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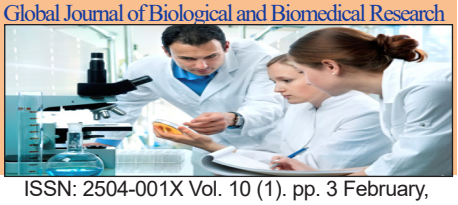

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Cell migration, cell invasion and cell proliferation in colonic epithelial cells

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ABOUT THE STUDY

Chemotherapy can improve quality of life and survival for several of chronic diseases. The development of anticancer drugs requires specific targeted signaling pathways or receptors. Endoplasmic reticulum stress (ERS) has been discussed for its multiple roles in can-cer. ERS causes apoptosis and helps cells adapt to harsh environments. ERS, on the other hand, helps cells avoid cell death and gain viability through up regulation of adaptive means. The endoplasmic re-ticulum stress response is an organized response program in the event of stimulating stress in the ER due to the accumulation of proteins. In cancer, UPR is postulated to set the paradoxical environment and aid in cancer therapy resistance. In addition, ERS has been linked to persistent inflammation in a variety of immunological and inflammatory disorder.

Cell migration refers to the ability of cells to move be-tween tissues or organs, whereas cell invasion refers to cancer cells' ability to overcome tissue barriers. Cancer metastasis is caused by a combination of cell movement and invasion, which results in cancer death. Therefore, it is obvious that the development of novel anticancer and anti Meta static drugs can be made possible only upon knowledge of metasta-sis and their mediated mechanisms. In this regard, screening for cell migration and infiltration during can-cer progression is paramount. We demonstrated the role of the heat shock protein Hspa5 in cell migration and infiltration and correlated the effects of ERS on cancer.

Glycosylation, on the other hand, plays a major role in glycoprotein folding, stability, intracellular localiza-tion, and biological function. Aberrant glycosylation has been identified as an essential hallmark of cancer and correlates with tumor development, progression, metastasis, and chemotherapy resistance. TUN blocks N-linked glycosylation by inhibiting the transfer of UD-PN-acetyl glucosamine to dichlorophosphate in the en-doplasmic reticulum (ER) of eukaryotic cells, thereby inhibiting protein maturation.

However, new evidence suggests that (ER) stress may have a role in several other aspects of the can-cer progression, including metastasis and proliferation. ER proteins implicated in ER stress, such as XBP1, PERK, ATF6, and ATF4, for example, have been linked to tumour growth and metastasis. Furthermore, no pre-vious work has reported on the time point-mediated detrimental consequences of TUN-induced ERS.

The role of ERS in cancer growth and metastasis has long been known, and it has been linked to the PI3K/AKT pathway. PI3K/AKT acts upstream of ER stress to affect fibroblast proliferation, resulting in bleomycin induced pulmonary fibrosis in a previous study. Treat-ment with endoplasmic reticulum stress inhibitors or PI3K inhibitors has been shown to normally reduce fi-broblast proliferation and improve lung function. Use of angiotensin inhibitors used to treat hypertensive patients, prolonging survival in patients with metastat-ic renal cell carcinoma, reducing tumor fibrosis, and improving chemotherapeutic effects in experimental cancer models, perhaps growth factor-stimulated PI3K signals Due to inhibition of transmission. These results also showed that ERS suppression reduced cancer cell proliferation and metastasis.