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Research Article

Lipid-Derived Extracellular Particles from Obese Murine Fat Promote Viability in Irradiated Genomically Unstable Systems

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Abstract

The interaction between obesity-associated biological processes and radiation-induced cellular damage has emerged as a significant area of investigation in biomedical research. In particular, lipid-derived extracellular particles, including exosome-like vesicles released from adipose tissue, have been implicated in modulating cellular responses under stress conditions such as genomic instability induced by ionizing radiation. This study presents an analytical and conceptual exploration of how extracellular vesicles originating from obese murine adipose tissue influence cell viability in irradiated systems exhibiting genomic instability. Drawing upon existing experimental and theoretical frameworks, the research integrates molecular biology, radiation oncology, and cellular signaling perspectives to elucidate the mechanisms underlying enhanced cellular survival.

The study synthesizes evidence indicating that extracellular vesicles function as carriers of bioactive molecules, including mitochondrial DNA, non-coding RNAs, and proteins that regulate apoptosis, proliferation, and stress responses. The role of obesity in reshaping the adipose microenvironment is critically examined, emphasizing how altered lipid metabolism and inflammatory signaling contribute to the production of vesicles with distinct functional properties. Furthermore, radiation-induced bystander effects are explored as a mediating mechanism through which vesicle-based intercellular communication influences non-irradiated or partially damaged cells.

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A conceptual model is proposed to explain how lipid-derived vesicles enhance cellular resilience in genomically unstable environments, integrating pathways related to oxidative stress modulation, DNA repair facilitation, and anti-apoptotic signaling. The findings suggest that these vesicles may play a dual role, promoting short-term survival while potentially contributing to long-term oncogenic risks. The study also discusses methodological considerations, including experimental models such as murine adipose-derived vesicles and in vitro genomic instability assays.

This research contributes to the growing body of knowledge on extracellular vesicle biology and highlights the need for further investigation into their role in radiation response and cancer progression. The implications extend to therapeutic strategies, including targeted modulation of vesicle signaling to enhance radiotherapy outcomes or mitigate adverse effects.

KEYWORDS

Extracellular vesicles, obesity, adipose tissue, radiation-induced genomic instability, cell survival, exosomes, lipid signaling, bystander effect, apoptosis regulation, murine models.

INTRODUCTION

The increasing prevalence of obesity has significantly altered the landscape of biomedical research, particularly in relation to cancer biology, metabolic disorders, and cellular stress responses. Adipose tissue is no longer regarded as a passive energy reservoir but is recognized as a metabolically active organ that secretes a wide range of signaling molecules, including cytokines, hormones, and extracellular vesicles. These secreted factors influence systemic physiological processes and contribute to disease progression in various pathological contexts (Quail and Dannenberg, 2019; Donohoe et al., 2017).

One of the emerging areas of interest is the role of adipose-derived extracellular vesicles, particularly in environments subjected to external stressors such as ionizing radiation. Radiation exposure is known to induce genomic instability, characterized by DNA damage, chromosomal aberrations, and altered cellular function. While the direct effects of radiation on targeted cells are well documented, increasing attention has been directed toward indirect effects, including the radiation-induced bystander phenomenon. This phenomenon describes how non-irradiated cells exhibit radiation-like damage due to signaling from irradiated neighboring cells, often mediated by extracellular vesicles (Ariyoshi et al., 2019).

Simultaneously, obesity has been linked to altered cellular microenvironments that favor tumorigenesis and disease progression. Adipose tissue in obese individuals undergoes structural and functional changes, including chronic inflammation, hypoxia, and increased lipid accumulation. These conditions promote the

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release of extracellular vesicles enriched with bioactive molecules capable of modulating cellular behavior (Bhaskaran et al., 2014; Demark-Wahnefried et al., 2012). Notably, these vesicles can influence cellular survival pathways, particularly under stress conditions such as radiation-induced genomic instability.

The intersection of these two domains—radiation biology and obesity-associated vesicle signaling—presents a complex yet critical area for investigation. While studies have demonstrated that extracellular vesicles can enhance cell survival following radiation exposure, the specific contribution of lipid-derived vesicles from obese adipose tissue remains insufficiently understood. This gap is particularly significant given the potential implications for cancer therapy, where radiotherapy is a primary treatment modality.

The primary objective of this study is to analyze the role of lipid-derived extracellular particles from obese murine adipose tissue in promoting cell viability within irradiated, genomically unstable systems. The research aims to develop a conceptual framework that integrates molecular signaling pathways, vesicle-mediated communication, and radiation-induced cellular responses. Additionally, the study seeks to evaluate the dual implications of enhanced cell survival, considering both protective and potentially oncogenic outcomes.

The scope of this research encompasses theoretical synthesis, mechanistic modeling, and critical analysis of existing empirical findings. By focusing on murine models, the study leverages controlled experimental insights while acknowledging translational limitations. The significance of this work lies in its potential to inform therapeutic strategies, particularly in optimizing radiotherapy protocols and developing interventions targeting extracellular vesicle signaling.

Literature Review

The study of extracellular vesicles, particularly exosomes and exosome-like structures, has expanded rapidly in recent years due to their critical role in intercellular communication. These vesicles, typically ranging from 30 to 150 nanometers in size, are secreted by various cell types and carry a diverse array of biomolecules, including proteins, lipids, and nucleic acids. Their ability to transfer functional cargo between cells positions them as key mediators in both physiological and pathological processes (Jeurissen et al., 2017).

Ariyoshi et al. (2019) demonstrated that radiation-induced bystander effects are mediated by mitochondrial DNA contained within exosome-like vesicles. This finding highlights the importance of vesicle-mediated signaling in transmitting stress responses across cellular populations. Furthermore, subsequent work by Ariyoshi et al. (2021) showed that extracellular vesicles released from irradiated tissues can enhance cell survival, suggesting a protective mechanism that may counteract radiation-induced damage.

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The role of adipose tissue in producing extracellular vesicles has also been extensively studied. Zhu et al. (2021) reported that vesicles derived from adipose stem cells can reduce apoptosis in hypoxic conditions, indicating their potential to modulate cell survival pathways. Similarly, Pan et al. (2022) emphasized the regulatory role of exosomal non-coding RNAs in metabolic and inter-tissue communication, particularly in obesity-related conditions.

Obesity itself has been identified as a critical factor influencing cancer development and progression. Bhaskaran et al. (2014) provided large-scale epidemiological evidence linking body mass index to increased cancer risk, while Demark-Wahnefried et al. (2012) highlighted the impact of obesity on cancer survival and recurrence. These findings are supported by mechanistic insights from Quail and Dannenberg (2019), who described the obese adipose tissue microenvironment as a pro-inflammatory and tumor-promoting niche.

In addition to its role in cancer biology, adipose-derived vesicle signaling has been implicated in cellular crosstalk across different tissue types. Rios-Colon et al. (2020) demonstrated that exosomes facilitate communication between adipocytes and liver cancer cells, influencing tumor behavior. This intercellular communication underscores the systemic impact of adipose-derived vesicles.

The concept of genomic instability is central to understanding radiation-induced cellular damage. Sasaki and Kodama (1987) established foundational models for studying mutational characteristics in mouse cell lines, while Schmid (1975) introduced the micronucleus test as a method for detecting chromosomal damage. These methodologies remain relevant for assessing the effects of radiation and vesicle-mediated modulation.

Despite significant advancements, several research gaps persist. First, while the protective effects of extracellular vesicles have been demonstrated, the specific contribution of lipid-derived vesicles from obese adipose tissue remains underexplored. Second, the long-term implications of enhanced cell survival, particularly in the context of oncogenesis, are not well understood. Third, the integration of obesity-related metabolic alterations with radiation-induced cellular responses requires further theoretical development.

This study addresses these gaps by synthesizing existing knowledge and proposing a comprehensive framework that links adipose-derived vesicle signaling with radiation-induced genomic instability. By focusing on the interplay between lipid metabolism, vesicle biology, and cellular stress responses, the research aims to provide a nuanced understanding of this complex biological system.

CONCEPTUAL FOUNDATIONS AND THEORETICAL FRAMEWORK

1 Extracellular Vesicle Biology in Adipose Systems

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Extracellular vesicles (EVs) represent a heterogeneous population of membrane-bound structures that facilitate intercellular communication through the transfer of molecular cargo. In adipose tissue, EV production is significantly influenced by metabolic state, particularly under conditions of obesity. Adipocytes, stromal cells, and immune cells within adipose tissue collectively contribute to a vesicle-rich microenvironment characterized by altered lipid composition and inflammatory signaling (Quail and Dannenberg, 2019).

The biogenesis of EVs involves endosomal pathways leading to the formation of multivesicular bodies, which subsequently fuse with the plasma membrane to release vesicles into the extracellular space. These vesicles encapsulate lipids, proteins, and nucleic acids reflective of the physiological state of the originating cells. In obese conditions, the cargo profile of EVs is altered, often enriched with pro-inflammatory cytokines, lipid metabolites, and regulatory RNAs (Pan et al., 2022).

Functionally, adipose-derived EVs act as signaling intermediaries that influence cellular processes such as proliferation, apoptosis, and metabolic regulation. Their lipid-rich composition enables efficient membrane fusion and cargo delivery, enhancing their impact on recipient cells. This property is particularly relevant in stress environments, where rapid cellular adaptation is required.

2 Radiation-Induced Genomic Instability and Cellular Stress Response

Radiation-induced genomic instability represents a sustained alteration in the genetic integrity of cells following exposure to ionizing radiation. This phenomenon extends beyond immediate DNA damage and includes delayed mutations, chromosomal rearrangements, and altered gene expression patterns that persist across cell generations. Foundational experimental models, such as near-diploid murine cell lines, have demonstrated that radiation exposure induces both direct and indirect genomic perturbations (Sasaki and Kodama, 1987).

The cellular response to radiation is governed by a complex network of signaling pathways that regulate DNA repair, apoptosis, and cell cycle progression. DNA double-strand breaks activate repair mechanisms such as homologous recombination and non-homologous end joining, while checkpoint proteins determine whether damaged cells undergo repair or programmed cell death. However, when repair mechanisms are insufficient or dysregulated, genomic instability becomes a persistent characteristic.

A critical extension of radiation biology is the concept of the bystander effect, wherein non-irradiated cells exhibit radiation-like responses due to signals received from irradiated neighbors. Ariyoshi et al. (2019) demonstrated that mitochondrial DNA contained within extracellular vesicles plays a central role in mediating this effect. These vesicles act as carriers of stress signals, propagating damage responses across cellular populations. This mechanism is particularly relevant in heterogeneous tissue environments, where only a

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subset of cells may be directly exposed to radiation.

In the context of obesity, the presence of metabolically active adipose tissue introduces additional complexity. The altered biochemical environment influences how cells respond to radiation-induced stress, potentially modifying both the magnitude and persistence of genomic instability.

3 Obesity-Driven Microenvironmental Modulation

Obesity fundamentally alters the structural and functional characteristics of adipose tissue, transforming it into a pro-inflammatory and metabolically dysregulated microenvironment. This transformation is characterized by increased adipocyte size, hypoxia, immune cell infiltration, and elevated secretion of inflammatory mediators (Donohoe et al., 2017).

One of the key consequences of this altered microenvironment is the enhanced production of extracellular vesicles with distinct biochemical properties. These vesicles are enriched with lipids, cytokines, and nucleic acids that reflect the inflammatory and metabolic state of the tissue. Quail and Dannenberg (2019) emphasized that such changes contribute to tumor-promoting conditions, particularly by facilitating cellular communication that supports proliferation and survival.

The relationship between obesity and cancer progression has been well established. Epidemiological evidence indicates a strong association between increased body mass index and elevated risk of multiple cancer types (Bhaskaran et al., 2014). Additionally, obesity has been linked to poorer cancer outcomes, including higher recurrence rates and reduced survival (Demark-Wahnefried et al., 2012). These findings suggest that the obese microenvironment not only initiates but also sustains pathological processes.

From a mechanistic perspective, adipose-derived vesicles act as mediators of this microenvironmental influence. They enable the transfer of regulatory molecules that can alter gene expression, modulate immune responses, and influence cellular metabolism in distant tissues. This systemic impact underscores the importance of understanding vesicle-mediated communication in the context of obesity.

4 Integration of Vesicle Signaling with Radiation Response

The intersection of extracellular vesicle signaling and radiation-induced cellular responses represents a complex and underexplored domain. While radiation primarily affects DNA integrity, vesicle-mediated communication introduces an additional layer of regulation that can either exacerbate or mitigate cellular damage.

Ariyoshi et al. (2021) demonstrated that extracellular vesicles released from irradiated tissues can enhance cell survival, suggesting a protective mechanism that operates alongside traditional DNA repair pathways. These vesicles may contain factors that promote cell cycle progression, inhibit apoptosis, or enhance stress

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tolerance. In obese systems, the composition of vesicles is further modified, potentially amplifying these protective effects.

The integration of lipid-derived vesicles into this framework is particularly significant. Lipids play a crucial role in membrane dynamics, signaling pathways, and energy metabolism. Vesicles enriched with lipid molecules can influence cellular responses by modulating membrane fluidity, receptor activation, and intracellular signaling cascades. This lipid-mediated modulation may enhance the ability of cells to withstand radiation-induced stress.

However, this protective effect presents a paradox. While increased cell survival may be beneficial in preventing tissue damage, it may also allow genomically unstable cells to persist, increasing the risk of oncogenic transformation. This duality highlights the need for a nuanced understanding of vesicle-mediated signaling in radiation biology.

MECHANISTIC MODEL OF LIPID-DERIVED VESICLE-MEDIATED SURVIVAL

1 Molecular Composition and Functional Implications

Lipid-derived extracellular vesicles from obese adipose tissue exhibit a unique molecular composition that distinguishes them from vesicles produced under normal physiological conditions. These vesicles are enriched with saturated and unsaturated fatty acids, cholesterol derivatives, and bioactive lipid mediators. Additionally, they carry proteins involved in signaling pathways and nucleic acids that regulate gene expression (Pan et al., 2022).

The presence of non-coding RNAs, including microRNAs, enables these vesicles to modulate post-transcriptional gene regulation in recipient cells. For example, microRNAs can suppress pro-apoptotic genes or enhance the expression of survival-related proteins. This regulatory capacity allows vesicles to influence cellular fate decisions in response to stress.

Moreover, mitochondrial DNA contained within vesicles has been shown to activate innate immune pathways and stress responses. Ariyoshi et al. (2019) demonstrated that mitochondrial DNA-mediated signaling contributes to the propagation of radiation-induced bystander effects. This mechanism may also play a role in enhancing cellular resilience by activating adaptive responses.

2 Pathways of Enhanced Cellular Viability

The enhancement of cellular viability in irradiated systems can be attributed to several interconnected pathways mediated by extracellular vesicles. First, vesicles can activate anti-apoptotic signaling pathways, such as the PI3K/Akt pathway, which promotes cell survival by inhibiting apoptotic processes. Second, they can enhance DNA repair mechanisms by upregulating proteins involved in damage recognition and repair.

Another critical pathway involves the modulation of oxidative stress. Radiation exposure generates reactive oxygen species (ROS), which contribute

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to cellular damage. Vesicles containing antioxidant molecules or regulatory RNAs can reduce oxidative stress, thereby mitigating damage and enhancing survival.

Additionally, vesicles can influence cellular metabolism by altering energy production pathways. Lipid-rich vesicles may provide alternative energy substrates, enabling cells to maintain metabolic function under stress conditions. This metabolic support is particularly important in environments characterized by hypoxia and nutrient deprivation, which are common in obese tissues.

3 Hypothetical Model of Interaction

A comprehensive model of vesicle-mediated survival in irradiated systems can be conceptualized as a multi-stage process. Initially, radiation exposure induces DNA damage and stress signaling in targeted cells. Concurrently, adipose tissue releases lipid-derived vesicles enriched with bioactive molecules. These vesicles interact with both irradiated and non-irradiated cells, delivering cargo that modulates cellular responses.

In recipient cells, vesicle-derived molecules activate survival pathways, enhance DNA repair, and reduce oxidative stress. This results in increased cellular viability despite the presence of genomic instability. Over time, however, the persistence of damaged cells may lead to cumulative genetic alterations, increasing the risk of malignant transformation.

This model highlights the dynamic interplay between protective and pathological processes, emphasizing the need for targeted interventions that can modulate vesicle signaling.

EXPERIMENTAL AND ANALYTICAL CONSIDERATIONS

1 Model Systems and Methodological Approaches

The study of vesicle-mediated effects in radiation biology relies on a combination of in vitro and in vivo models. Murine systems are particularly valuable due to their genetic and physiological similarities to humans. Sasaki and Kodama (1987) established foundational cell models that enable the study of mutational characteristics and genomic instability.

The micronucleus test, introduced by Schmid (1975), remains a widely used method for assessing chromosomal damage and genomic instability. This assay provides a quantitative measure of radiation-induced damage and can be used to evaluate the protective effects of extracellular vesicles.

Isolation and characterization of extracellular vesicles are critical methodological steps. Techniques such as ultracentrifugation and size-exclusion chromatography are used to isolate vesicles, while electron microscopy and molecular assays are employed to analyze their composition (Jeurissen et al., 2017).

2 Analytical Challenges and Limitations

Despite significant advancements, several challenges remain in the study of extracellular

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vesicles. One major limitation is the heterogeneity of vesicle populations, which complicates the identification of specific functional components. Additionally, the dynamic nature of vesicle production and uptake introduces variability in experimental outcomes.

Another challenge is the translation of findings from murine models to human systems. While murine studies provide valuable insights, differences in physiology and metabolism may limit the applicability of results. Furthermore, the long-term effects of vesicle-mediated survival, particularly in the context of cancer development, require longitudinal studies that are difficult to conduct.

Results

The analytical synthesis of the reviewed literature and conceptual modeling reveals several key findings regarding the role of lipid-derived extracellular vesicles in irradiated, genomically unstable systems. First, there is consistent evidence that extracellular vesicles act as critical mediators of intercellular communication, particularly under stress conditions. Vesicles derived from adipose tissue exhibit enhanced bioactivity in obese environments, characterized by altered molecular cargo that influences cellular behavior (Pan et al., 2022; Quail and Dannenberg, 2019).

Second, radiation-induced genomic instability is not confined to directly exposed cells but extends to neighboring populations through vesicle-mediated bystander effects. The involvement of mitochondrial DNA and other signaling molecules in this process underscores the complexity of radiation responses (Ariyoshi et al., 2019). Importantly, vesicles released under these conditions can promote cellular survival, suggesting a compensatory mechanism that mitigates damage (Ariyoshi et al., 2021).

Third, lipid-derived vesicles from obese adipose tissue appear to enhance this survival effect through multiple pathways, including anti-apoptotic signaling, oxidative stress reduction, and metabolic support. These mechanisms collectively increase the resilience of cells exposed to radiation, enabling them to maintain viability despite genomic instability.

However, the findings also indicate a potential trade-off. While increased survival may protect tissues from immediate damage, it may also facilitate the persistence of genetically unstable cells. This dual effect raises concerns regarding long-term outcomes, particularly in relation to cancer development and progression (Demark-Wahnefried et al., 2012).

Overall, the results highlight a complex interplay between protective and pathological processes, emphasizing the need for targeted approaches that can balance these effects in clinical applications.

Discussion

The findings of this study provide a critical perspective on the dualistic role of lipid-derived extracellular vesicles in modulating cellular

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responses to radiation-induced genomic instability. At a fundamental level, the results reinforce the concept that extracellular vesicles function not merely as passive carriers but as active regulators of cellular fate. Their ability to transfer bioactive molecules enables them to orchestrate complex signaling networks that determine whether a cell undergoes repair, apoptosis, or survival under stress conditions.

One of the central implications of this study is the recognition that obesity significantly amplifies vesicle-mediated signaling effects. The altered adipose microenvironment in obese systems produces vesicles with enhanced pro-survival properties, which can influence both local and systemic cellular responses. This observation aligns with previous findings that obesity contributes to tumor-promoting conditions through chronic inflammation and metabolic dysregulation (Quail and Dannenberg, 2019; Donohoe et al., 2017). However, the present analysis extends this understanding by demonstrating how these conditions specifically interact with radiation-induced stress mechanisms.

A critical aspect of the discussion involves the radiation-induced bystander effect. The evidence suggests that extracellular vesicles serve as primary mediators of this phenomenon, facilitating the transmission of stress signals between cells. Ariyoshi et al. (2019) identified mitochondrial DNA as a key component of this signaling process, highlighting the role of vesicles in propagating genomic instability beyond directly irradiated cells. The present study builds on this insight by suggesting that lipid-rich vesicles from obese adipose tissue may further enhance this signaling, thereby intensifying both protective and potentially harmful effects.

From a therapeutic perspective, the implications are multifaceted. On one hand, the ability of vesicles to enhance cell survival could be leveraged to protect normal tissues during radiotherapy. For instance, targeted delivery of beneficial vesicle components may reduce collateral damage in surrounding healthy tissues. On the other hand, the same mechanisms may undermine the efficacy of cancer treatment by promoting the survival of malignant or pre-malignant cells. This paradox underscores the importance of selective modulation, where beneficial effects are preserved while adverse outcomes are minimized.

The study also highlights important methodological considerations. The reliance on murine models provides controlled experimental conditions but introduces limitations in terms of translational applicability. Differences in metabolic regulation, immune responses, and tissue architecture between mice and humans may influence the behavior of extracellular vesicles. Additionally, the heterogeneity of vesicle populations complicates the identification of specific functional components responsible for observed effects.

Another limitation lies in the temporal dimension of vesicle-mediated effects. While short-term survival benefits are evident, the long-term consequences remain uncertain. The persistence

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of genomically unstable cells may contribute to cumulative genetic alterations, increasing the risk of oncogenesis. This concern is supported by epidemiological evidence linking obesity to higher cancer incidence and poorer outcomes (Bhaskaran et al., 2014; Demark-Wahnefried et al., 2012). Therefore, future research must adopt longitudinal approaches to fully understand these dynamics.

Comparatively, the findings align with existing literature on extracellular vesicle function but introduce a novel integrative perspective that combines obesity, radiation biology, and vesicle signaling. While previous studies have examined these domains independently, the present work emphasizes their interconnectedness, providing a more comprehensive understanding of cellular responses under complex physiological conditions.

Conclusion

This study provides a comprehensive analytical framework for understanding the role of lipid-derived extracellular vesicles from obese murine adipose tissue in promoting cellular viability within irradiated, genomically unstable systems. By integrating insights from radiation biology, extracellular vesicle research, and obesity-related metabolic studies, the research elucidates the mechanisms through which vesicle-mediated signaling influences cellular outcomes under stress conditions.

The findings demonstrate that extracellular vesicles act as critical mediators of intercellular communication, capable of enhancing cell survival through multiple pathways, including anti-apoptotic signaling, oxidative stress reduction, and metabolic support. In obese systems, these effects are amplified due to the altered composition of vesicles, which are enriched with bioactive molecules reflective of the inflammatory and metabolic state of adipose tissue.

However, the study also highlights a significant paradox. While vesicle-mediated signaling may protect cells from immediate radiation-induced damage, it may simultaneously facilitate the persistence of genomically unstable cells, thereby increasing the risk of long-term pathological outcomes such as cancer development. This dual role underscores the complexity of biological systems and the need for carefully balanced therapeutic strategies.

The research contributes to the existing body of knowledge by proposing a conceptual model that integrates vesicle biology with radiation-induced genomic instability. It identifies key pathways and mechanisms that can serve as targets for future experimental investigation and therapeutic intervention. Moreover, the study emphasizes the importance of considering systemic factors, such as obesity, in the design and evaluation of radiotherapy protocols.

Future research should focus on empirical validation of the proposed model, particularly through longitudinal studies that assess long-term outcomes of vesicle-mediated survival. Additionally, advancements in vesicle isolation

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and characterization techniques will be essential for identifying specific molecular components responsible for observed effects. Translational studies are also needed to bridge the gap between murine models and human clinical applications.

In conclusion, lipid-derived extracellular vesicles represent a critical yet complex factor in the modulation of cellular responses to radiation. Their role as both protectors and potential facilitators of disease progression highlights the need for nuanced approaches in biomedical research and clinical practice.

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