# Recent post-translational modification research progress in oncology.

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## Abstract

**The genetic code that makes up a protein determines its core structure, but the specific functional form that a protein takes on is mostly the result of a dynamic interaction between numerous enzymes that are involved in post-translational modifications. Cells frequently use this diverse repertory to control transcription, protein localisation, and proteostasis as well as how they respond to external stimuli. In this article, post-translational changes that clearly have the ability to cause prostate cancer are investigated. Proteome-wide and individual protein post-translational changes are listed in detail, along with how they affect phenotypic consequences. This kind of information is extremely effective when the differential activity of cancer cells is controlled at the level of post-translational modifications but cancer cells do not differ in the expression or mutational status of a protein. Post-translational alterations are extensively researched in an effort to better prostate cancer treatment due to their driving roles in the disease. The promise of post-translational alterations in prostate cancer therapy is currently being presented by current methods.**

**Keywords**: Oncology, Post-translational modification, Protein, Mass spectrometry, Sumoylation.

## Introduction

Oncogenes, tumour suppressor genes, apoptotic genes, and DNA repair genes are only a few of the groups of genes that are affected by cancer, which is a manifestation of both genetic and epigenetic modifications that cause genomic instability. Most cancer researchers have recently focused their emphasis on the topic of cancer genetics, which includes the study of point mutation, deletion, insertion, gene amplification, chromosomal deletion/inversion/translocation, and allelic loss/gain. Although various studies have already demonstrated that in addition to numerous genetic mutations, human malignancies also harbour widespread epigenetic aberrations, the understanding of cancer epigenetics is more recent [1]. In cancer cells, PTMs adjust the state of effector proteins that are involved in regulation of cell survival, cell cycle and proliferation. Several unrestrictive PTM identification methods, such as large mass tolerance searches, have been developed to simultaneously search for both recognised and unidentified PTM kinds. These methods, however, have drawbacks, such as the inability to be easily applied to large proteome data sets the need to detect both changed and unmodified forms of peptides in the sample or the need to give up either sensitivity or confidence when detecting modified peptides. Biochemical and biophysical analysis has been used to identify more distinct PTMs. Nearly every element of protein function is known to be regulated by PTMs either alone or in combination. Numerous histone PTMs, the enzymes that transfer and remove many PTMs have been identified thanks to tremendous scientific advancements. Our ability to identify PTMs now outpaces our knowledge of how they work biologically. Many people consider the name "code" deceptive because there isn't yet a universally accepted set of guidelines for cracking these codes. The identification and quantification of individual PTMs serves as the first step in the investigation of PTM codes. The more PTMs in a system that can be precisely measured, the better a "code" can be defined, obviously. The most effective analytical method for identifying combinatorial PTM is Mass Spectrometry (MS) [2].

Identification of the sites of post-translational modifications (PTMs) in protein, RNA, and DNA sequences is currently a very hot topic. The information obtained is very useful for in-depth understanding the biological processes at the cellular level. It can also be used to develop effective drugs against major diseases including cancers. The identification and quantification of individual PTMs serves as the first step in the investigation of PTM codes [3]. The more PTMs in a system that can be precisely measured, the better a "code" can be defined, obviously. The most effective analytical method for identifying combinatorial PTM is mass spectrometry (MS). Through a variety of regulatory mechanisms, SUMO modification may increase the stability of complicated signalling circuits. The majority of malignancies have a high up regulation of Sumoylation, making it a viable target for cancer therapy. A reversible post-translational modification known as Sumoylation has become an important molecular regulatory mechanism, controlling processes such as DNA

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damage repair, immunological responses, carcinogenesis, cell cycle progression, and apoptosis. To confirm these results and offer helpful information for the diagnosis and prognosis of cancer, more clinical trials are required [4,5].

## Conclusion

Deciphering functional PTM codes would certainly benefit from systems-level analysis and network theory, which become more effective with further integration of proteomic technologies and combinatorial biophysical experiments, given the ostensibly limitless amounts of data arising from MS-based PTM research. In fact, many initiatives that have already begun to apply quantitative proteomic approaches have been aimed towards unravelling functional PTM networks in the context of human disease and therapy. It is hoped that one day it will be possible to determine the contribution of each PTM on any protein to a particular PTM network and biological process.

## References


1. Hollander N, Haimovich J. Altered N-linked glycosylation in follicular lymphoma and chronic lymphocytic leukemia: involvement in pathogenesis and potential therapeutic targeting. Front in Immunol. 2017;8:912.
2. Suzuki O. Glycosylation in lymphoma: Biology and glycotherapy. Pathol Int. 2019;69(8):441-9.
3. Yang Y, Staudt LM. Protein ubiquitination in lymphoid malignancies. Immunoll Rev. 2015;263(1):240-56.
4. Sun X, Liu M, Hao J, et al. Parkin deficiency contributes to pancreatic tumorigenesis by inducing spindle multipolarity and misorientation. Cell Cycle. 2013;12(7):1133-41.
5. Lee SB, Kim JJ, Nam HJ, et al. Parkin regulates mitosis and genomic stability through Cdc20/Cdh1. Molecular Cell. 2015;60(1):21-34.



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