J Stem Cell Res Ther 2017, 7:9 (Suppl)
DOI: 10.4172/2157-7633-C1-030

Genetic and epigenetic clues: a promising insights to tame carcinoma drug resistance

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Background: In recent, several notions as genetic and epigenetic deregulation are proposed to contribute in existing cancer therapeutic options and their linked problems as resistance and relapse. One of genetic approaches has been suggested to look at the level of DNA repair responses towards genotoxic drugs. Among epigenetic contribution, epigenetic microenvironment and small regulatory RNAs are being explored to evaluate their therapeutic implications.

Methods: We have focused on genotoxic drugs such as cisplatin, doxorubicin and temozolomide. On the other side, DNA repair protein inhibitors including KU-55933, L189 and SCR-7. We have also employed microenvironment from breast carcinoma to test their inhibition towards HeLa. We have also tested small regulatory RNAs from maize corn in HeLa proliferation. To dissect out these genetic and epigenetic approaches for better cancer cytotoxicity, authors have employed cellular and molecular based techniques such as MTT, PI based cell estimation, FACS based cell cycle and cell death determination, confocal microscopy and immunoblotting assays.

Results and Discussion: Based on cellular and molecular analysis, present data strongly suggest the synergistic effect of drugs and inhibitors especially Dox plus DNA ligase inhibitors. Cisplatin plus KU-55933 and TMZ plus L189 could be better option in chemotherapy with significant reduction in dose of drugs. Our data from maize derived regulatory RNAs demonstrated strong inhibition of HeLa cell growth. In case of tumor microenvironment based experiment, non-cellular factors obtained from *in vitro* and clinical tissue samples, data suggest non-cellular microenvironment components of breast cancer that may block the growth of HeLa.

Conclusion: In conclusion, besides genotoxic drug therapy, utilizing genetic and epigenetic aspects could open up better responsiveness in carcinoma.

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