Crimson Publishers
Wings to the Research

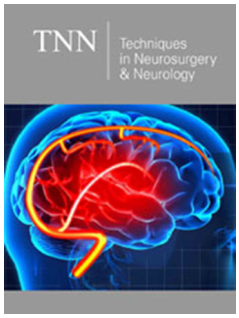
Mini Review

KIAA1217 (SKT) in Spinal Integrity and Neuroplasticity: A Hypothesis Linking Structural and Cytoskeletal Mechanisms to Neural Function

Mosab Nouraldein Mohammed Hamad*

Assistant professor of Microbiology, Excellence Research Center, Elsheikh Abdallah Elbadri University, Sudan

ISSN: 2637-7748



***Corresponding author:** Mosab Nouraldein Mohammed Hamad, Assistant Professor of Microbiology, Excellence Research Center, Elsheikh Abdallah Elbadri University, Berber, Sudan

Submission: February 13, 2026

Published: March 09, 2026

Volume 6 - Issue 2

How to cite this article: Mosab Nouraldein Mohammed Hamad*. KIAA1217 (SKT) in Spinal Integrity and Neuroplasticity: A Hypothesis Linking Structural and Cytoskeletal Mechanisms to Neural Function. Tech Neurosurg Neurol. 6(2). TNN. 000635. 2026.
DOI: 10.31031/TNN.2026.06.000635

Abstract

KIAA1217 (SKT) is a protein-coding gene located on chromosome 10p12.2-p12, primarily implicated in vertebral development and intervertebral disc integrity. Beyond its musculoskeletal role, emerging molecular evidence indicates that KIAA1217 encodes a scaffold protein involved in cytoskeletal regulation, including actin filament dynamics and centrosomal organization. Public transcriptomic datasets demonstrate measurable KIAA1217 expression in human brain regions, including the cerebral cortex and midbrain and single-cell RNA sequencing suggests neuronal cell-type association. Given that actin remodeling underlies dendritic spine morphogenesis and synaptic plasticity, we propose that KIAA1217 may influence neural plasticity through cytoskeletal mechanisms. Although direct functional evidence in neurons remains limited, current molecular and transcriptomic data provide preliminary support for investigating a potential structural-neural interface mediated by KIAA1217.

Keywords: KIAA1217; SKT; Spinal integrity; Actin remodeling; Neuroplasticity; Cytoskeletal proteins; Hypothesis

Introduction

KIAA1217 (SKT) is a protein-coding gene associated with vertebral development, Inter Vertebral Disc (IVD) formation and skeletal integrity [1-3]. Genetic studies have linked KIAA1217 polymorphisms to lumbar disc herniation and vertebral malformations [2,3]. These findings support its established developmental role in axial skeletal architecture.

At the molecular level, KIAA1217 functions as a scaffold protein involved in cytoskeletal regulation. Recent cellular studies indicate that SKT localizes to centrosomal regions and microtubule plus-end structures, participates in focal adhesion organization and modulates actin polymerization dynamics through Src-dependent signaling pathways [4]. Disruption of KIAA1217 alters actin organization and cytoskeletal stability in cultured cells [4]. Additionally, functional studies in epithelial and cancer cell models demonstrate that KIAA1217 regulates cytoskeleton-dependent processes such as migration and structural remodeling [5]. These findings indicate that KIAA1217 is not merely a structural developmental protein but an active regulator of cytoskeletal architecture.

Actin filament dynamics are central to neuronal structure and function. Dendritic spine formation, maturation and synaptic plasticity depend on tightly regulated actin remodeling [6]. Structural plasticity driven by actin turnover underlies learning, memory consolidation and adaptive neural network refinement. Therefore, proteins involved in actin scaffolding and centrosomal organization may plausibly contribute to neural plasticity mechanisms.

Importantly, transcriptomic data provide preliminary support for neural relevance. According to the National Center for Biotechnology Information Gene database, KIAA1217 is broadly expressed across tissues, including central nervous system regions [1]. Data from the Human Protein Atlas demonstrate measurable RNA expression of KIAA1217 in human cerebral cortex and midbrain tissues [7]. Furthermore, single-cell RNA sequencing datasets curated by the Human Protein Atlas indicate expression within neuronal and glial cell clusters [7]. Cross-species expression databases such as Bgee report cortical expression of KIAA1217 orthologs in mammalian models [8]. While expression alone does not confirm function, these findings support biological plausibility for neural investigation.

Hypothesis

We hypothesize that KIAA1217 may influence neural plasticity through cytoskeletal regulation mechanisms. Specifically:

A. Cell-autonomous pathway: If expressed in neurons, KIAA1217 may regulate actin filament organization within dendritic spines, thereby influencing synaptic stability and plasticity.

B. Centrosomal coordination pathway: Given its reported centrosomal localization [4], KIAA1217 may participate in cytoskeletal polarity and intracellular transport mechanisms relevant to neuronal morphology.

C. Structural-neural interface pathway: Through its established role in spinal development and structural integrity [2,3], KIAA1217 may indirectly influence sensorimotor circuit adaptation, potentially contributing to neuroplastic responses to biomechanical changes.

Discussion

Current evidence supporting this hypothesis includes:

a) Genetic association with vertebral development and disc pathology [2,3].

b) Demonstrated involvement in cytoskeletal and actin remodeling pathways [4,5].

c) Established importance of actin remodeling in synaptic plasticity and higher neural function [6].

d) Documented expression in human brain regions and neuronal cell clusters [7].

e) Cross-species cortical expression supporting evolutionary conservation [8].

Although no direct experimental studies have yet examined KIAA1217 in neurons, the convergence of cytoskeletal regulatory function and neural tissue expression provides a testable framework. Investigating KIAA1217 in neuronal models may uncover previously unrecognized links between structural scaffold proteins and synaptic regulation.

Conclusion

KIAA1217, traditionally studied in musculoskeletal development, exhibits molecular properties and brain expression patterns that support investigation into potential roles in neural plasticity. Its involvement in actin remodeling and centrosomal architecture provides a mechanistic basis for exploring contributions to dendritic spine dynamics and synaptic regulation. Future experimental validation is required to determine whether KIAA1217 directly modulates neuronal structure and function.

References

1. (2026) National Center for Biotechnology Information (NCBI). Gene ID: 56243, KIAA1217.
2. Al Dhaheri N, Wu N, Zhao S, Wu Z, Blank RD, et al. (2020) KIAA1217: A novel candidate gene associated with isolated and syndromic vertebral malformations. Am J Med Genet A 182(7): 1664-1672.
3. Dai J, Jiang H, Cheng Z, Li Y, Tang X (2024) Genetic polymorphism of KIAA1217 is functionally associated with lumbar disc herniation in the Chinese population. Neurochirurgie 70(2): 101538.
4. Greibill L, Raimbourg L, Siegfried H, Bonaventure J, Defilippi P, et al. (2025). KIAA1217/SKT is a centrosomal protein that regulates ciliogenesis and integrates src dependent signaling with actin remodeling (preprint). bioRxiv.
5. Wang Y, Li N, Zheng Y, Wang A, Yu C, et al. (2021) KIAA1217 promotes epithelial-mesenchymal transition and hepatocellular carcinoma metastasis by interacting with and activating STAT3. Int J Mol Sci 23(1): 104.
6. Zhuravlev AV (2025) Neuronal actin remodeling and its role in higher nervous activity. Int J Mol Sci 26(22): 11215.
7. (2026) Human protein atlas. Tissue and single-cell RNA expression data for KIAA1217.
8. Bastian F, Parmentier G, Roux J, Moretti S, Laudet V, et al. (2008) Bgee: Integrating and comparing heterogeneous transcriptome data among species. In: Berger SD (Ed.), International Workshop on Data Integration in the Life Sciences, Springer, Heidelberg, Berlin, Germany, pp. 124-131.