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Antitumor Activity of β-glucan Isolated from *Phoenix Dactylifera* L. Fruits on Cancer and Normal Cell Lines

Hiba Muhammed Al-Khuzaay[1*], Yasir Hussein Al-Juraisy[2], Yousif Hussein Mohammed[3], Ali Hussein Alwan[4]

1. *Department of Sciences, College of Basic Education, Mustansiriyah University, Baghdad, Iraq*
2. *Department of Biology, College of Science, Mustansiriyah University, Baghdad, Iraq*
3. *Department of Mathematics, College of Basic Education, Mustansiriyah University, Baghdad, Iraq*
4. *Iraqi Center for Cancer and Medical Genetics Research, Mustansiriyah University, Baghdad, Iraq*

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Corresponding Information:
Hiba Muhammed Al-Khuzaay,
Department of Sciences, College of Basic Education, Mustansiriyah University, Baghdad, Iraq

Email:
hibamuhammed@uomustansiriyah.edu.iq

ABSTRACT

Background & Objective: Recent research has focused on β-glucan from *Phoenix dactylifera* L. fruits as a potential anti-tumor agent. This study investigates its cytotoxic effects on cancer and normal cell lines, exploring its ability to inhibit tumor cell proliferation and activate immune responses, thus evaluating its potential as a selective anticancer treatment.

Materials & Methods: β-glucan was extracted from *Phoenix dactylifera* fruits using hot water extraction (glucan-P1), ion exchange chromatography (glucan-P2), and gel filtration chromatography (glucan-P3). Concentrations ranging from 31.25 to 1000 µg/mL were prepared. MCF-7, AMJ13, and REF cell lines were cultured in RPMI-1640 medium. Cytotoxicity was assessed using the MTT assay, measuring absorbance at 540 nm. Inhibition rates were calculated, and statistical analysis was performed using ANOVA and Dunn's test.

Results: The study evaluated the cytotoxic effects of three β-glucan preparations (glucan-P1, glucan-P2, and glucan-P3) on breast cancer cell lines MCF-7 and AMJ13, as well as normal REF cells. Glucan-P3 demonstrated the highest growth inhibition across all-time points (24, 48, and 72 hours) for both cancer cell lines. The most significant effect was observed on MCF-7 cells after 48 hours of exposure, with glucan-P3 at the highest concentration (1000 µg/ml) achieving 87.774% growth inhibition. AMJ13 cells showed similar trends, with glucan-P3 exhibiting the strongest inhibitory effect. Importantly, all β-glucan preparations showed minimal cytotoxicity towards normal REF cells after 72 hours of exposure, suggesting a selective anti-cancer effect.

Conclusion: β-glucan from *Phoenix dactylifera* showed significant antitumor activity against breast cancer cells with no effect on normal cells, highlighting its therapeutic potential.

Keywords: Breast Cancer, Phoenix Dactylifera, Selective Cytotoxicity, β-glucan

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1. Introduction

The exploration of natural compounds with antitumor properties has garnered significant attention in recent years, particularly in the context of enhancing cancer treatment efficacy and minimizing side effects. Among these compounds, β-glucan**,** a polysaccharide found in various plants and fungi, has emerged as a promising candidate due to its immunomodulatory effects. Specifically, β-glucan derived from *Phoenix dactylifera* L. (date palm) fruits has shown potential in preclinical studies for its ability to inhibit tumor cell proliferation and activate immune responses against cancer cells. This article aims to investigate the antitumor activity of β-glucan isolated from date palm fruits on both cancerous and normal cell lines, contributing to the understanding of its therapeutic potential **(1, 2)**.

The significance of β-glucan in cancer therapy lies in its multifaceted role as an immune modulator. Research indicates that β-glucan can enhance the activity of various immune cells, including macrophages and natural killer (NK) cells, which are crucial for the body's defense against tumors. By binding to specific receptors on immune cells, such as Dectin-1, β-glucan triggers a cascade of immune responses that can lead to increased production of pro-inflammatory cytokines and enhanced cytotoxicity against tumor cells. The ability of β-glucan to stimulate

both innate and adaptive immunity positions it as a valuable adjuvant in cancer treatment strategies **(3, 4)**.

Previous studies have demonstrated the anticancer properties of β-glucan from different sources, but research focusing specifically on the β-glucan extracted from *Phoenix dactylifera* is limited. Initial findings suggest that this specific β-glucan not only inhibits cancer cell growth, but may also improve the overall quality of life for patients undergoing conventional treatments such as chemotherapy. The dual action of β-glucan-acting directly on tumor cells while simultaneously modulating the immune system-highlights its potential as a complementary therapy in oncology. Moreover, the purification and characterization processes for extracting β-glucan from date palm fruits are critical for ensuring its efficacy and safety. Techniques such as hot water extraction followed by ethanol precipitation have been employed to obtain high molecular weight β-glucanase without altering their structural integrity. Understanding these methodologies is essential for optimizing the therapeutic use of β-glucanase in clinical settings **(5, 6)**.

Cancer is an abnormal growth of cells brought on by abnormal cell proliferation and has the ability to spread to other parts of the body through circulation **(7, 8)**. 1.41 million new cases of breast cancer are diagnosed each year, and 46,000 people pass away from the disease **(9)**. Breast cancer is the second-biggest cause of death for women and has a high rate of morbidity and mortality **(10, 11)**. One of the oldest forms of medicine is herbal medicine. Independent plant-based healing systems have evolved all across the world over time, including traditional Chinese medicine (TCM), Ayurveda, Kampo medicine, and Sa-sang in Korea **(12)**.

Phoenix dactylifera (date palm) belongs to the family Asteraceae; date trees can grow to a height of 21–23 meters, with leaves that can measure 4–6 meters in length. The trees typically spread out in clusters or independently of a single root system. There are over 100 million date trees in cultivation, 90% of which are in the Middle East **(13)**.

The date's fruits are where it gets the majority of its nutrients. The fruits of dates are rich in minerals, vitamins, carbohydrates and amino acids **(14)**. β-glucan is a structural component of the plant and cell walls of many microorganisms, including bacteria, yeast, and fungi **(15)**. In this study, investigate the cytotoxic effect of β-glucan that was isolated from *Phoenix dactylifera* fruits by water extraction and ethanol precipitation (glucan-P1), partially purified by DEAE (glucan-P2) and completely purified by Sephadex G-100 column chromatography (glucan-P3) on cancer and normal cell lines. This article aims to systematically evaluate the antitumor activity of β-glucan isolated from *Phoenix dactylifera* fruits by conducting in vitro studies on various cancer cell lines alongside normal cell lines. By comparing the effects on both types of cells, we seek to elucidate the selective cytotoxicity of β-glucan and its potential implications for future cancer therapies. Ultimately, this research could pave the way for innovative treatment modalities that harness natural compounds like β-glucan to improve patient outcomes in cancer care.

2. Materials and Methods

β-glucan Preparation

β-glucan was previously isolated from dried fruits of *Phoenix dactylifera* by hot water extraction (glucan-P1), partial purified by ion exchange chromatography (glucan-P2) then completely purified by gel filtration chromatography (glucan-P3) **(9,16)**.

Preparation of β-glucan Concentrations

β-glucan (glucan-P1, glucan-P2 and glucan-P3) stock was prepared by dissolving 200 mg of prepared β-glucan powder in 10 mL phosphate puffer saline and filtering through a Millipede filter (0.22µm). Using sterile serum-free medium for various concentrations' preparation starting from concentration 1000 µg/mL to 31.25 µg/ml.

Cell Culture and Maintenance

Human breast cancer cells (MCF-7 and AMJ13) and normal cell lines were graciously provided by The Iraqi Center for Cancer and Medical Genetics Research (ICCMGR), Mustansiriyah University. In RPMI-1640 (Sigma Aldrich, USA) supplemented with 10 % fetal bovine serum, cells were maintained and cultivated. In tissue culture flasks, cells (3×10^4 cells mL^{-1}) were plated and given 24 to 48 hours to create an 80–90% confluent monolayer. Cultures were kept in a humid environment at 37°C using a CO_2 incubator. The cells were extracted using a mild atropinization procedure (50 mg/mL trypsin) **(17)**.

MTT cytotoxicity assay

The cytotoxic effect of β-glucan on human breast cancer cell lines (MCF-7 and AMJ13) and normal cell lines (REF) was calculated using the 3-(4,5-Dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) assay. Cells were cultivated and incubated in 96-well plates until they achieved 80% confluence. After discarding the medium, 200 µL of various β-glucan concentrations (31.25, 62.5, 125, 250, 500, and 1000) µg/mL were added to the appropriate wells containing the cells. As the adverse control in wells, cells that had not been treated were employed. After 24 hours, 20 µL of MTT was added to each well. At 37°C and 5% CO_2, the plates underwent an additional four hours of incubation. Each well received 100 µL of dimethyl sulfoxide after the media had been carefully removed, and each was then given 5 minutes to incubate. Using a microplate reader for an ELISA was to quantify absorbance at 540 nm **(18)**. The following formula was used to compute the inhibition percentage (%)**:**

IR (%) = (ODc− ODt) / OD Control ×100

IR = Inhibitor rate, ODc = the optical density of control, ODt= the optical density of test

Statistical Analysis

Data were presented as means with standard deviations (SD) and evaluated using Graph Pad Prism's one-way analysis of variance (ANOVA) and Dunn's test (Graph Pad Software Inc.). A statistically significant difference between groups was defined as $p< 0.05$ **(19)**.

3. Results

Cytotoxic effect of β-glucan (glucan-P1, glucan-P2 and glucan-P3) on breast cell line (MCF-7)

The results in Figure 1 revealed that the glucan-P3 had the greatest growth inhibition effect on MCF-7 cells than the other tested β-glucan after 24 hours of exposure. The results in Figure 2 revealed that glucan-P3 had the greatest growth inhibition effect on the MCF-7 cell line than glucan-P1 and glucan-P2 after 48 hours of exposure. Figure 3 showed that glucan-P3 had the greatest growth inhibition effect on the MCF-7 cell line than the glucan-P1 and glucan-P2 after 72 hours of exposure. The highest growth inhibition percentage for glucan-P3 at the highest concentration was 87.774 % after 48 hours of exposure.

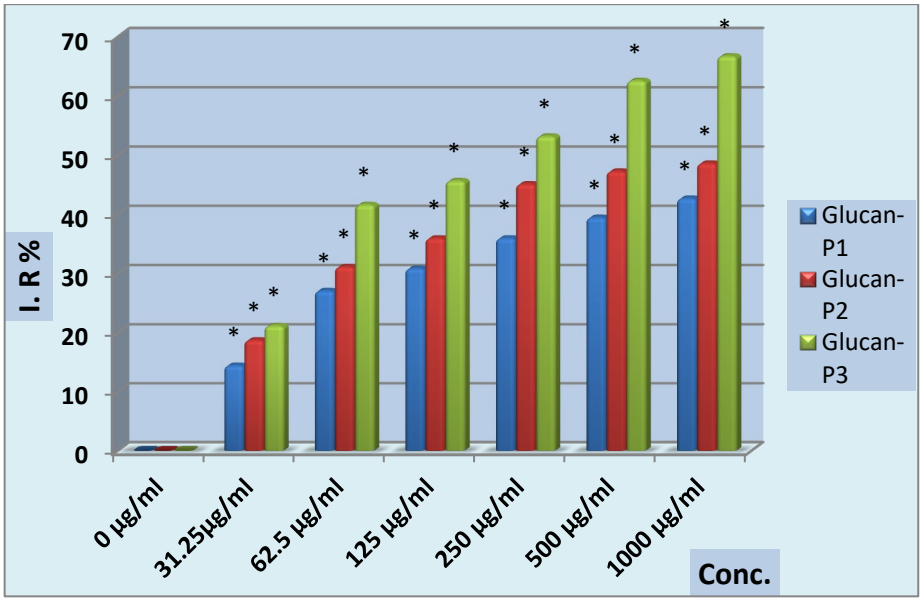


Figure 1. A comparison of growth inhibition percentages on MCF-7 cell line by glucan-P1, glucan-P2, and glucan-P3 after 24 hours of exposure.

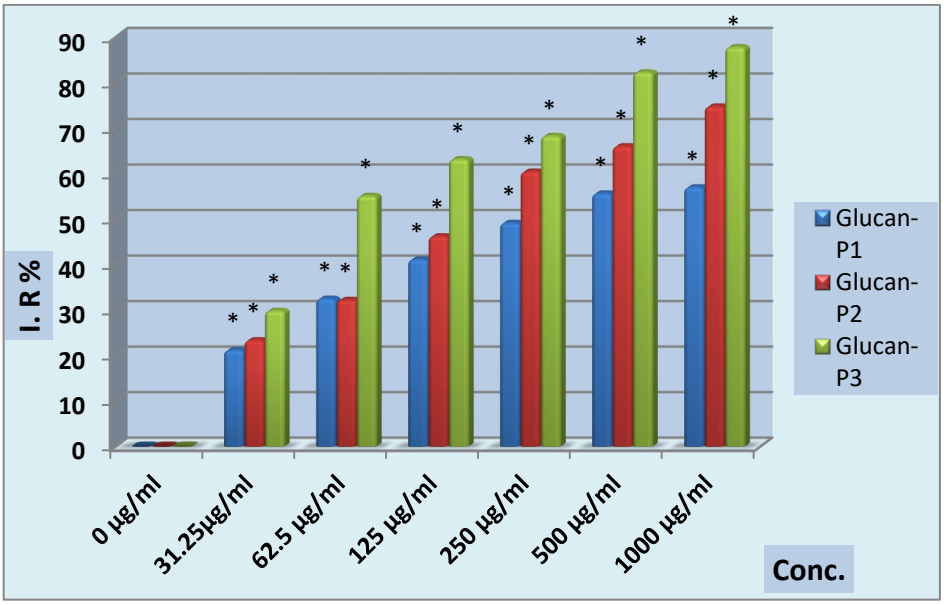


Figure 2. A comparison of growth inhibition percentages on MCF-7 cell line by glucan-P1, glucan-P2, and glucan-P3 after 48 hours of exposure.

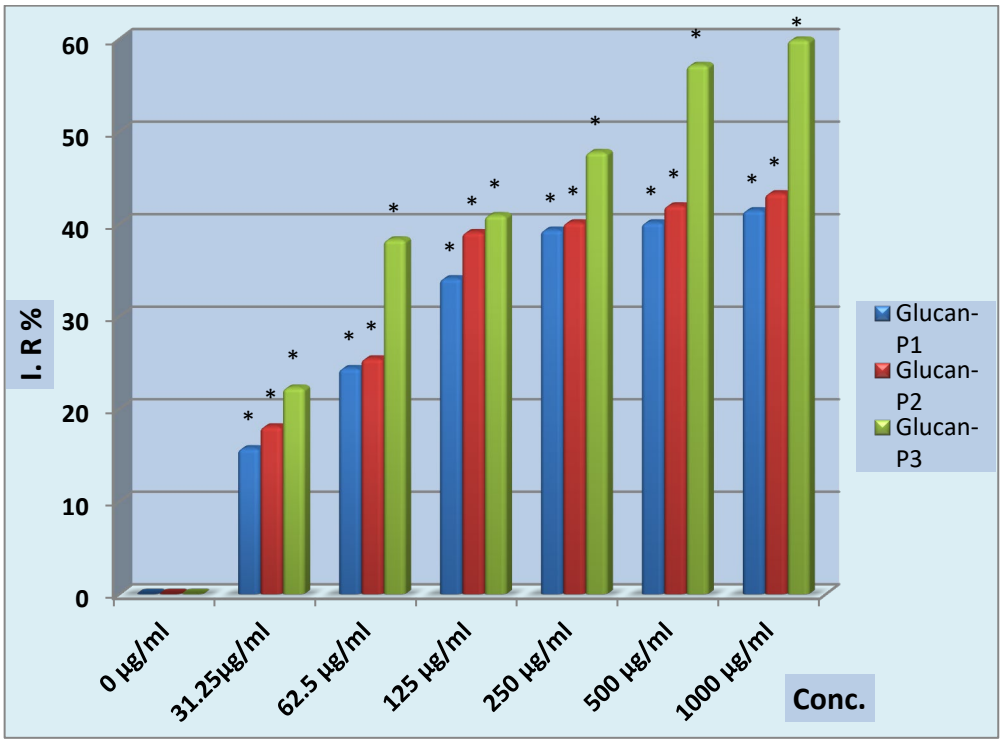


Figure 3. A comparison of growth inhibition percentages on MCF-7 cell line by glucan-P1, glucan-P2, and glucan-P3 after 72 hours of exposure.

Cytotoxic effect of β-glucan (glucan-P1, glucan-P2 and glucan-P3) on Breast cancer cell line (AMJ13)

The effect of different concentrations of β-glucan (glucan-P1, glucan-P2 and glucan-P3) (31.25, 62.5, 125, 250, 500, and 1000) µg/ml on the AMJ13 cell line after 24 hours of exposure is shown in Figure (4), and after 48 hours of exposure are shown in figure 5, and after 72 hours of exposure are shown in Figure 6. The results revealed significant cytotoxic effects at levels ($P<0.05$) for all concentration. Completely purified β-glucan (glucan-P3) had the highest inhibitory growth on MCF-7 at the highest concentration, 1000 µg/ml after 48 hours of exposure.

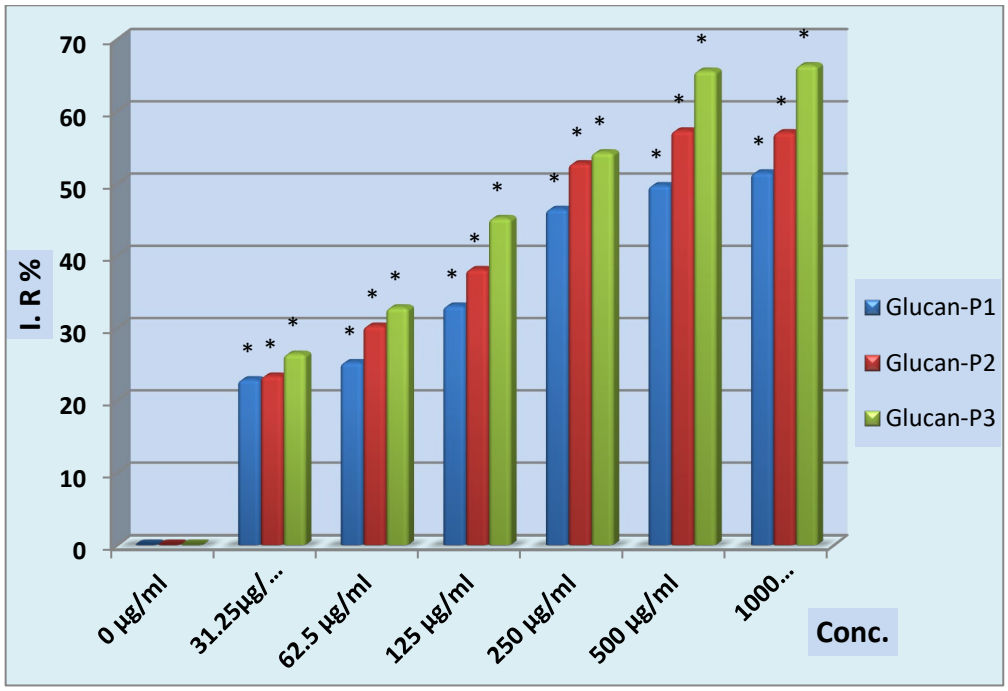


Figure 4. A comparison of growth inhibition percentages on the AMJ13 cell line by glucan-P1, glucan-P2, and glucan-P3 after 24 hours of exposure.

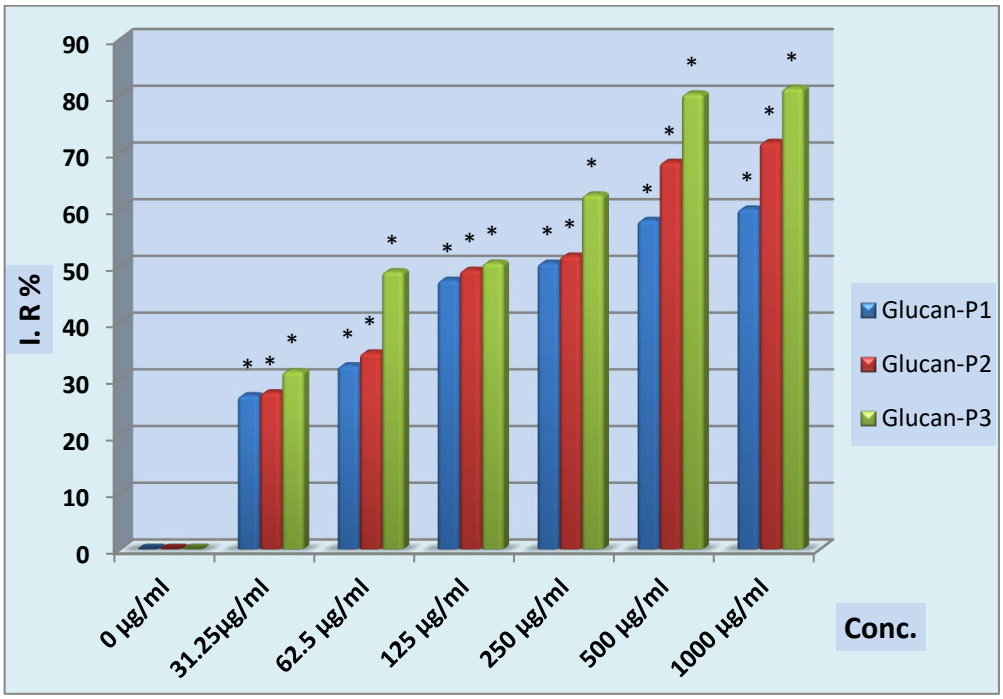


Figure 5. A comparison of growth inhibition percentages on the AMJ13 cell line by glucan-P1, glucan-P2, and glucan-P3 after 48 hours of exposure.

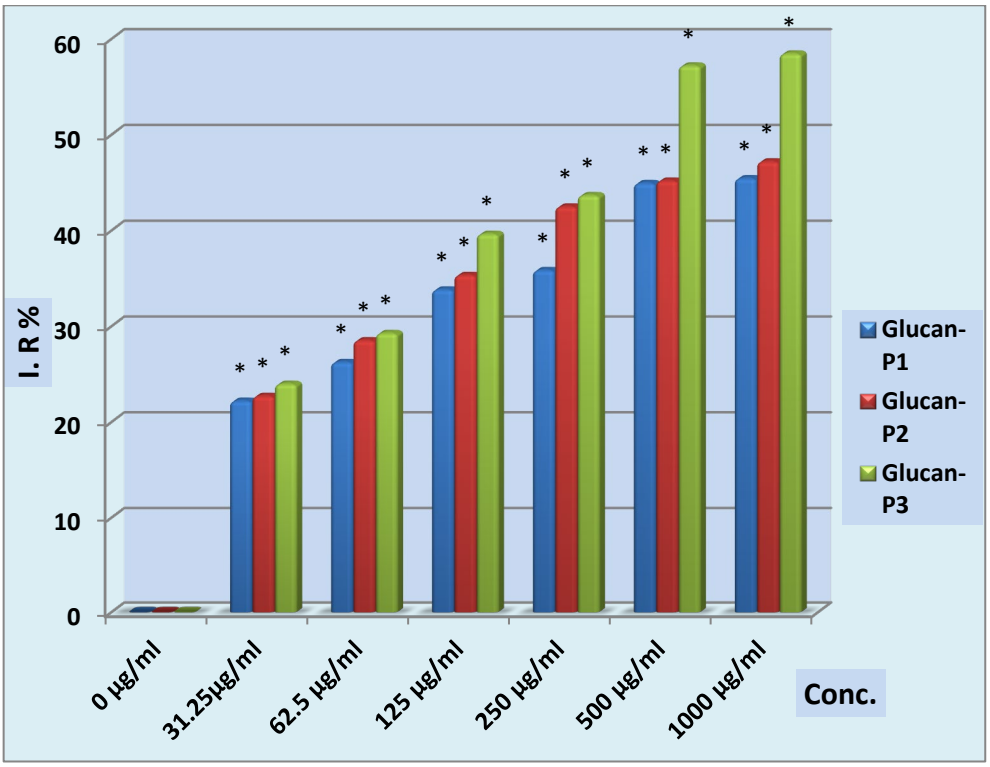


Figure 6. A comparison of growth inhibition percentages on the AMJ13 cell line by glucan P1, glucan P2, and glucan P3 after 72 hours of exposure.

Cytotoxic effect of β-glucan (glucan-P1, glucan-P2 and glucan-P3) on normal cell lines (REF)

Figure (7) showed a very low degree of growth inhibition of β-glucan (glucan-P1, glucan-P2 and glucan-P3) on REF cells after 72 hours of exposure.

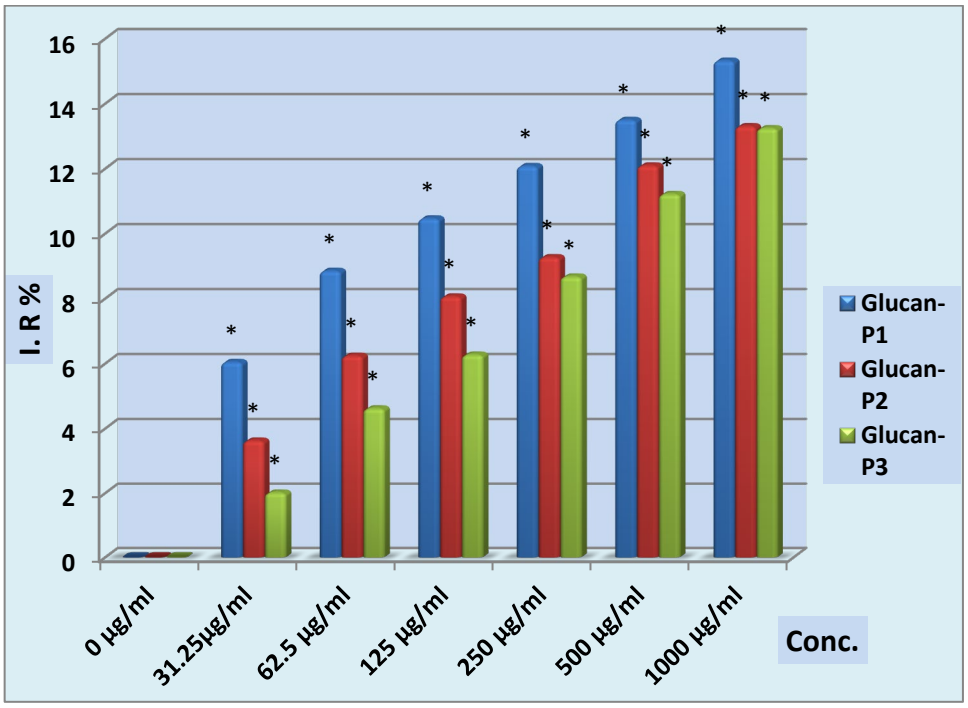


Figure 7. A comparison of growth inhibition percentages on REF cell line by glucan-P1, glucan-P2, and glucan-P3 after 72 hours of exposure.

4. Discussion

After being exposed to various concentrations of β-glucan for 24, 48, and 72 hours, the stained cell lines' optical densities (OD) revealed variances between the concentrations, with the high concentration resulting in a low OD value, indicating the highest response. Additionally, the low concentration resulted in a high OD value, indicating a minimal reaction in contrast to a high percentage of viable cells. The findings of this study showed that β-glucan selectively affected the vitality of different cell lines, and that this selective effect of β-glucan may manifest itself in cell adhesion. These findings suggest that β-glucan can cause cells to detach from a plate by preventing them from adhering to it.

The study of the cytotoxic effects of β-glucan isolated from *Phoenix dactylifera* L. on breast cancer cell lines, specifically MCF-7 and AMJ13, reveals significant insights into its potential as an antitumor agent. The results indicate that glucan-P3 exhibits the most potent growth inhibition across both cancer cell lines after 48 hours of exposure, achieving an impressive growth inhibition percentage of 87.774% at the highest concentration tested (1000 µg/ml). This finding is consistent with previous research that highlights the importance of molecular structure and purity in determining the efficacy of β-glucan as an anticancer agent. The superior performance of glucan-P3 suggests that its specific structural characteristics may enhance its interaction with tumor cells, leading to increased cytotoxicity **(1, 20)**.

The differential effects observed among the three β-glucan variants (glucan-P1, glucan-P2, and glucan-P3) underscore the importance of purification methods and molecular weight in the biological activity of polysaccharides. Glucan-P3's significant inhibitory effect on MCF-7 cells after 24, 48, and 72 hours indicates sustained action that could be leveraged in therapeutic applications. These results align with existing literature that demonstrates how β-glucanase can modulate immune responses and directly inhibit cancer cell proliferation through various mechanisms, including apoptosis induction and cell cycle arrest. The findings suggest that glucan-P3 could serve as a valuable adjunct to traditional cancer therapies by enhancing their effectiveness **(20, 21)**. In contrast to the pronounced effects on cancer cell lines, the minimal growth inhibition observed in normal REF cells after exposure to β-glucanase suggests a degree of selectivity in their action. This selectivity is crucial for therapeutic applications, as it implies that β-glucanase may spare normal cells while targeting malignant ones. Such properties are essential for reducing the side effects commonly associated with conventional cancer treatments. Previous studies have indicated that β-glucanase can stimulate immune responses without adversely affecting healthy tissues, making them attractive candidates for combination therapies aimed at improving patient outcomes **(22, 23)**.

The significant cytotoxicity observed in both MCF-7 and AMJ13 cell lines further supports the hypothesis that β-glucanase may play a role in overcoming resistance mechanisms often encountered in breast cancer treatment. The ability of glucan-P3 to inhibit growth effectively even at lower concentrations

suggests potential for use in clinical settings where maximizing therapeutic efficacy while minimizing toxicity is paramount. Additionally, the findings raise intriguing questions about the underlying mechanisms by which β-glucanase exert their effects—whether through direct cytotoxicity or by modulating immune responses that enhance tumor surveillance **(24, 25)**.

β-glucan has anticancer properties and the ability to boost the immune system. Cancer cells are destroyed by β-glucan in a complex and poorly understood manner. Numerous intriguing studies have shown that β-glucan can affect cancer cells both *in vitro* and *in vivo*. Even so, there isn't any conclusive evidence that it can be utilized as a cancer preventative (26). Due to their global anticancer properties, β-glucan has received a lot of interest lately **(27)**. Many anticancer drugs function by placing tumor cells under oxidative stress, which is regarded to be the main factor in the majority of macromolecular alterations in the cell. Proteins, membrane lipids, and large molecules like DNA can all be harmed by reactive oxygen species **(28)**. After incubation with β-glucan, we noticed a proportional decline in the viability of all tested MCF-7 and AMJ13 cancer cell lines. In order to validate the cytotoxicity of β-glucan, Hong and Jung **(29)** concentrated on the anticancer effect of β-glucan produced by microorganisms on four cancer lines. Hela and Sarcoma 180 cells were unquestionably toxic.

5. Conclusion

In the present study, β-glucan that was isolated from *Phoenix dactylifera* fruits exhibited a significant antitumor activity against breast cancer cell lines (MCF-7 and AMJ13) in concentration and time dependent manner. The cytotoxic effect of β-glucan was more on MCF-7 than on AMJ13, while not affect on normal cell line (REF). In conclusion, this study contributes valuable data to the growing body of evidence supporting the antitumor activity of β-glucanase derived from *Phoenix dactylifera*. The pronounced effects of glucan-P3 on breast cancer cell lines highlight its potential as a therapeutic agent, warranting further investigation into its mechanisms of action and clinical applications. Future research should focus on elucidating these mechanisms and exploring the synergistic effects of combining β-glucanase with existing cancer therapies to optimize treatment protocols for breast cancer patients. The promising results pave the way for innovative approaches to cancer treatment that harness natural compounds for enhanced efficacy and reduced side effects.

6. Declarations

Acknowledgments

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Ethical Considerations

The Institutional Animal Care and Use Committee (IACUC-SCI-TU-00179) had the capability to underwrite this study which was guided by the rules provided by the Moral Committee of the Faculty of Science at Tanta University.

Authors' Contributions

All authors contributed to the study conception and design. Material preparation, data collection and analysis were performed by Hiba Mohammed Al-Khuzaay, Yasir Hussein Al-Juraisy, Yousif Hussein Mohammed and Ali Hussein Alwan. The first draft of the manuscript was written by Hiba Mohammed Al-Khuzaay and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

Conflict of Interest

The authors declare no conflict of interest. The authors have no relevant financial or non-financial interests to disclose.

Fund or Financial Support

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Data Availability Statement

All data generated or analyzed during this study are included in this published article.

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