

A REVIEW PAPER ON NANOPARTICLE AND TARGETED SYSTEMS FOR THE CANCER TREATMENT

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Abstract

This analysis examines recent work aimed at more focused cancer care, either through more precise anticancer agents or through distribution methods. Such fields include dissemination by avoiding the reticuloendothelial system, using the improved permeability and retention effect and tumor-specific targeting. There are summarized therapeutic options using antibody-targeted therapies. It also addresses the potential to cure cancer by targeting delivery via angiogenesis and introduces antiangiogenic drugs in clinical trials. Methods of distribution that use nanoparticles directly are also illustrated, for both degradable and non-degradable polymers. Current cancer treatment typically includes invasive procedures, including catheter application to allow chemotherapy, initial chemotherapy to minimize any cancer present, surgery to remove the tumor(s) if necessary, followed by further chemotherapy and radiation. The aim of chemotherapy and radiation is to destroy tumor cells because, due to their growth at a much faster pace than healthy cells, these cells are more vulnerable to the actions of these drugs and methods, at least in adults.

Keywords: Cancer, Clinical, Cells, Chemotherapy, Treatment.

I. INTRODUCTION

Over the past 25 years, clinical attempts to improve chemotherapy have contributed to increased patient survival, but there is still a need for progress. Current research areas include the production of alternative dosing channels for carriers, new therapeutic targets such as tumor growth fuelling blood vessels and targeted therapeutics that are more precise in their action[1]. Clinical studies have shown that patients are open to new treatment approaches and that the purpose of these new chemotherapeutics is to improve cancer patients' survival time and quality of life. In both cases,



drug efficacy is directly connected to the ability of the treatment to target and destroy cancer cells while affecting as few healthy cells as possible[2].

Generic name	Trade name	Manufacturer, year approved	Target and indication	
Rituximab	Rituxan®	IDEC Pharmaceuticals, 1997	Anti-CD20 antibody or relapsed/refractory CD-20 positive B-call non-Hodgkin's lymphoma and low-grade or follicular-type lymphoma	
Trastuzumab	Herceptin [®]	Genentech, 1998	Blocks HER2 receptor for HER-2 positive metastatic breast cancer	
Gemtuzumabozogamicin	Mylotarg [®]	Wyeth Pharmaceuticals, 2000	Anti-CD33 antibody for relapsed/refractory acute myelogenous leukemia	
Alemtuzumab	Campath®	Berlex Laboratories, 2001	Anti-CD52 antibody for B-call chronic lymphocytic leukemia	
Ibritumomab tiuxetan	Zevalin®	IDEC Pharmaceuticals, 2002	Anti-CD20 antibody for Rituximab-failed non-Hodgkins lymphoma	
Gefitinib	Iressa	AstraZeneca, 2003	Blocks epidermal growth factor receptors and tyrosine kinase activity for advanced non-small cell lung cancer	

Table 1 Illustrates the cancer treatment using antibodies[2].

The degree of improvement in the quality of life of the patient and subsequent life expectancy is directly related to this treatment's targeting capacity. The only selectivity in the care of most current cancer patients is due to the intrinsic existence of chemotherapeutic drugs to work more intensively on a specific type of cancer cell than on healthy cells. However, certain side effects can often arise and are often so severe by systematically administering bolus doses of these aggressive drugs that the patient must discontinue treatment before the drugs have a chance to kill the cancer[3]. Unfortunately, even if carried out to the oncology requirements, not all therapies are successful in killing the cancer until the patient is killed by the cancer. Advances in cancer care are evolving steadily in terms of both new cancer agents and new methods of delivering both old and new agents. Hopefully, we will move away from near-toxic doses of non-specific agents with this development[4]. This analysis will mainly discuss new therapy delivery approaches, both old and new, with an emphasis on formulations of nanoparticles and those that directly target tumors[5].





Layer of healthy tissue



One cell acquires enough mutations to become cancerous



Cancerous cell will divide at accelerated rate and displace healthy tissue



Eventually, normal cells will be eliminated and organ function is compromised



Tumor cells will continue to grow and die as steady state size exists until new blood vessels form

Fig. 1 Illustrates the tumor development from initial carcinogenesis to diffusionlimited maximal size[6].

Cancers may be intermittent or inherited, i.e. caused by different carcinogenic agents. Several changes in cell physiology are associated with neoplastic development, including growth signal enhancement, non-responsiveness to anti-growth signals, infinite replicative capacity, metastasis, angiogenesis, and apoptosis escape[7].

II. DISCUSSION

Paclitaxel is a stabilizing agent for microtubules that encourages tubululin polymerization, causing cell death by disrupting the dynamics required for cell division. In particular, it has neoplastic activity against primary ovarian epithelial carcinoma, breast, colon, and non-small cell lung cancers. Paclitaxel is poorly soluble but soluble in certain organic solvents, such as alcohols, in aqueous solutions. Therefore, it lends itself well to more sophisticated methods for formulation[8].

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2ME2	EMD 121974	Photopoint
Angiostatin	Endostatin	PI-88
Angiozyme	Flavopiridol	Prinomastat (AG-3340)
Anti-VEGF RhuMAb	Genistein (GCP)	PTK787 (ZK22584)
Apra (CT-2584)	Green Tea	RO317453
	Extract	
Avicine	IM-862	Solimastat
Benefin	ImmTher	Squalamine
BMS275291	Interferon alpha	SU 101
Carboxyamidotriazole	Interleukin-12	SU 5416
CC4047	Iressa (ZD1839)	SU 6668
CC5013	Marimastat	Suradista (FCE 26644)
CC7085	Metastat (Col-3)	Suramin (Metaret)
CDC801	Neovastat	Tetrathiomolybdate
CGP-41251 (PKC 412)	Octreotide	Thalidomide
CM101	Paclitaxel	TNP-470
Combretastatin A-4	Penicillamine	Vitaxin
Prodrug	Photofrin	

Table 2 Antiangiogenic drugs currently in clinical trials for cancer[10].



Table 1 illustrates the cancer treatment using antibodies. Table 2 antiangiogenic drugs currently in clinical trials for cancer. Figure 1 illustrates the tumor development from initial carcinogenesis to diffusionlimited maximal size. Figure 2 illustrates tumor development.

III. CONCLUSION

In the last 5 years or so, scientific work aimed at achieving specific and selective delivery of anticancer agents has grown exponentially with new ways to guide drugs to tumors as well as new forms of drugs. The first of these innovative models of treatment have entered the clinic and are hopefully well on their way to increasing the length and quality of life for patients with cancer. However, by treating it as early as possible, there is a great deal more that can be done to treat and even avoid advanced cancer. This will involve superior methods of detection and targeting, which will certainly be sought and hopefully accomplished by many of the researchers listed here. To target imaging agents, several of the same methods used to target the delivery of drugs to cancerous tissues can also be used. In reality, when targeted delivery systems reach the stage where they can be used clinically, primary evaluation of the usefulness of a specific formulation in a specific patient can be performed with imaging agents to verify that before any drug regimen is initiated, the delivery system goes primarily to the cancerous tissues.

IV. REFERENCES

- [1] S. Kumar, A. Gupta, and A. Arya, *Triple Frequency S-Shaped Circularly Polarized Microstrip Antenna with Small Frequency-Ratio.* International Journal of Innovative Research in Computer and Communication Engineering (IJIRCCE)/ISSN(Online): 2320-9801, 2016.
- [2] L. Brannon-Peppas and J. O. Blanchette, "Nanoparticle and targeted systems for cancer therapy," *Advanced Drug Delivery Reviews*. 2012, doi: 10.1016/j.addr.2012.09.033.
- [3] S. A. A. Rizvi and A. M. Saleh, "Applications of nanoparticle systems in drug delivery technology," *Saudi Pharmaceutical Journal*. 2018, doi: 10.1016/j.jsps.2017.10.012.
- [4] E. N. Kumar and E. S. Kumar, "A Simple and Robust EVH Algorithm for Modern Mobile Heterogeneous Networks- A MATLAB Approach," 2013.
- [5] J. D. Byrne, T. Betancourt, and L. Brannon-Peppas, "Active targeting schemes for nanoparticle systems in cancer therapeutics," *Advanced Drug Delivery Reviews*. 2008, doi: 10.1016/j.addr.2008.08.005.
- [6] F. Alexis, E. M. Pridgen, R. Langer, and O. C. Farokhzad, "Nanoparticle technologies for cancer therapy," *Handbook of Experimental Pharmacology*. 2010, doi: 10.1007/978-3-642-00477-3_2.
- [7] H. Sun, X. Zhu, P. Y. Lu, R. R. Rosato, W. Tan, and Y. Zu, "Oligonucleotide aptamers: New tools for targeted cancer therapy," *Molecular Therapy - Nucleic Acids*. 2014, doi: 10.1038/mtna.2014.32.



- [8] S. Wilhelm *et al.*, "Analysis of nanoparticle delivery to tumours," *Nature Reviews Materials*. 2016, doi: 10.1038/natrevmats.2016.14.
- [9] V. Bagalkot *et al.*, "Quantum dot-aptamer conjugates for synchronous cancer imaging, therapy, and sensing of drug delivery based on Bi-fluorescence resonance energy transfer," *Nano Lett.*, 2007, doi: 10.1021/nl071546n.
- [10] A. Babu, A. K. Templeton, A. Munshi, and R. Ramesh, "Nanodrug delivery systems: A promising technology for detection, diagnosis, and treatment of cancer," AAPS *PharmSciTech*. 2014, doi: 10.1208/s12249-014-0089-8.