Exploring the Effects of WJ-MSC-Derived Exosomes on the Viability and Metabolic Activity of Lung, Pancreatic and Liver Cancer Cells*

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Abstract

Aim: Mesenchymal stem cells (MSCs) act on cancer cells in the same microenvironment by secreting cytokines, chemokines, exosomes, or by direct contact. The purpose of this study is to analyse the impact of WJ-MSC-derived exosomes on the viability and metabolic activity of lung, pancreatic, and liver cancer cells.

Method: In this study, Wharton's Jelly (WJ)-MSCs-derived exosomes were added to A549, Panc-1, and HepG2 cells as 1-5-10-15 and 20 million particle exosomes. The effects on viability were evaluated by MTT analysis at 48 and 96 hours. The effects on metabolic activity were evaluated by calcein staining at the end of the 96th hour. Cell metabolic activity was calculated using fluorescence intensity.

Results: At the end of the 96th hour, 62% increase in A549 cells, 95% increase in Panc-1 cells and 67% increase in HepG2 cells were detected in the control group. In the group which 20 million exosomes were applied, 36% increase in A549 cells, 43% increase in Panc-1 cells and 31% increase in HepG2 cells were observed. The fluorescence intensity of 20 million exosomes was calculated as: A549: 61%, Panc-1: 64% and HepG2: 68%. Preliminary findings indicate that WJ-MSC-derived exosomes do not increase the viability and metabolic activity of cancer cells when applied to them. It was also shown that the viability and metabolic activity can decrease depending on time and concentration.

Conclusion: WJ-MSC-derived exosomes may be effective on cell viability and metabolic activity *in vitro* in tumors with high stroma content, and their effects on other mechanisms on tumor cells need to be investigated.

Keywords: WJ-MSC, MSC derived exosome, cancer cell, tumor stroma.

Wharton Jölesi Kökenli Mezenkimal Kök Hücrelerden Elde Edilen Eksozomların Akciğer, Pankreas ve Karaciğer Kanseri Hücrelerinin Canlılığı ve Metabolik Aktivitesi Üzerindeki Etkilerinin Araştırılması

Öz

Amaç: Mezenkimal kök hücreler (MKH) sitokinler, kemokinler, eksozomlar salgılayarak veya doğrudan temas yoluyla aynı mikroçevredeki kanser hücreleri üzerinde etki gösterir. WJ-MKH kökenli eksozomların akciğer, pankreas ve karaciğer kanseri hücre hatlarının canlılığı ve metabolik aktivitesi üzerindeki etkileri incelenmesi amaçlanmıştır.

Özgün Araştırma Makalesi (Original Research Article)
Geliş / Received: 16.08.2024 **Kabul / Accepted:** 10.11.2025
DOI: https://doi.org/10.38079/igusabder.1534054

* Parts of the data in this study was presented as a poster declaration titled "Evaluation of Antiproliferative Effects of WJ-MSC-Derived Exosomes on Lung, Pancreatic and Liver Cancer Cells" at the 1st Extracellular Vesicles Conference 2024.

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Yöntem: Bu çalışmada Wharton Jölesi (WJ)-MKH kökenli eksozomlar A549, Panc-1 ve HepG2 hücrelerine 1-5-10-15 ve 20 milyon partikül eksozom olarak uygulandı. Canlılık üzerindeki etkiler 48 ve 96. saatlerde MTT analizi ile değerlendirilmiştir. Metabolik aktivitesi üzerindeki etkiler 96. saatin sonunda kalsein boyaması ile floresan yoğunluğu ile değerlendirilmiştir. Hücre metabolik aktivitesi floresan yoğunluğu kullanılarak hesaplanmıştır.

Bulgular: Kontrol grubunda 96. saatin sonunda A549 hücrelerinde % 62, Panc-1 hücrelerinde %95 ve HepG2 hücrelerinde %67 artış gözlemlenirken 20 milyon eksozom uygulanan grupta ise A549 hücrelerinde %36, Panc-1 hücrelerinde %43 ve HepG2 hücrelerinde %31 artış gözlenmiştir. Proliferasyon oranı 20 milyon eksozom uygulanan grupta kontrol grubuna göre (% 100) A549: %61, Panc-1: %64 ve HepG2: %68 olarak hesaplanmıştır. Ön bulgular, WJ-MSC kökenli eksozomların kanser hücrelerine uygulandığında kanser hücrelerinin canlılığını ve metabolik aktivitesini artırmadığını ve zaman-konsantrasyona bağlı olarak azalabildiği de gösterilmiştir.

Sonuç: WJ-MKH türevi eksozomlar, yüksek stroma içeriğine sahip tümörlerde *in vitro* hücre canlılığı ve metabolik aktivite üzerinde etkili olabilir ve tümör hücreleri üzerindeki diğer mekanizmalar üzerindeki etkilerinin araştırılması gerekmektedir.

Anahtar Sözcükler: WJ-MKH, MKH kökenli eksozom, kanser hücresi, tümör stroması.

Introduction

Mesenchymal stem cells (MSCs) are fibroblast-like stromal cells with high regeneration potential and therapeutic properties such as anti-apoptotic, pro-angiogenic and chemoattractant[1]. MSCs are therapeutically used in graft-versus-host disease, autoimmune diseases, haematological malignancies, cardiovascular diseases, neurological diseases, organ transplants, diabetes and regenerative medicine[2]. Even though MSCs show antitumour effects in both *in vitro* and *in vivo* studies, there are also studies showing that they support tumour cells[3]. As the direct impact of mesenchymal stem cells in cancer treatment varies according to the source, dose, and cancer type, they are utilised for therapeutic aims in cancer by being modified in different ways[4]. MSCs are also an essential modulator of the microenvironment in tumours consisting of a high amount of stroma. The interplay with cancer cells and MSCs regulates many crucial functions of cancer cells, such as proliferation, metastasis and epithelial-mesenchymal transition, and can secrete exosomes that can modulate the tumour microenvironment[5]. Recently, the therapeutic use of the extracellular vesicles of MSCs in acute tissue damage of different organs such as heart, brain, kidney, lung and liver, their effects on tumour progression and their possible potential in cancer treatment have been investigated[6].

Exosomes are known to carry a wide variety of biomolecules, including membrane proteins, cytosolic and nuclear proteins, extracellular matrix components, cytokines, metabolites, and nucleic acids such as mRNA, non-coding RNAs, and DNA[7]. Mesenchymal stem cell (MSC)-derived exosomes have been shown to influence key cancer-related processes, including tumour growth, metastasis, apoptosis, angiogenesis, chemosensitivity, and drug resistance. For example, exosomes from bone marrow-derived MSCs have been reported to increase the sensitivity of glioblastoma multiforme cells to temozolomide through the delivery of miR-9[8]. Similarly, exosomes derived from human umbilical cord MSCs have been associated with reduced proliferation and

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increased apoptosis in bladder cancer cells[9]. Furthermore, MSCs treated with paclitaxel demonstrated strong anti-tumour activity in pancreatic cancer models by absorbing and subsequently releasing the drug[10]. However, while several studies highlight the tumour-inhibitory properties of MSC-derived exosomes, others report contradictory findings, indicating tumour-promoting effects in various *in vitro* and *in vivo* models[11]. This inconsistency suggests that the biological effects of MSC exosomes are highly context-dependent and influenced by factors such as the source of MSCs and the type of cancer studied. Indeed, a meta-analysis revealed that only 26% of studies on bone marrow-derived MSCs and 46% on adipose tissue-derived MSCs reported oncosuppressive effects, whereas 88% of studies on Wharton's jelly-derived MSCs supported a tumour-suppressive role[12]. These findings underscore the heterogeneity of exosome content depending on their cellular origin and highlight the need for more systematic and comparative studies to fully understand the dual role of MSC-derived exosomes in cancer biology.

MSC-derived exosomes exhibit context-dependent effects on tumors. Bone marrow and umbilical cord MSC-exosomes promote tumor progression through EMT induction, activation of signaling pathways, and miRNA-mediated mechanisms, whereas Wharton's jelly and umbilical cord MSC-exosomes demonstrate tumor-suppressive effects by inducing apoptosis, inhibiting proliferation, and enhancing drug sensitivity (Table 1). This dual role highlights the importance of detailed characterization of the source and cargo of MSC-exosomes for therapeutic applications[21]. Accordingly, the effects of MSC-derived exosomes on tumors are summarized in Table 1.

Table 1. Effects of MSC-derived exosomes (MSC-Exos) on tumor cells

MSC Source	Effect Type	Mechanism / Content	Affected Cancer / Cell Type	Reference
Umbilical Cord MSC-Exos	Tumor-promoting	Induction of EMT, activation of Akt	Gastric cancer	(13)
Bone Marrow MSC-Exos	Tumor-promoting	ERK1/2 signaling pathway and VEGF expression	Gastric cancer	(14)
Bone Marrow MSC-Exos	Tumor-promoting	Decreased miR-15a, supports clonal expansion	Multiple myeloma	(15)
Bone Marrow MSC-EVs	Tumor-promoting	Activation of Hedgehog signaling	Osteosarcoma, Gastric cancer	(16)
MSC-Exos	Tumor-suppressive	circ_6790 → downregulation of S100A11, DNA hypermethylation via CBX7	Pancreatic ductal adenocarcinoma (PDAC)	(17)
Umbilical Cord MSC-Exos	Tumor-suppressive	Inhibition of proliferation, induction of apoptosis	Bladder tumor cells	(9)

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Bone Marrow MSC-Exos	Tumor-suppressive	miR-16-5p → regulation of integrin α2, induction of apoptosis	Colorectal cancer	(18)
Bone Marrow MSC-Exos	Tumor-suppressive	miR-124 → reduction of proliferation, EMT, and drug resistance	Pancreatic cancer	(19)
Bone Marrow MSC-Exos	Tumor-suppressive	hsa-miR-143-3p → induction of apoptosis, inhibition of growth and invasion	Pancreatic cancer	(20)

WJ-MSC-derived exosomes might have an impact on *in vitro* cell metabolism in tumours with a high stroma content and other effects on tumour cells need to be explored. The objective of our present study was to assess the viability and metabolic activity effects of exosomes obtained from WJ-MSCs on lung, pancreatic and liver cancer cell lines at different amounts and times. This study is among the first reports to evaluate the effects of WJ-MSC derived exosomes on these cancer cells in relation to both dose and time parameters. In this regard, our findings provide the existing literature with a more detailed and comparative perspective on the potential therapeutic use of WJ-MSC derived exosomes.

Material and Methods

Characterization of WJ-MSC-Derived Exosomes

Exosomes obtained from WJ-MSCs were obtained from Curecell, Osteopharma (Ankara, Turkey). For exosome characterisation, exosome surface markers were determined by flow cytometry and size and quantity were determined by Nanoparticle tracking analysis (NTA). For exosome characterisation by immunophenotypic cytometry, exosome surface markers were labelled with relevant antibodies. ExoStepTM Kit (Immunostep, Salamanca, Spain) was used for this. The labelled exosomes were analysed for marker positivity in a flow cytometer (Beckman Coulter, California, USA). A specific antibody detectable by the device consisting of 6 µm diameter magnetic beads coated with CD63 capture antibodies was used as secondary antibodies with CD9 detector antibodies labelled with red fluorescence and detected by the flow cytometer fluorescence detector[22]. The size and quantity analyses of exosomes were conducted using a NanoSight NTA 3.4 instrument (Malvern Panalytical, UK).

Cell Culture

Lung adenocarcinoma cells (A549/ATCC:CCL-185), pancreatic adenocarcinoma cells (Panc-1/ATCC:CRL-1469) and hepatocellular carcinoma (HepG2/ATCC:HB-8065) cell lines were used. All cells were cultured in T-25 flasks containing 1% L-Glutamine, 10% Fetal Bovine Serum, 1% penicillin/streptomycin and high glucose DMEM (Capricorn, Germany) at 37°C and 5% CO_2 incubator.

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Cell Viability Analysis

MTT was performed to assess the viability of WJ-MSC-derived exosomes on cancer cells. For each experiment, 5000 cells were seeded in 96-well plates for 48 and 96 hours evaluation. Three controls were used for each group. After the cells adhered, 1-5-10-15 and 20 million exosomes were added to each well in saline. Each 1 µL saline contained 1 million exosomes. At 48 and 96 hours, 10 µL MTT solution (Biotium, California, USA) with a final concentration of 0.5 mg/mL Absorbance was then recorded at a wavelength of 570 nm using a microplate reader (BioTek Synergy H1, BioTek Instruments, Winooski, VT, USA) and the viability rates of the experimental groups were determined by accepting the OD value as 100%.

Cell Metabolic Activity Analysis

To investigate the metabolic activity of WJ-MSC-derived exosomes on cancer cells, calcein staining was performed and analysed by fluorescence intensity. For analyses at 96 hours, 5000 cells were seeded in 96-well plates. After cells attached, 1-5-10-15 and 20 million exosomes were added to each well in saline. At the end of 96 h, 1µM calcein-acetoxymethyl ester (Santa Cruz Biotechnology, Inc., Dallas, TX, USA) dye was added and incubated at 37°C in the dark for 30 min. A Leica DM IL fluorescence inverted microscope (Leica, Germany) was utilise to visualise live cells. The fluorescence intensity (Excitation: 488 nm /Emission: 520 nm) was then measured in a microplate reader. Cell metabolic activity was expressed as a percentage compared to the control group, which was adjusted to 100% using fluorescence intensity.

Statistical Analysis

Statistical analyses and graphical representations were performed using GraphPad Prism version 8.1 (GraphPad Software, San Diego, CA, USA). Results are presented as mean ± standard deviation (SD). Due to the small sample size and violation of normality assumptions, non-parametric alternatives were used for two-way comparisons. Specifically, the Kruskal-Wallis test was applied for group comparisons based on each independent factor, followed by Dunn's post hoc test for multiple comparisons. Untreated cells were used as the control group. A p-value of less than 0.05 was considered statistically significant and indicated with an asterisk (*). The significance levels were denoted as follows: *$p<0.05$, **$p<0.01$, ***$p<0.001$, ****$p<0.0001$.

Results

Characterization of WJ-MSC-Derived Exosomes

In this section, Nanoparticle Tracking Analysis instrument and flow cytometry equipment were used to display the characterisation of exosomes. The size of exosomes in ml and the number of particles per ml were also determined by this analysis in five separate measurements of the exosome sample with the NTA instrument (Figure 1a). NTA results showed that the average exosome size was 129.4 +/- 1.1 nm and the graph showing the average concentration is presented (Figure 1b). Dot plot of the density and size of exosome particles is presented (Figure 1c). The graph showing the setting of the

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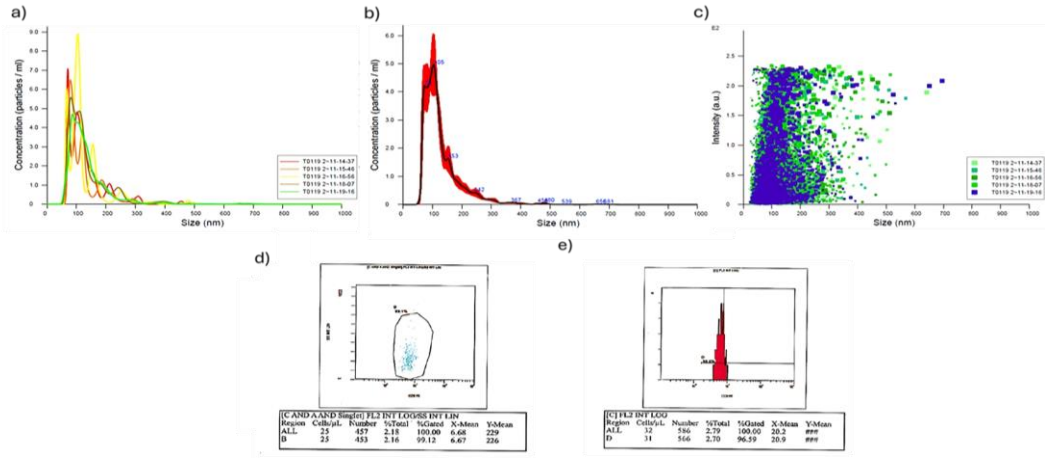
gate of the target cell cluster is presented (Figure 1d). CD63 and CD9 surface markers were positive above 96.59% (Figure 1e). All experiments conducted for characterization confirmed that the obtained Exracellular vesicules were exosomes.

Figure 1. NTA analysis result and flow cytometry analysis of exosomes. Graph indicating the size and concentration per ml in five separate analyses of the exosome sample by the instrument (a). Graph showing the average concentration (b). Dot plot of density and size of exosome particles (c). Graph display the gating of the target cell cluster (d). Graphical representation of positive cells within the cell cluster (e).



Effects of WJ-MSC-Derived Exosomes on Cancer Cells Viability

The analyses in this section were performed at 48 and 96 hours by MTT method to determine the viability of WJ-MSC-derived exosomes on cancer cells. At the end of 48th hour In A549 cell line; 32% increase was observed in the control and saline groups. In A549 cell line; 1 million: 35%, 3-5 million: 29.6%, 10 million: 25.3%, 15 million: 23.3% and 20 million: 20.3% increase was observed. At the end of 96th hour, 62% increase was observed in the control group and 58% increase was observed in the saline group. In A549 cell line; 1 million: 61%-5 million: 61.3%-10 million: 54.3%-15 million: 53.3% and 20 million: 36% increase was observed (Figure 2a). In Panc-1 cell line; 64.3% increase was observed in the control group and 63% increase in the saline group at 48 hours. At 48th hour; 1 million: 60-5 million: 61,3- 10 million: 56%- 15 million: 50,6% and 20 million: 48% increase was observed. At the end of 96th hour, 95% and 93% increase was observed in the saline group. At 96th hour; 1 million: 91.6% -5 million: 90.3% -10 million: 63.3% -15 million: 46% and 20 million: 43.6% increase was observed (Figure 2b). In HepG2 cell line; 36.6% increase was observed in control group and 36% increase in saline group at 48th hour. At 48th hour; 1 million: 33%, 3-5 million: 32.6% - 10 million: 32.3% - 15 million: 29% and 20 million: 25.3% increase was observed. At the end of 96th hour, 67% and 65% increase was observed in the saline group. At 96th hour; 1 million: 67% - 5 million: 64% - 10 million: 60,3% - 15 million: 40,3% and 20 million: 31% increase was observed (Figure 2c). In general, when the results at 48 hours were evaluated, a significant decrease in viability was observed in 15 and 20 million experimental groups.

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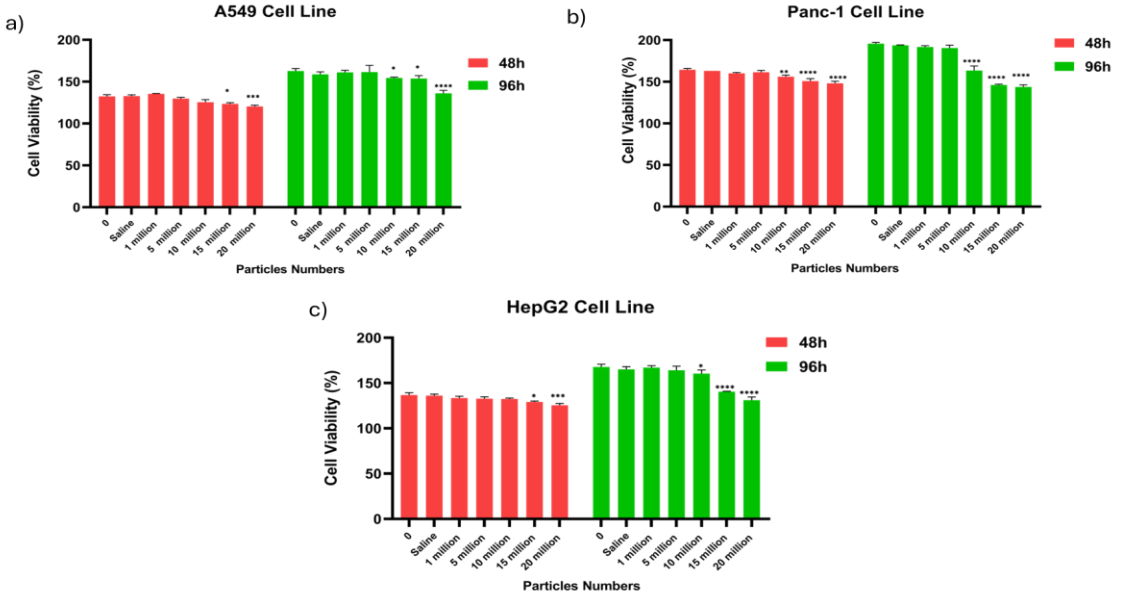
At 96 hours, significant results were also observed in cells treated with 10 million exosomes. It can be stated that WJ-MSC-derived exosomes may reduce viability depending on the amount.

Figure 2. The effects of 1, 5, 10, 15, and 20 million WJ-MSC exosomes treated with 5000 cells on the viability of A549 (a), Panc-1 (b) and HepG2 (c) cells at 48 and 96 hours. Values are expressed as mean ± SD with n=3.



Effects of WJ-MSC-Derived Exosomes on Cancer Cells Metabolic Activity

The experiments in this part were performed at 96 h by calcein staining method to determine the metabolic activity of WJ-MSC-derived exosomes on cancer cells. The images of the control group cells were taken by fluorescence microscope with calcein staining (Figure 3a). At 96 hour calcein staining, the fluorescence intensity of the control group (Excitation: 488 nm /Emission: 520 nm) was accepted as 100%. At the end of 96th hour in A549 cell line; 1 million: 98%, 5 million: 96.5%, 10 million: 83%, 15 million: 72.5% and 20 million: 64% if the control group is accepted as 100%. At the end of 96th hour in HepG2 cell line; 1 million: 99%; 5 million: 97%, 10 million: 89%, 15 million: 74.5% and 20 million: 68.5% if the control group is accepted as 100%. In overall metabolic activity results at 96th hour, no significant results were observed in the 1 and 5 million groups in all three cancer cells. Significant results were observed in cells treated with 10 million, 15 million and 20 million exosomes. It can be considered that WJ-MSC-derived exosomes may decrease metabolic activity depending on the number.

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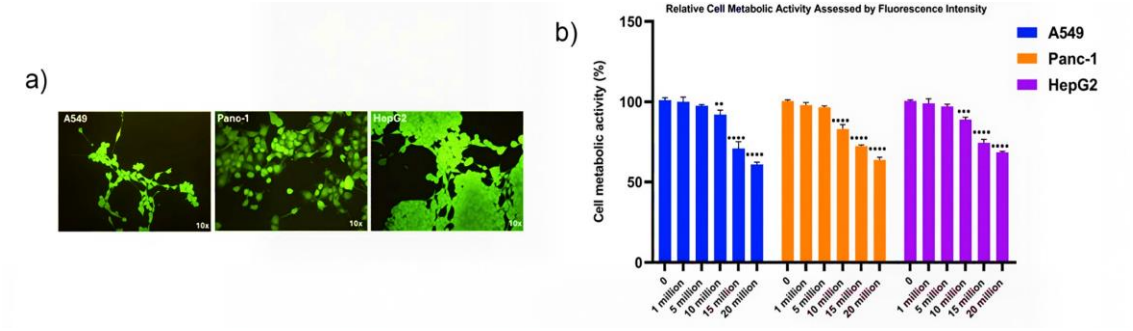
Figure 3. The metabolic activity results of 1, 5, 10, 15 and 20 million WJ-MSC exosomes treated with cells and images of control cell. Images of A549, Panc-1 and HepG2 cell lines stained with calcein under 10x magnification (a). Metabolic activity graph of WJ-MSC exosome on cancer cells (b). Values are expressed as mean ± SD with n=3. Only results compared to the control group are presented.



Discussion

In this experiment, we have demonstrated that WJ-MSC-derived exosomes did not promote the viability and metabolic activity of cancer cells when treated with A549, Panc-1 and HepG2 cancer cells in 96-well plates. It was also revealed that it could decrease in a time-concentration exosome numbers dependent manner.

MSC-derived exosomes have garnered significant attention as dual regulators of tumor progression, with their effects largely dependent on their molecular cargo, cellular origin, and the tumor microenvironment. Several studies have confirmed the pro-tumorigenic roles of MSC-Exos. For instance, Gu et al. reported that exosomes from human MSCs promote gastric cancer cell proliferation and migration through activation of the Akt signaling pathway[13], while Zhu et al. showed similar tumor-promoting effects in vivo using BM-MSC-derived exosomes[14]. Additionally, Qi et al. demonstrated that BM-MSC-Exos can activate the Hedgehog signaling pathway, further supporting tumor growth[16]. These findings indicate that, under certain conditions, MSC-derives exosomes may contribute to tumor progression, possibly by transferring oncogenic proteins, RNAs, or signaling molecules that modulate cell proliferation, angiogenesis, and immune evasion. In contrast, accumulating evidence supports the anti-tumorigenic potential of MSC exosomes. For example, Gao et al. identified circ_0006790 in BM-MSC-exosomes as a crucial regulator that suppresses pancreatic ductal adenocarcinoma progression via S100A11 DNA methylation[17]. Likewise, WJ-MSC exosomes have been shown to reduce migration in U87 glioblastoma cells, associated with anti-tumor miRNA content[23]. In chronic myeloid leukemia models, UC-MSC exosomes exert synergistic apoptotic effects when combined with imatinib[24]. Furthermore, adipose-derived MSC exosomes have been reported to promote caspase-dependent apoptosis in prostate cancer by downregulating BCL-xl[25]. Interestingly, while some studies, such as that by Karaöz et al., reported minimal changes in proliferation following treatment with WJ-MSC-exosomes[26], others have demonstrated their capacity to modulate gene expression

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related to EMT, angiogenesis, and apoptosis in a variety of genitourinary cancers[27]. These heterogeneous results suggest that the biological effects of MSC exosomes are highly context-specific and may depend on dose, tumor type, and experimental conditions. In our study, we similarly observed that MSC exosomes influence cancer cell viability and metabolic activity, further reinforcing their potential role as therapeutic agents or targets depending on their bioactive cargo.

Our results align with recent findings suggesting that MSC-derived exosomes can suppress tumor viability in a dose- and time-dependent manner. Specifically, we observed that WJ-MSC-derived exosomes decreased the metabolic activity of multiple cancer cell lines, with Panc-1 cells exhibiting a stronger and earlier response compared to lung and liver cancer cells. This observation is consistent with prior studies reporting that BM-MSC exosomes enriched with miR-16-5p and miR-124 suppress proliferation and induce apoptosis in colorectal and pancreatic cancer cells, respectively[18,19]. Furthermore, miR-143-containing exosomes have been shown to promote apoptosis and inhibit growth in pancreatic cancer via targeted gene regulation[20]. Supporting these findings, a recent review by Kacaroglu and Gurbuz (2025) emphasized the therapeutic advantages of MSC-versus macrophage-derived exosomes in pancreatic cancer, highlighting the allogeneic applicability and immunomodulatory benefits of WJ-MSC-derived exosomes[21]. Our study corroborates these observations by demonstrating a pronounced anti-tumor effect of WJ-MSC exosomes on pancreatic cancer cells, likely due to their unique cargo composition which may enhance therapeutic efficacy.

Although we applied up to 20 million exosome particles, we did not reach the IC50 value in our study. In contrast, other research using adipose-derived MSC exosomes reported IC50 values between 15 and 20 million particles[27]. This discrepancy may be attributed to source-dependent heterogeneity of MSC exosomes and differences in cancer cell line sensitivities. For example, when cytotoxic effects of exosomes between 0 and 25 million particles were evaluated, IC50 values of approximately 15 million particles were observed for PC3, LNCaP, and 5637 cell lines, and 20 million particles for ACHN cells, with apoptosis suggested as the main mode of cell death[27]. Despite not reaching IC50, our results confirmed that exosome treatment significantly decreased cell viability in a dose- and time-dependent manner. Moreover, exosomes containing miR-145-5p derived from human umbilical cord MSCs suppressed proliferation and induced apoptosis in an in vivo pancreatic ductal adenocarcinoma model[28], while BM-MSC exosomes enriched with miR-1231 inhibited invasion and metastasis in pancreatic cancer both in vitro and in vivo[29]. Notably, we found that WJ-MSC-derived exosomes reduced the metabolic activity of Panc-1 cells starting from 10 million particles, with a higher and earlier response compared to lung and liver cancer cells, indicating the potential for further investigation in pancreatic cancer models.

Exosomes play critical roles in tumor microenvironment formation and modulation due to their diverse molecular cargo, which varies based on the cell of origin and target cells. MSC-derived exosomes have been identified as key regulators of carcinogenesis by mediating signaling within the tumor microenvironment, influencing cell growth, and migration[30,31]. For example, adipose tissue-derived MSC exosomes have been shown to

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induce Wnt signaling in breast cancer cells, promoting proliferation[32]. Additionally, both WJ-MSC and BM-MSC exosomes induce apoptosis via sub-G1 phase arrest in malignant glioblastoma U87MG cells and reduce proliferation in vitro and in vivo[33]. Bone marrow MSC exosomes also modulate paracrine signaling, WNT/β-catenin pathways, and epithelial-mesenchymal transition in hepatoma cell lines[34]. Taken together, these studies illustrate the heterogeneity of exosomal content depending on their cellular origin. Considering that WJ-MSC-derived exosomes have demonstrated superior therapeutic effects in the literature and possess allogeneic use potential, they represent a promising avenue for future cancer therapies.

The lack of content analysis of the exosomes used in this study constitutes one of our main limitations, which limits the comparison of the observed biological effects. In addition, apoptosis, necrosis, cell cycle and related signaling pathways were not investigated in exosome-treated cells, which is another limitation of our study. However, it is of great importance to investigate these mechanisms in future studies in this field. In addition, the fact that our study was conducted only at the *in vitro* level prevents the confirmation of the findings obtained under *in vivo* conditions and causes limited biological validity. Further studies in *in vivo* models are of great importance to evaluate the biological effects, distribution, target specificity and safety of exosomes at the organismal level.

Conclusion

Preliminary findings reveal that between 1 and 20 million WJ-MSC-derived exosomes of 5000 (per well in 96-well plate) when treated with A549, HepG2 and Panc-1 cells do not promote cell viability and metabolic activity. In the present study, understanding the role of WJ-MSCs exosomes on viability in the cancer cells and elucidating other molecular mechanisms will be an important step to develop potential therapeutic approaches.

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