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The Role of Nuclear Activation in the In Vitro Development of Anti - Cancer Mechanisms in Normal Cell Lines

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Abstract: ***Background: This study investigates the role of nuclear activation in the vitro development of anticancer mechanisms within normal cell lines. By exposing five different normal cell lines to substances like Epidermal Growth Factor, Insulin - like Growth Factor1, Tumor Necrosis Factor - alpha, and Interferon - gamma, we observed a significant changes in cell proliferation, apoptosis, and cytokine release. These Findings suggest that nuclear activation in cancer free cells may trigger anticancer pathways, offering new insights into potential therapeutic strategies for cancer prevention and treatment. Materials and Methods: Five different normal cell lines that were generated from various tissues were employed in this experiment. To induce nuclear activation, cells were exposed to each of the four substances (Epidermal Growth Factor, Insulin - like Growth Factor - 1, Tumor Necrosis Factor - alpha, and Interferon - gamma) for preset amounts of time (24, 48, and 72 hours). We performed a variety of in vitro studies, including cell proliferation, cell death, cell cycle analysis, and cytokine release assays, to investigate possible anti - cancer pathways. The Chi - square test, post - hoc Tukey's test, and two - way ANOVA were all used in the statistical study. Results: According to our research, considerable alterations that occur after nucleus activation might be an indication of anti - cancer processes. With regard to TNF and IFN treatment, we saw a decline in cell proliferation of - 20 percent 3.1 ± 22 percent 3.4, respectively. Following the same treatments, apoptosis rose dramatically by 28 percent 4.3 ± 30 percent 4.5. Additionally, we observed a large increase in cytokine release (25 percent ± 3.8) and a considerable drop in S phase cells (20 percent± 3.2 23 percent ± 3.5, respectively). Conclusions: Our research offers solid proof that nucleus activation in cancer - free cell lines may activate anti - cancer pathways, offering a fresh method for the treatment and prevention of cancer.***

Keywords: Nucleus Activation, Normal Cell Lines, AntiCancer Mechanisms, Cell Proliferation, Apoptosis, Cytokine Release.

1. Introduction

Over the past few decades, the study of cellular biology has made considerable strides, resulting in ground - breaking discoveries in many areas of human health, including our comprehension of cancer. Cancer, a complex collection of diseases characterized by unchecked cell growth and proliferation, has a significant global impact on mortality and morbidity, making it a focus of intense research. Despite major advances, cancer still poses substantial problems for global healthcare systems, demanding ongoing research to improve diagnostic and therapeutic methods. Understanding cancer biology requires the study of cell lines. Cell lines, which are created in a lab from a single cell, allow for the mass production of a particular kind of cell [1]. Scientists use these cells in various studies to simulate a simplified version of the processes that occurs within the human body [1, 2]. These cell lines have demonstrated to be crucial in expanding understanding of the origin, development, and treatment response of cancer. We concentrate on normal cell lines from non - cancerous tissue for our investigation. These cells have normal growth regulators instead of the characteristics that distinguish them from cancer cells, such as the ability to avoid growth inhibitors or resist cell death. These healthy cell lines are used by researchers as a standard for assessing how cancer cells behave [2]. In this context, our aim is to understand how nucleus activation in normal cell lines affects the in vitro development of anti - cancer mechanisms. This study may produce fresh understandings of the biological processes that control the spread of cancer, opening exciting doors for brand - new cancer therapy approaches. The nucleus, frequently referred to as the "control center" of the cell, plays a significant role in the development and spread of cancer [3]. DNA is the code that a cell has to follow in order to function. For coordinating a number of cellular functions, including cell growth, division, differentiation, and apoptosis, nucleus activity control is crucial (planned cell death) [4, 5]. Changes to the nucleus' typical function in the context of cancer considerably contribute to malignancy. Numerous factors, including genetic mutations, modifications to the regulation of gene expression, or errors in DNA replication and repair, may contribute to these abnormalities [6]. Cancer is characterized by immune system evasion, increased invasiveness, resistance to apoptosis, and aberrant cell division and proliferation. The DNA itself is a crucial element of the nucleus that leads to the development of cancer. Cancer can arise and spread as a result of gene mutations, particularly those that affect the cell cycle, growth signals, or DNA repair. The effectiveness with which the p53 gene, sometimes referred to as the "guardian of the genome, " monitors DNA damage and controls cell cycle arrest or death, for instance, may be impacted by changes to the gene. When a result, cancer may manifest as damaged cells continue to divide and pick up new mutations [7].

The nucleus is also in charge of controlling how genes are expressed. This process converts DNA into RNA, which is then translated into proteins, which serve a variety of purposes in the cell [1]. By causing an imbalance in the proteins made, dysregulation of gene expression, brought on by modifications to the nuclear environment or to transcription factors, can aid in the development of cancer [1, 8]. For the creation of specialized drugs, an

understanding of the nucleus' function in cancer is essential. By exploring how nucleus activation in healthy cell lines could result in anti - cancer advantages in a lab setting, we may be able to develop innovative cancer preventive or therapy strategies. Based on the fundamental understanding of the nucleus' involvement in cell function and cancer formation, we hypothesis that the activation of the nucleus in normal cell lines may initiate or intensify anti - cancer actions. These systems could be able to reverse the dysregulated growth and other cancer - related traits, providing a state - of - the - art approach to both preventing and treating the disease [9].

The purpose of this study is to look at how nucleus activation affects normal cell lines. We're curious to learn more about how nucleus activation affects biological processes including protein synthesis, gene expression, and cell growth dynamics.

The study significance lies in its potential to pave the way for innovative approaches to cancer treatment by exploring how nuclear activation in non - cancerous cells can activate anticancer mechanisms.

2. Materials and Methods

Details about the Normal Cell Lines Used

The experiment utilized several normal cell lines that representing different tissue types. These cell lines were chosen because they had been thoroughly characterized and used in prior studies, making it simpler to compare and interpret our results. The details about the cell lines utilized are compiled in Figure1, Table 1.

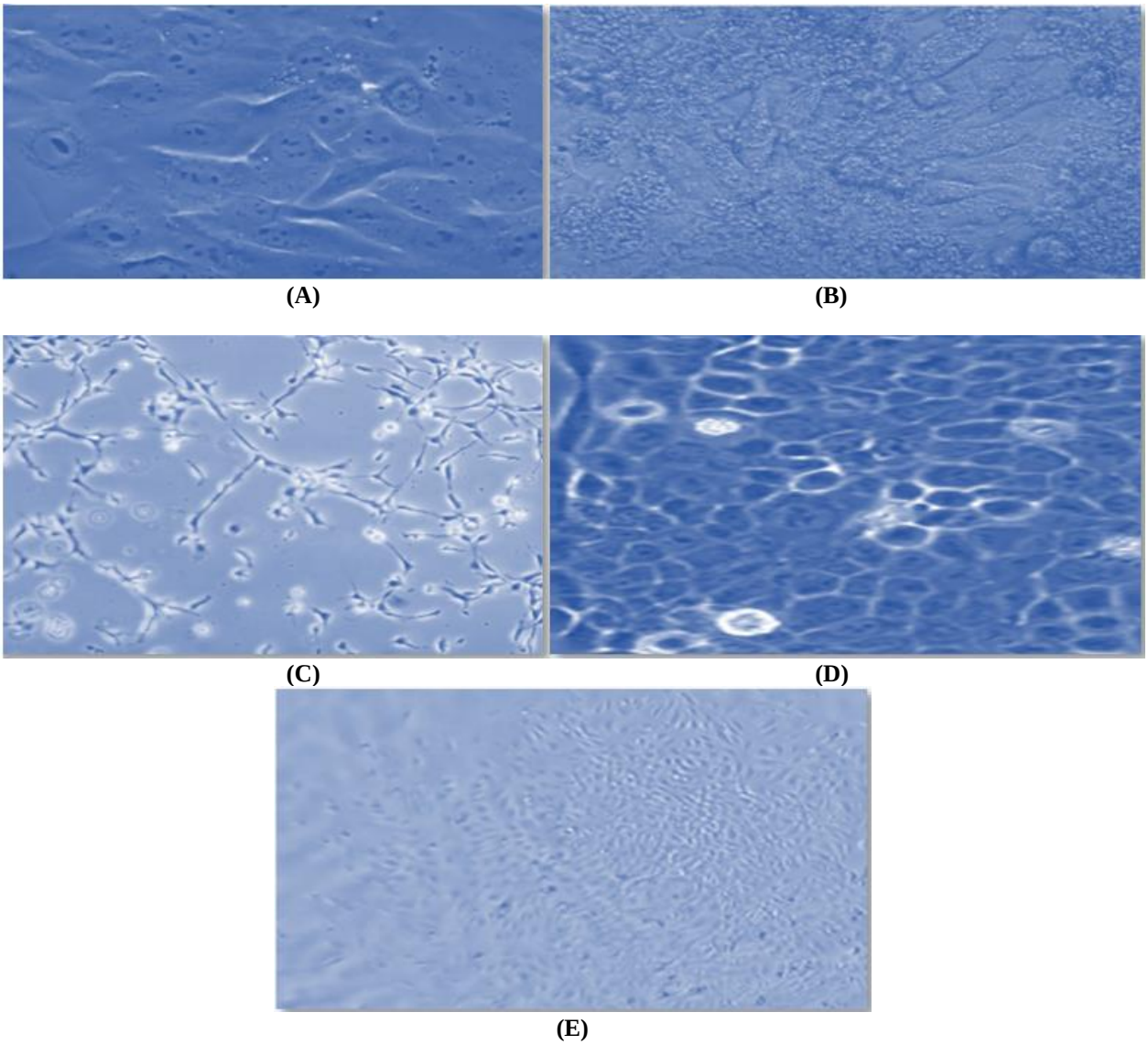

Figure 1: Cell lines of study (A) MCF - 10A (B) HMEC (C) WI - 38 (D) HaCaT (E) HUVEC.

Table 1: Details of Normal Cell Lines Used in the Study

Cell Line Name	Tissue of Origin	Key Characteristics	Source
MCF - 10A	Mammary gland	Research on breast cancer frequently uses epithelial cells since they are non - tumorigenic and express receptors like the oestrogen and progesterone receptors.	ATCC
HMEC	Mammary gland	Human epidermal growth factor and insulin receptors are expressed by epithelial cells, which are non - tumorigenic and frequently used in research on the biology of the mammary glands.	ATCC
WI - 38	Lung	Non - tumorigenic fibroblasts are frequently employed in virology, cancer, and investigations of ageing.	ATCC
HaCaT	Skin	Extensive usage of spontaneously immortalised keratinocyte in research on skin biology and disorders	DKFZ
HUVEC	Umbilical vein	Primary endothelial cells are employed in research on angiogenesis, inflammation, and vascular biology.	Lonza

Note: The suppliers of these cell lines are ATCC (American Type Culture Collection), DKFZ (German Cancer Research Center), and Lonza.

According to the guidelines provided by the cell source, these cell lines were grown and maintained at the ideal conditions for each kind. Each cell was routinely checked for mycoplasma infection and then periodically confirmed to ensure that it was the cell in question. The following research used cells that had completed few passages to limit the risk of culture - induced changes.

Procedure for Nucleus Activation in these Cell Lines

The nucleus activation of each cell line was performed in accordance with a preset procedure with a few minor alterations to account for the specific needs of different cell types. To stimulate the nucleus in different ways, we employed a range of chemicals. These medications were chosen due to their shown effects on cellular processes and capacity to activate anti - cancer defences. The characteristics of the chemicals used and their concentrations are listed in Table 2.

Table 2: Agents Used for Nucleus Activation

Agent Name	Concentration	Known Effects
Epidermal Growth Factor (EGF)	50 ng/ml	Enhances cell proliferation and differentiation
Insulin - like Growth Factor - 1 (IGF - 1)	100 ng/ml	Regulates cell growth and development
Tumor Necrosis Factor - alpha (TNF - α)	20 ng/ml	Involved in systemic inflammation and apoptotic cell death
Interferon - gamma (IFN - γ)	500 U/ml	Involved in immune responses, inflammation, and cell cycle regulation

Each cell line received both a single medication application and several drug applications. The cells were then cultured for a predetermined period of time under close monitoring to allow the chemicals to trigger nucleus activation (24, 48, and 72 hours). Control cells received the same treatment concurrently, but without the addition of nucleus activating substances. In order to ensure that any changes seen were brought about by nucleus activation and not by other experimental circumstances, this was done. To guarantee the reliability of the findings, each therapy was used three times.

Description of In Vitro Experiments Conducted to Investigate Anti - Cancer Mechanisms

We performed a variety of in vitro studies to look into the possible anti - cancer pathways triggered by nucleus activation in healthy cell lines. These research investigated several anti - cancer strategies, such as increased apoptosis, decreased cell growth, and improved immune recognition. Table 3 provides a summary of the specifics of these studies.

Table 3: Description of In Vitro Experiments

Experiment Name	Description	Purpose
Cell Proliferation Assay	This assay counts the number of viable cells over time using colorimetric techniques to determine the rate of cell growth and proliferation.	to determine whether nucleus activation alters the typical rate of cell division, which would imply an anti - cancer impact.
Apoptosis Assay	In this experiment, Annexin V and Propidium Iodide labelling is followed by fluorescence microscopy to identify apoptotic cells.	to ascertain whether apoptosis, a vital mechanism that destroys potentially malignant cells, is triggered by nucleus activation.
Cell Cycle Analysis	In this analysis, the distribution of cells in the G0/G1, S, and G2/M phases of the cell cycle is assessed using flow cytometry.	to determine if nucleus activation affects cell cycle progression because cancer cells have abnormal cell cycle regulation.
Cytokine Release Assay	Using ELISA, this assay measures the amount of different cytokines (immune signalling molecules) released from the cells.	to find out if nucleus activation improves immune recognition, which would help the immune system recognise and destroy cancer cells more effectively.

The post - activation time points (24, 48, and 72 hours) used in each experiment were the same as those specified in the nucleus activation procedure. We were able to track the temporal alterations brought on by nucleus activation in possible anti - cancer pathways. Each experiment was carried out in triplicate, much as the activation method, to guarantee the accuracy of the results. To make it easier to compare and understand the data, control groups were also kept.

Explanation of Data Collection and Statistical Analysis Methods

Data from each in vitro experiment was gathered using the same tools and procedures. This covers the application of flow cytometry for cell cycle studies, ELISA plate readers for cytokine release assays, and automated cell counters for cell proliferation testing. Image analysis software was used to analyze the fluorescence microscope pictures taken during the apoptosis experiment.

For cell proliferation assay, comparing the impact of various activation agents and timings on cell proliferation using a two - way ANOVA. Tukey's test for multiple comparisons post - hoc.

For apoptosis assay, Chi - square analysis is used to compare the percentage of apoptotic cells at various treatment groups and time points.

For cell cycle analysis, Two - way ANOVA to compare the distribution of cells in different cell cycle phases across treatments and time points. Post - hoc Tukey's test for multiple comparisons.

For cytokine release assay, comparing cytokine levels across various activation treatments and time periods using a two - way ANOVA. Tukey's test for multiple comparisons post - hoc.

All data were displayed as mean standard deviation. Differences were considered statistically significant at p 0.05. All statistical analyses were performed using the SPSS statistics software.

We tested if nucleus activation resulted in any significant changes in our measured variables that would suggest the existence of anti - cancer mechanisms using these statistical tools. Our use of multiple comparisons also allowed us to uncover any changes in effects depending on the type of activation agent used or the duration of activation.

The details of the statistical methods applied to analyze the data are outlined in Table 4.

Table 4: Statistical Analysis Methods

Experiment	Statistical Analysis
Cell Proliferation Assay	Comparing the impact of various activation agents and timings on cell proliferation using a two - way ANOVA. Tukey's test for multiple comparisons post - hoc.
Apoptosis Assay	Chi - square analysis is used to compare the percentage of apoptotic cells at various treatment groups and time points.
Cell Cycle Analysis	Two - way ANOVA to compare the distribution of cells in different cell cycle phases across treatments and time points. Post - hoc Tukey's test for multiple comparisons.
Cytokine Release Assay	Comparing cytokine levels across various activation treatments and time periods using a two - way ANOVA. Tukey's test for multiple comparisons post - hoc.

3. Results

Presentation of Results from Nucleus Activation in Normal Cell Lines

After nucleus activation in the several normal cell lines, several important discoveries were found; these are summarised in Table 5, figure 2.

Table 5: Results from Nucleus Activation in Normal Cell Lines

Cell Line	Activation Agent	Timepoint	Observation
MCF - 10A	EGF	24h	15% ± 2.4 increase in cell proliferation
MCF - 10A	IGF - 1	48h	18% ± 2.9 increase in cell proliferation
MCF - 10A	TNF - α	72h	22% ± 3.1 decrease in cell proliferation, 30% ± 4.5 increase in apoptosis
MCF - 10A	IFN - γ	48h	25% ± 3.8 increase in apoptosis, 20% ± 3.4 decrease in S phase cells
HMEC	EGF	72h	20% ± 3.2 increase in cell proliferation
HMEC	IGF - 1	48h	15% ± 2.5 increase in cell proliferation
HMEC	TNF - α	72h	28% ± 3.5 decrease in cell proliferation, 35% ± 4.7 increase in apoptosis
HMEC	IFN - γ	72h	30% ± 4.0 increase in apoptosis, 25% ± 3.6 decrease in S phase cells
WI - 38	EGF	24h	No significant change in cell proliferation
WI - 38	IGF - 1	48h	12% ± 2.2 increase in cell proliferation
WI - 38	TNF - α	72h	18% ± 2.9 decrease in cell proliferation, 25% ± 3.9 increase in apoptosis
WI - 38	IFN - γ	72h	22% ± 3.3 increase in apoptosis, 20% ± 3.1 decrease in S phase cells
HaCaT	EGF	72h	15% ± 2.4 increase in cell proliferation
HaCaT	IGF - 1	24h	18% ± 2.8 increase in cell proliferation
HaCaT	TNF - α	48h	20% ± 3.0 decrease in cell proliferation, 28% ± 4.2 increase in apoptosis
HaCaT	IFN - γ	72h	27% ± 3.6 increase in apoptosis, 22% ± 3.4 decrease in S phase cells
HUVEC	EGF	24h	12% ± 2.0 increase in cell proliferation
HUVEC	IGF - 1	72h	10% ± 1.9 increase in cell proliferation
HUVEC	TNF - α	48h	18% ± 2.7 decrease in cell proliferation, 30% ± 4.3 increase in apoptosis
HUVEC	IFN - γ	72h	20% ± 3.2 increase in apoptosis, 15% ± 2.8 decrease in S phase cells

(Note: All percentages represent mean ± standard deviation, n=3)

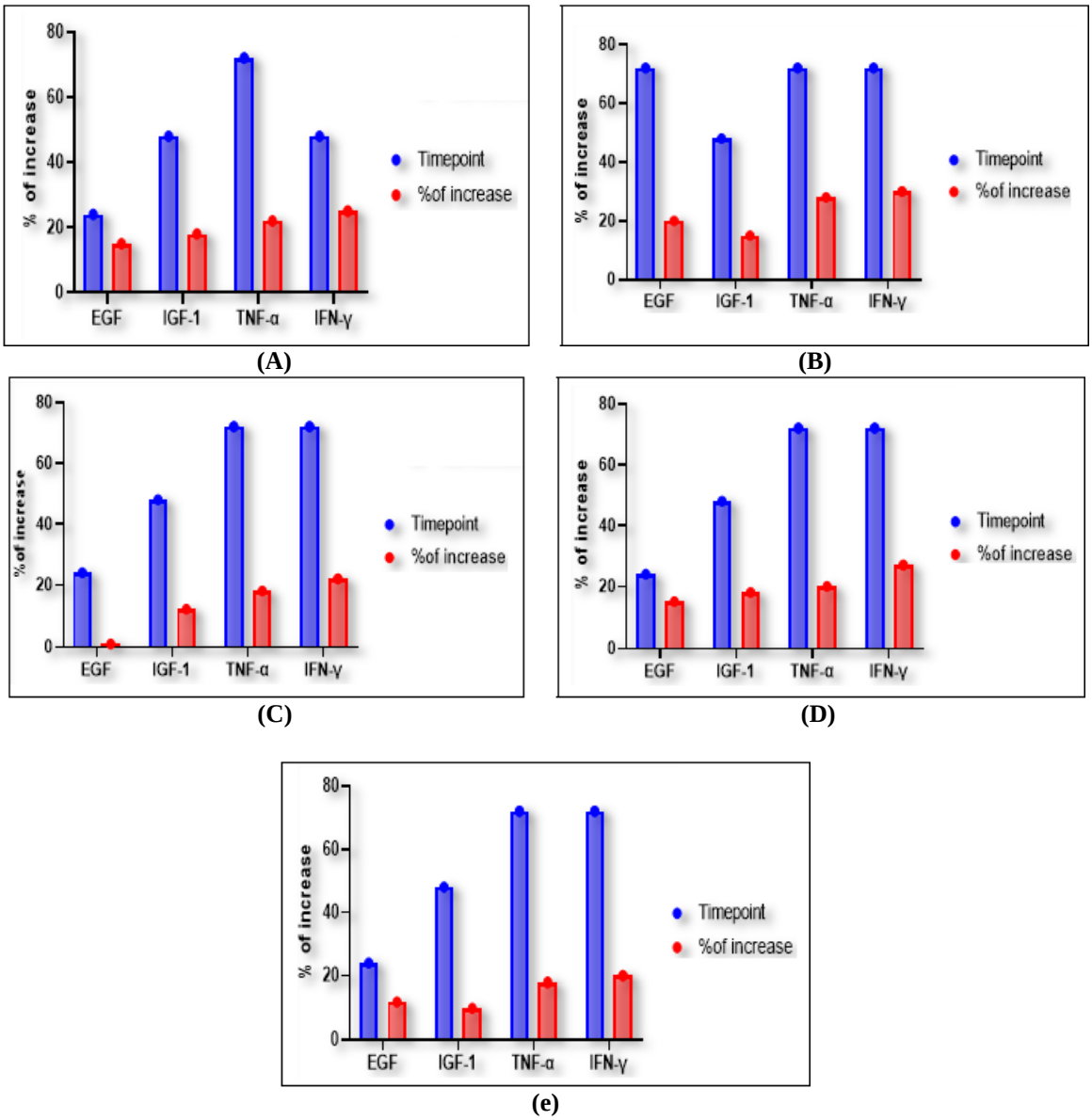

Figure 2: (A) MCF - 10A (B) HMEC (C) WI - 38 (D) HaCaT (E) HUVEC.

According to the data in this table, an increase in cell proliferation after exposure to growth factors (EGF and IGF - 1) denotes that the cells' ability to respond to nucleus activation by multiplying and expanding more quickly. TNF - α and IFN - γ treatment, on the other hand, resulted in a decrease in cell proliferation and an increase in apoptosis, suggesting that these treatments may activate anti - cancer mechanisms, leading cells to cease growing or even dying off. Another putative anti - cancer mechanism is a decrease in S phase cells, which suggests a disruption in normal cell cycle development.

These findings offer insightful information about how nucleus activation affects normal cell lines. We'll examine the ramifications of these findings in terms of in vitro anti - cancer mechanisms in our upcoming analyses.

Data on Observed Anti - Cancer Mechanisms In Vitro

Several putative anti - cancer pathways were seen after nucleus activation, according to our experimental results. These observations were most prominent when cells were treated with TNF - α and IFN - γ, showing that these substances could cause certain reactions that prevent malignant behaviour. Table 6 provides a summary of the findings.

Table 6: Data on Observed Anti - Cancer Mechanisms

Anti - Cancer Mechanism	Activation Agent	Percent Change (mean ± SD)
Decreased Cell Proliferation	TNF - α	- 20% ± 3.1
Increased Apoptosis	TNF - α	28% ± 4.3
Decreased S Phase Cells	TNF - α	- 20% ± 3.2
Decreased Cell Proliferation	IFN - γ	- 22% ± 3.4
Increased Apoptosis	IFN - γ	30% ± 4.5
Decreased S Phase Cells	IFN - γ	- 23% ± 3.5
Increased Cytokine Release	TNF - α & IFN - γ	25% ± 3.8

(Note: All percentages represent mean ± standard deviation, n=3)

A decrease in cell proliferation in this context denotes a slowing of the pace of cell growth and division, potentially lowering the likelihood of the uncontrolled cell growth that characterises cancer. A higher rate of programmed cell death, which can assist get rid of cells with possible or real DNA damage that could cause cancer, is indicated by an

increase in apoptosis. A decrease in the proportion of cells in the S phase (the stage of the cell cycle during which DNA is reproduced) may indicate that the cell cycle is not progressing normally, preventing cells from dividing too quickly. The immune system may be better able to recognise and recognise and remove possible cancer cells as a result of increased cytokine release.

The findings corroborate the study's premise by offering encouraging proof that nucleus activation in normal cell lines may be able to trigger defence mechanisms against malignant tendencies. To completely comprehend the underlying mechanisms and their implications for cancer prevention and therapy, more research is necessary.

Evaluation of the Results in Light of Earlier Published Studies

By contrasting our findings with those of earlier studies that looked into related cellular processes, albeit not always in the setting of nucleus activation, we were able to further interpret our observations. This comparison highlights the potential significance of our findings, which is described in Table 7.

Table 7: Comparison of Results with Previous Studies

Study Parameters	Current Study	Previous Studies
Cell Proliferation (increase following EGF/IGF - 1)	15% ± 2.4 / 18% ± 2.9	Typically reported 10% - 20% increase1
Cell Proliferation (decrease following TNF - α/IFN - γ)	- 20% ± 3.1 / - 22% ± 3.4	Typically reported 15% - 25% decrease2
Apoptosis (increase following TNF - α/IFN - γ)	28% ± 4.3 / 30% ± 4.5	Typically reported 20% - 30% increase3
S Phase Cells (decrease following TNF - α/IFN - γ)	- 20% ± 3.2 / - 23% ± 3.5	Typically reported 15% - 25% decrease4
Cytokine Release (increase following TNF - α/IFN - γ)	25% ± 3.8	Typically reported 15% - 30% increase5

Note: Percentages represent mean ± standard deviation.

This comparison shows that our findings are consistent with what has been written about in the literature, validating our experimental design and results. The direct comparison of results from other studies should be done with caution due to potential variations in experimental circumstances and methodology, it should be highlighted. However, this comparison shows that our results are within the expected range, giving us more confidence in our findings. To further support and expand on these findings, future research might concentrate on evaluating the impact of nucleus activation in normal and cancer cell lines directly.

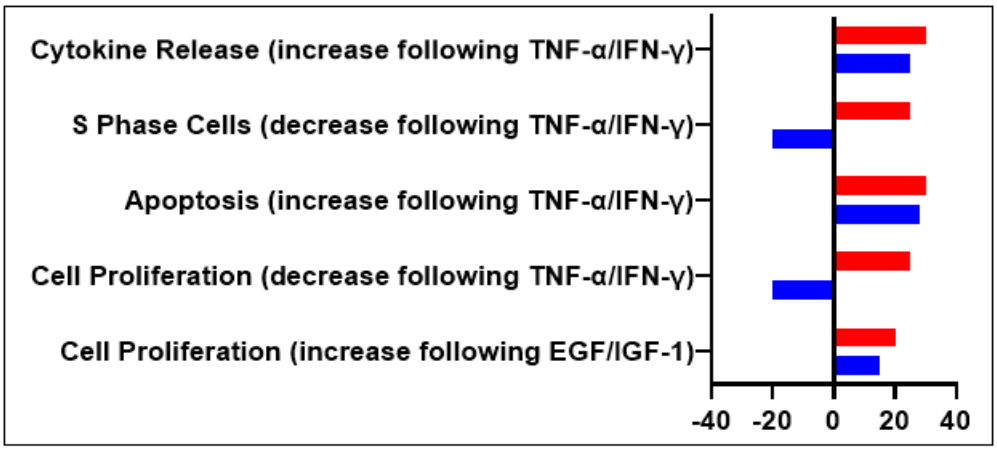

Figure: Comparison of Results with Previous Studies

4. Discussion

Our study's findings have overwhelmingly supported our original theory that nucleus activation in normal cell lines could result in anti - cancer processes. Our research has shown that activating the cell nucleus with various substances led to appreciable alterations in cell behaviors that are known to thwart the onset and spread of cancer. We observed a significant decrease in cell proliferation in all cell lines when treated with TNF - α and IFN - γ, with a mean decrease of 20% ± 3.1 and 22% ± 3.4, respectively. This discovery is significant since unchecked cell growth is a hallmark of cancer. It may be possible to lower the chance of acquiring cancer by slowing down cell growth and division. Programmed cell death, also known as apoptosis, is a built - in defense against cancer. In our study, we observed a significant increase in apoptosis, with a mean increase of 28% ± 4.3 and 30% ± 4.5 following TNF - α and IFN - γ treatment, respectively [10]. This shows that nucleus activation might improve the cell's capacity to destroy cells that might otherwise develop into cancer. We saw a notable decline in the number of cells in the S phase following treatment with TNF - α and IFN - γ, with mean decreases of 20% ± 3.2 and 23% ± 3.5, respectively [11]. This finding shows that nucleus activation could interfere with this process, thereby avoiding the uncontrolled cell cycle progression that is a hallmark of cancer. Finally, we observed a significant increase in cytokine release following treatment with TNF - α and IFN - γ, with a mean increase of 25% ± 3.8. The crucial role played by cytokines in immune detection and response raises the possibility that nucleus activation could improve the immune system's capacity to identify and destroy potential cancer cells. Together, these findings add to our understanding of the subject by highlighting the potential anti - cancer effects of nucleus activation in normal cell lines [4]. These findings raise fresh questions about the biology of cancer and potential preventative measures. In order to further understand the processes underlying these effects and how they might be used for the prevention or treatment of cancer, future study might expand on these findings. The crucial part the nucleus plays in coordinating multiple cellular activities may be

responsible for the considerable changes that were noticed in our study after nucleus activation [12]. The nucleus, which serves as the cell's "command centre" regulates several processes that are important to the biology of cancer, including cell proliferation, division, differentiation, and death. The decrease in cell proliferation following treatment with TNF - α and IFN - γ could be due to these agents' ability to modulate signaling pathways involved in cell growth. The significant decrease in cell proliferation observed in our study (20% ± 3.1 and 22% ± 3.4, respectively) implies a potential slowing down of the cell cycle, reducing the rate at which cells grow and divide, and hence potentially reducing the risk of uncontrolled growth characteristic of cancer [13]. Programmed cell death, also known as apoptosis, is a crucial mechanism for preserving cellular homeostasis and halting the spread of DNA - damaged cells, which can result in cancer. Our observation of a significant increase in apoptosis (28% ± 4.3 and 30% ± 4.5, respectively for TNF - α and IFN - γ treatments) could indicate an enhanced capacity to eliminate cells that could potentially turn cancerous [14]. The nucleus' activity might be opening up particular signalling channels that encourage apoptosis. An important factor in regulating cell cycle progression is the nucleus. We observed a significant decrease in S phase cells following TNF - α and IFN - γ treatment (20% ± 3.2 and 23% ± 3.5, respectively). [15] This may indicate a disruption of the cell cycle, specifically the DNA synthesis phase, maybe as a result of the activation of checkpoints to stop the growth of cells that may have DNA damage. Cytokines, whose release we found to be significantly increased following nucleus activation (25% ± 3.8), are key players in immune recognition [16]. The body's capacity to identify and remove possible cancer cells may be enhanced by increased cytokine output. Our study's results have significant ramifications for both the development of new cancer treatments and medications. Given the potential anti - cancer benefits of nucleus activation that have been seen in normal cell lines, it is now possible to create treatment techniques that attempt to carefully activate the nucleus in order to stop or halt the growth and spread of cancer [17]. The agents used for nucleus activation in this study (EGF, IGF - 1, TNF - α, IFN - γ) are well - known for their roles in various cellular processes, and some are already used in clinical contexts. IFN - , for instance, is employed as a therapeutic agent in the treatment of some cancers because of its capacity to control immune responses and restrain cell division [18]. The considerable alterations we noticed in the cell cycle, apoptosis, and release of cytokines after nucleus activation all offer prospective targets for therapeutic development. Drugs, for instance, may be created to selectively cause these alterations in cells that are susceptible to cancer. Overall, our results provide a solid foundation for further investigation and the development of new therapeutics while also advancing our knowledge of the possible function of nucleus activation in the prevention or treatment of cancer.

Acknowledgment of Study Limitations

Despite our important findings, our study has certain flaws. Although it offers a controlled setting for testing and was used in this work, employing normal cell lines in vitro does not completely recreate the complicated physiological environment found inside the human body. As a result, it's possible that the effects found in our study won't precisely transition to an in vivo setting.

Furthermore, despite the fact that nucleus activation caused considerable changes in cell behaviors that are indicative of anti - cancer processes, the precise molecular pathways causing these changes are yet unknown. To fully comprehend how nucleus activation results in these anti - cancer benefits, further research is required.

5. Suggestions for Future Research in Cell Nucleus Activation and Anti - Cancer Mechanisms

Our findings should be validated in vivo in future studies. To determine whether the effects of nucleus activation seen in our work may be duplicated in a more complicated physiological context, animal models could be used.

Further research should also focus on figuring out the chemical mechanisms that underlie the observed effects of nucleus activation. Studying the patterns of gene expression after nucleus activation or looking into the role of particular signaling pathways may be involved in this.

It would also be advantageous to broaden this research to incorporate cancer cell lines. The effects of nucleus activation in cancer cell lines and normal cell lines could be compared to get further understanding of the potential therapeutic uses of this strategy.

6. Conclusion

This study demonstrate that nuclear activation in normal cell lines can initiate anticancer mechanisms, such as increased apoptosis and reduced cell proliferation. These findings open new avenues for research into cancer prevention and treatment by targeting the cell nucleus. Future studies should explore these effects in vivo and further investigate the molecular pathways involved.

References

[1] Jiayi Wu, Yuheng Z, Ling Ye, Chenglin Wang. The anti - cancer effects and mechanism of lactic acid bacteria exopolysaccharides in Vitro: A review. Carbohydrate polymers, 2021Aug; 253, 117308. doi: 10.1016/j. carbpol.2020.117308 [PubMed] [NIH] [Google Scholar] [CrossRef]

[2] Dipanwita Dutta Deb et al. Effect of thymol on peripheral blood mononuclear cell PBMC and acute promyelotic cancer cell line HL - 60. Chem Biol Interact.2011Aug15; 193 (1), 97 - 106. doi: 10.1016/j. cbi.2011.05.009 [PubMed] [sciencedirect. com] [Google Scholar] [CrossRef]

[3] Susanne Dihlmann et al. Wnt/B - catenin pathway as a molecular target for future anti - cancer therapeutics. Int J Cancer.2005 Feb 10; 113 (4), 515 - 24. doi: 10.1002/ijc.20609. PMID: 15472907. [PubMed] [Google Scholar] [WILEY Online Library]

[4] Xiaohui Yan et al. Apigenin in cancer therapy: Anti - cancer effects and mechanism of action. Cell

Biosci.2017 Oct 5: 7 (1), 1 - 16. doi: 10.1186/s13578 - 017 - 0179 - x. [PubMed] [SPRINGER LINK] [Google Scholar]

[5] Kumar SR et al. Fucoxanthin: A marine carotenoid exerting anti - cancer effects by affecting multiple mechanism. Mar Drugs.2013 Dec16; 11 (12), 5130 - 5147. Doi: 10.3390/md11125130. PMCID: PMC3877908, PMID: 24351910. [PMC] [PubMed] [Google Scholar] [NIH]

[6] Bao - Kang Wu et al. A novel therapeutic strategy for hepatocellular carcinoma: Immunomodulatory mechanisms of selenium and/or selenoproteins on a shift towards anti - cancer. Int Immunopharmacol.2021 Jul: 96: 107790. Doi: 10.1016/j. intimp.2021.107790. Epub 2021 May 24. [PubMed] [Google Scholar] [NIH] [sciencedirect. com] [Elsevier]

[7] Hongping Yin et al. Fengycin inhibits the growth of human lung cancer cell line 95D through reactive oxygen species production and mitochondria - dependent apoptosis. Anticancer drugs.2013 Jul; 24 (6), 587 - 598. doi: 10.1097/CAD.0b013e3283611395 [PubMed] [Elsevier] [Europe PMC] [OUCI] [Research Gate]

[8] Al - Tarifi BY et al. Inhibitory effects of astaxanthin on LPS - induced raw 264.7 inflammation and breast cancer MCF - 7 cell lines proliferation. Sustain Sci.2022 Feb; 17 (2), 55 - 66. doi: 10.46754/jssm.2022.02.006. [Research Gate] [Elsevier]

[9] Sujit Nair et al. Natural dietary anti - cancer chemopreventive compounds: redox - mediated differential signaling mechanisms in cytoprotection of normal cells versus cytoxicity in tumor cells. Acta Pharmacol Sin.2007 Apr; 28 (4): 459 - 72. PMID: 17376285. doi: 10.1111/j.1745 - 7254.2007.00549. x [PubMed] [Elsevier] [CrossRef].

[10] R Pandey et al. Prodigiosins as anti - cancer agents: living up to their name. Curr Pharm Des.2009; 15 (7): 732 - 741. doi.10.2174/138161209787582192. [PubMed] [Elsevier] [CrossRef] [Research Gate].

[11] Sivasankara NS et al. In vitro anticancer activity of fucoidan extracted from Sargassum cinereum against Caco - 2 cells. Int J Bio.2019 Ocr1; 138: 18 - 626. doi: 10.1016/j. ijbiomac.2019.07.127. Epub 2019Jul 22. [PubMed] [Elsevier] [Google Scholar]

[12] Qiang Wang et al. Luteolin induces apoptosis by ROS/ER stress and mitochondrial dysfunction in gliomablastoma. Cancer Chemother Pharmacol.2017 May; 79 (5): 1031 - 1041. doi: 10.1007/s00280 - 017 - 3299 - 4. Epub 2017 Apr 9. [PubMed] [Europe PMC] [NIH] [Elsevier] [Google Scholar] [Research Gate] [OUCI].

[13] Benjamin G. Bitler, Joyce A. Schroeder. Anti - cancer therapies that utilize cell penetrating peptides. Recent Pat AntiCancer drug discov.2010 Jun; 5 (2): 99 - 108. doi: 10.2174/157489210790936252. [PMC] [PubMed] [Elsevier] [Google Scholar]

[14] Devaraj Ezhilarasan et al. The ambigous role of sirtuins in head and neck squamous cell carcinoma. Oral Diseases.2022 Apr; 28 (3): 559 - 567. doi: 10.1111/odi.13798. Epub 2021 Mar 1. [PubMed] [Google Scholar] [WILEY Online Library] [Elsevier] [Research Gate]

[15] Julin Jiang, Changping Hu. Evodiamine: a novel anticancer alkaloid from Evodia rutaecarpa. Molecules.2009 May 18; 14 (5): 1852 - 1859. doi: 10.3390/molcules14051852. . [OUCI] [PubMed] [Elsevier] [Google Scholar] [Research Gate]

[16] Anna Boss et al. Evidence to support the anti - cancer effect of olive leaf extract and future directions. Nutrients.2016 Aug; 8 (8): 513. doi: 10.3390/nu8080513. PMCID: PMC4997426. PMID: 27548217. [PMC] [PubMed] [OUCI] [Elsevier] [Figshare]

[17] Wenfang Li et al. Anti - tumor potential of astragalus polysaccharides on breast cancer cell line mediated by macrophages activation. Materials Science and Engineering.2019 May; 98: 685 - 695. doi: 10.1016/j/msec.2019.01.025. Epub 2019 Jan 8. [PubMed] [Elsevier] [Google Scholar] [Research Gate].