SCOPING REVIEW

Role of Circular RNAs in Epithelial-Mesenchymal Transition of Oral Squamous Cell Carcinoma; A Scoping Review

Jiveswara Vijiakumar[1,2], Thomas George Kallarakkal[1,2], Karen Ng lee Peng[1,2], Wanninayake Mudiyanselage Tilakaratne[1,2]

Abstract

Background: CircRNAs are non-coding RNAs found to contribute various functions in cellular processes and have been associated with EMT regulation in OSCC.

Objective: To review the latest findings of circRNAs in relation to EMT in OSCC.

Methodology: A systematic search with the following MeSH terms was conducted: "circular RNA" AND "epithelial mesenchymal transition" OR "epithelial-mesenchymal transition" AND "oral squamous cell carcinoma" in Pubmed/MEDLINE, SCOPUS, and EMBASE.

Results: Nineteen articles were included in this review for analysis, comprising of in-vivo and in-vitro studies. 19 circRNAs were identified to be involved in EMT of OSCC, of which 16 were identified as EMT-promoting circRNA while 3 were identified as EMT-suppressing circRNAs.

Conclusion: Based on the current review, Circ_0058063, circEPSTI1, and Circ_0001971 seem to be the most potential EMT-promoting circRNAs while circRNA_0000140 seems to be the most potential EMT-suppressing circRNA. However, further in-depth studies are needed to elucidate their accurate roles in EMT and as possible use in therapeutic interventions.

Keywords: Circular RNAs, epithelial-mesenchymal transition, malignant transformation, oral squamous cell carcinoma

Introduction

Oral squamous cell carcinoma (OSCC) is the most prevalent malignancy of the oral cavity, accounting for over 90% of oral cancers worldwide and contributing significantly to morbidity and mortality despite advances in surgical and adjuvant therapies (Bray et al., 2024). Its aggressive behaviour, characterised by early invasion, high metastatic potential, and resistance to treatment, underscores the need to better understand the molecular drivers of disease progression.

Epithelial–mesenchymal transition (EMT) is a reversible cellular programme in which epithelial cells lose polarity and adhesion, acquiring mesenchymal traits that enhance motility, invasion, and metastatic potential. In OSCC, EMT is strongly associated with aggressive clinical behaviour, poor prognosis, and reduced treatment responsiveness, making the regulation of EMT a critical area of investigation.

Circular RNAs (circRNAs) are covalently closed, single-stranded RNA molecules generated through back-splicing. Once considered non-functional splicing by-products, they are now recognised as stable and conserved regulators capable of influencing gene expression through mechanisms such as miRNA sponging, protein interaction, and transcriptional modulation. Emerging evidence suggests that circRNAs contribute to cancer progression and may play important roles in EMT-related processes in OSCC.

Although individual studies have reported links between circRNA dysregulation and EMT-related phenotypes in OSCC, existing evidence is fragmented, with studies differing widely in methodology, mechanistic scope, and EMT endpoints. To date, no scoping review has synthesized circRNAs specifically associated with EMT regulation in OSCC.

Department and Institution Affiliation: [1]Department of Oral & Maxillofacial Clinical Sciences, Faculty of Dentistry, Universiti Malaya, Kuala Lumpur 50603, Malaysia; [2]Oral Cancer Research & Coordinating Centre (OCRCC), Faculty of Dentistry, Universiti Malaya, Kuala Lumpur 50603, Malaysia

Corresponding author: Wanninayake Mudiyanselage Tilakaratne, Department of Oral & Maxillofacial Clinical Sciences, Faculty of Dentistry, Universiti Malaya, Kuala Lumpur 50603, Malaysia, Email: wmtilak@um.edu.my

How to cite the article: Vijiakumar J, Kallarakkal T G, Peng K N L, Tilakaratne W M.Role of Circular RNAs in Epithelial-Mesenchymal Transition of Oral Squamous Cell Carcinoma; A Scoping Review. Oral Maxillofac Pathol J 2026; 17(1); 125-131.

Source of Support: Nil

Conflict of Interest: None

A consolidated overview is therefore required.

This scoping review aims to summarize current evidence on circRNAs implicated in EMT regulation in OSCC, describe their proposed mechanisms of action, and identify knowledge gaps to guide future research.

Methodology

PRISMA-ScR guidelines and the methodological framework by Peters et al. (2015). A systematic search of PubMed/MEDLINE, SCOPUS, and EMBASE was performed to identify studies published between January 2010 and December 2024 using the following MeSH terms and keywords: "circular RNA" AND "epithelial mesenchymal transition" OR "epithelial-mesenchymal transition" AND "oral squamous cell carcinoma," with additional searches using "circular RNA" AND "epithelial mesenchymal transition" AND "oral." Only peer-reviewed, English-language studies were included, while grey literature such as theses, conference abstracts, and non–peer-reviewed sources were excluded. Titles and abstracts were screened based on relevance to circRNAs, OSCC, and EMT-related outcomes, followed by full-text assessment using predefined inclusion and exclusion criteria. The search initially identified 52 records, of which 22 duplicates were removed. The remaining 30 articles underwent title and abstract screening, leading to the exclusion of 11 records that did not meet the criteria. A total of 19 full-text articles were assessed for eligibility, all of which were included in the final qualitative synthesis. The study selection process is summarised in the updated PRISMA flow diagram (Figure 1).

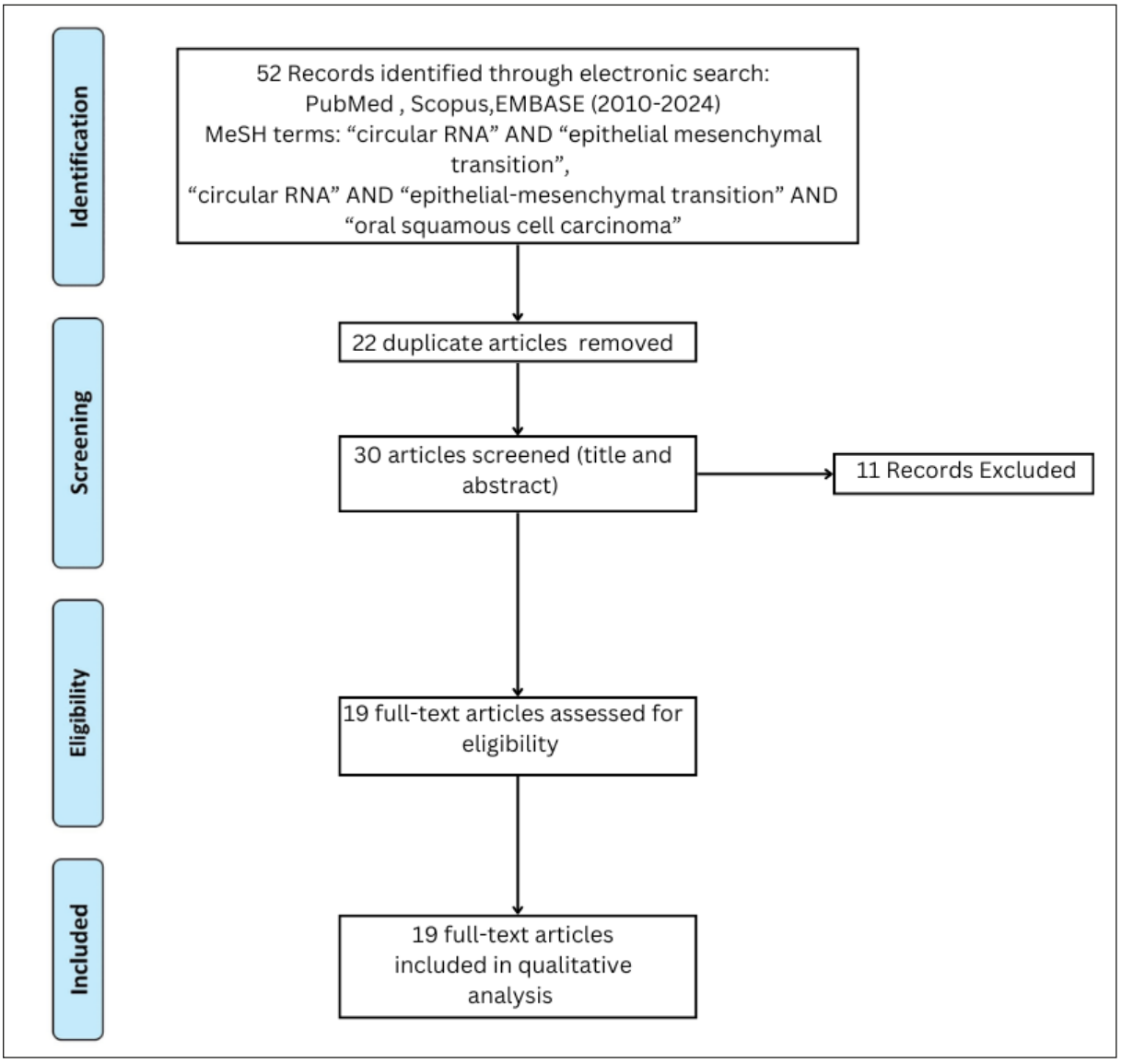

Fig. 1- PRISMA

Results

The initial search query yielded 52 articles based on the specified key terms. After removing 22 duplicate articles, a total of 30 articles were assessed for relevance to the study objectives and screened based on their title and abstract. Following this process, 11 articles were excluded for the following reasons: as they are not relevant to circular RNA and/or OSCC (n=10) and done article retracted (n=1). 19 articles were deemed suitable for inclusion in the study (Figure 1).

A total of 19 circRNAs were examined across these studies, with 16 identified as EMT-promoting circRNAs and 3 acting as EMT-suppressing circRNAs in OSCC. Promoter circRNAs exhibited increased expression, while suppressors showed reduced expression in OSCC cell lines compared to normal oral tissue (Table 1). Fourteen studies investigated circRNA function as miRNA sponges through bioinformatics and in-vitro analyses, while the remaining studies assessed EMT marker expression in relation to circRNA levels in OSCC versus normal oral tissue in vivo.

One of the identified functions of CircRNAs is miRNA sponges which bind specific miRNAs and reduce their availability, thereby altering key regulatory pathways in OSCC. Many of these miRNAs serve as tumor suppressors by targeting mRNAs that promote EMT. When circRNAs sequester these

Table 1: Summary of circRNAs found to be involved in EMT of OSCC.

CircRNA	EMT Role	Mechanistic Target	Observed Response	OSCC Regulation vs Normal Oral Mucosa	Reference
Circ_0004771	Promoter	Not explored	N-Cadherin, Vimentin upregulation, E-cadherin downregulation	Upregulation	(Shi & Zhang, 2024)
Circ-OMAC	Promoter	Not explored	Vimentin, N-cadherin upregulation, E-cadherin downregulation	Upregulation	(B. Li et al., 2024)
Circ_0058063	Promoter	miR-145 sponge	PI3K/Akt/mTOR signalling pathway activation	Upregulation	(J. Zhou & Jin, 2023)
aCircFNDC3B	Promoter	miR-181c-5p Sponge	SERPINE1 & PROX 1 upregulation	Upregulation	(X. Li et al., 2023)
Hsa_ Circ_0009128	Promoter	Upregulates MMP9	E-cadherin downregulation, N-cadherin upregulation	Upregulation	(Hong Zhang et al., 2023)
Circ_0000311	Promoter	miR-876-5p sponge	EZH2 upregulation	Upregulation	(Xu et al., 2023)
Circ_0014359	Promoter	miR-149 Sponge	MAPK pathway upregulation	Upregulation	(X. Chen et al., 2022)
CircZDBF2	Promoter	miR-362-5p, miR-500b-5p sponge	RNF145 upregulation	Upregulation	(Rong et al., 2022)
circFAM126A	Promoter	miR-186 Sponge	miR-186/FUS/RAB41 stabilization	Upregulation	(Jun Wang et al., 2022)
circANKS1B	Promoter	miR-515-5p Sponge	TGF-β upregulation	Upregulation	(Yan & Xu, 2021)
Circ-LRP6	Promoter	Not explored	E-cadherin downregulation, N-cadherin upregulation	Upregulation	(Q. Zhang et al., 2021)
CircKRT1	Promoter	miR-495-3p sponge	PD-L1 upregulation	Upregulation	(Yang et al., 2021)
CircIGHG	Promoter	miR-142-5p sponge	IGF2BP3 upregulation	Upregulation	(J. Liu et al., 2021)
circEPSTI1	Promoter	mir-942-5p sponge	PI3K/Akt/mTOR signaling pathway activation, LTBP2 upregulation	Upregulation	(Jie Wang et al., 2020)
Circ_0001971	Promoter	miR-194/miR-204 sponge	PI3K-AKT-Fox3a pathway activation	Upregulation	(Tan et al., 2020)
circUHRF1	Promoter	miR-526b-5p sponge	c-Myc upregulation/ TGF- β & ESRP1 upregulation	Upregulation	(Zhao et al., 2020)
Hsa_ circ_0072387	Suppressor	miR-503-5p sponge	Reduced proliferation, migration, and glycolysis	Downregulation	(Han et al., 2021)
circRNA_0000140	Suppressor	miR-31	Upregulate LATS2, Hippo pathway suppression	Downregulation	(Peng et al., 2020)
hsa_ circ_0036988	Suppressor	Not explored	Vimentin downregulation, E-cadherin upregulation	Downregulation	(Hanyu Zhang et al., 2021)

miRNAs, tumor-suppressive effects are diminished, leading to the activation of EMT-related pathways such as PI3K/Akt/mTOR and MAPK. Circ-OMAC, circ_0058063, circEPSTI1, and Circ_0001971 were found to upregulate EMT via activation of PI3K/Akt/mTOR pathway by regulating PI3K, AKT and mTOR phosphorylation while Circ_0014359 was theorized by the authors to upregulate EMT via activation of MAPK signaling pathway (B. Li et al., 2024; Tan et al., 2020; Jie Wang et al., 2020; J. Zhou & Jin, 2023). CircANKS1B and circUHRF1 were found to increase TGF-β activity by sequestering miRNAs miR-515-5p and miR-526b-5p which typically binds to and suppresses TGF-β and prevents EMT (Yan & Xu, 2021; Zhao et al., 2020).

Other than pathway activation, known EMT regulators were also found to be regulated by circRNAs. CircFNDC3B was found to increase the activity of SERPINE1 and PROX1 which facilitates EMT of OSCC cells(X. Li et al., 2023). CircIGHG was found to increase activity of IGF2BP3 by sequestering miR-142-5p, which facilitates EMT progression (J. Liu et al., 2021).

Correlation studies indicated that elevated circRNAs Circ_0004771, Circ-OMAC, Hsa_Circ_0009128 and Circ-LRP6 expression were associated with increased levels of mesenchymal markers and EMT-associated components, including N-cadherin, vimentin MMP9, EZH2, RNF145, and TGF-β. Meanwhile, epithelial marker E-cadherin expression was downregulated (B. Li et al., 2024; Shi & Zhang, 2024; Hong Zhang et al., 2023; Q. Zhang et al., 2021). CircKRT1 was found to upregulate PD-L1 expression by sequestering miR-495-3p, which indirectly promotes EMT via facilitating immune evasion and increasing cell survivalduring transformation (Yang et al., 2021).

Conversely, hsa_circ_0072387and circRNA_0000140 were found to suppress OSCC by binding to EMT-promoting miRNAs miR-503-5p and miR-31 (Han et al., 2021; Peng et al., 2020). hsa_circ_0036988 expression was correlated with the reduced expression of mesenchymal markers Vimentin, Bcl-2, Cyclin D1 followed by increased expression of epithelial marker E-cadherin (Hanyu Zhang et al., 2021).

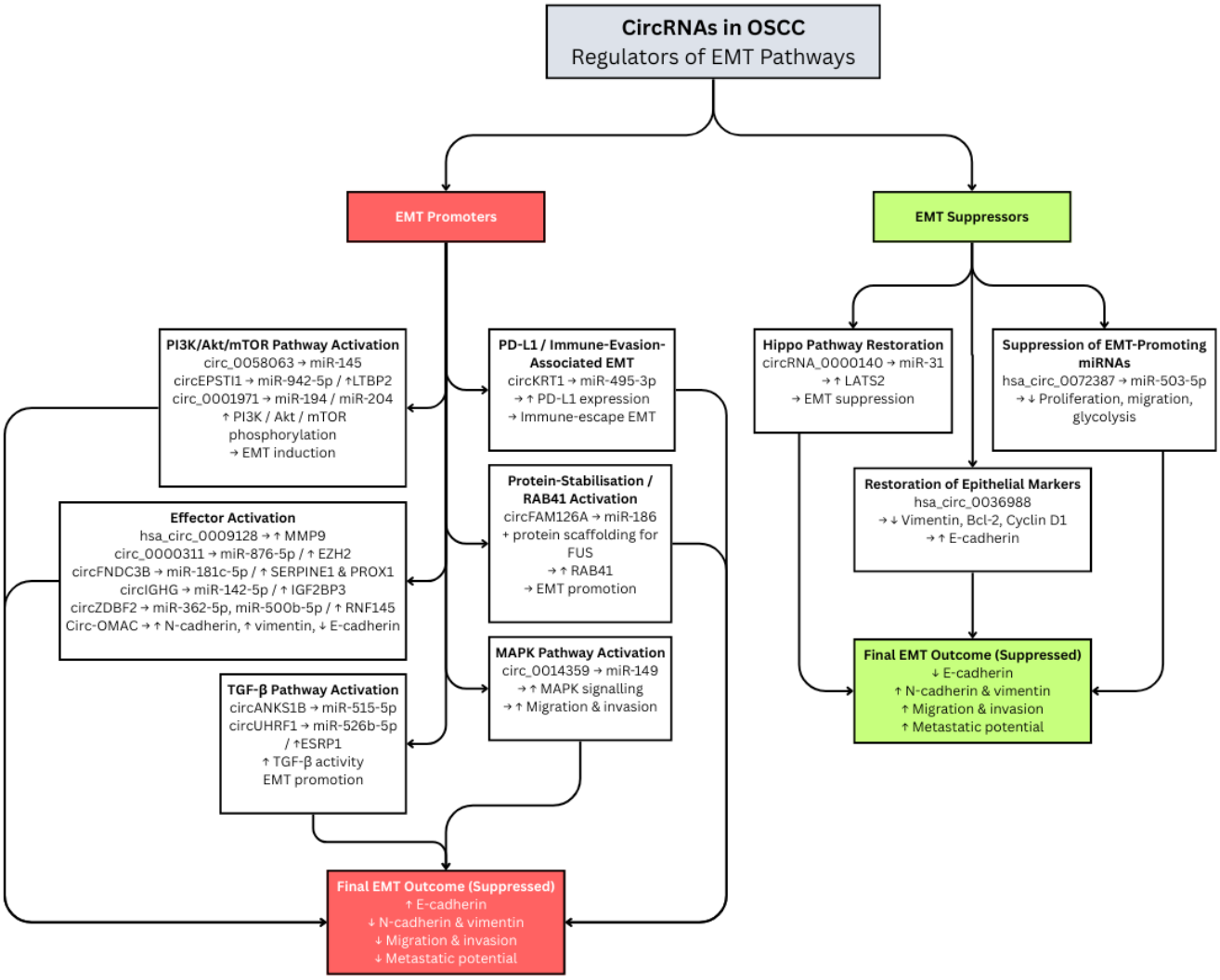

Fig. 2- CircRNAs in OSCC Regulators of EMT Pathways

Discussion

Epithelial-mesenchymal transition (EMT) is a fundamental process in the progression of oral squamous cell carcinoma (OSCC), enabling tumor cells to acquire invasive and metastatic properties. By transitioning from an epithelial to a mesenchymal phenotype, OSCC cells lose cell-cell adhesion, gain motility, and enhance their ability to invade surrounding tissues and establish distant metastases. EMT is closely associated with poor prognosis, therapeutic resistance, and increased aggressiveness in OSCC, making it a critical area of study for understanding disease progression and identifying potential therapeutic targets. The EMT process is highly intricate, involving a coordinated interplay of transcription factors, signaling pathways, and molecular markers. Key transcription factors such as Snail, Slug, Twist, and ZEB1/2 drive EMT by repressing epithelial markers like E-cadherin while upregulating mesenchymal markers such as N-cadherin and vimentin. This transition is further modulated by signaling pathways, including TGF-β, Wnt/β-catenin, PI3K/Akt, and NF-κB, which collectively regulate the EMT process at multiple levels (Bai et al., 2020; Chaudhury et al., 2010). Additionally, non-coding RNAs, including microRNAs (miRNAs) and long non-coding RNAs (lncRNAs), have been shown to fine-tune EMT by targeting key regulators and modifying gene expression dynamics (D. Li et al., 2018; Lin et al., 2014; Xie et al., 2018; Zeng et al., 2016).

Emerging evidence suggests that circular RNAs (circRNAs) play a crucial role in cancer progression, including EMT regulation in OSCC. CircRNAs have been identified as important modulators of gene expression, primarily through their function as competing endogenous RNAs (ceRNAs) that sponge miRNAs, thereby preventing miRNA-mediated repression of EMT-associated genes(Conn et al., 2024). Despite their growing recognition, circRNAs represent a relatively new area of study in cancer research, as they were previously thought to be non-functional by-products of splicing. However, recent studies have demonstrated their diverse regulatory roles in various malignancies, including lung, breast, colorectal, and hepatocellular carcinoma, where they influence tumor proliferation, invasion, metastasis, and drug resistance(Z. Chen et al., 2019; Hang et al., 2018; Nanishi et al., 2020). The increasing recognition of circRNAs as key players in cancer progression highlights their potential as biomarkers and therapeutic targets, particularly in EMT-driven malignancies like OSCC.

A key pattern emerging across studies is the dominance of the PI3K/Akt/mTOR pathway, which was activated by several circRNAs including circ_0058063, circEPSTI1, and circ_0001971. This pathway is known to enhance cell survival, proliferation, metabolic adaptation, and pro-EMT transcriptional activity. The consistency with which PI3K/Akt/mTOR appears suggests that circRNAs may preferentially exploit this signalling cascade to drive EMT in OSCC. Another notable group involves circRNAs that regulate TGF-β signalling, including circANKS1B and circUHRF1. TGF-β is a central inducer of EMT and metastasis, and its activation through circRNA-mediated miRNA suppression highlights a potential mechanism contributing to both EMT progression and chemoresistance.

A smaller but important subset of circRNAs influences EMT through MAPK activation (e.g., circ_0014359) or Hippo pathway inhibition (e.g., circRNA_0000140), the latter of which leads to increased LATS2 expression and suppression of mesenchymal transition. Additionally, emerging evidence supports a link between circRNAs and immune-evasion–associated EMT, particularly through the circKRT1–miR-495-3p–PD-L1 axis. This finding extends the role of EMT beyond invasion and metastasis by connecting it with immune checkpoint regulation, an area with significant therapeutic implications.

Although most circRNAs exert their effects through miRNA sponging, this review identified mechanistic diversity such as protein scaffolding, as demonstrated by circFAM126A, which stabilizes FUS and enhances RAB41 expression. This highlights the need to explore circRNA functions beyond ceRNA activity and to consider broader multi-layered regulatory interactions.

Despite the insights provided, the current body of evidence has several limitations. Many studies employ in-vitro models without in-vivo or patient-derived validation, restricting clinical generalisability. Sample sizes are often small, and OSCC stage or grade is rarely considered, despite known associations between EMT activation and tumour differentiation. Most studies investigate only a single circRNA–miRNA–mRNA axis at a time, without examining potential synergistic or antagonistic relationships between circRNAs. Furthermore, EMT characterisation commonly relies on a limited set of markers, with inconsistent inclusion of functional assays such as migration, invasion, or 3D culture systems. Multi-omics approaches integrating circRNA expression with proteomic or transcriptomic EMT signatures are also lacking.

Overall, the current evidence suggests that circRNAs play substantial and diverse roles in regulating EMT in OSCC, acting through several converging pathways summarised in the accompanying signalling flow diagram (Figure 2). However, more rigorous and comprehensive studies are needed to clarify the biological significance and translational relevance of these findings. Future research should incorporate larger clinical cohorts, multi-omic profiling, patient-derived models, and longitudinal EMT tracking to better define the potential of circRNAs as biomarkers or therapeutic targets in OSCC.

Conclusion

The discovery of circRNAs, particularly their roles in various cellular processes, remains in its early stages. As a result, their interactions, especially in EMT, have not been fully explored. Current studies reviewed here are largely exploratory, focusing on potential interactions between specific circRNAs and EMT components, particularly miRNAs and signaling pathway proteins. However, there is a lack of comprehensive research examining circRNAs alongside multiple EMT components, including transcription factors, signaling pathways, and proteomic expression, within a single study. Each study primarily aimed to identify circRNAs that may influence EMT in OSCC and assessed their impact through gain or loss of function, correlating these changes with alterations in EMT markers such as N-cadherin, E-cadherin, and vimentin, relative to adjacent normal oral mucosa. While these studies provide valuable insights into circRNA involvement in EMT, several limitations

must be addressed in future research to enhance accuracy and clinical relevance. These include small sample sizes and the absence of OSCC grading, which could impact EMT marker expression (Rai K & Ahmed, 2019).

References

Akhurst, R. J., & Derynck, R. (2001). TGF-beta signaling in cancer--a double-edged sword. Trends in Cell Biology, 11(11), S44-51.

Amano, Y., Matsubara, D., Kihara, A., Yoshimoto, T., Fukushima, N., Nishino, H., Mori, Y., & Niki, T. (2024). The significance of Hippo pathway protein expression in oral squamous cell carcinoma. Frontiers in Medicine, 11, 1247625.

Bai, Y., Sha, J., & Kanno, T. (2020). The role of carcinogenesis-related biomarkers in the Wnt pathway and their effects on epithelial-mesenchymal transition (EMT) in oral squamous cell carcinoma. Cancers, 12(3), 555.

Bray, F., Laversanne, M., Sung, H., Ferlay, J., Siegel, R. L., Soerjomataram, I., & Jemal, A. (2024). Global cancer statistics 2022: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA: A Cancer Journal for Clinicians, 74(3), 229–263.

Chaudhury, A., Hussey, G. S., Ray, P. S., Jin, G., Fox, P. L., & Howe, P. H. (2010). TGF-beta-mediated phosphorylation of hnRNP E1 induces EMT via transcript-selective translational induction of Dab2 and ILEI. Nature Cell Biology, 12(3), 286–293.

Chen, X., Chen, J., Fu, L., Chen, J., Chen, Y., & Liu, F. (2022). Circ_0014359 promotes oral squamous cell carcinoma progression by sponging miR-149. Acta Biochimica Polonica, 69(2), 315–320.

Chen, Z., Zuo, X., Pu, L., Zhang, Y., Han, G., Zhang, L., Wu, J., & Wang, X. (2019). circLARP4 induces cellular senescence through regulating miR-761/RUNX3/p53/p21 signaling in hepatocellular carcinoma. Cancer Science, 110(2), 568–581.

Conn, V. M., Chinnaiyan, A. M., & Conn, S. J. (2024). Circular RNA in cancer. Nature Reviews. Cancer, 24(9), 597–613.

Fathima J H, S., Jayaram, S., Veeraraghavan, V. P., & Karobar, M. I. (2024). miRNA99a as a Potential target in P13K/Akt1/mTOR signaling pathway in progression of OSCC. Advances in Biomarker Sciences and Technology, 6, 242–259.

Fujibayashi, E., Mukai, S., Torigata, K., Ando, Y., Uchihashi, T., Nozaki, M., Tanaka, S., Okada, M., Kogo, M., Nojima, H., & Yabuta, N. (2022). LATS kinases and SLUG regulate the transition to advanced stage in aggressive oral cancer cells. Scientific Reports, 12(1), 12363.

Greeshma, L. R., Joseph, A. P., Sivakumar, T. T., Raghavan Pillai, V., & Vijayakumar, G. (2023). Correlation of PD-1 and PD-L1 expression in oral leukoplakia and oral squamous cell carcinoma: an immunohistochemical study. Scientific Reports, 13(1), 21698.

Han, L., Cheng, J., & Li, A. (2021). Hsa_circ_0072387 suppresses proliferation, metastasis, and glycolysis of oral squamous cell carcinoma cells by downregulating miR-503-5p. Cancer Biotherapy & Radiopharmaceuticals, 36(1), 84–94.

Hang, D., Zhou, J., Qin, N., Zhou, W., Ma, H., Jin, G., Hu, Z., Dai, J., & Shen, H. (2018). A novel plasma circular RNA circFARSA is a potential biomarker for non-small cell lung cancer. Cancer Medicine, 7(6), 2783–2791.

Jayanthi, P., Varun, B. R., & Selvaraj, J. (2020). Epithelial-mesenchymal transition in oral squamous cell carcinoma: An insight into molecular mechanisms and clinical implications. Journal of Oral and Maxillofacial Pathology: JOMFP, 24(1), 189.

Jiang, Y., & Zhan, H. (2020). Communication between EMT and PD-L1 signaling: New insights into tumor immune evasion. Cancer Letters, 468, 72–81.

Kristensen, L. S., Jakobsen, T., Hager, H., & Kjems, J. (2022). The emerging roles of circRNAs in cancer and oncology. Nature Reviews. Clinical Oncology, 19(3), 188–206.

Li, B., Xie, S., Pan, G., Lin, Z., Ren, S., Chen, S., Fan, S., & Li, J. (2024). Circ-OMAC drives metastasis in oral squamous cell carcinoma. Oral Diseases, 30(2), 140–148.

Li, D., Liu, K., Li, Z., Wang, J., & Wang, X. (2018). miR-19a and miR-424 target TGFBR3 to promote epithelial-to-mesenchymal transition and migration of tongue squamous cell carcinoma cells. Cell Adhesion & Migration, 12(3), 236–246.

Li, X., Wang, C., Zhang, H., Li, Y., Hou, D., Liu, D., Xu, R., Cheng, J., Liu, L., Fu, Y., Ye, J., & Jiang, H. (2023). CircFNDC3B accelerates vasculature formation and metastasis in oral squamous cell carcinoma. Cancer Research, 83(9), 1459–1475.

Lin, Z., Sun, L., Chen, W., Liu, B., Wang, Y., Fan, S., Li, Y., & Li, J. (2014). miR-639 regulates transforming growth factor beta-induced epithelial-mesenchymal transition in human tongue cancer cells by targeting FOXC1. Cancer Science, 105(10), 1288–1298.

Liu, J., Jiang, X., Zou, A., Mai, Z., Huang, Z., Sun, L., & Zhao, J. (2021). CircIGHG-induced epithelial-to-mesenchymal transition promotes oral squamous cell carcinoma progression via miR-142-5p/IGF2BP3 signaling. Cancer Research, 81(2), 344–355.

Liu, X., Yun, F., Shi, L., Li, Z.-H., Luo, N.-R., & Jia, Y.-F. (2015). Roles of signaling pathways in the epithelial-mesenchymal transition in cancer. Asian Pacific Journal of Cancer Prevention: APJCP, 16(15), 6201–6206.

Mortazavi, H., Baharvand, M., & Mehdipour, M. (2014). Oral potentially malignant disorders: an overview of more than 20 entities. Journal of Dental Research, Dental Clinics, Dental Prospects, 8(1), 6–14.

Nanishi, K., Konishi, H., Shoda, K., Arita, T., Kosuga, T., Komatsu, S., Shiozaki, A., Kubota, T., Fujiwara, H., Okamoto, K., Ichikawa, D., & Otsuji, E. (2020). Circulating circERBB2 as a potential prognostic biomarker for gastric cancer: An investigative study. Cancer Science, 111(11), 4177–4186.

Peng, Q.-S., Cheng, Y.-N., Zhang, W.-B., Fan, H., Mao, Q.-H., & Xu, P. (2020). circRNA_0000140 suppresses oral squamous cell carcinoma growth and metastasis by targeting miR-31 to inhibit Hippo signaling pathway. Cell Death & Disease, 11(2). https://doi.org/10.1038/s41419-020-2273-y

Peters, M. D. J., Godfrey, C. M., Khalil, H., McInerney, P., Parker, D., & Soares, C. B. (2015). Guidance for conducting systematic scoping reviews. International Journal of Evidence-Based Healthcare, 13(3), 141–146.

Rai K, H., & Ahmed, J. (2019). A Correlative Study of N-Cadherin Expression with Different Grades of Oral Squamous Cell Carcinoma Projecting as a Marker of Epithelial to Mesenchymal Transition in Tumor Progression. Asian Pacific Journal of Cancer Prevention: APJCP, 20(8), 2327–2332.

Rong, L., Chen, B., Liu, K., Liu, B., He, X., Liu, J., Li, J., He, M., Zhu, L., Liu, K., Shi, X., Shuai, Y., & Jin, L. (2022). CircZDBF2 up-regulates RNF145 by ceRNA model and recruits CEBPB to accelerate oral squamous cell carcinoma progression via NFκB signaling pathway. Journal of Translational Medicine, 20(1), 148.

Saeed, S., Rauf, F., Iqbal, F., Khan, A. S., Khan, A. H., & Alamgeer, R. (2022). Comparison of PD-L1 expression in oral squamous cell carcinoma and premalignant lesions of oral cavity. Asian Pacific Journal of Cancer Prevention: APJCP, 23(12), 4039–4045.

Shen, T., Yang, T., Yao, M., Zheng, Z., He, M., Shao, M., Li, J., & Fang, C. (2022). BTC as a novel biomarker contributing to EMT via the PI3K-AKT pathway in OSCC. Frontiers in Genetics, 13, 875617.

Shi, R., & Zhang, L. (2024). Circ_0004771 regulates malignant biological behaviors and has clinical significance in oral squamous cell carcinoma. Journal of Oral Pathology & Medicine: Official Publication of the International Association of Oral Pathologists and the American Academy of Oral Pathology, 53(8), 502–510.

Tan, X., Zhou, C., Liang, Y., Lai, Y.-F., & Liang, Y. (2020). Circ_0001971 regulates oral squamous cell carcinoma progression and chemosensitivity by targeting miR-194/miR-204 in vitro and in vivo. European Review for Medical and Pharmacological Sciences, 24(5), 2470–2481.

Wang, Jie, Jiang, C., Li, N., Wang, F., Xu, Y., Shen, Z., Yang, L., Li, Z., & He, C. (2020). The circEPSTI1/mir-942-5p/LTBP2 axis regulates the progression of OSCC in the background of OSF via EMT and the PI3K/Akt/mTOR pathway. Cell Death & Disease, 11(8). https://doi.org/10.1038/s41419-020-02851-w

Wang, Jun, Ouyang, S., Zhao, S., Zhang, X., Cheng, M., Fan, X., Cai, Y., & Liao, L. (2022). SP1-mediated upregulation of circFAM126A promotes proliferation and epithelial-mesenchymal transition of oral squamous cell carcinoma via regulation of RAB41. Frontiers

in Oncology, 12, 715534.

Xie, N.-N., Liu, Z.-X., Wu, C., Wang, P.-L., Song, G.-T., & Chen, Z. (2018). MicroRNA-200c suppresses tumor metastasis in oral squamous carcinoma by inhibiting epithelial-mesenchymal transition. European Review for Medical and Pharmacological Sciences, 22(11), 3415–3422.

Xu, J., Lin, Q., & Zhao, X. (2023). Circular RNA 0000311 aggravates the aggressiveness of oral squamous cell carcinoma via miR-876-5p/EZH2 axis. Journal of Environmental Pathology, Toxicology and Oncology: Official Organ of the International Society for Environmental Toxicology and Cancer, 42(3), 43–52.

Yan, J., & Xu, H. (2021). Regulation of transforming growth factor-beta1 by circANKS1B/miR-515-5p affects the metastatic potential and cisplatin resistance in oral squamous cell carcinoma. Bioengineered, 12(2), 12420–12430.

Yang, Z., Chen, W., Wang, Y., Qin, M., & Ji, Y. (2021). CircKRT1 drives tumor progression and immune evasion in oral squamous cell carcinoma by sponging miR-495-3p to regulate PDL1 expression. Cell Biology International, 45(7), 1423–1435.

Zeng, G., Xun, W., Wei, K., Yang, Y., & Shen, H. (2016). MicroRNA-27a-3p regulates epithelial to mesenchymal transition via targeting YAP1 in oral squamous cell carcinoma cells. Oncology Reports, 36(3), 1475–1482.

Zhang, Hanyu, Zhang, B., Chen, Y., Zhang, Y., Qian, M., Yuan, L., Shen, Y., & Yang, H. (2021). Downregulated hsa_circ_0036988 promotes proliferation and metastasis in oral squamous cell carcinoma. Cancer Biomarkers: Section A of Disease Markers, 31(4), 375–383.

Zhang, Hong, Wang, Z., & Zhang, Z. (2023). Hsa_circ_0009128 mediates progression of oral squamous cell carcinoma by influencing MMP9. Oral Diseases, 29(2), 661–671.

Zhang, Q., Jiang, C., Ren, W., Li, S., Zheng, J., Gao, Y., Zhi, K., & Gao, L. (2021). Circ-LRP6 mediates epithelial-mesenchymal transition and autophagy in oral squamous cell carcinomas. Journal of Oral Pathology & Medicine: Official Publication of the International Association of Oral Pathologists and the American Academy of Oral Pathology, 50(7), 660–667.

Zhao, W., Cui, Y., Liu, L., Qi, X., Liu, J., Ma, S., Hu, X., Zhang, Z., Wang, Y., Li, H., Wang, Z., Liu, Z., & Wu, J. (2020). Splicing factor derived circular RNA circUHRF1 accelerates oral squamous cell carcinoma tumorigenesis via feedback loop. Cell Death and Differentiation, 27(3), 919–933.

Zhou, J., & Jin, S. (2023). Circ_0058063 contributed to oral squamous cell carcinoma development by sponging miR-145 and regulating PI3K/AKT pathway. Molecular Biotechnology, 65(12), 2049–2060.

Zhou, W.-Y., Cai, Z.-R., Liu, J., Wang, D.-S., Ju, H.-Q., & Xu, R.-H. (2020). Circular RNA: metabolism, functions and interactions with proteins. Molecular Cancer, 19(1), 172.