ACCSCIENCE PUBLISHING

Eurasian Journal of Medicine and Oncology

PERSPECTIVE ARTICLE

Perspectives on the involvement of NCAPD3 in prostate cancer

Hongzhen Wang[1]*, Kaixin Zhang[1], Liang Ma[2], and Yingjie Yu[1]*

[1]Department of Biological Sciences, College of Life Sciences, Jilin Normal University, Siping, Jilin, China

[2]Department of General Surgery, Siping Central People's Hospital, Siping, Jilin, China

Abstract

Prostate cancer is one of the most common malignant tumors in men worldwide. Its pathogenesis is complex and involves the abnormal regulation of multiple genes and signaling pathways. Non-SMC condensin II complex subunit D3 (NCAPD3), a core component of the condensin II complex, was initially identified as being primarily involved in chromosome condensation and segregation during mitosis and in maintaining genomic stability. In recent years, advances in research on the molecular mechanisms of tumors have increasingly revealed the aberrant expression and oncogenice role of NCAPD3 in prostate cancer and other malignancies, suggesting that NCAPD3 may represent a potential target for molecularly targeted therapy in prostate cancer. Based on the existing evidence, this article reviews the biological functions, expression characteristics, associations with clinicopathological characteristics and prognosis, regulatory mechanisms, and potential clinical applications of NCAPD3 in prostate cancer.

Keywords: Condensin; Non-SMC condensin II complex subunit D3; Prostate cancer

***Corresponding authors:**
Hongzhen Wang (wanghz@jlnu.edu.cn);
YingjieYu (yingjie0518@163.com)

Citation: Wang H, Zhang K, Ma L, Yu Y. Perspectives on the involvement of NCAPD3 in prostate cancer. *Eurasian J Med Oncol.* 2026;10(4):026230261. doi: 10.36922/EJMO026230261

Received: June 6, 2026

Revised: July 30, 2026

Accepted: July 31, 2026

Published online: August 6, 2026

1. Introduction

Non-SMC condensin II complex subunit D3 (NCAPD3) was originally identified in nuclear extracts of HeLa cells as a non-SMC subunit of human condensin II and belongs to the huntingtin-elongation-A-subunit-TOR (HEAT)-repeat protein family.[1-3] In recent years, several studies have shown that aberrant NCAPD3 expression is associated with PC development and patient prognosis. PC remains the most commonly diagnosed malignancy in males worldwide and the second leading cause of cancer-related deaths in men in Western countries.[4,5] This article firstly reviews the current understanding of the role of aberrant NCAPD3 expression in PC. It then discusses the involvement of NCAPD3 in PC from three perspectives.

2. Basic biological functions of NCAPD3

Non-SMC condensin II complex subunit D3 is a key subunit of the condensin II complex. The condensin II complex is a highly conserved, five-subunit protein complex primarily involved in chromosome assembly, condensation, and segregation during mitosis and meiosis in eukaryotic cells.[6-8] NCAPD3 binds to the other four subunits (i.e., NCAPG2, NCAPH2, SMC2, and SMC4) to form a functionally intact condensin II complex.

The resulting complex is localized in the nucleus during interphase and participates in chromosome condensation during mitosis, thereby ensuring accurate chromosome segregation and preventing chromosomal aberrations.

In addition to its canonical function in regulating cell division, recent studies have shown that NCAPD3 is also involved in the maintenance of mitochondrial function and the transcriptional regulation of genes related to histone modification.[9-11] NCAPD3 dysfunction may lead to cell-cycle disruption and increased genomic instability, thereby contributing to tumorigenesis and progression.

3. Expression characteristics of NCAPD3 in prostate cancer

NCAPD3 mRNA and protein levels have been evaluated using RNA sequencing (RNA-seq), quantitative real-time polymerase chain reaction (qRT-PCR), Western blotting, and immunohistochemistry in PC tissues and cell lines. The results showed that NCAPD3 expression was significantly higher in PC tissues than in adjacent noncancerous prostate tissues.[12-16] NCAPD3 expression in PC cell lines, such as PC-3, DU145, 22Rv1, and LNCaP, was also significantly higher than that in the prostatic stromal myofibroblast cell line WPMY-1 and the benign prostatic epithelial cell line BPH-1, suggesting that elevated NCAPD3 expression may be closely related to the development of PC.[12-16] For example, RNA-seq analysis of clinical specimens by Zhang *et al.*[12] showed that NCAPD3 expression was significantly higher in PC tissues than in adjacent noncancerous tissues, whereas the expression levels of other condensin subunits, such as NCAPD2 and NCAPG2, did not differ significantly. Jing *et al.*[13] also reported that NCAPD3 was upregulated in PC tissues and cell lines and that its expression level was associated with tumor aggressiveness. Collectively, studies using RNA-seq, qRT-PCR, Western blotting, and immunohistochemistry have confirmed that NCAPD3 is upregulated in PC cells and tissues.[12,13,17] These findings suggest that NCAPD3 may be a candidate biomarker for PC and provide a basis for further studies of its diagnostic value.

NCAPD3 expression is also regulated by the androgen receptor (AR) signaling pathway.[14] Studies showed that AR directly upregulated NCAPD3 expression, whereas AR knockdown reduced NCAPD3 levels, indicating that NCAPD3 may be involved in androgen-dependent PC progression. This finding provides a basis for investigating the role of NCAPD3 in hormone-sensitive PC and its potential relevance to PC treatment.[14,16,17]

4. Association with clinicopathological characteristics and prognosis

Conflicting findings have been reported regarding the relationship between NCAPD3 expression and the clinicopathological characteristics and prognosis of PC. An early study by Lapointe *et al.*[14] identified NCAPD3 as a marker of molecular subtype 1 of PC, which was characterized by indolent clinical behavior and an androgen-signaling signature. NCAPD3 expression was associated with a significantly lower postoperative tumor recurrence rate, independently of pathological stage, Gleason score, and preoperative prostate-specific antigen level, suggesting an association with a favorable prognosis.

However, more recent studies have indicated that elevated NCAPD3 expression promotes PC progression. Jing *et al.*[13] found that high NCAPD3 expression promoted the proliferation and migration of PC cells and is closely related to the activation of carcinogenic signaling pathways, such as STAT3 and E2F1, suggesting that NCAPD3 may drive PC progression. Zhang *et al.*[12] further showed that NCAPD3 promoted PC cell growth *in vitro* and tumor growth *in vivo* by suppressing the expression of tumor-suppressor miR-30a-5p. These findings support a tumor-promoting role for NCAPD3.

A 2025 study by Zhang *et al.*[18] further support for this conclusion. In a nude mouse xenograft model, NCAPD3 overexpression significantly accelerated PC tumor growth *in vivo*, and elevated NCAPD3 expression was positively correlated with tumor volume and weight. Clinical sample analysis also demonstrated that patients with elevated NCAPD3 expression had significantly shorter progression-free survival, further supporting the association between high NCAPD3 expression and poor prognosis in patients with PC. These findings provide additional experimental evidence regarding the conflicting results of previous studies.

The discrepancy among these findings may be related to the heterogeneity of PC molecular subtypes, and the role of NCAPD3 may differ among these subtypes. Larger studies incorporating analyses stratified by molecular subtype are therefore needed.

5. Regulatory mechanisms of NCAPD3 in prostate cancer

The tumor-promoting effects of NCAPD3 in PC are mainly mediated through multiple signaling pathways, non-coding RNA (ncRNA) networks, and transcription

factors. These regulatory mechanisms involve complex and overlapping pathways. The currently identified mechanisms are summarized in Figure 1.

5.1. Regulation of STAT3-related signaling

Signal transducer and activator of transcription 3 (STAT3) is a key oncogenic transcription factor that is aberrantly activated in PC. It promotes tumor cell proliferation, inhibits apoptosis, and enhances invasive and metastasis capacities. Existing studies have confirmed that NCAPD3 increases STAT3 expression and activation through multiple mechanisms, thereby contributing to PC progression.[12,13,18]

Non-SMC condensin II complex subunit D3 increases STAT3 expression and phosphorylation at Tyr705, thereby enhancing the transcription of downstream STAT3 target genes.[19] NCAPD3 also co-localizes and interacts with STAT3 in the nucleus, facilitating the recruitment of STAT3 to the promoter regions of downstream target genes. For example, Jing *et al.*[13] found that NCAPD3 interacted with STAT3 and promoted the recruitment of STAT3 to the promoter regions of enhancer of zeste 2 polycomb repressive complex 2 subunit (EZH2) and metastasis-associated lung adenocarcinoma transcript 1 (MALAT1), thereby increasing their expression. Zhang *et al.*[12] also confirmed that NCAPD3 promoted MALAT1 transcription by upregulating STAT3, thereby contributing to the regulation of miR-30a-5p. They further clarified the mechanism underlying the interaction between NCAPD3 and STAT3. NCAPD3 activated STAT3, thereby upregulating the long non-coding RNA (lncRNA) MALAT1 and contributing to PC progression. These findings provide additional insight into the mechanism by which NCAPD3 regulates STAT3 signaling.[18]

Non-SMC condensin II complex subunit D3 may also enhance oncogenic signaling through a positive feedback loop between STAT3 and Janus kinase 2 (JAK2).[18] STAT3 acts as a transcription factor of JAK2 by binding to the JAK2 promoter and promoting its expression. NCAPD3 may therefore increase JAK2 expression indirectly by upregulating STAT3. In turn, JAK2 phosphorylates and activates STAT3, forming a JAK2/STAT3 positive feedback loop that sustains the malignant phenotype of PC cells.

5.2. Regulation of AKT-related Signaling Pathway

The protein kinase B (AKT) signaling pathway is one of the most frequently dysregulated pathways in PC. AKT activation, which involves phosphorylation at Thr308 and Ser473, promotes tumor cell proliferation, migration, and metastasis. NCAPD3 increases AKT phosphorylation at these two sites through two distinct upstream pathways.[12,13,18]

Non-SMC condensin II complex subunit D3 regulates AKT Thr308 through the JAK2/phosphoinositide 3-kinase (PI3K) axis. PI3K catalyzes the conversion of phosphatidylinositol 4,5-bisphosphate to phosphatidylinositol 3,4,5-trisphosphate, thereby facilitating AKT recruitment and phosphorylation at Thr308. NCAPD3 overexpression increased PI3K phosphorylation, whereas NCAPD3 silencing reduced it. The PI3K inhibitor LY294002 significantly inhibited NCAPD3-induced AKT Thr308 phosphorylation. NCAPD3 also activated PI3K/AKT signaling by increasing JAK2 expression, whereas the JAK2 inhibitor Z3 blocked NCAPD3-induced PI3K and AKT Thr308 phosphorylation. These findings suggest that NCAPD3 regulates AKT Thr308 phosphorylation through the JAK2/PI3K axis.

Non-SMC condensin II complex subunit D3 regulates AKT Ser473 phosphorylation through the STAT3/EZH2/

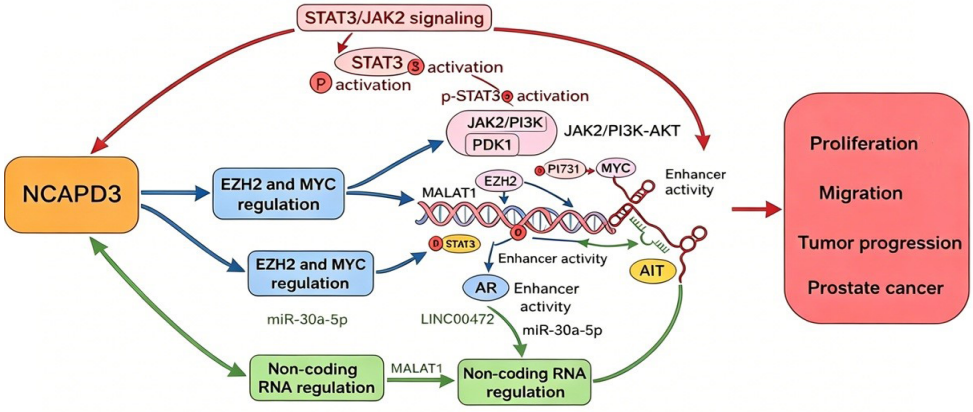

Figure 1. Regulatory mechanisms of NCAPD3 in prostate cancer. NCAPD3 promotes prostate cancer progression through the STAT3/JAK2 signaling pathway, AKT-related signaling pathway, E2F1/EZH2 signaling axis, and MALAT1/miR-30a-5p axis.

nuclear receptor-binding SET domain protein 2 (NSD2)/mechanistic target of rapamycin complex 2 (mTORC2) axis. mTORC2 promotes AKT phosphorylation at Ser473, and rapamycin-insensitive companion of mechanistic target of rapamycin (RICTOR) is an essential component of this complex. NCAPD3 upregulates EZH2 through STAT3; EZH2 subsequently promotes NSD2 expression, and NSD2 regulates RICTOR expression, thereby enhancing mTORC2 activity and AKT Ser473 phosphorylation. The EZH2 inhibitor GSK126 significantly reduced NCAPD3-induced increases in NSD2 and RICTOR expression and AKT Ser473 phosphorylation, supporting the involvement of this regulatory axis.[12,13,18]

Non-SMC condensin II complex subunit D3 is primarily localized in the nucleus, whereas AKT is mainly localized in the cytoplasm. Therefore, NCAPD3 may regulate AKT phosphorylation indirectly through upstream regulatory factors, although the precise molecular mechanisms remain to be clarified.

5.3. Regulation of E2F1/EZH2 signaling axis

Enhancer of zeste homolog 2 is the pivotal catalytic subunit of polycomb repressive complex 2. It represses tumor-suppressor gene expression through histone H3K27 trimethylation and is highly expressed in PC, thereby promoting tumor progression. Jing *et al.*[13] found that NCAPD3 promoted EZH2 transcription by upregulating E2F1 and STAT3.

Specifically, NCAPD3 overexpression significantly increased the mRNA and protein levels of E2F1 and STAT3, both of which bound to the EZH2 promoter region and enhanced EZH2 transcription. The E2F1 inhibitor HLM006474 and the STAT3 inhibitor Stattic significantly inhibited NCAPD3-induced EZH2 expression, whereas combined treatment with both inhibitors completely blocked this effect. These findings suggest that NCAPD3 regulates EZH2 expression through STAT3 and E2F1, thereby contributing to PC progression.

5.4. Regulation of non-coding RNA network: The MALAT1/miR-30a-5p axis

In recent years, the regulatory role of ncRNAs in PC has attracted wide attention. NCAPD3 participates in a competing endogenous RNA (ceRNA) network involving the lncRNA MALAT1 and miR-30a-5p, thereby contributing to PC progression. The MALAT1/miR-30a-5p axis is one of the best-characterized ncRNA-related pathways regulated by NCAPD3.

Metastasis-associated lung adenocarcinoma transcript 1 is a highly conserved lncRNA that is upregulated in PC and promotes tumor cell proliferation, migration, and metastasis. Zhang *et al.*[12] found that NCAPD3 regulated MALAT1 expression through STAT3. STAT3 promoted MALAT1 transcription by binding to its promoter region. MALAT1 subsequently acted as a ceRNA that sequestered miR-30a-5p, thereby reducing miR-30a-5p expression. miR-30a-5p is a tumor-suppressive microRNA, and its reduced expression is related to poor prognosis in PC.

In addition, NCAPD3 can suppress miR-30a-5p transcription by upregulating MYC.[12] MYC binds to the promoter region of LINC00472, the host gene of miR-30a-5p, and represses its transcription, thereby reducing miR-30a-5p expression. Thus, NCAPD3 suppresses miR-30a-5p expression through two mechanisms. First, it upregulates STAT3, which promotes MALAT1 transcription, allowing MALAT1 to sequester miR-30a-5p. Second, it upregulates MYC, which binds to the LINC00472 promoter and represses miR-30a-5p transcription. The study also confirmed that miR-30a-5p overexpression significantly attenuated NCAPD3-induced increases in PC cell viability and migration, supporting the functional relevance of this regulatory network. In summary, NCAPD3 reduces miR-30a-5p expression through STAT3–MALAT1-mediated sequestration and MYC-mediated transcriptional repression, thereby weakening the tumor-suppressive effects of miR-30a-5p and promoting PC progression.

6. Biological functions of NCAPD3 in prostate cancer

Based on the regulatory mechanisms described above, NCAPD3 exerts tumor-promoting effects in PC cells by promoting tumor cell proliferation, migration, invasion, and tumor growth *in vivo*. NCAPD3 dysregulation may therefore contribute to PC progression. Studies investigating the role of NCAPD3 in PC are summarized in Table 1.

Table 1. Summary of studies on the involvement of NCAPD3 in prostate cancer

Reported role of NCAPD3 in PC	Reference
Function as a diagnostic and prognostic marker for prostate cancer	Lapointe *et al.*;[14] Jung *et al.*[16]
Function as a tumor-promoting factor, prognostic marker, and potential target for prostate cancer	Zhang *et al.*;[12] Jing *et al.*;[13] Yin *et al.*;[17] Zhang *et al.*[18]

6.1. Promotion of prostate cancer cell proliferation

Multiple studies using Cell Counting Kit-8, 5-ethynyl-

2'-deoxyuridine staining, colony formation assays, and other methods have shown that NCAPD3 upregulation significantly increased the proliferation of PC cells, including DU145, PC-3, and LNCaP cells, whereas NCAPD3 silencing significantly inhibited cell proliferation.[12-14,18] The proliferation-promoting effects of NCAPD3 are mainly related to the regulation of cell-cycle-related signaling pathways. For example, NCAPD3 promotes the expression of cell-cycle-related genes by activating the STAT3/E2F1/EZH2 and JAK2/PI3K/AKT axes, thereby promoting cell-cycle progression from the G1 to the S phase. NCAPD3 also suppresses miR-30a-5p expression, thereby relieving miR-30a-5p-mediated repression of proliferation-related target genes (such as *PCLAF*) and further promoting cell proliferation.

6.2. Promotion of prostate cancer cell migration and invasion

Wound-healing and Transwell assays showed that NCAPD3 upregulation significantly enhanced the migration and invasion of PC cells, whereas NCAPD3 silencing significantly reduced these effects.[12,14,18] The migration- and invasion-promoting effects of NCAPD3 are mainly related to the regulation of epithelial–mesenchymal transition (EMT)-related signaling pathways and ncRNA networks. On the one hand, NCAPD3 activates AKT signaling, increases the expression of EMT-related proteins (such as N-cadherin and vimentin), and decreases E-cadherin expression, thereby inducing EMT and enhancing migration and invasion. On the other hand, NCAPD3 regulates the MALAT1/miR-30a-5p axis, allowing MALAT1 to sequester miR-30a-5p and thereby promoting the expression of migration-related genes.

6.3. Promotion of prostate cancer tumor growth *in vivo*

Subcutaneous xenograft experiments in nude mice provided further evidence of the tumor-promoting role of NCAPD3.[13] Subcutaneous injection of NCAPD3-overexpressing PC cells (such as DU145 and PC-3) resulted in significantly greater tumor volume and weight than those observed in the control group. By contrast, 22Rv1 cells with NCAPD3 silencing formed no detectable tumors.

In vivo experiments also confirmed that NCAPD3 overexpression significantly increased the expression of STAT3, MYC, EZH2, and MALAT1 and reduced miR-30a-5p expression. These findings were consistent with the *in vitro* experimental results, suggesting that NCAPD3 plays a key role in promoting the *in vivo* growth of PC. NCAPD3 overexpression also significantly accelerated xenograft growth and suppressed miR-30a-5p transcription, further supporting the role of NCAPD3 in PC progression *in vitro* and *in vivo*.[18]

7. Clinical application prospects of NCAPD3 in prostate cancer

Based on the aberrant expression, proposed tumor-promoting mechanisms, and biological functions of NCAPD3 in PC, NCAPD3 may have potential clinical applications in diagnosis, prognosis assessment, and targeted therapy.

7.1. NCAPD3 as a potential diagnostic and prognostic marker for prostate cancer

Non-SMC condensin II complex subunit D3 is upregulated in PC tissues and cell lines, and its expression has been associated with tumor aggressiveness, clinical stage, and patient prognosis. These findings suggest that NCAPD3 may be a candidate diagnostic and prognostic biomarker for PC.[12-18]

Zinc-alpha_2-glycoprotein 1 (AZGP1) is an adipokine associated with lipid metabolism and vascular fibrosis. In 2004, Lapointe *et al.*[15] first identified AZGP1 as a strong predictor of tumor recurrence in PC subtype 1. Subsequently, Lapointe *et al.*[14] characterized NCAPD3, also known as hCAP-D3, as another candidate tissue biomarker expressed in subtype 1 prostate tumors. In 2014, Jung *et al.*[16] reported that AZGP1 and hCAP-D3 were strong predictors of tumor recurrence in PC. Overall, AZGP1 was first identified as a predictor in subtype I PC, and NCAPD3 was subsequently found to have a similar predictive value. The combination of these markers was evaluated for its ability to improve the prediction of PC.[16] However, the association between NCAPD3 expression and prognosis remains controversial.[14,18] Multi-center studies with larger sample sizes are needed to clarify its prognostic value in different molecular subtypes of PC.

7.2. NCAPD3 as a potential therapeutic target for prostate cancer

As a potential tumor-promoting factor in PC, NCAPD3 regulates several pathways that contain established or potential therapeutic targets, such as STAT3 and AKT signaling and the MALAT1/miR-30a-5p axis. NCAPD3 may therefore represent a potential therapeutic target in PC. However, research on NCAPD3-targeted therapy remains at the preclinical stage. Current studies have mainly focused on silencing NCAPD3 expression using small interfering RNA or short hairpin RNA to inhibit PC cell proliferation and migration or combining NCAPD3 silencing with inhibition of its downstream pathways (such as STAT3 inhibitors, AKT inhibitors, and MALAT1

inhibitors).[12-14,17,18]

Developing specific NCAPD3 inhibitors may provide a potential therapeutic strategy. The successful development of inhibitors targeting other tumor-promoting proteins may provide a conceptual basis for developing specific NCAPD3 inhibitors. For example, sanguinarine, a novel EphB4 inhibitor, was reported to inhibit β-catenin signaling.[19] Similarly, STAT3 inhibitor Stattic, AKT inhibitor MK-2206, and MALAT1 inhibitor MALAT1-IN-1 have been reported to significantly inhibit NCAPD3-induced malignant phenotypes in PC cells, providing an experimental basis for NCAPD3-targeted therapy.

Mechanisms through which NCAPD3 contributes to other cancers may also operate in PC. For example, NCAPD3 has been reported to promote colorectal cancer and papillary thyroid carcinoma progression by enhancing the Warburg effect.[10,20] Whether NCAPD3 similarly regulates the Warburg effect in PC remains to be determined. In addition to PC, NCAPD3 has been implicated in hepatocellular carcinoma and non-small cell lung cancer through PI3K/AKT signaling.[21,22] The differences and similarities among the mechanisms through which NCAPD3 regulates PI3K/AKT signaling in these cancers warrant further investigation.

Future studies should investigate whether mechanisms identified in other cancers also operate in PC and develop specific inhibitors of NCAPD3. Subsequent clinical trials will be required to evaluate the efficacy and safety of NCAPD3-targeted treatments.

8. Summary and outlook

In summary, NCAPD3 is overexpressed in PC. It promotes PC cell proliferation, migration, and tumor growth *in vivo* by regulating multiple signaling pathways, such as STAT3, AKT, and E2F1/EZH2 signaling, as well as the MALAT1/miR-30a-5p ncRNA network. Its expression level has been associated with the clinicopathological characteristics and prognosis of PC, suggesting that it may have potential value as a diagnostic or prognostic marker and therapeutic target. The two mechanisms through which NCAPD3 suppresses miR-30a-5p in PC provide a new direction for mechanistic research.

However, current research on NCAPD3 in PC has several limitations. First, the specific molecular mechanism through which NCAPD3 regulates AKT phosphorylation (especially how nuclear-localized NCAPD3 regulates AKT activation in the cytoplasm) is not fully understood. Second, differences in NCAPD3 expression and regulatory mechanisms among PC molecular subtypes require further investigation to clarify the conflicting findings concerning its association with prognosis. Finally, NCAPD3-targeted therapy remains at the preclinical stage, and specific inhibitors and clinical data are lacking.

Future studies should further explore the precise regulatory mechanism of NCAPD3 in PC and clarify its interaction with other oncogenic factors. Multi-center clinical studies with larger sample sizes are also needed to evaluate the clinical utility of NCAPD3. In addition, the development of specific NCAPD3-targeted drugs and the investigation of combination treatment strategies may provide new approaches to the precision treatment of PC.

Acknowledgments

None.

Funding

The work was supported by funds from the National Natural Science Foundation of China (32101448).

Conflict of interest

The authors declare no competing interests.

Author contributions

Conceptualization: Hongzhen Wang
Writing–original draft: Hongzhen Wang, Yingjie Yu
Writing–review & editing: All authors

Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Availability of data

Not applicable.

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