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EDITORIAL

New targets for cancer promotion and therapy in gliomas: Scinderin

Xi Wang, Lian-Xiang Luo

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Abstract

Glioma is one of the most common primary intracranial tumors, characterized by invasive growth and poor prognosis. Actin cytoskeletal rearrangement is an essential event in tumor cell migration. Scinderin (SCIN), an actin severing and capping protein that regulates the actin cytoskeleton, is involved in the proliferation and migration of certain cancer cells. However, its biological role and molecular mechanism in glioma remain unclear. Lin *et al* explored the role and mechanism of SCIN in gliomas. The results showed that SCIN mechanically affected cytoskeleton remodeling and inhibited the formation of lamellipodia *via* RhoA/FAK signaling pathway. This study identifies the cancer-promoting role of SCIN and provides a potential therapeutic target for SCIN in glioma treatment.

Key Words: Glioma; Scinderin; Actin cytoskeleton; RhoA/FAK signaling; 1p/19q co-deletion

Core Tip: The role of scinderin (SCIN) in cancer progression has been studied, but its role in glioma remains unknown. Lin *et al* found that the expression level of *SCIN* mRNA was positively correlated with the tumor grade of glioma. SCIN affected cytoskeletal remodeling and inhibited lamellipodia formation through RhoA/FAK signaling pathway, thereby facilitating the migration and invasion of glioma cells.

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INTRODUCTION

Gliomas, tumors arising from glial cells or precursor cells, are the most common primary intracranial tumors, accounting for 26% of all primary brain and other central nervous system tumors and 81% of malignant brain tumors. Although relatively rare, they cause significant mortality and morbidity[1]. Though they are most common in the brain and glial tissue, they can arise anywhere in the central nervous system[2]. Gliomas can be categorized as Grades 1-4 based on malignant behavior, according to the fifth edition of the World Health Organization Classification of Tumors of the Central Nervous System, where Grades 1 and 2 are low-grade gliomas (LGG) and Grades 3 and 4 are high-grade gliomas [3]. 3 to 6 out of 100000 people suffer from glioma in China, the majority of which are Grades 3 and 4 gliomas [glioblastoma multiforme (GBM)]. For patients with intact brain function, surgical excision is still the preferred course of treatment. For high-grade gliomas, chemotherapy and radiation therapy come next. Glioma sufferers still have a dismal outlook, nonetheless. Thus, research into the molecular mechanisms underlying the growth of gliomas as well as the creation of novel prognostic markers and therapeutic targets are becoming increasingly important.

SCIN'S PRO-ONCOGENIC FUNCTION

An initial and fundamental step in the process of tumor metastasis is the active migration of tumor cells, which depends critically on the reorganization of the actin cytoskeleton, which drives the cellular motor signaling cascade downstream of the complex. Factors that regulate actin assembly begin to emerge as potential targets to prevent the spread and invasion of tumor cells[4]. Scinderin (SCIN), a member of the calcium-dependent gelatin superfamily, controls the tissue of actin by cutting off and capping actin. The SCIN protein consists of six homologous structural domains (*i.e.*, A1-A6). The analysis of the X-ray crystal structure indicates that calcium binding at the N-terminal end of SCIN inhibits the activation process, thereby exposing the F actin binding site on A2[5]. Prior research has documented that SCIN impacts several cellular functions, such as cancer differentiation, migration, and proliferation. For example, SCIN promotes the survival of prostate cancer cells by stabilizing epidermal growth factor receptor and MEK/ERK signaling[6]. Furthermore, filopodia formation, Cdc42 expression, and the proliferation and migration of gastric cancer cells are all inhibited by SCIN knockdown[7]. Lung cancer's progression and dissemination are likewise encouraged by SCIN[8]. Uncertainty persists regarding the molecular mechanism and biological function of SCIN in gliomas. Lin *et al*[9] were the first to investigate how SCIN works in gliomas and to search for new targets for the treatment of gliomas. Based on the GEPIA database, Lin *et al*[9] found that there was a notable increase in SCIN abundance in both LGG and GBM compared to normal tissues, and there was also an increase in SCIN expression in different types of gliomas. Recent studies have identified three particularly noteworthy molecular changes that occur early in glioma formation are prevalent in gliomas, and are tumor markers[10]. The first molecular alteration identified in gliomas is codeletion of chromosome arms 1p and 19q (1p/19q codeletion), which is believed to be associated with the diagnosis of oligodendrogliomas, greater sensitivity to chemotherapy, and better prognosis[11]. In addition, the other two are telomerase reverse transcriptase promoter mutations and indole dehydrogenase (IDH) mutations[12,13]. Mutant IDH genes have been shown to generate the proteins that change α-ketoglutarate into 2-hydroxyglutarate (2HG), a possible tumor metabolite[14]. One of the earliest known processes in the creation of gliomas has been shown to involve IDH mutations; nevertheless, the ensuing 2HG appears to promote broad epigenetic alterations, changing cellular differentiation and perhaps stimulating tumorigenesis [15]. According to Lin *et al*[9], the expression of SCIN was found to be significantly reduced in gliomas with both IDH mutations and 1p/19q chromosome co-deletion. They also observed a positive correlation between the expression level of SCIN mRNA and the tumour grade of gliomas. Lin *et al*[9] experimentally demonstrated decreased cell proliferation after silencing endogenous SCIN. Wound healing experiments demonstrated that SCIN-deficient cells migrated significantly more slowly to the scratch area than to the sh-NC group. In addition, SCIN knockdown inhibited the invasive ability of cells. The findings indicate that SCIN exerts a stimulatory influence on the migration and invasion of glioma cells. To explore this even further, Lin *et al*[9] experimentally demonstrated that SCIN prevented the RhoA/FAK signaling pathway from being activated. Interestingly, it is commonly recognized that F-actin polymerization is correlated with the inhibition of the RhoA/FAK signaling pathway, which indicates weak F-actin polymerization[16], which implies that SCIN regulates F-actin polymerization through RhoA/FAK signaling pathway. Moreover, cell motility was inhibited after the selective FAK inhibitor pf573228 and the RhoA inhibitor CCG1423SCIN were employed, indicating that SCIN promotes the malignant behavior of glioma cells through RhoA/FAK signaling pathway. In summary, lack of SCIN prevented glioma cells from proliferating, invading, and migrating. Mechanistically, SCIN blocked the RhoA/FAK signaling pathway to prevent plate foot formation and altered cytoskeletal remodeling[9]. This work establishes SCIN's pro-oncogenic function and offers a possible therapeutic target for the management of gliomas.

CONCLUSION

A correlation has been identified between the presence of wild-type IDH and an advanced tumour grade, and an elevated expression of SCIN. The absence of SCIN has been demonstrated to inhibit the development, invasion, and migration of glioma cells. Mechanistically, SCIN influences the RhoA/FAK signaling pathway, which in turn influences cytoskeletal remodeling and plate foot formation. As a possible therapeutic target for the treatment of gliomas, this study highlights the pro-oncogenic activity of SCIN.

FOOTNOTES

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REFERENCES

1 **Ostrom QT**, Gittleman H, Truitt G, Boscia A, Kruchko C, Barnholtz-Sloan JS. CBTRUS Statistical Report: Primary Brain and Other Central Nervous System Tumors Diagnosed in the United States in 2011-2015. *Neuro Oncol* 2018; **20**: iv1-iv86 [PMID: 30445539 DOI: 10.1093/neuonc/noy131]
2 **Ostrom QT**, Price M, Neff C, Cioffi G, Waite KA, Kruchko C, Barnholtz-Sloan JS. CBTRUS Statistical Report: Primary Brain and Other Central Nervous System Tumors Diagnosed in the United States in 2015-2019. *Neuro Oncol* 2022; **24**: v1-v95 [PMID: 36196752 DOI: 10.1093/neuonc/noac202]
3 **Louis DN**, Perry A, Wesseling P, Brat DJ, Cree IA, Figarella-Branger D, Hawkins C, Ng HK, Pfister SM, Reifenberger G, Soffietti R, von Deimling A, Ellison DW. The 2021 WHO Classification of Tumors of the Central Nervous System: a summary. *Neuro Oncol* 2021; **23**: 1231-1251 [PMID: 34185076 DOI: 10.1093/neuonc/noab106]
4 **Nürnberg A**, Kollmannsperger A, Grosse R. Pharmacological inhibition of actin assembly to target tumor cell motility. *Rev Physiol Biochem Pharmacol* 2014; **166**: 23-42 [PMID: 24193252 DOI: 10.1007/112_2013_16]
5 **Chumnarnsilpa S**, Lee WL, Nag S, Kannan B, Larsson M, Burtnick LD, Robinson RC. The crystal structure of the C-terminus of adseverin reveals the actin-binding interface. *Proc Natl Acad Sci U S A* 2009; **106**: 13719-13724 [PMID: 19666531 DOI: 10.1073/pnas.0812383106]
6 **Lai X**, Su W, Zhao H, Yang S, Zeng T, Wu W, Wang D. Loss of scinderin decreased expression of epidermal growth factor receptor and promoted apoptosis of castration-resistant prostate cancer cells. *FEBS Open Bio* 2018; **8**: 743-750 [PMID: 29744289 DOI: 10.1002/2211-5463.12412]
7 **Liu JJ**, Liu JY, Chen J, Wu YX, Yan P, Ji CD, Wang YX, Xiang DF, Zhang X, Zhang P, Cui YH, Wang JM, Bian XW, Qian F. Scinderin promotes the invasion and metastasis of gastric cancer cells and predicts the outcome of patients. *Cancer Lett* 2016; **376**: 110-117 [PMID: 27033455 DOI: 10.1016/j.canlet.2016.03.035]
8 **Liu H**, Shi D, Liu T, Yu Z, Zhou C. Lentivirus-mediated silencing of SCIN inhibits proliferation of human lung carcinoma cells. *Gene* 2015; **554**: 32-39 [PMID: 25303873 DOI: 10.1016/j.gene.2014.10.013]
9 **Lin X**, Zhao Z, Sun SP, Liu W. Scinderin promotes glioma cell migration and invasion *via* remodeling actin cytoskeleton. *World J Clin Oncol* 2024; **15**: 32-44 [PMID: 38292665 DOI: 10.5306/wjco.v15.i1.32]
10 **Eckel-Passow JE**, Lachance DH, Molinaro AM, Walsh KM, Decker PA, Sicotte H, Pekmezci M, Rice T, Kosel ML, Smirnov IV, Sarkar G, Caron AA, Kollmeyer TM, Praska CE, Chada AR, Halder C, Hansen HM, McCoy LS, Bracci PM, Marshall R, Zheng S, Reis GF, Pico AR, O'Neill BP, Buckner JC, Giannini C, Huse JT, Perry A, Tihan T, Berger MS, Chang SM, Prados MD, Wiemels J, Wiencke JK, Wrensch MR, Jenkins RB. Glioma Groups Based on 1p/19q, IDH, and TERT Promoter Mutations in Tumors. *N Engl J Med* 2015; **372**: 2499-2508 [PMID: 26061753 DOI: 10.1056/NEJMoa1407279]
11 **Fontaine D**, Vandenbos F, Lebrun C, Paquis V, Frenay M. [Diagnostic and prognostic values of 1p and 19q deletions in adult gliomas: critical review of the literature and implications in daily clinical practice]. *Rev Neurol (Paris)* 2008; **164**: 595-604 [PMID: 18565359 DOI: 10.1016/j.neurol.2008.04.002]
12 **Killela PJ**, Reitman ZJ, Jiao Y, Bettegowda C, Agrawal N, Diaz LA Jr, Friedman AH, Friedman H, Gallia GL, Giovanella BC, Grollman AP, He TC, He Y, Hruban RH, Jallo GI, Mandahl N, Meeker AK, Mertens F, Netto GJ, Rasheed BA, Riggins GJ, Rosenquist TA, Schiffman M, Shih IeM, Theodorescu D, Torbenson MS, Velculescu VE, Wang TL, Wentzensen N, Wood LD, Zhang M, McLendon RE, Bigner DD, Kinzler KW, Vogelstein B, Papadopoulos N, Yan H. TERT promoter mutations occur frequently in gliomas and a subset of tumors derived from cells with low rates of self-renewal. *Proc Natl Acad Sci U S A* 2013; **110**: 6021-6026 [PMID: 23530248 DOI: 10.1073/pnas.1303607110]
13 **Cancer Genome Atlas Research Network**, Brat DJ, Verhaak RG, Aldape KD, Yung WK, Salama SR, Cooper LA, Rheinbay E, Miller CR, Vitucci M, Morozova O, Robertson AG, Noushmehr H, Laird PW, Cherniack AD, Akbani R, Huse JT, Ciriello G, Poisson LM, Barnholtz-Sloan JS, Berger MS, Brennan C, Colen RR, Colman H, Flanders AE, Giannini C, Grifford M, Iavarone A, Jain R, Joseph I, Kim J, Kasaian K, Mikkelsen T, Murray BA, O'Neill BP, Pachter L, Parsons DW, Sougnez C, Sulman EP, Vandenberg SR, Van Meir EG, von Deimling A, Zhang H, Crain D, Lau K, Mallery D, Morris S, Paulauskis J, Penny R, Shelton T, Sherman M, Yena P, Black A, Bowen J, Dicostanzo K, Gastier-Foster J, Leraas KM, Lichtenberg TM, Pierson CR, Ramirez NC, Taylor C, Weaver S, Wise L, Zmuda E, Davidsen T, Demchok JA, Eley G, Ferguson ML, Hutter CM, Mills Shaw KR, Ozenberger BA, Sheth M, Sofia HJ, Tarnuzzer R, Wang Z, Yang L, Zenklusen JC, Ayala B, Baboud J, Chudamani S, Jensen MA, Liu J, Pihl T, Raman R, Wan Y, Wu Y, Ally A, Auman JT, Balasundaram M, Balu S, Baylin SB,

Beroukhim R, Bootwalla MS, Bowlby R, Bristow CA, Brooks D, Butterfield Y, Carlsen R, Carter S, Chin L, Chu A, Chuah E, Cibulskis K, Clarke A, Coetzee SG, Dhalla N, Fennell T, Fisher S, Gabriel S, Getz G, Gibbs R, Guin R, Hadjipanayis A, Hayes DN, Hinoue T, Hoadley K, Holt RA, Hoyle AP, Jefferys SR, Jones S, Jones CD, Kucherlapati R, Lai PH, Lander E, Lee S, Lichtenstein L, Ma Y, Maglinte DT, Mahadeshwar HS, Marra MA, Mayo M, Meng S, Meyerson ML, Mieczkowski PA, Moore RA, Mose LE, Mungall AJ, Pantazi A, Parfenov M, Park PJ, Parker JS, Perou CM, Protopopov A, Ren X, Roach J, Sabedot TS, Schein J, Schumacher SE, Seidman JG, Seth S, Shen H, Simons JV, Sipahimalani P, Soloway MG, Song X, Sun H, Tabak B, Tam A, Tan D, Tang J, Thiessen N, Triche T Jr, Van Den Berg DJ, Veluvolu U, Waring S, Weisenberger DJ, Wilkerson MD, Wong T, Wu J, Xi L, Xu AW, Zack TI, Zhang J, Aksoy BA, Arachchi H, Benz C, Bernard B, Carlin D, Cho J, DiCara D, Frazer S, Fuller GN, Gao J, Gehlenborg N, Haussler D, Heiman DI, Iype L, Jacobsen A, Ju Z, Katzman S, Kim H, Knijnenburg T, Kreisberg RB, Lawrence MS, Lee W, Leinonen K, Lin P, Ling S, Liu W, Liu Y, Lu Y, Mills G, Ng S, Noble MS, Paull E, Rao A, Reynolds S, Saksena G, Sanborn Z, Sander C, Schultz N, Senbabaoglu Y, Shen R, Shmulevich I, Sinha R, Stuart J, Sumer SO, Sun Y, Tasman N, Taylor BS, Voet D, Weinhold N, Weinstein JN, Yang D, Yoshihara K, Zheng S, Zhang W, Zou L, Abel T, Sadeghi S, Cohen ML, Eschbacher J, Hattab EM, Raghunathan A, Schniederjan MJ, Aziz D, Barnett G, Barrett W, Bigner DD, Boice L, Brewer C, Calatozzolo C, Campos B, Carlotti CG Jr, Chan TA, Cuppini L, Curley E, Cuzzubbo S, Devine K, DiMeco F, Duell R, Elder JB, Fehrenbach A, Finocchiaro G, Friedman W, Fulop J, Gardner J, Hermes B, Herold-Mende C, Jungk C, Kendler A, Lehman NL, Lipp E, Liu O, Mandt R, McGraw M, Mclendon R, McPherson C, Neder L, Nguyen P, Noss A, Nunziata R, Ostrom QT, Palmer C, Perin A, Pollo B, Potapov A, Potapova O, Rathmell WK, Rotin D, Scarpace L, Schilero C, Senecal K, Shimmel K, Shurkhay V, Sifri S, Singh R, Sloan AE, Smolenski K, Staugaitis SM, Steele R, Thorne L, Tirapelli DP, Unterberg A, Vallurupalli M, Wang Y, Warnick R, Williams F, Wolinsky Y, Bell S, Rosenberg M, Stewart C, Huang F, Grimsby JL, Radenbaugh AJ. Comprehensive, Integrative Genomic Analysis of Diffuse Lower-Grade Gliomas. *N Engl J Med* 2015; **372**: 2481-2498 [PMID: 26061751 DOI: 10.1056/NEJMoa1402121]

14 **Pirozzi CJ**, Yan H. The implications of IDH mutations for cancer development and therapy. *Nat Rev Clin Oncol* 2021; **18**: 645-661 [PMID: 34131315 DOI: 10.1038/s41571-021-00521-0]

15 **Chen R**, Smith-Cohn M, Cohen AL, Colman H. Glioma Subclassifications and Their Clinical Significance. *Neurotherapeutics* 2017; **14**: 284-297 [PMID: 28281173 DOI: 10.1007/s13311-017-0519-x]

16 **Tang J**, Kang Y, Huang L, Wu L, Peng Y. TIMP1 preserves the blood-brain barrier through interacting with CD63/integrin β 1 complex and regulating downstream FAK/RhoA signaling. *Acta Pharm Sin B* 2020; **10**: 987-1003 [PMID: 32642407 DOI: 10.1016/j.apsb.2020.02.015]

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