# Different Treatments to Cure Cancer Pain

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## Abstract

In most parts of the world, cancer is still one of the top causes of mortality. The impact of cancer-related mortality and morbidity on health-care systems and socioeconomic development is a pressing issue in both emerging and industrialized countries with ageing populations. Although our understanding of cancer genes has vastly advanced in the last three decades, this has not translated into comparable advantages for cancer patients. Indeed, greater survival is more often due to early discovery or prevention than to better therapy. Furthermore, the efficacy of traditional cancer treatments like chemotherapy and radiation therapy has reached a plateau in the treatment of many malignancies, including colon, prostate, ovarian, lung, and cervical cancers, and new approaches are needed to enhance outcomes.

**Keywords:** Chemotherapy • Radiation therapy • Malignancies • Mental health

## Introduction

Despite the fact that there is no shortage of prospective cancer treatment targets, there are only a few molecules that are differently expressed in cancer and cross many pathways essential for tumour maintenance. Survivin is a member of the Inhibitor of Apoptosis (IAP) gene family that has been linked to a variety of important functions, including cell division, apoptosis (programmed cell death), the cellular stress response, and genomic integrity checkpoint mechanisms. To begin addressing the structural basis of survivin's function, an X-ray crystal structure of a recombinant form of full length survivin with a resolution of 2.58 A has been described. Survivin is made up of two distinct domains: A binding BIR domain at the N-terminus and a 65 A amphipathic C-terminal alpha-helix. The crystal structure of each survivin monomer's BIR domain displays an extended dimerization interface along a hydrophobic surface. Functional protein-protein interaction surfaces are suggested by a basic patch working as a sulfate/phosphate-binding module, an acidic cluster extending off the BIR domain, and a solvent-accessible hydrophobic surface lying on the C-terminal amphipathic helix. Survivin, potentially the mitochondrial fraction rather than the cytosol fraction, is thought to suppress apoptosis via interfering with caspases. Survivin also helps cells live longer by interfering with cell cycle kinases and microtubule networks. Survivin overexpression suppressed both microtubule dynamics instability in mitotic spindles and bidirectional growth of microtubulesin midbodies during cytokinesis by reducing centrosomal microtubule nucleation and suppressing both microtubule dynamics instability in mitotic spindles and bidirectional growth of microtubulesin midbodies during cytokinesis. The creation of global pathway inhibitors with unique therapeutic potential could result from pursuing the disruption of survivin's nodal roles in cancer.

The major goal of this article is to explain the current developments of survivin-targeted therapy in both preclinical and clinical settings, since the molecular biology of survivin has been studied in numerous literatures. We'll also talk about the problems that come with targeting survivin in malignancies.

However, a new idea is emerging that examines what it means to be a cancer patient, where cancer is no longer viewed merely as an acute sickness, but as a chronic condition that must be cured. A prominent result of the increased survival rate indicated above is a rise in cancer patients' life expectancy, which leads to a lengthening of the patients' cohabitation with the disease's or treatment's collateral effects or issues. To put it another way, increasing cancer patients' life expectancy is just as important as improving their quality of life. Improving patients' quality of life through a comprehensive therapy strategy is one of the most important goals in cancer care. In the medical profession, it is now widely accepted that the patient must be treated in all facets of the illness. The American Society of Clinical Oncology (ASCO) and the European Society for Medical Oncology (ESMO), two of the most influential oncology societies, have established the necessity of treating patients' illnesses holistically, including biologically, psychologically, and sociologically. Aside from changes in the endocrine, circulatory, and neurological systems, the burden of cancer and its treatment can also cause physical changes in patients. In fact, due to scarring, disfiguring operations, hair loss, or weight loss/gain, individuals with cancer or a history of cancer frequently suffer dissatisfaction with their personal image. Changes in physical appearance have been identified as one of the most major reasons of distress in women with breast cancer. As a result, paying more attention to a patient's body image becomes critical in order to improve their quality of life. Dermatologists are now taking a more active part in preventing and treating therapy-related side events, as well as maintaining patients' self-image by minimizing stigmatization and any reminder of the condition. Stimulating a positive self-image in the patient has a significant positive impact on self-esteem, mental health, and personal connections, increasing the patient's reaction to therapy and prognosis. The demand for aesthetic therapies among cancer patients is steadily increasing in this area. These aesthetic procedures are generally safe; nonetheless, it should be noted that, as with any medical treatments, there is always the risk of side effects. The cancer patient, who is considered "fragile" by definition, requires special treatment. The major goal of this study is to assess the safety of different aesthetic therapies in cancer patients, such as fillers, botulinum toxin, and laser treatment. In Europe, an estimated 4 million new cancer cases were diagnosed in 2018. Prostate cancer is the most common cancer in males, whereas breast cancer is the most common disease in women. 5 Although cancer incidence is on the rise, cancer death is on the decline. In Italy, about 5% of the population has been diagnosed with cancer, with 3% of those instances being "long-surviving" individuals who have had their cancer for more than 5 years. Around 27% of persons who survive a cancer diagnosis have a life expectancy comparable to those who have never had a cancer diagnosis and, as a result, can be considered "cured." The number of cancer survivors in Italy has nearly quadrupled since 1992, owing to an increase in new cases (due to the ageing population), a higher prevalence of particular malignancies, and longer survival after the disease. People who are disease-free after treatment, people who continue to receive treatment to reduce the risk of cancer recurrence (also known as adjuvant therapy), and finally, people with a well-controlled disease and minimal symptoms receiving treatment to keep the cancer under control, which often manifests itself as a chronic disease, are all considered "cancer survivors." A cancer survivor, on the other hand, is far more vulnerable than someone who has never had cancer. A tumor's and its therapies can have a long-term effect, increasing susceptibility to infections, altering immune response, or worsening pre-existing illnesses like diabetes or heart disease. When a cancer patient's immune system is compromised, the tumour can manipulate the normal immune response to its benefit by increasing the production of pro-inflammatory cytokines and the formation of su-

-ppressive cell populations that dampen the immune response (regulatory T-cells and myeloid-derived suppressor cells). Furthermore, the stress of a cancer diagnosis and treatment can have a significant impact on the immune system. Chronic cancer-related stress inhibits the protective immune response in two ways: first, by increasing mediators such catecholamines and glucocorticoids, and second, by decreasing the cell-mediated immune response, antibody response, and the activity of Natural Killer (NK) cells. Both chemotherapy and target treatment can cause immunosuppression, which is undeniable. Cancer patients are also more vulnerable to infections such bacterial, viral, and fungal infections. Alterations in the skin and mucosal barrier, neutropenia, and, as previously indicated, immunosuppression are the key risk factors. Invasive procedures (e.g., catheters, surgery) and treatments frequently impair the skin and mucous barrier, which serves as the first line of defense against infections in cancer patients [1-3].

Chemotherapy has been shown to cause many changes in skin composition in cancer patients, including a drop in sebum content, water content, and TEWL (trans epidermal water loss), affecting skin barrier function. Radiotherapy has also been shown to harm barrier function by causing epidermal necrosis and apoptosis, which reduces the generation of natural moisturizing agents and intercellular lipids. Radiation also causes the stratum corneum to become alkaline, which encourages bacterial and fungal development. It's worth noting that cancer patients are frequently elderly, with comorbidities like malnutrition, kidney failure, heart disease, or diabetes adding to their fragility. Cancer-Related Fatigue (CRF) is one of the most common and incapacitating side effects experienced by cancer patients during and after treatment. Cancer-related fatigue can last for years after treatment has ended, and it is exacerbated by co-occurring cancer-related side symptoms such depression, anxiety, insomnia, and pain. Cancer-related fatigue impairs a patient's ability to complete cancer treatments and engage in important and valued life activities, lowering quality of life and potentially reducing overall survival. The National Cancer Institute has recognized cancer-related fatigue as a high-priority research subject, and it is one of the five highest-priority research areas identified by the National Cancer Institute Clinical Oncology Research Program in the United States. Exercise, psychological, exercise plus psychological, and pharmacological therapies for the treatment of CRF have all been investigated in randomized clinical trials. The results of these RCTs are promising; however, due to the lack of a direct meta-analytic comparison of the four most often suggested behavioral and pharmacological treatments for CRF, developing and implementing guidelines for clinical practice is difficult. Although there are clinical practice guidelines for the management of CRF, it is uncertain whether therapeutic method is the most successful.

To our knowledge, no previous review of CRF has used meta-analytic methods to compare the efficacy of all four major types of treatments recommended for managing CRF, nor has any prior review systematically investigated factors associated with treatment effectiveness (e.g., age, type of cancer, primary cancer treatment during vs.completed primary cancer treatment, study quality) when managing CRF. This knowledge can be used to improve a personalised medicine strategy to treating CRF and to guide future research. Currently, a cancer patient's therapy is determined mostly by a combination of the primary tumor's location and histological characterization. While this has been a partially successful strategy, it is now obvious that combining these criteria with an awareness of the underlying molecular abnormalities found in tumours could improve the process of selecting a cancer therapy. The genomic instability that characterizes nearly all solid tumours and adult-onset leukemias is a growing focus for cancer therapy. Genomic DNA, whether in normal or tumour cells, is constantly attacked by a wide range of DNA-damaging chemicals. The DNA lesions that occur as a result of this damage are generally repaired by a complex network of genome surveillance systems and DNA repair pathways to preserve the integrity of the genome and thus the fitness and viability of cells. Non-Homologous End Joining (NHEJ), Homologous Recombination (HR), Base Excision Repair (BER), Nucleotide Excision Repair (NER), Mismatch Repair (MMR), and Translesion Synthesis (TLS) are some of these processes. In a nutshell, the NHEJ pathway is responsible for repairing Double-Strand Breaks quickly (DSBs) [4,5].

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