Research & Reviews: Journal of Medical and Health Sciences

Gene Therapy for Cancer Treatment

Chetna Thakur*

Department of Biotechnology, Lovely Professional University, Phagwara, Punjab, India

Review Article

Received date: 01/08/2016 Accepted date: 05/08/2016 Published date: 12/08/2016

*For Correspondence

Chetna Thakur, Department of Biotechnology, Lovely Professional University, Phagwara, Punjab, India, Tel: 9862871315.

E-mail: chetna.chts@gmail.com

Keywords: Cancer, Tumour, Vectors, Hereditary, DNA immunizations, Gene therapy.

ABSTRACT

Cancer is an unusual development of cells the proximate reason for which is unevenness in cell expansion and demise getting through the ordinary physiological governing rules framework and a definitive reason for which are one or even more an assortment of quality adjustments. These modifications can be basic, e.g. transformations, insertions, erasures, intensifications, combinations and translocations, or utilitarian (heritable changes without changes in nucleotide succession). No single genomic change is found in all diseases and different changes (heterogeneity) are normally found in every tumour by and large free of histology. In solid grown-ups, the safe framework may perceive and kill the malignancy cells or permit a non-hindering hosttumour balance; sadly, growth cells can some of the time get away from the safe framework bringing about extension and spread of these malignancy cells prompting genuine life debilitating sickness. Ways to deal with malignancy quality treatment incorporate three primary procedures: the insertion of a typical quality into disease cells to supplant a changed (or generally adjusted) quality, hereditary alteration to quiet a transformed quality, and hereditary ways to deal with straightforwardly slaughter the tumour cells.

INTRODUCTION

The possibility of quality treatment is one of a kind; it works by the expansion of a solid quality set up of inadequate or flawed applicant qualities so that the adjustments in the quality capacity in the patient's body are held and re-established ^[1]. It speaks to a way to treat an illness in a more inventive manner by utilization of hereditary materials prompting articulation of proteins in the cells which meddle with the amalgamation of proteins to treat maladies ^[2-25]. Distinctive vector frameworks perform quality exchange with specific focal points and detriments. A few specialized issues must be overcome before productive and complete cure are conceivable, and innovations should consistently be made strides. Like all medicinal treatments, quality treatment will improve a few, however not all sort of illnesses. Different methodologies are being analysed in clinical trials for quality treatment. Focusing on hereditary injuries of tumor cells, immunoaulation by quality treatment, hereditarily adjusted tumor antibodies in quality treatment, co-stimulatory particles, DNA immunizations, "Suicide" quality treatment, and so forth are the critical ones. Before quality treatment can turn into the system of decision for a wide assortment of clinical settings, there will be a change in the proficiency of quality move into target cells ^[26-37]. The issue of productive quality exchange will require not just further research to enhance viable conveyance frameworks and vector development additionally a parallel push to comprehend the science of the objective cells. A superior comprehension of allment etiology, pathogenesis particularly in acquired, mutagenic or gained infections will positively affect the treatment systems ^[38-40].

Concerning malignancy, introductory endeavors to deactivate oncogenes and supplant non-working tumor silencer qualities were scarcely fruitful. Consequently, new methodologies have been created to exchange hereditary materials (transgenes) specifically into target cells expecting to temporarily or for all time change their phenotypes. Target cells might be typical cells, carcinogenic cells, safe interceded cells, or pluripotent undifferentiated organisms ^[41,42]. Once the transgene enters a growth cell, it can then help with its passing or re-establish ordinary cell capacities, though for typical cells, the transgene can shield them from medication instigated toxicities, or enact a resistant cell to dispose of the malignancy cell. Quality and vector-based sub-atomic treatments for growth involve an extensive variety of treatment modalities to adjust disease cells, ordinary cells, and/or a tumor microenvironment ^[43-50].

e-ISSN:2319-9865 p-ISSN:2322-0104

PHYSICAL INTERVENED QUALITY EXCHANGE

DNA hereditary material that is covered with nanoparticles from gold or different minerals, and with their motor vitality supplemented by packed air or liquid (quality weapon), or utilizing ultrasound, can constrain the hereditary material into the objective cell, trailed by the arrival of DNA into its core ^[51-63]. They are most appropriate for quality conveyance into tissue or in the event of quality inoculation. The electroporation quality treatment approach plans to accomplish cell film disturbance with high-voltage electrical heartbeats, bringing about the arrangement of Nano pores through which stripped DNA, remote hereditary materials, and even chemotherapeutic specialists can enter cells. This methodology is most appropriate for plasmid DNA-based quality exchange treatment with the benefit of viability in an endless exhibit of cell sorts, simplicity of its organization, absence of genome incorporation with the danger of harm, and additionally the low potential for undesirable immunogenicity. Electroporation is in the blink of an eye being tried in a few clinical trials, particularly on patients with threatening melanoma, prostate disease, colorectal malignancy, and leukaemia ^[64-75].

SUBSTANCE INTERVENED QUALITY EXCHANGE

Cationic liposomes are minuscule vesicles of engineered phospholipids and cholesterol that can go into cells by endocytosis, with the capacity of conveying an assortment of atoms, for example, drugs, nucleotides, proteins, plasmids and extensive qualities ^[76-80]. Their favourable position is selectivity to endothelial cells, a generally high rate of quality exchange effectiveness, an expansive application as transporters for some qualities, and the absence of serious symptoms. At the point when consolidated with little meddling RNA (siRNA), cationic liposomes may prompt the hindrance of tumour multiplication, incitement of apoptosis, and improvement of radio sensitivity to tumour cells. Manufactured infections have been created to misuse the effectiveness of viral vectors and the benefit of liposomes. When they enter the objective cell, DNA is discharged from the endosome. This technique has indicated promising results in preclinical studies. Transposons can likewise transport hereditary material inside the cell and in addition into the core ^[81.87].

BACTERIAL INTERCEDED QUALITY EXCHANGE

A few microorganisms have the ability of particularly focusing on tumour cells, prompting RNA obstruction (RNAi) and quality hushing with blockage of RNA capacities, including cell digestion system and protein union. Illustrations incorporate *Escherichia coli, Salmonella typhimurium*, Clostridium and Listeria. Bacterial vectors can convey professional medication changing over chemicals and cytotoxic specialists into tumour cells, and can intercede the host invulnerable reaction ^[88-90]. They can be built to convey attractive or fluorescent material to upgrade the utility of demonstrative methodologies in tumour restriction, for example, with attractive reverberation imaging (MRI) and even in the improvement of growth immunizations. Notwithstanding, the result has been far less proclaimed contrasted with other RNA obstruction quieting strategies. Generally, hereditarily designed microscopic organisms going about as vectors for RNA obstruction are moderately protected, powerful, handy and less expensive to fabricate contrasted with viral vectors. They specifically colonize and develop inside the tumour. They can likewise be regulated orally, thus their utilization in the administration of gastrointestinal issue.

VIRAL INTERCEDED QUALITY EXCHANGE

Infections are little particles that contain either ribonucleic corrosive (RNA) or deoxyribonucleic corrosive (DNA), and might be single-stranded (ss) or twofold stranded (ds) ^[91.94]. The viral structure comprises of a genome encompassed by a defensive protein coat (viral capsid) which helps the infection append to host cell receptors, and avoids viral annihilation by cell nuclease catalysts. Some infections may likewise have a lipid bilayer envelope got from the host cell's film, and an external layer of viral envelope made of glycoprotein ^[95-98]. A complete viral molecule (virion) without anyone else can't reproduce. For spread, the infection needs to embed its hereditary material into a host cell, with a specific end goal to secure metabolic and biosynthetic items for viral translation and replication.

REFERENCES

- 1. Judson G and Genard H. Extending life: From stem cells to gene therapy. J Clin Med Genom 2016;4:140.
- 2. Amin SH and Muhammad TMB. Genetic counselling, pharmacogenetics and gene therapy: The paving- stones leading to brighter futures. Adv Genet Eng 2016;5:151.
- 3. Shivani K and Sivakumar JTG. Gene therapy: An overview. Gene Technol 2016;5:e116.
- 4. Liu X, et al. Evaluation of AAV-mediated gene therapy with reduced vector volume in Cngb3 knockout mice, a model of achromatopsia. Hereditary Genet 2016;5:163.
- 5. Zhang X, et al. Reflection on the efficacy of gene therapy in the treatment of inherited retinal degeneration. Clon Transgen 2015;5:e122.
- 6. Salam M, et al. Nano-materials for gene therapy: An efficient way in overcoming challenges of gene delivery. J Biosens Bioelectron 2016;7:195.

RRJMHS | Volume 5 | Issue 3 | September, 2016

- 7. Liu G. Non-viral reprogramming genes accelerate formation of neurons from murine embryonic brain cells: Synergistic effect of brain derived neurotrophic factor gene therapy. J Cell Sci Ther. 2015.
- 8. Shi D, et al. Gene therapy: Current situation and application prospect. Med Aromat Plants. 2016;5:E167.
- 9. Paul LH. Waking the sleeping giant: Gene therapy in decline? Clon Transgen. 2015;4:e115.
- 10. Navya P. Genetic syndromes and Involvement of gene therapy, cellular biology, microbial cell biology, signal transduction. Journal of Microbiology and Biotechnology. 2015.
- 11. Adriana C, et al. Genetic test and gene therapy for Krabbe disease: An update. Gene Technol. 2015;4:118.
- 12. Kajhoj TQ, et al. Test of critical steps towards a combined cell and gene therapy approach for the treatment of duchenne muscular dystrophy. J Mol Genet Med. 2015;9:160.
- 13. Cestmir A. Prodrug gene therapy for cancer mediated by mesenchymal stem/stromal cells engineered to express yeast Cytosinedeaminase::Uracilphos phoribosyltransferase. J Stem Cell Res Ther. 2015;5:264.
- 14. Makoto Y, et al. Successful induction of pluripotent stem cells from a Fabry disease mouse model: toward the development of safe lentiviral gene therapy. J Stem Cell Res Ther. 2015;20.
- 15. Maria G, et al. Molecular genetics and gene therapy aspects of phenylalanine hydroxylase (PAH) related hyperphenylalaninemias. J Genet Syndr Gene Ther. 2014;5:e125.
- 16. Hermonat PL, et al. Improving AAV gene therapy: Graduating from transgene expression? Everywhere, all the time? To? Disease-specific? Clon Transgen. 2014;3:e114.
- 17. Fumikazu K, et al. Adenovirus-mediated Bcl-XI gene therapy combined with pronase treatment protects the small intestine from radiation-induced enteritis in mouse model. J Genet Syndr Gene Ther. 2015;5:239.
- 18. Foldvari M. Nanopharmaceutics innovations in gene therapy: Moving towards non-viral and non-invasive delivery methods. J Nanomedine Biotherapeutic Discov. 2014;4:e135.
- 19. Daniel LJB. Delivery techniques in gene therapy: A brief overview. J Phys Chem Biophys. 2014;4:147.
- 20. Irfan A and Reshman S. Autologous gene therapy A proactive approach to cancer critique. J Cancer Sci Ther. 2014;6:4.
- 21. Julia T, et al. Gene therapy in rodents models of traumatic peripheral nerve injury. J Cell Sci Ther. 2014;5:156.
- 22. Xinhua S. Gene therapy for x-linked retinitis pigmentosa. Clon Transgen. 2014;3:e108.
- 23. Massimo C. Gene therapy gets its momentum through the marketing of an engineered virus to treat lipoprotein lipase deficiency. Adv Genet Eng. 2013;2:e104.
- 24. Michio N, et al. Gamma-delta T cells may function as carrier vehicles in adenovirus vector-based gene therapy. J Cancer Sci Ther. 2013;5:384-390.
- 25. Konrad R, et al. Suicide gene therapy against cancer. J Genet Syndr Gene Ther. 2013;4:187.
- 26. John MP and Grant DT. Identification of hematopoietic stem cell engraftment genes in gene therapy studies. J Stem Cell Res Ther. 2013;S3:4.
- 27. Alexander F, et al. Gene therapy using a secreted single chain variable fragment targeting CCR5 to inhibit HIV infection. J Antivir Antiretrovir. 2013;5:085.
- 28. Tuppurainen L, et al. Functional MRI measurements to predict early adenoviral gene therapy response in ovarian cancer mouse model. J Genet Syndr Gene Ther. 2013;4:171.
- 29. Paul Z, et al. Suicide gene therapy for cancer? Current strategies. J Genet Syndr Gene Ther. 2013;4:139.
- 30. Pedro MC and Maria CP. Viral and non-viral gene therapy for glioblastoma: New insights into the treatment of malignant brain tumors. J Genet Syndr Gene Ther. 2013;4:161.
- 31. Dabernat S, et al. Gene therapy of pancreatic cancer. J Genet Syndr Gene Ther. 2013;4:138.
- 32. Brunetti-Pierri N and Philip Ng. Adenoviral vectors for hemophilia gene therapy. J Genet Syndr Gene Ther. 2013;S1:017.
- 33. Connell MT, et al. Genetic syndromes and genes involved in the development of the female reproductive tract: A possible role for gene therapy. J Genet Syndr Gene Ther. 2013;4:127.
- 34. Jianhua M, et al. *In vitro* and *in vivo* model systems for hemophilia A gene therapy. J Genet Syndr Gene Ther. 2013;S1-014.
- 35. Fumiaki U and Sei-ichi T. Anticipation of a novel gene therapy inspired by a concept of iPS Cells. Pharm Anal Acta. 2012;3:196.
- 36. Ji-jing P. Gene therapy to treat inherited retinal diseases. Hereditary Genet. 2013;2:e107.
- 37. Marek M. Frontiers in suicide gene therapy of cancer. J Genet Syndr Gene Ther. 2012;3:119.

- 38. Patricia QL. Engineered factor VII, factor IX and factor X variants for hemophilia gene therapy. J Genet Syndr Gene Ther. 2013;S1-013.
- 39. Masato Y. Direction of gene therapy and virotherapy. J Cancer Sci Ther. 2013;5:1.
- 40. Marvin R, et al. Tumor compensation associated with gene therapy. J Genet Syndr Gene Ther. 2012;3:e117.
- 41. Marek M. Cancer suicide gene therapy. J Genet Syndr Gene Ther. 2012;3:e114.
- 42. Geoffrey LR and Brad EH. Optimal immunofluorescent staining for human factor IX and infiltrating T cells following gene therapy for hemophilia B. J Genet Syndr Gene Ther. 2013;S1-012.
- 43. Tung W, et al. The need for gene therapy for the effective treatment of hemophilia. J Genet Syndr Gene Ther. 2013;S1:016.
- 44. Kyung-Chul Y and Kyung KK. Gene therapy for corneal neovascularization. J Clin Exp Ophthalmol. 2012;3:e110.
- 45. Sanxia L, et al. Phase II study of post-surgery radiotherapy combined with recombinant adeno-viral human P53 gene therapy in treatment of oral cancer. J Cancer Sci. 2015.
- 46. Tong ML. Gene therapy for articular cartilage repair. Pharm Anal Acta. 2012;3:e112.
- 47. Denise ES and Valder RA. Muscle gene therapy for hemophilia. J Genet Syndr Gene Ther. 2013;S1-010.
- 48. Michael WL. Viral gene therapy in skeletal muscle: A work in progress. J Genet Syndr Gene Ther. 2012;3:e109.
- 49. Nicole S, et al. Safety modality for X-linked severe combined immunodeficiency gene therapy. J Cell Sci Ther. 2012;3:121.
- 50. Samir AF. Anti-metastatic gene therapy in patients with advanced epithelial ovarian cancer (EOC). J Cell Sci Ther. 2013;S15-001.
- 51. Ann P, et al. Corneal gene therapy in veterinary medicine: A review. J Veterinar Sci Technol. 2013;S8-001.
- 52. David MM and Roland WH. Liver-directed adeno-associated viral gene therapy for hemophilia. J Genet Syndr Gene Ther. 2013;S1-009.
- 53. Takayoshi W, et al. Double β-alanine substitutions incorporated in 12-ring pyrrole-imidazole polyamides for lengthened DNA minor groove recognition. Adv Tech Biol Med. 2016;4:175.
- 54. Esteban OP. Novel findings in familial non-medullary thyroid cancer genetics. Thyroid Disorders Ther. 2015;4:e119.
- 55. Rao AA, et al. Computational analysis of mutations in colon cancer genes reveals a possible role of micro satellite in mutagenesis. J Proteomics Bioinform. 2008.
- 56. Wang X. An Exploration of mutation status of cancer genes in breast cancers. Next Generat Sequenc & Applic. 2014;1:103.
- 57. Ponizovskiy MR. Biophysical and biochemical transmutation of mitochondrial function in cancer genesis. Biochem Anal Biochem. 2013;2:137.
- 58. Breast cancer genetic testing awareness, attitudes and intentions of Latinas living along the US-Mexico border: A qualitative study. J Community Med Health Edu. 2012;2:152.
- 59. Bonucci M. Integrated cancer therapy: Treat the person to cure the cancer. Interdiscip J Microinflammation. 2016.
- 60. Lay FD and Liang G. Rethinking demethylating agents in epigenetic cancer therapy. J Mol Pharm Org Process Res. 2016;4:133.
- 61. Khalid A and Javaid MA. Matrix metalloproteinases: New targets in cancer therapy. J Cancer Sci Ther/Vol.8.6 143. 2016.
- 62. Hussen RSD and Heidelberg T. Drug carriers in cancer therapy: Administration, formulation and characterization. IJPR. 2016.
- 63. Dong J, et al. Targeting ROS for cancer therapy. Chemotherapy (Los Angel). 2016;5:199.
- 64. Vaze OS. Pharmaceutical nanocarriers (Liposomes and Micelles) in cancer therapy. J Nanomed Nanotechnol. 2016;7:e138.
- 65. Varol M. Ultrasound-mediated cancer therapy as a non-invasive and repeatable treatment strategy. J App Pharm. 2016;8:2.
- Efferth T and Shan L. Natural products for cancer therapy â€Â is economic success Reachable? Med Aromat Plants. 2016;5:E174.
- 67. Rosaria CM. Addressing the potential role of fingolimod in cancer therapy. Med Chem (Los Angeles). 2016;6:195.
- 68. Fernández A. Anticancer therapy based on suppression of pathways recruited to cope with metabolic stress. Metabolomics. 2016;6:e144.
- 69. Her SC and Her C. Targeting DNA double-strand break repair in cancer therapy. J Mol Genet Med. 2015;9:E106.
- 70. Faes S and Dormond O. Systemic buffers in cancer therapy: The example of sodium bicarbonate; stupid idea or wise remedy? Med Chem (Los Angeles). 2015;5:540.
- 71. Delgado Y, et al. Development of HAMLET-like cytochrome c-oleic acid nanoparticles for cancer therapy. J Nanomed Nanotechnol. 2015;6:303.

- 72. Lu DY, et al. Cancer bioinformatics, its impacts on cancer therapy. Metabolomics. 2015;5:e133.
- 73. Molina AM, et al. Redox-sensitive cross-linking enhances albumin nanoparticle function as delivery system for photodynamic cancer therapy. J Nanomed Nanotechnol. 2015;201.
- 74. Tripathi V. Part of tumor suppressor protein p53 in apoptosis and cancer therapy. RRJMHS. 2015.
- 75. Revathi B and Prashanth K. Potential Hsp90 inhibitors: A novel target for cancer therapy. Chemotherapy (Los Angel). 2015;4:146.
- 76. Liu B, et al. Seven protective miRNA signatures for prognosis of cervical cancer. Oncotarget. 2016.
- 77. Zhao S, et al. BTG1 might be employed as a biomarker for carcinogenesis and a target for gene therapy in colorectal cancers. Oncotarget. 2016.
- 78. Wang M and Ma H Paired box gene 2 is associated with estrogen receptor α in ovarian serous tumors: Potential theory basis for targeted therapy. Mol Clin Oncol. 2016;5:323-326.
- 79. Goto A, et al. Genotype frequencies for polymorphisms related to chemotherapy-induced nausea and vomiting in a Japanese population. J Pharm Health Care Sci. 2016;21;2:16.
- Chung T, et al. Dihydropyrimidine dehydrogenase is a prognostic marker for mesenchymal stem cell-mediated cytosine deaminase gene and 5-fluorocytosine prodrug therapy for the treatment of recurrent gliomas. Theranostics. 2016;6:1477-1490.
- 81. Yang X, et al. Cooperative antiproliferative effect of coordinated ectopic expression of DLC1 tumor suppressor protein and silencing of MYC oncogene expression in liver cancer cells: Therapeutic implications. Oncol Lett. 2016;12:1591-1596.
- 82. Luo G, et al. Expression levels of JNK associated with polymorphic lactotransferrin haplotypes in human nasopharyngeal carcinoma. Oncol Lett. 2016;12:1085-1094.
- 83. Sun B, et al. Combined treatment with everolimus and fulvestrant reversed anti-HER2 resistance in a patient with refractory advanced breast cancer: A case report. Onco Targets Ther. 2016;9:3997-4003.
- 84. Dirican E and Akkiprik M Functional and clinical significance of SALL4 in breast cancer. Tumour Biol. 2016
- 85. Judson PL, et al. Complementary and alternative medicine use in individuals presenting for care at a comprehensive cancer center. Integr Cancer Ther. 2016.
- Li J, et al. Reversal of multidrug resistance in breast cancer MCF-7/ADR cells by h-R3-siMDR1-PAMAM complexes. Int J Pharm. 2016;S0378-5173:30669-X.
- 87. Yap TA, et al. Drug discovery in advanced prostate cancer: translating biology into therapy. Nat Rev Drug Discov. 2016.
- 88. Yan W, et al. Genetic alteration and mutation profiling of circulating cell-free tumor DNA (cfDNA) for diagnosis and targeted therapy of gastrointestinal stromal tumors. Chin J Cancer. 2016;35:68.
- 89. Gregson EM, et al. Genetic progression of Barrett's oesophagus to oesophageal adenocarcinoma. Br J Cancer. 2016;21.
- 90. Dhanik A, et al. *In silico* discovery of cancer-specific peptide-HLA complexes for targeted therapy. BMC Bioinformatics. 2016;17:286.
- 91. Effenberger KA, et al. Modulating splicing with small molecular inhibitors of the spliceosome. Wiley Interdiscip Rev RNA. 2016.
- 92. Ahmed S, et al. Enhanced protein internalization and efficient endosomal escape using polyampholyte-modified liposomes and freeze concentration. Nanoscale. 2016.
- 93. Viswanathan P, et al. Thalidomide promotes transplanted cell engraftment in the rat liver by modulating inflammation and endothelial integrity. J Hepatol. 2016;S0168-8278:30332-30334.
- 94. Walline HM, et al. Genomic integration of high-risk HPV alters gene expression in oropharyngeal squamous cell carcinoma. Mol Cancer Res. 2016;0105.2016
- 95. Campo C, et al. Genetic susceptibility to bortezomib-induced peripheral neuroropathy: Replication of the reported candidate susceptibility loci. Neurochem Res. 2016.
- 96. Burga RA, et al. Improving efficacy of cancer immunotherapy by genetic modification of natural killer cells. Cytotherapy. 2016;pii:S1465-3249:30411-X.
- 97. Liang X, etv al. A folate receptor-targeted lipoplex delivering interleukin-15 gene for colon cancer immunotherapy. Oncotarget. 2016.
- 98. Ye H, et al. Ranking novel cancer driving synthetic lethal gene pairs using TCGA data. Oncotarget. 2016.