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*Short Communication*

# Evolving Nature of Gene Therapies in Cancer Treatment

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## Introduction

Any cancer gene therapy will be based on its ability to target just cancer cells and not harm healthy cells. Therefore, precautions are required to stop its activation in healthy cells. We created a minimal p14ARF promoter including transcription start site, TATA box, Ap1 and E2F enhancer elements upstream, and MDR1 inhibitory element downstream (hereafter p14ARFmin). When produced, the bicistronic P14 and truncated BID (tBID) genes connected to the modified p14ARFmin promoter resulted in synergistic apoptosis *via* the intrinsic and extrinsic routes of apoptosis. Only cells harbouring both mutant Ras and mutant p53 would activate the construct, since the promoter was made to be preferentially activated by mutant Ras and fully blocked by wild-type p53. This design exhibits selective benefits over the majority of p53 gene treatments. Our construct does not induce p21WAF/CIPI in contrast to other p53-based gene therapies, which can induce cell cycle arrest increasing chemotherapy resistance; the modified construct (p14ARFmin-p14-tBID) exhibits bidirectional control of its promoter, which is completely repressed by wild-type p53 and activa; and the p53-based gene therapies with a constitutive CMV promoter cannot distinguish between normal cells and cancer cells and can be toxic to normal cells.

Cellular metabolism is a finely regulated process that keeps cells growing and surviving. There are obvious sex differences in lung cancer, with female patients having higher survival rates than male patients. Cancer risk, prognosis, and response to various medicines all show evidence of sex differences, but a sex-specific strategy to cancer studies is not frequently addressed. It is crucial to include viral and hormonal impacts because of the many tumour features associated with sex that affect disease outcome, including constitutional genetic and somatic molecular variations.

## Description

Lung cancer is accompanied by changes in the expression of many genes, which have been linked to genetic, biochemical, demographic, environmental, and even behavioural variables as well as carcinogenesis exposure. It has been challenging to distinguish between core processes and their relationships with prognosis due to the size and variety of these components. We were able to find differentially expressed genes in both phenotypes in the current study by differential gene analysis. We discovered differences in signalling pathways, metabolic processes, immunological response, and cellular homeostasis between the phenotypes. We wanted to know if smoking or not smoking had an impact on the regulation of oestrogen and androgen transcriptional targets in both males and females. To evaluate oestrogen protection in the comparison groups, we also included males and pre- and post-menopausal females. Environmental variables that influence cancer susceptibility have previously been the subject of several articles; nevertheless, there is currently no conclusive information about whether lung cancer is more common in men than women. Cancers such colon cancer and hormonally-dependent tissue cancer are known to be influenced by food and hormonal condition (BRCA and PRAD). In the current study, we also had shown that environmental variables, such as nutrition, have a direct correlation with the development and prognosis of lung cancer and should be taken into consideration.

In GBM, metabolic rewiring and immune suppression are two aspects that are intimately associated to one another and support pathogenesis and an aggressive pattern, limiting the effectiveness of both conventional treatments and

cutting-edge therapeutic strategies like immunotherapy. The Tumour Microenvironment (TME) is significantly altered by the dynamic metabolic situation that characterises GBM, making it hostile to T cell survival and proliferation and adversely influencing the host immune response. The relationship that tumour cells form with other cell populations in the TME, which adopt strategies to escape, being recognised and destroyed by immune cells, is strongly correlated with the growth of GBM, according to a number of lines of evidence. Similar to how glial cells may affect stromal cells' activity under physiological circumstances, GBM cells can also mediate the homing, the recruitment, and the differentiation of invading cells in a paracrine manner or through direct cell-cell interaction [1-4].

## Conclusion

Steady states in an organism can be disrupted by external and internal stressful stimuli such bacteria, viruses, metabolite build-up, dead cells, and unchecked growing cells. The immune system's innate and adaptive response, which spans the spectrum of "self" vs "non-self" interactions, is crucial in preserving tissue homeostasis. Given the extensive web of contacts created between stromal, parenchymal, and TME sites, cancer immune-surveillance is especially challenging for highly aggressive tumours like GBM.

## Acknowledgement

Authors do not have acknowledgments currently.

## Conflict of Interest

There are no conflicts of interest.

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