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LETTER TO THE EDITOR

Harnessing traditional medicine and biomarker-driven approaches to counteract Trichostatin A-induced esophageal cancer progression

Heng-Rui Liu

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Abstract

The recent study by Chen *et al* highlights the paradoxical role of the histone deacetylase inhibitor (HDACi) Trichostatin A (TSA) in esophageal squamous cell carcinoma (ESCC), revealing its promotion of epithelial-mesenchymal transition (EMT) and tumor migration *via* the BRD4/c-Myc/endoplasmic reticulum (ER)-stress pathway. While HDACis are traditionally considered anti-tumor agents, these findings underscore the need for alternative therapeutic strategies. In this commentary, we discuss the potential of traditional medicine-derived compounds, such as berberine, curcumin, and resveratrol, in modulating epigenetic regulators and mitigating TSA-induced oncogenic pathways. Additionally, we emphasize the prognostic significance of histone acetylation markers, particularly acetylated histone H3, which could serve as predictive biomarkers for ESCC progression and HDACi therapy responsiveness. Further, we explore the role of ER stress in tumor aggressiveness and suggest that compounds like quercetin and baicalein, known for their ER stress-alleviating properties, warrant further investigation. Integrating traditional medicine-based interventions with biomarker-driven targeted therapy may enhance ESCC treatment efficacy while minimizing HDACi-associated risks. We advocate for future research focusing on the interplay between epigenetic modulation, natural compounds, and biomarker identification to refine personalized therapeutic strategies for ESCC.

Key Words: Traditional Chinese medicine; biomarkers; Esophageal squamous cell carcinoma; Epithelial-mesenchymal transition

Core Tip: Trichostatin A, a histone deacetylase inhibitor (HDACi), paradoxically promotes esophageal squamous cell carcinoma (ESCC) progression *via* the BRD4/c-Myc/endoplasmic reticulum stress pathway. Traditional medicine-derived compounds like berberine, curcumin, and resveratrol may counteract these oncogenic effects by modulating epigenetic regulators. Additionally, histone acetylation markers, particularly acetylated histone H3, hold promise as predictive biomarkers for ESCC progression and HDACi therapy response. Integrating biomarker-driven approaches with natural compounds may enhance ESCC treatment efficacy while mitigating HDACi-associated risks.

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TO THE EDITOR

We read with great interest the recent study by Chen *et al*[1] investigating the role of Trichostatin A (TSA) in promoting epithelial-mesenchymal transition (EMT) and migration in esophageal squamous cell carcinoma (ESCC) *via* the BRD4/c-Myc/ER-stress pathway. The findings provide valuable insights into the molecular mechanisms underlying tumor progression and suggest new therapeutic targets for ESCC treatment.

The study highlights the paradoxical role of histone deacetylase inhibitor (HDACi) in ESCC, where TSA, rather than suppressing tumor progression, facilitates migration and EMT through histone acetylation-mediated BRD4 recruitment and c-Myc activation. These findings underscore the need for alternative therapeutic strategies to mitigate the potential tumor-promoting effects of HDACi. Given the intricate relationship between epigenetic modifications and tumor progression, we believe that natural compounds derived from traditional medicine could offer a promising complementary approach. Traditional Chinese medicine (TCM) has long been recognized for its holistic approach to disease management, and many herbal compounds exhibit anti-cancer properties. For instance, ion channel has been suggested as a cancer biomarker and to be a TCM target for glioma[2-5]. These natural compounds, either alone or in combination with conventional therapies, may provide a more nuanced approach to cancer treatment by targeting multiple molecular pathways.

Another crucial aspect of the study is the identification of biomarkers associated with poor prognosis in ESCC patients. Previous studies reported biomarkers for head and neck squamous cell carcinoma[6] has similarly suggested the biomarkers can potentially be therapeutic targets. The authors demonstrated that elevated histone acetylation levels, particularly acetylated histone H3 (acH3), correlated with enhanced migration and poor survival outcomes. This finding suggests that acH3 could serve as a predictive biomarker for ESCC progression and response to HDACi therapy. Further research into the role of histone acetylation in ESCC pathophysiology is warranted, particularly in the context of integrating biomarker-driven approaches into personalized treatment regimens.

The suggestion to use acH3 as a biomarker for ESCC progression and treatment response is compelling and warrants further exploration. To validate acH3 as a predictive biomarker, preclinical studies involving cell lines and animal models of ESCC should be conducted, assessing the correlation between acH3 levels and key markers of EMT, tumor progression, and survival outcomes. In parallel, clinical studies using patient samples could further confirm the association between acH3 levels and clinical parameters, such as metastasis, disease progression, and response to HDACi therapies. Additionally, the integration of acH3 into clinical practice should be considered, including the development of accessible diagnostic techniques such as liquid biopsy or tissue biopsy analysis to monitor acH3 levels in patients. Monitoring acH3 levels over time could also serve as a tool for evaluating patient response to therapy, enabling more personalized and effective treatment strategies in ESCC.

The suggestion to use acH3 as a biomarker for ESCC progression and treatment response is indeed intriguing[7]. However, further exploration is needed to validate acH3 as a predictive biomarker. This validation can be approached from two key aspects: First, through preclinical studies involving cell lines and animal models, where the correlation between acH3 levels and key markers of EMT, tumor progression, and survival outcomes should be examined. These studies would provide a solid foundation for understanding the biological relevance of acH3 in ESCC. Second, clinical studies involving patient samples are crucial to confirm the association between acH3 expression and clinical parameters, such as metastasis, disease progression, and response to HDACi-based therapies[8]. Moreover, to integrate acH3 into clinical practice, diagnostic methods such as liquid biopsy or tissue biopsy analysis should be developed for monitoring acH3 levels in patients. This would allow for dynamic tracking of acH3 levels as a potential tool for predicting disease progression and assessing treatment response, ultimately facilitating personalized treatment strategies in ESCC.

Furthermore, ER stress has emerged as a key mediator of TSA-induced EMT[9], with upregulation of GRP78, CHOP, and ATF6 contributing to tumor aggressiveness. Given that certain TCM-derived compounds, such as quercetin[10] and baicalein[11], have been reported to alleviate ER stress and inhibit EMT, their therapeutic potential in counteracting HDACi-induced tumor progression should be further explored. Future studies should aim to validate the efficacy of these compounds in preclinical and clinical settings to determine their role in modulating ER stress pathways in ESCC.

In addition to TCM-derived compounds, several current and emerging strategies in cancer therapeutics aim to target ER stress pathways, which could complement the proposed TCM-based approach. One such strategy involves the use of

small molecule inhibitors targeting key ER stress mediators such as GRP78, IRE1α, PERK, and ATF6. For example, chemical chaperones like 4-Phenylbutyric acid[12] and tauroursodeoxycholic acid[13] have shown promise in alleviating ER stress and improving the therapeutic response in various cancers. Additionally, selective inhibitors targeting IRE1α, such as STF-083010[14,15] and PERK inhibitors like GSK2656157[16] are under investigation for their potential to modulate ER stress and sensitize tumors to chemotherapy and immunotherapy. Comparing the efficacy of these emerging ER stress inhibitors with TCM-derived compounds, such as quercetin and baicalein, could provide a broader context for evaluating their respective therapeutic potentials. While TCM compounds offer a more holistic approach with multi-target effects, conventional ER stress inhibitors may provide more specific, targeted interventions. The integration of both strategies may enhance treatment outcomes by simultaneously modulating ER stress and other key oncogenic pathways, offering a promising avenue for cancer therapy.

ER stress is intricately linked with several key oncogenic pathways, including the p53 pathway, autophagy, and oxidative stress, and plays a critical role in tumor progression. For instance, the p53 tumor suppressor is activated in response to severe ER stress through the unfolded protein response (UPR) and acts to promote cell cycle arrest, apoptosis, or senescence[17-22]. However, in many cancers, p53 mutations or dysregulation allow cells to evade these protective responses, leading to enhanced survival under conditions of chronic ER stress. Similarly, ER stress is tightly connected to autophagy, a cellular process that helps maintain homeostasis during stress conditions. When ER stress is excessive, autophagy is often upregulated to alleviate protein aggregation and restore cellular function. However, the dysregulation of autophagy in cancer cells can also contribute to tumor survival by enabling the recycling of damaged cellular components and maintaining energy homeostasis. Moreover, ER stress can modulate oxidative stress by promoting the accumulation of ROS, which, in turn, can drive tumorigenesis and metastasis[23].

The tumor microenvironment (TME) is also profoundly affected by ER stress, influencing factors such as hypoxia, immune evasion, and inflammation. ER stress-induced UPR signaling can activate hypoxia-inducible factors, promoting the adaptation of tumor cells to low oxygen conditions and enhancing their survival. Furthermore, chronic ER stress may trigger immune evasion by altering immune cell interactions or by inducing immunosuppressive molecules. Inflammation is another key feature of the TME that can be exacerbated by ER stress, as the UPR signaling pathway induces the expression of pro-inflammatory cytokines and chemokines. This creates a favorable microenvironment for tumor progression and metastasis. Exploring how targeting ER stress affects these TME factors, such as by modulating hypoxia, immune evasion, and inflammation, could provide valuable insights into how ER stress-targeting therapies may enhance cancer treatment efficacy and limit cancer progression.

While TCM compounds are generally considered safe, especially when used in low doses and under proper guidance, their potential side effects and interactions with other therapies, particularly cancer treatments, warrant careful consideration. Many TCM compounds have been shown to possess anti-cancer properties, but some may carry toxicity risks, particularly when used at higher doses or in combination with other therapies. For instance, compounds like quercetin and baicalein have demonstrated therapeutic potential in modulating ER stress and inhibiting tumor growth, but at high doses, they could lead to hepatotoxicity, gastrointestinal disturbances, or allergic reactions. Additionally, certain TCM compounds may interact with chemotherapy drugs, either enhancing or reducing their effectiveness. For example, some herbal compounds may affect cytochrome P450 enzymes[24], which play a crucial role in drug metabolism, potentially leading to altered drug levels and unexpected toxicity or reduced efficacy. Moreover, while TCM compounds often have immune-modulating effects, their combination with immune checkpoint inhibitors or other immunotherapies may result in immune-related adverse events. It is crucial to conduct thorough pharmacological studies, including assessing the pharmacokinetics and pharmacodynamics of TCM compounds, to identify potential toxicities and interactions when used alongside conventional therapies. These studies will help ensure that TCM-based strategies are both effective and safe, maximizing therapeutic benefits while minimizing risks.

In conclusion, the work by Chen *et al*[1] provides a compelling foundation for understanding the paradoxical effects of TSA in ESCC and highlights the potential of histone acetylation and ER stress as key therapeutic targets. Integrating traditional medicine-derived compounds with biomarker-driven targeted therapy could enhance the efficacy of current ESCC treatment modalities while mitigating the risks associated with HDACi monotherapy. We commend the authors for their insightful research and encourage future studies to explore complementary therapeutic strategies, particularly those incorporating natural compounds with established epigenetic and anti-metastatic properties. Recent studies have highlighted the growing application of TCM in the treatment of various human diseases[25,26]. The therapeutic potential of TCM compounds, often derived from natural herbs, lies in their ability to modulate complex biological pathways and offer holistic treatment options. However, despite promising results, more rigorous studies are needed to fully understand the mechanisms of action, efficacy, and safety profiles of these compounds, especially in the context of cancer treatment. Given the increasing interest in integrating TCM with modern cancer therapies, we urge the scientific community to invest more resources into this field. Well-designed preclinical and clinical trials are essential to validate the potential of TCM compounds, identify their optimal usage in combination with conventional therapies, and explore their long-term safety. Expanding our understanding of TCM's role in disease treatment could significantly contribute to the development of more effective, personalized, and sustainable therapeutic strategies.

FOOTNOTES

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