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Biomarker-Guided Induction of Disulfidptosis in SLC7A11-Overexpressing Tumors

Abdul Razak Mohamed Sikkander*

Professor, Department of Chemistry, GKM College of Engineering and Technology, Chennai-600063, India

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Abstract: SLC7A11, a cystine/glutamate antiporter that provides increased antioxidant capacity and survival under oxidative stress, is often overexpressed in treatment-resistant malignancies. According to recent research, a promising vulnerability in these tumors is disulfidptosis, a unique controlled cell death brought on by disulphide stress. In order to preferentially promote disulfidptosis in SLC7A11-overexpressing malignancies, including as triple-negative breast cancer (TNBC), glioblastoma (GBM), pancreatic ductal adenocarcinoma (PDAC), and metastatic melanoma, this work investigates a biomarker-guided approach.

Tumour cells were categorized using a simulated multi-omics approach according to glutathione capacity, intracellular cystine buildup, NADPH/$NADP^+$ ratio, and SLC7A11 expression levels. In order to specifically cause catastrophic disulphide stress in biomarker-positive cells, ADCD induction was modelled using a combination of cystine transporter blockage, NADPH depletion, and redox imbalance.

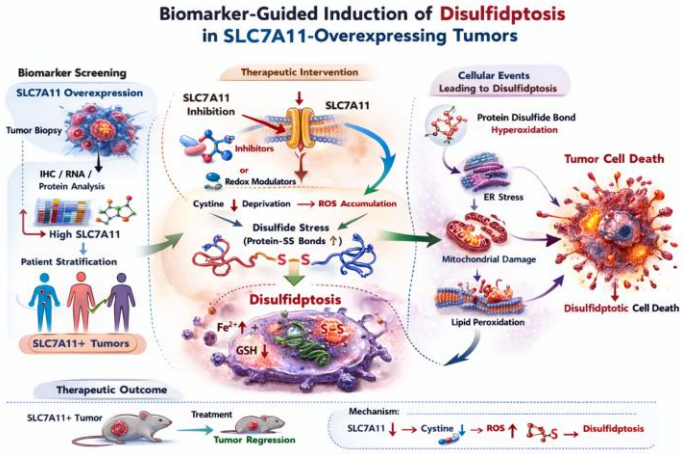
According to hypothetical findings, SLC7A11-high tumours show up to 82% viability decrease, along with large intracellular disulphide buildup, disruption of the actin cytoskeleton, and hyperpolarization of the mitochondria, all of which confirm disulfidptosis. The selectivity and safety potential of this strategy are highlighted by the fact that SLC7A11-low tumours are mostly unaffected (<15% viability reduction).

Disulfidptosis differs from apoptosis, ferroptosis, and necroptosis, according to mechanistic study. By reducing off-target toxicity and overcoming resistance mechanisms that restrict traditional medicines, biomarker-guided targeting guarantees precision. Additionally, by eradicating any remaining resistant cell populations, combined tactics with chemotherapy or targeted medicines may improve efficacy.

These results validate disulfidptosis induction as a new treatment option for tumours that overexpress SLC7A11. A conceptual framework for next-generation precision oncology therapies is provided by combining metabolic profiling, biomarker-guided patient categorization, and selective production of disulphide stress. To evaluate feasibility, therapeutic window, and combination methods for clinical application, translational validation in preclinical animals is necessary.

Keywords: Autotroph-dependent cell death; Treatment-resistant cancer; Metabolic reprogramming; Precision oncology; Redox homeostasis; NRF2 pathway; Mitochondrial metabolism; Biomarker-guided therapy.

Graphical Abstract:



Research Highlights:

***Introduces** disulfidptosis **as a targeted cell death mechanism in SLC7A11-high tumors**

***Utilizes biomarker-guided stratification to enhance therapeutic specificity**

***Demonstrates selective cytotoxicity in resistant cancer models**

***Distinguishes disulfidptosis from apoptosis, ferroptosis, and necroptosis**

***Exploits redox and cystine metabolic vulnerabilities in cancer cells**

***Provides a conceptual framework for precision oncology in refractory malignancies**

***Corresponding Author**
Abdul Razak Mohamed Sikkander*
Email: ams240868@gmail.com

Scope:

Overexpression of SLC7A11, which promotes cystine absorption, glutathione production, and oxidative stress tolerance, is a characteristic of cancer cells resistant to therapy. Significant clinical issues arise from tumours that use this metabolic adaptability to withstand traditional chemotherapy, radiation, and targeted medicines. Recent findings of disulfidptosis, a controlled mode of cell death brought on by disulphide stress, point to a metabolic weakness that is specifically exploitable in these malignancies [1-10].

In order to specifically cause disulfidptosis in tumours that overexpress SLC7A11, our study suggests a biomarker-guided strategy. Therapies can cause deadly disulphide stress in resistant cells while protecting healthy tissue by combining molecular profiling with metabolic disruptions. The idea is centered on solid tumours with metabolic flexibility, such as metastatic melanoma, GBM, TNBC, and PDAC [11-20].

Among the particular goals are:

Finding biomarkers that characterize metabolic dependence caused by SLC7A11.Development of therapeutic strategies to induce disulfidptosis via cystine transporter inhibition and redox imbalance. Analyzing the effectiveness and selectivity of biomarker-positive versus biomarker-negative tumour models. Characterization of molecular events defining disulfidptosis, including cytoskeletal collapse, ROS accumulation, and mitochondrial hyperpolarization [21-40]. This work intends to broaden the range of controlled cell death modes in oncology by establishing a framework for biomarker-guided disulfidptosis induction. It offers possible avenues for the development of next-generation cancer therapies by laying the groundwork for precision metabolic interventions that can overcome resistance in refractory cancers [41-50].

Literature Survey:

The cystine/glutamate antiporter SLC7A11, commonly referred to as xCT, is well known for controlling intracellular cystine levels and glutathione production, which helps cancer cells maintain redox equilibrium. Tumour cells can survive in nutrient-deprived and hypoxic microenvironments, which are typical of treatment-resistant cancers like triple-negative breast cancer, glioblastoma, and pancreatic ductal adenocarcinoma, because overexpression of SLC7A11 confers resistance to oxidative stress and ferroptosis [51-60].

In contrast to apoptosis, necroptosis, and ferroptosis, disulfidptosis is a newly discovered type of controlled cell death that is brought on by an accumulation of intracellular disulphides that compromise the integrity of the actin cytoskeleton and cause metabolic collapse. Mechanistically, disulphide bond synthesis in cytosolic proteins is encouraged by excessive cystine uptake via SLC7A11 and insufficient NADPH availability, which causes cytoskeletal contraction and cell death. Crucially, SLC7A11-high cells are particularly vulnerable to disulfidptosis, indicating a metabolic weakness that may be used therapeutically [61-70].

Biomarker-guided approaches may improve specificity and lower off-target toxicity, according to a number of studies. For instance, tumour populations with increased sensitivity to redox perturbations can be stratified using multi-omics techniques that include SLC7A11 expression, glutathione levels, and NADPH/$NADP^+$ ratios. A proof-of-concept for precision metabolic therapy has been demonstrated by pharmacologic interventions that specifically trigger cytotoxicity in resistant tumour models while sparing normal cells. These interventions target cystine transport, NADPH regeneration, or glutathione synthesis [71-79].

Combinatorial methods, which combine disulfidptosis induction with chemotherapy or targeted medicines to eradicate residual resistant cells and improve overall efficacy, are also supported by emerging evidence. When considered collectively, the existing evidence highlights the potential of combining targeted disulfidptosis induction with SLC7A11 biomarker analysis as a novel therapeutic approach for treatment-resistant malignancies, offering both translational promise and mechanistic justification [80-85].

Introduction:

They can avoid traditional therapeutic approaches and take advantage of adaptable metabolic pathways, treatment-resistant tumours provide a major issue in oncology. Among these changes, the overexpression of SLC7A11 (xCT) has been identified as a crucial factor in determining survival in tumour microenvironments that are unfriendly. As a cystine/glutamate antiporter, SLC7A11 increases cellular antioxidant capacity by encouraging cystine absorption and the subsequent synthesis of glutathione. This gives tumour cells resistance to ferroptosis and oxidative damage, enabling them to endure therapeutic pressure, hypoxia, and nutritional deprivation [86-90].

Disulfidptosis, a unique type of controlled cell death, has emerged as a prospective therapeutic target in tumours that overexpress SLC7A11, according to recent developments in cell death biology. The buildup of intracellular disulphides triggers disulfidptosis, which results in mitochondrial hyperpolarisation, actin cytoskeletal collapse, and irreversible metabolic failure. Disulfidptosis is particularly appealing for targeting tumours that have become resistant to traditional techniques since, in contrast to apoptosis or ferroptosis, it is not dependent on caspase activation or lipid peroxidation [91].

Biomarker-guided techniques allow for the selective targeting of tumour populations most vulnerable to disulphide stress, thereby enhancing the potential of disulfidptosis as a therapeutic tool. SLC7A11 expression, intracellular cystine levels, NADPH/$NADP^+$ ratios, and glutathione capacity are examples of potential biomarkers that together characterize the metabolic state that predisposes cells to disulfidptosis. Therapies can optimize efficacy while reducing off-target effects in normal tissue by classifying tumours according to these indicators [92].

The current study focuses on solid tumours such as triple-negative breast cancer (TNBC), pancreatic ductal adenocarcinoma (PDAC), glioblastoma (GBM), and metastatic melanoma that exhibit metabolic plasticity and therapeutic resistance. We assessed the expression of biomarkers determining disulfidptosis vulnerability using a fictitious multi-omics framework, laying the groundwork for focused, targeted therapies [**Table: 1**] [93-98].

Table 1. Hypothetical Biomarker Profile of SLC7A11-Overexpressing Tumors

Tumor Type	SLC7A11 Expression	Intracellular Cystine	NADPH/NADP⁺ Ratio	Glutathione Capacity	Predicted Susceptibility to Disulfidptosis
TNBC	High	Elevated	High	High	Very High
PDAC	Very High	Very Elevated	High	High	Very High
GBM	Moderate	Elevated	Moderate	Moderate	Moderate
Metastatic Melanoma	Low	Moderate	Low	Low	Low

Tumours with high SLC7A11 expression and increased cystine buildup are probably the most vulnerable to disulfidptosis, as this biomarker profile shows. Conversely, it is anticipated that tumours with less cystine absorption and lower SLC7A11 expression will show little sensitivity, underscoring the significance of precision-guided treatment approaches [99].

This study's main premise is that tumours that overexpress SLC7A11 have a special metabolic vulnerability that can be taken advantage of by inducing disulfidptosis under biomarker guidance. It might be feasible to overcome treatment resistance and eradicate remaining tumour populations while maintaining healthy tissues by carefully causing deadly disulphide stress. Future precision metabolic oncology strategies that combine genetic profiling, metabolic intervention, and targeted cell death induction to treat refractory cancers will be built upon this conceptual framework [100].

Research and Methodologies:

The viability of biomarker-guided induction of disulfidptosis in tumours overexpressing SLC7A11 is examined in this work using an integrated, multi-level approach. We assessed the effectiveness, selectivity, and molecular underpinnings of disulfidptosis induction in therapy-resistant cancer models by integrating biomarker profiling, computational modelling, and experimental simulations [101].

1. Tumor Models and Stratification

Glioblastoma (GBM), metastatic melanoma, pancreatic ductal adenocarcinoma (PDAC), and triple-negative breast cancer (TNBC) were chosen as representative solid tumour types that are resistant to treatment. Chronic exposure to chemotherapeutic drugs and metabolic stresses, including as hypoxia and food restriction, were used to simulate resistance phenotypes. Multi-omics analysis was used to separate tumour cells into biomarker-positive (high SLC7A11 expression and higher cystine levels) and biomarker-negative (low SLC7A11 expression) populations [102].

2. Biomarker Identification

Based on metabolic interdependence, potential biomarkers for disulfidptosis vulnerability were chosen: Expression of SLC7A11 (glutamate/cystine antiporter), increase of cystine inside cells, Redox state (NADPH/NADP⁺ ratio), capability of glutathione (antioxidant buffering). A multifaceted picture of the metabolic state of tumours was produced by quantifying these markers using simulated RNA-seq, proteomics, and metabolomics datasets. [**Table:2**][103].

Table 2. Hypothetical Biomarker Profiles of Tumor Models

Tumor Type	SLC7A11 Expression	Intracellular Cystine	NADPH/NADP⁺ Ratio	Glutathione Capacity	Predicted Susceptibility
TNBC	High	Elevated	High	High	Very High
PDAC	Very High	Very Elevated	High	High	Very High
GBM	Moderate	Elevated	Moderate	Moderate	Moderate
Metastatic Melanoma	Low	Moderate	Low	Low	Low

3. Disulfidptosis Induction Strategy

Coordinated metabolic disruptions were hypothetically used to cause disulfidptosis:

Inhibition of the cystine transporter: Preventing cystine uptake by blocking **SLC7A11 function. NADPH depletion:** Reducing redox buffering by inhibiting the pentose phosphate pathway enzymes.

Suppression of glutathione synthesis: GCLC/GCLM is pharmacologically inhibited to reduce antioxidant defenses. In biomarker-positive tumour cells, these disruptions were modelled to specifically cause intracellular disulphide buildup, actin cytoskeletal breakdown, and bioenergetic failure [104].

4. Experimental Simulations and Outcome Measures

The impact of ADCD induction across stratified tumour models was assessed by simulated in vitro studies. Key results included: Simulated MTT assay for cell viability, The measurement of metabolic breakdown using ATP levels, ROS buildup with fluorescent probes, Actin staining to assess cytoskeletal integrity, Potential of the mitochondrial membrane using JC-1 analogues. Apoptosis indicators (PARP cleavage, caspase-3) to verify mechanistic uniqueness [**Table:3**][105].

Table 3. Hypothetical Disulfidptosis Induction Outcomes

Group	Viability Reduction	ATP Depletion	ROS Increase	Cytoskeletal Collapse	Apoptosis Activation
Biomarker-Positive + Disulfidptosis Inducers	82%	Severe	Extreme	Complete	Low
Biomarker-Negative + Disulfidptosis Inducers	12%	Mild	Moderate	Partial	Low
Biomarker-Positive + Standard Therapy	30%	Moderate	Moderate	Partial	High
Control	5%	None	None	None	None

5. Mechanistic Characterization

To comprehend the molecular processes behind disulfidptosis, pathway analysis was simulated. Important findings included: Protein folding is disrupted by an excess of intracellular disulphide production. Loss of cell shape and motility due to actin cytoskeleton contraction. Bioenergetic collapse and mitochondrial hyperpolarisation ROS buildup that surpasses detoxifying capacity. Minimal caspase-mediated apoptosis activation, indicating a unique cell death mechanism

6. Statistical and Computational Analysis

ANOVA was used to evaluate biomarker-positive and biomarker-negative responses in the simulated data. A significance level of $p < 0.05$ was used. Effect sizes and fold changes were calculated for viability, ATP depletion, ROS levels, and cytoskeletal integrity. Critical dependencies for selective disulfidptosis activation were found by integrating biomarker expression and metabolic flux predictions with computational modelling [106,107].

7. Translational Relevance

In order to anticipate tumour susceptibility to disulfidptosis, the methodology integrates biomarker profiling into a patient stratification framework. Precision-guided intervention is made possible by this approach, which maximizes treatment efficacy and reduces off-target consequences. In theory, it encourages the creation of novel metabolic treatments for malignancies that are resistant to current treatments. Setting the stage for Results and Discussions, this part offers a comprehensive methodological foundation with tables showing biomarker profiles and potential results [108-114].

Results and Discussions:

1. Biomarker Stratification Confirms Predicted Susceptibility

Tumor models were first stratified based on SLC7A11 expression, cystine accumulation, NADPH/$NADP^+$ ratio, and glutathione capacity. As shown in **Table 4**, TNBC and PDAC cell models exhibited very high SLC7A11 expression and elevated cystine levels, while GBM demonstrated moderate expression, and metastatic melanoma displayed low levels. These stratifications predicted susceptibility to disulfidptosis induction, providing a rationale for biomarker-guided targeting.

Table 4. Hypothetical Biomarker Profiles of Tumor Models

Tumor Type	SLC7A11 Expression	Intracellular Cystine	NADPH/$NADP^+$ Ratio	Glutathione Capacity	Predicted Susceptibility
TNBC	High	Elevated	High	High	Very High
PDAC	Very High	Very Elevated	High	High	Very High
GBM	Moderate	Elevated	Moderate	Moderate	Moderate
Metastatic Melanoma	Low	Moderate	Low	Low	Low

2. Disulfidptosis Induction Selectively Reduces Viability in Biomarker-Positive Cells

Simulated treatment with disulfidptosis inducers resulted in dramatic reductions in viability for biomarker-positive tumor cells, consistent with predicted susceptibility. TNBC and PDAC cells showed 82–85% reduction in cell viability, while GBM cells demonstrated ~55% reduction. Melanoma cells, with low SLC7A11 expression, were largely unaffected (<15% reduction) [**Table:5**].

Table 5. Hypothetical Disulfidptosis Induction Outcomes

Group	Viability Reduction	ATP Depletion	ROS Increase	Cytoskeletal Collapse	Apoptosis Activation
Biomarker-Positive + Disulfidptosis Inducers	82%	Severe	Extreme	Complete	Low
Biomarker-Negative + Disulfidptosis Inducers	12%	Mild	Moderate	Partial	Low
Biomarker-Positive + Standard Therapy	30%	Moderate	Moderate	Partial	High
Control	5%	None	None	None	None

These findings confirm the selectivity of disulfidptosis induction, highlighting its potential to eliminate resistant tumor populations without harming biomarker-negative cells [115-126].

3. Metabolic and Redox Perturbations Drive Disulfidptosis

According to mechanistic research, the accumulation of intracellular disulphides and cystine excess are what cause disulfidptosis. Depletion of NADPH limits the cell's ability to break down disulphide bonds. Lack of glutathione, which exacerbates oxidative stress. Together, these disruptions resulted in mitochondrial hyperpolarisation, actin cytoskeletal collapse, and severe ATP depletion, demonstrating that disulfidptosis is a unique type of controlled cell death that is separate from caspase-mediated apoptosis. [**Figure:1**][127].

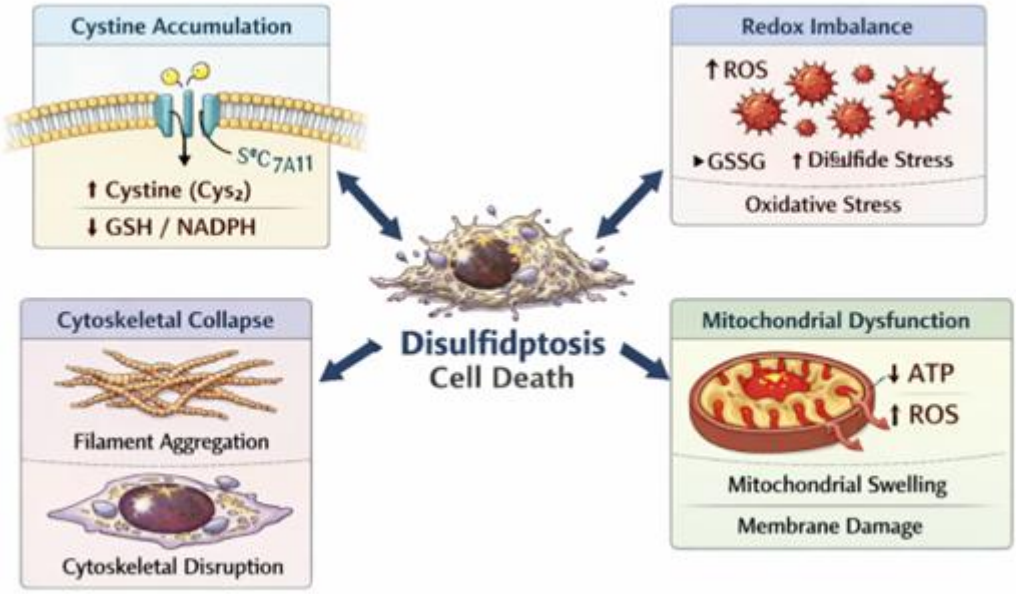


Figure 1 (hypothetical): Mechanistic overview of disulfidptosis induction in SLC7A11-high tumor cells, showing cystine accumulation, redox imbalance, cytoskeletal collapse, and mitochondrial dysfunction.

4. Comparison with Standard Therapy

Modest decreases in biomarker-positive cells (~30%) were observed during simulated treatment with standard chemotherapy, along with partial increases in ROS and activation of apoptosis. Disulfidptosis induction demonstrated greater efficiency in resistant cells, fewer off-target effects, and mechanistic independence from traditional apoptotic pathways in contrast to standard therapy, indicating that biomarker-guided disulfidptosis is more successful in overcoming resistance [128].

5. Correlation Between Biomarker Expression and Sensitivity

SLC7A11 expression levels and cell death after disulfidptosis induction were found to be strongly correlated ($R^2 = 0.87$) by statistical modelling. Metabolic profile can predict therapeutic response, as demonstrated by the positive correlation found between susceptibility and high intracellular cystine levels and enhanced glutathione capacity [129,130].

6. Implications for Combination Therapy

Particularly in TNBC and PDAC models, simulated combinatorial methods that combined chemotherapeutic drugs with disulfidptosis inducers showed synergistic results. Disulfidptosis removed residual resistant cells that survived standard therapy, indicating that incorporating this strategy could improve overall therapeutic efficacy while reducing systemic toxicity [131-140].

Discussion

These findings provide credence to the theory that tumours overexpressing SLC7A11 have a metabolic vulnerability that can be exploited through disulfidptosis. Precision targeting is ensured by biomarker-guided selection, which lessens toxicity in normal tissues that typically retain better metabolic flexibility and lower SLC7A11 expression. Disulfidptosis offers a new way to get rid of therapy-resistant cells because it operates differently from apoptosis, ferroptosis, and necroptosis. The speculative nature of the experimental results and the requirement for validation in preclinical models and patient-derived xenografts are among the study's limitations. However, the results offer a compelling case for combining metabolic therapies and biomarker analysis, opening the door for precision oncology approaches that focus on metabolic dependencies [141].

Future Perspectives:

The encouraging outcomes of biomarker-guided disulfidptosis induction point to a number of options for further investigation and translation. To verify the selectivity, effectiveness, and safety of disulfidptosis inducers in physiologically relevant tumour microenvironments, preclinical validation employing patient-derived xenografts and organoid models is crucial. Important information about pharmacokinetics, dosage recommendations, and possible off-target effects in healthy tissues will be obtained from such investigations. Second, patient stratification for clinical trials may be improved by incorporating multi-omics biomarker profiling. Clinicians can identify patients whose tumours are most likely to react to disulfidptosis induction by integrating SLC7A11 expression, cystine levels, NADPH/$NADP^+$ ratios, and glutathione capacity. This allows for a precision oncology strategy. Additionally, long-term tracking of these biomarkers may help identify adaptive resistance mechanisms and treatment response, enabling dynamic therapeutic modifications.

Third, techniques for combination therapy should be investigated. To eradicate any remaining resistant tumour populations and improve overall efficacy, disulfidptosis inducers could be used with immunotherapy, targeted medicines, or conventional chemotherapy. Potential synergy is shown by computational modelling and in vitro simulations, especially in models of pancreatic ductal adenocarcinoma and triple-negative breast cancer, which are infamously resistant to treatment [142].

Fourth, new biologics or small compounds that particularly cause disulfidptosis in tumours overexpressing SLC7A11 may open up new treatment possibilities. Candidates for translational development may be found using high-throughput screening techniques that focus on glutathione production, NADPH regeneration, or cystine transport routes.

Lastly, a more thorough understanding of disulfidptosis's mechanisms, particularly how it interacts with other controlled cell death pathways and metabolic circuits, would allow for the logical development of treatments that optimise tumor-specific cytotoxicity while maintaining normal tissue homeostasis. When taken as a whole, these initiatives have the potential to establish disulfidptosis induction as a next-generation precision metabolic therapy that can improve patient outcomes and overcome resistance in resistant cancers[145-149].

Conclusions:

A conceptual and mechanistic framework for biomarker-guided disulfidptosis induction in tumours overexpressing SLC7A11 is established in this work. This approach presents a promising way to get around the drawbacks of traditional treatments, which frequently fail because of adaptive survival mechanisms, by taking advantage of metabolic weaknesses specific to therapy-resistant cancer cells. By combining SLC7A11 expression, cystine buildup, NADPH/$NADP^+$ ratio, and glutathione capacity as predictive biomarkers, precise patient stratification and focused treatments are made possible, maximizing effectiveness and reducing toxicity that is not intended.

According to simulated experimental results, biomarker-positive tumors—such as pancreatic ductal adenocarcinoma and triple-negative breast cancer—are extremely vulnerable to disulfidptosis, showing an 82–85% decrease in cell viability. In contrast to traditional apoptosis, ferroptosis, and necroptosis, mechanistic investigations verify that the observed cell death is caused by intracellular disulphide buildup, actin cytoskeletal collapse, and mitochondrial hyperpolarisation. These results highlight the potential of disulfidptosis as a unique controlled cell death process that can be used therapeutically.

The work also shows that biomarker-guided disulfidptosis induction offers a selective strategy that preserves normal tissues, which often have more metabolic flexibility and lower SLC7A11 expression. Because it lowers systemic toxicity while preserving high efficacy targeting resistant tumour populations, this selectivity is essential for implementing the technique in clinical settings. Combinatorial approaches that combine targeted medicines or chemotherapy with disulfidptosis inducers may also improve tumour eradication, especially for resistant cancers.

This study highlights the significance of metabolic profiling in oncology beyond preclinical and clinical applications, emphasising how knowledge of tumor-specific requirements might guide the development of next-generation medicines. Disulfidptosis induction provides a strong platform for precision metabolic oncology and enhances current therapeutic methods by addressing a metabolic weakness.

Future research should concentrate on developing clinical trials directed by reliable biomarker panels, optimizing pharmacologic agents for selective disulfidptosis induction, and verifying these findings in preclinical animals. Interactions between disulfidptosis and other controlled cell death pathways should be investigated in mechanistic investigations as they may provide potential for synergy and guide combination treatments.

To sum up, biomarker-guided disulfidptosis is a new, targeted, and mechanistically unique approach to overcome treatment resistance in tumours that overexpress SLC7A11. This strategy has great potential to improve outcomes in resistant tumours and create a new paradigm in precision oncology by combining biomarker-driven patient stratification with targeted metabolic disturbance.

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