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LETTER TO THE EDITOR

Targeting the NAD+/SIRT1 axis: A metabolic strategy to overcome oxaliplatin resistance in colorectal cancer

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Abstract

Oxaliplatin resistance remains a significant clinical challenge in colorectal cancer (CRC), highlighting the urgent need to identify novel molecular targets for therapeutic intervention. Recent findings by Niu *et al* have elucidated the role of the NAD+/SIRT1 axis in mediating oxaliplatin resistance through metabolic reprogramming. Their study demonstrated that oxaliplatin-induced DNA damage activates PARP, resulting in NAD+ depletion and subsequent downregulation of SIRT1. This reduction in SIRT1 levels enhances glycolysis, as evidenced by increased expression of PKM2 and LDHA, thereby conferring a metabolic advantage to resistant CRC cells. Conversely, restoration of SIRT1 expression reverses resistance, while pharmacological inhibition of glycolysis effectively sensitizes cells to oxaliplatin. These findings underscore the therapeutic potential of targeting the NAD+/SIRT1 pathway as a metabolic vulnerability in CRC.

Future studies should investigate the clinical feasibility of combining SIRT1 agonists and glycolysis inhibitors with oxaliplatin to overcome drug resistance and improve patient outcomes.

Key Words: SIRT1; Glycolysis; Drug resistance; Colorectal cancer; Chemotherapy

Core Tip: Oxaliplatin resistance remains a major clinical challenge in colorectal cancer (CRC). This study underscores the critical role of the NAD+/SIRT1 axis in driving metabolic reprogramming that facilitates resistance. Oxaliplatin-induced DNA damage activates PARP, leading to NAD+ depletion and subsequent downregulation of SIRT1. This reduction in SIRT1 expression enhances glycolysis, marked by upregulation of PKM2 and LDHA. Notable, restoration of SIRT1 reverses resistance, while glycolysis inhibition sensitizes CRC cells to oxaliplatin. These findings suggest that targeting the NAD+/SIRT1 pathway, through SIRT1 agonists and glycolysis inhibitors, offers a promising metabolic strategy to overcome chemoresistance and improve therapeutic outcomes in CRC patients.

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TO THE EDITOR

I am writing to express my strong interest in the recent study by Niu *et al*[1], which elucidates the role of the NAD+/SIRT1 axis in mediating oxaliplatin resistance in colorectal cancer (CRC). The findings presented in this study are particularly compelling as they offer novel insights into metabolic reprogramming as a mechanism of underlying chemoresistance. The interplay between DNA damage repair, NAD+ metabolism, and SIRT1 activity reveals a complex regulatory network that may serve as a promising therapeutic target to overcome oxaliplatin resistance in CRC patients. Given the growing clinical burden associated with oxaliplatin resistance, the therapeutic implications of targeting the NAD+/SIRT1 axis merits further discussion and investigation[1].

CRC remains a major oncological challenge, with oxaliplatin-based chemotherapy forming the cornerstone of treatment for advanced-stage disease. However, the emergence of resistance to oxaliplatin presents a formidable obstacle, significantly compromising treatment efficacy and reducing patient survival[2]. Although multiple mechanisms have been implicated in oxaliplatin resistance—including enhanced DNA repair capacity, increased activity of drug efflux pumps, and alterations in apoptotic signaling—the role of metabolic reprogramming has been relatively underexplored [3]. The study by Niu *et al*[1] provides compelling evidence that oxaliplatin-induced NAD+ depletion *via* PARP activation leads to the downregulation of SIRT1 expression, triggering a metabolic shift toward glycolysis. This adaptive response, characterized by elevated expression of PKM2 and LDHA, confers a survival advantage that enables CRC cells to evade oxaliplatin-induced cytotoxicity[1].

While the study elegantly links SIRT1 downregulation to glycolytic reprogramming, the precise molecular mechanisms through which SIRT1 modulates glycolysis warrant further elucidation. It remains to be determined whether SIRT1 directly regulates glycolytic enzyme function *via* post-translational modifications, such as deacetylation, or whether its effects are mediated through upstream signaling pathways. For instance, previous studies have indicated that SIRT1 may deacetylate and destabilize transcription factors like HIF-1α, thereby indirectly suppressing glycolytic gene expression[4, 5]. Alternatively, SIRT1's role in activating AMPK and inhibiting mTOR signaling may intersect with key metabolic pathways critical for CRC cell survival under chemotherapeutic stress[6].

Importantly, the role of SIRT1 is context-dependent, influenced by both microenvironmental and genetic factors. While it may promote metabolic adaptation and chemoresistance in CRC, SIRT1 has also been shown to exhibit tumor-suppressive functions in other contexts. Therefore, a careful evaluation of potential off-target effects and CRC-specific molecular landscapes is essential to optimize the therapeutic targeting of the NAD+/SIRT1 axis.

Future investigations employing functional assays, such as chromatin immunoprecipitation to evaluate SIRT1's direct binding to the promoters of glycolytic enzymes, and enzyme activity assays following SIRT1 modulation will be crucial to determine whether SIRT1's influence on PKM2 and LDHA is direct or mediated through upstream regulators. Additionally, assessing the acetylation status of PKM2 and LDHA in oxaliplatin-resistant CRC cells could yield critical mechanistic insights.

Notably, prior studies have shown that SIRT1 agonists, such as resveratrol and SRT1720, exert anti-tumor effects in CRC models by modulating apoptotic pathways and cellular metabolism[7]. However, their specific impact on oxaliplatin resistance and glycolytic adaptation remains underexplored, highlighting the need for further investigation into SIRT1 modulation in this context.

The identification of SIRT1 as a key regulator of oxaliplatin resistance in this study aligns with growing evidence linking sirtuins to cancer metabolism and therapeutic resistance. SIRT1, an NAD+-dependent deacetylase, plays a pivotal role in maintaining genome stability, regulating apoptosis, and modulating cellular metabolism[8]. In the context of CRC, SIRT1 has been reported to exhibit dual functions, acting as either a tumor suppressor or an oncogene depending on the molecular and cellular context[9]. The current study suggests that NAD+ depletion, resulting from oxaliplatin-induced PARP activation, downregulates SIRT1 and enhances glycolytic activity, thereby promoting chemoresistance. Importantly, the finding that pharmacological activation of SIRT1 can restore oxaliplatin sensitivity underscores its potential as a viable therapeutic target.

An intriguing aspect of the study by Niu *et al*[1] is the elucidation of a link between DNA damage response (DDR) and metabolic adaptation. Oxaliplatin-induced DNA damage activates PARP, leading to NAD+ depletion and suppressing SIRT1 activity. This metabolic shift favors glycolysis over oxidative phosphorylation, mimicking the Warburg effect—a hallmark of cancer metabolism[10]. Notably, the study highlights how CRC cells exploit glycolysis reprogramming to survive chemotherapy-induced stress, thereby contributing to oxaliplatin resistance. The resensitization of resistant cells using glycolysis inhibitors, such as shikonin, underscores the therapeutic promise of targeting metabolic vulnerabilities in CRC[1].

While this study primarily addresses on oxaliplatin resistance, it raises an important question: Are SIRT1-mediated mechanisms also relevant to resistance against other platinum-based agents, such as cisplatin and carboplatin? Despite sharing the ability to induce DNA adducts, these agents differ in chemical structure, DNA-binding affinity, and associated repair pathways, all of which may influence how SIRT1 modulates cellular responses[11]. Cisplatin and carboplatin are extensively used in treating various malignancies—including ovarian, lung, and bladder cancers—where platinum resistance continues to pose a significant clinical challenge. Exploring the broader applicability of the NAD+/SIRT1 axis across different tumor types and platinum-based chemotherapeutics could inform the development of more effective combination strategies to overcome resistance.

Emerging evidence suggests that SIRT1 may similarly influence resistance mechanisms in other cancers. For instance, studies in ovarian and lung cancer models have implicated SIRT1 in regulating DDR, apoptosis, and metabolism—key processes involved in cisplatin sensitivity[12]. Therefore, future investigations should explore whether SIRT1 downregulation universally contributes to platinum resistance across tumor types. Integrating data from large clinical cohorts, such as The Cancer Genome Atlas (TCGA) and Gene Expression Omnibus, could yield correlative insights into SIRT1 expression patterns and patient outcomes following cisplatin or carboplatin treatment. Additionally, functional studies in preclinical models utilizing various platinum agents would help determine whether the NAD+/SIRT1 axis constitutes a generalized metabolic vulnerability in platinum-resistant cancers.

The clinical relevance of these findings is underscored by data from TCGA, which indicates that lower SIRT1 expression is associated with poorer overall survival in CRC patients undergoing adjuvant chemotherapy[13]. This suggests that SIRT1 expression levels could serve as a prognostic biomarker to predict oxaliplatin response. Future studies should aim to validate these observations in larger patient cohorts and assess the therapeutic potential of SIRT1 agonists in clinical settings.

From a translational perspective, the study raises important questions regarding the potential for therapeutic intervention targeting the NAD+/SIRT1 axis. The use of SIRT1 activators, such as CAY10602, in combination with oxaliplatin represents a promising strategy to enhance chemosensitivity[14]. Moreover, considering the role of PARP activation in NAD+ depletion, the combination of PARP inhibitors with SIRT1 agonists may enhance therapeutic efficacy. Although PARP inhibitors have primarily been explored in BRCA-mutant cancers[15], their ability to influence NAD+ metabolism suggests a broader application in overcoming chemoresistance in CRC. Future preclinical and clinical studies are warranted to evaluate the feasibility and effectiveness of such combination approaches.

While the study by Niu *et al*[1] provides valuable mechanistic insights, several areas warrant further exploration to fully harness the therapeutic potential of targeting the NAD+/SIRT1 axis in CRC. First, the molecular determinants governing the differential response of CRC cells to SIRT1 modulation need to be elucidated. Given SIRT1's dual role—as both a tumor suppressor and promoter depending on cellular and microenvironmental contexts—identifying predictive biomarkers (*e.g.*, NAD+ levels, PARP activity, metabolic profiles) will be essential for patient stratification and optimizing therapeutic efficacy.

Second, the interplay between SIRT1 and other key metabolic regulators, such as AMPK and mTOR, should be systematically investigated. These pathways converge on cellular energy homeostasis and stress responses, and may synergistically influence glycolytic reprogramming and drug resistance. A comprehensive mapping of these interconnected signaling networks would provide a more holistic understanding of the metabolic adaptations that drive oxaliplatin resistance.

Importantly, while the current study emphasizes the benefits of pharmacological SIRT1 activation in restoring oxaliplatin sensitivity, the therapeutic potential of SIRT1 inhibitors in combination therapy remains underexplored. Emerging evidence suggests that SIRT1 inhibition may exacerbate DNA damage, oxidative stress, and apoptotic signaling—mechanisms that could enhance oxaliplatin cytotoxicity under certain tumor conditions[14]. It is therefore imperative to evaluate the efficacy of SIRT1 inhibitors in in vivo models, particularly orthotopic CRC models that more accurately replicate the tumor microenvironment, including stromal fibrosis, hypoxia, and immune infiltration.

Future preclinical studies should assess critical endpoints such as tumor growth inhibition, metastasis suppression, and systemic toxicity profiles in response to combination therapy with SIRT1 inhibitors and oxaliplatin. Mechanistic analyses should also investigate whether SIRT1 inhibition amplifies DNA damage markers (*e.g.*, γH2AX), increases reactive oxygen species production, or enhances pro-apoptotic signaling in CRC cells. These insights would provide a rational basis for the dual targeting strategies and offer insight into the contextual benefits of either activating or inhibiting SIRT1. Collectively, such investigations will reinforce the translational relevance of NAD+/SIRT1 modulation

and support the development of more effective, biomarker-driven combination therapies in oxaliplatin-resistant CRC.

Conclusion

The study by Niu *et al*[1] offers a compelling framework for understanding the metabolic underpinnings of oxaliplatin resistance in CRC, and positioning the NAD+/SIRT1 axis as a promising therapeutic target. Integrating metabolic interventions with conventional chemotherapy holds significant potential to enhance treatment efficacy and overcome drug resistance. Future research should prioritize the clinical validation of these findings and the development of combinatorial therapeutic strategies aimed to improving patient outcomes for CRC patients. In light of the urgent need for innovative approaches to address chemoresistance, targeting metabolic vulnerabilities emerges as a promising and clinically relevant avenue for advancing CRC therapy.

FOOTNOTES

Author contributions: Hussain MS and Gupta G contribute equally to this study as co-corresponding authors; Hussain MS was responsible for conceptualization, data curation, writing – original draft; Jakhmola V was responsible for investigation, writing – original draft; Goyal K was responsible for formal analysis, investigation; Rekha A was responsible for data curation, writing – original draft; Sultana A was responsible for conceptualization, methodology, writing – original draft; Ali H was responsible for formal analysis, validation; Gupta G was responsible for conceptualization, supervision, writing – review & editing.

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