



Advancing Therapeutic Limits and Modified Chitosan for Precision Gene Delivery

Aneesh Foged*

Department of Pharmacy, University of Copenhagen, Copenhagen, Denmark

DESCRIPTION

Gene therapy is efficient delivery system is important for the success and safety of the treatment. Modified chitosan, a natural polymer derived from chitin, has emerged as candidate for gene delivery technologies [1]. The innovative world of modified chitosan-based gene delivery, exploring its potential for therapeutic advancements, the modifications enhancing its efficacy, and the exciting avenues it opens for the future of precision medicine. Chitosan, derived from the exoskeletons of crustaceans, is known for its biocompatibility, biodegradability, and low toxicity. These inherent qualities make chitosan an attractive candidate for gene delivery [2]. However, to optimize its performance and overcome certain limitations are turning to modifications that enhance its ability to efficiently ferry genetic cargo into target cells. Advancing chitosan-based gene delivery is surface modification. By altering the surface characteristics of chitosan nanoparticles, can improve their stability, cellular uptake, and ability to protect genetic material during transit [3]. Surface modifications with Poly Ethylene Glycol (PEG) or other polymers enhance the stealthiness of chitosan nanoparticles, reducing their recognition by the immune system and prolonging circulation times in the bloodstream.

Chitosan's effectiveness as a gene delivery vehicle depends on its ability to navigate cellular barriers and reach the nucleus where genetic material exerts its therapeutic effects [4]. Modifications such as the incorporation of cell-penetrating peptides or ligands that target specific cell receptors enhance the cellular uptake of chitosan nanoparticles. This targeted approach not only improves delivery efficiency but also minimizes off-target effects. The physiological conditions within cells vary, and modified chitosan can be designed to respond to these changes [5]. PH-responsive modifications, where chitosan nanoparticles release their cargo in response to the acidic environment of endosomes or lysosomes, enhance the precision of gene delivery. This smart release mechanism minimizes premature release, ensuring that the therapeutic payload reaches its intended destination intact.

The stability of nucleic acids, such as DNA or RNA, during their journey from the site of administration to the target cells is a critical consideration in gene delivery [6]. Modified chitosan, through various modifications, can provide protection to the fragile genetic material. Encapsulation within chitosan nanoparticles shields nucleic acids from enzymatic degradation, making it possible to preserve the integrity of the therapeutic cargo until it reaches the target cells. For gene therapy to translate from laboratory studies to clinical applications, the delivery system must be tailored to navigate the complexities of the environment [7]. Modified chitosan offers versatility in adapting to the biological milieu. Surface modifications can be fine-tuned to optimize interactions with the immune system, ensuring minimal immune response and maximizing the therapeutic impact of delivered genes.

Modified chitosan's ability to facilitate targeted gene delivery opens new vistas for precision medicine. Targeted therapies, designed to address specific molecular characteristics of diseases, can benefit from approach enabled by chitosan modifications [8]. Whether targeting cancer cells with specific surface markers or addressing genetic mutations responsible for rare diseases modified chitosan holds the potential to revolutionize the landscape of targeted gene therapies. The regenerative potential of gene therapy finds modified chitosan-based delivery systems. In regenerative medicine, where the goal is to repair or replace damaged tissues, the controlled release of therapeutic genes can stimulate tissue regeneration. Modified chitosan's adaptability allows for the incorporation of growth factors or specific genes to promote tissue healing, making it a valuable tool in advancing regenerative therapies. While modified chitosan holds great promise, challenges remain [9]. Fine-tuning the balance between stability and release, optimizing surface modifications for specific applications, and addressing potential immunogenicity are areas. Future directions in chitosan-based gene delivery include exploring combination therapies, integrating advanced imaging techniques for monitoring, and refining delivery systems to achieve even greater precision.

Correspondence to: Aneesh Foged, Department of Pharmacy, University of Copenhagen, Copenhagen, Denmark, E-mail: aneeshfo@gmail.com

Received: 01-Mar-2024, Manuscript No. RDT-24-25266; **Editor assigned:** 04-Mar-2024, Pre QC No. RDT-24-25266 (PQ); **Reviewed:** 18-Mar-2024, QC No RDT-24-25266; **Revised:** 25-Mar-2024, Manuscript No. RDT-24-25266 (R); **Published:** 01-Apr-2024, DOI: 10.35248/2329-6682.24.13.276

Citation: Foged A (2024) Advancing Therapeutic Limits and Modified Chitosan for Precision Gene Delivery. Gene Technol.13:276.

Copyright: © 2024 Foged A. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Modified chitosan-based gene delivery technologies represent for effective and targeted gene therapies. The full potential of chitosan modifications, the landscape of precision medicine is undergoing a transformative shift. From overcoming cellular barriers to responding intelligently to the biological milieu, modified chitosan offers a versatile platform for advancing therapeutic interventions [10]. The journey from laboratory innovation to clinical reality is propelled by the modified chitosan, for a future where gene therapies can be precisely tailored to the unique characteristics of individual patients.

REFERENCES

1. Borst P, Schinkel AH. P-glycoprotein ABCB1: a major player in drug handling by mammals. *J Clin Invest.* 2013;123:4131-4133.
2. Cascorbi I. P-glycoprotein: tissue distribution, substrates, and functional consequences of genetic variations. *Handb Exp Pharmacol.* 2011:261-283.
3. Nakanishi T, Ross DD. Breast cancer resistance protein (BCRP/ABCG2): its role in multidrug resistance and regulation of its gene expression. *Chin J Cancer.* 2012;31:73.
4. Shitara Y. Clinical importance of *OATP1B1* and *OATP1B3* in drug-drug interactions. *Drug Metab Pharmacokinet.* 2011;26:220-227.
5. Kullak-Ublick GA, Ismair MG, Stieger B, Landmann L, Huber R, Pizzagalli F, et al. Organic anion-transporting polypeptide B (OATP-B) and its functional comparison with three other OATPs of human liver. *Gastroenterology.* 2001;120:525-533.
6. Hsiang B, Zhu Y, Wang Z, Wu Y, Sasseville V, Yang WP, et al. A novel human hepatic organic anion transporting polypeptide (OATP2): identification of a liver-specific human organic anion transporting polypeptide and identification of rat and human hydroxymethylglutaryl-CoA reductase inhibitor transporters. *J Biol Chem.* 1999;274:37161-37168.
7. Lau YY, Huang Y, Frassetto L, Benet LZ. Effect of OATP1B transporter inhibition on the pharmacokinetics of atorvastatin in healthy volunteers. *Clin Pharmacol Ther.* 2007;81(2):194-204.
8. Zamek-Gliszczynski MJ, Chu X, Cook JA, Custodio JM, Galetin A, Giacomini KM, et al. ITC commentary on metformin clinical drug-drug interaction study design that enables an efficacy-and safety-based dose adjustment decision. *Clin Pharmacol Ther.* 2018;104(5):781-784.
9. Masuda S, Terada T, Yonezawa A, Tanihara Y, Kishimoto K, Katsura T, et al. Identification and functional characterization of a new human kidney-specific H⁺/organic cation antiporter, kidney-specific multidrug and toxin extrusion 2. *J Am Soc Nephrol.* 2006;17(8):2127-2135.
10. Tanihara Y, Masuda S, Sato T, Katsura T, Ogawa O, Inui KI. Substrate specificity of MATE1 and MATE2-K, human multidrug and toxin extrusions/H⁺-organic cation antiporters. *Biochem Pharmacol.* 2007;74:359-371.