330

NTNG1 Modulates Progressions of Prostate Cancer Cells through JAK/STAT Signalling Pathway

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KEYWORDS Janus Kinase. Ki67. Netrin G1. Prostate Cancer. Signal Transducer. Activator of Transcription 3

ABSTRACT Netrin-G1 (NTNG1) is a glycosyl-phosphatidylinositol-affixed synaptic adhesion molecule that participates in carcinoma developments. However, its underlying mechanism in regulating prostate cancer (PCa) is uncertain. NTNG1 mRNA and protein expressions in prostate cancer tissue samples and cells were demonstrated to be promoted using RT-qPCR and western blot. Thereafter, using CCK-8, NTNG1 overexpression accelerated PCa cell viability while suppressed NTNG1 restrained cell viability. Additionally, transwell results indicated that migratory and invasive abilities of PCa cells were also facilitated by overexpressed NTNG1 but inhibited with NTNG1 suppression. Furthermore, using RT-qPCR, Janus kinase 1 (JAK1) has been detected to be upregulated by NTNG1 upregulation but suppressed with NTNG1 downregulation. Moreover, JAK1, JAK2, signal transducer and activator of transcription 3 (STAT3), Ki67 and E-cadherin protein expressions were also suppressed by the knockdown of NTNG1 but elevated with NTNG1 overexpression. In PCa cells, NTNG1 acted as an oncogene through activating JAK/STAT3 signalling pathway.