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Original Article

SERINC4 is dispensable for male fertility and spermatogenesis in mice

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Abstract: Objectives: Transmembrane proteins are known to play pivotal roles in spermatogenesis and male fertility. Recent transcriptomic analyses have suggested that the serine incorporator 4 (SERINC4), a multi-pass transmembrane protein, is highly expressed in testicular germ cells, yet its physiologic function in male reproduction remains unclear. This study aimed to investigate the *in vivo* role of *SERINC4* in spermatogenesis and male fertility using a *Serinc4*-knockout (KO) mouse model. Methods: Single-cell RNA sequencing (scRNA-seq) data were analyzed to determine the expression pattern of *SERINC4* in human and *Serinc4* in mouse testis. *Serinc4*-KO mice were generated by CRISPR/Cas9-mediated deletion of exon 5. Phenotypic analyses included fertility testing, testis histology, sperm evaluation using computer-assisted sperm analysis (CASA), immunofluorescence for germ and somatic cell markers, and TUNEL assay for germ cell apoptosis. Results: Although scRNA-seq analyses revealed high *SERINC4* expression in spermatids of human testes and high *Serinc4* expression in spermatocytes of mouse testes, its deficiency did not impair male fertility, testicular morphology, or spermatogenesis in mice. No significant differences were observed between wild-type (WT) and *Serinc4*-KO mice in terms of sperm count, motility, or testis to body weight ratio. In addition, TUNEL assay revealed no significant change in germ cell apoptosis. Conclusions: Under physiologic conditions, *Serinc4* appears to be dispensable for normal spermatogenesis and male fertility in mice. Future studies are needed to investigate its role under pathologic or aging-related conditions.

Keywords: *Serinc4*, male fertility, spermatogenesis, testis

Introduction

Spermatogenesis takes place within the seminiferous epithelium of the testis and is essential for male fertility. This intricate physiologic process is orchestrated by intrinsic genetic programs and extrinsic cues from the testicular microenvironment [1]. Among the key regulators, transmembrane proteins - including ion channels, transporters, receptors, and structural components - play indispensable roles by mediating molecular transport, transducing signals, and maintaining the integrity of the blood-testis barrier (BTB). For example, TMC7, a transmembrane channel-like protein, is critical for acrosome biogenesis, and its deficiency leads to male infertility in mice [2]. SUN5, a testis-specific nuclear envelope protein, facilitates the nuclear export of mRNA during spermatogenesis [3]. The CatSper calcium channel is essential for sperm capacitation and the acrosome reaction, while potassium channels are involved not only in capacitation but also in epididymal maturation [4, 5]. Additionally, NECL2, a cell adhesion molecule, regulates BTB homeostasis, and its deletion disrupts BTB protein levels, resulting in male infertility in mice [6]. Collectively, these findings highlight the critical

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roles of transmembrane proteins in regulating spermatogenesis and maintaining male reproductive function.

The serine incorporator (SERINC) family comprises five members (SERINC1-5), all of which are multi-pass transmembrane proteins [7]. Among them, SERINC2-4 can incorporate serine into cellular membranes and promote the synthesis of two serine-derived lipids: phosphatidylserine and sphingolipids [8]. The SERINC family is involved in the biological processes underlying various diseases. For instance, SERINC3 and SERINC5 are known to reduce the infectivity of Human Immunodeficiency Virus-1 (HIV-1), with SERINC5 exerting a more potent antiviral effect than SERINC3 [9, 10]. In cancer biology, *SERINC2* knockdown has been shown to inhibit the proliferation, migration, and invasion of lung adenocarcinoma cells [11]. SERINC4, on the other hand, is prone to proteasomal degradation in human cells; however, when stabilized and properly expressed, it can inhibit HIV-1 replication as effectively as SERINC5 [12]. Despite these emerging insights, the physiologic roles of SERINC proteins, particularly in non-viral contexts, remain largely unexplored.

In the context of male reproduction, transcriptomic analyses of Sertoli cells from patients with non-obstructive azoospermia (NOA) have revealed a marked downregulation of *SERINC4* expression [13], suggesting a link between *SERINC4* and male fertility. Interestingly, in our study, transcriptomic data from publicly available single-cell RNA sequencing (scRNA-seq) databases demonstrated that *SERINC4* is highly expressed in germ cells of both human and mouse testis, indicating a potential role in spermatogenesis. This high expression, despite the lack of a clearly defined function, underscores the value of studying such genes for insights into fertility preservation, reproductive toxicology, and contraceptive strategies. These observations prompted us to further investigate the physiologic significance of *SERINC4* in male reproductive biology.

Given this knowledge gap, we generated *Serinc4*-knockout (KO) mice using CRISPR/Cas9-mediated genome editing to investigate the role of *Serinc4* in spermatogenesis and male fertility. Through comprehensive phenotypic characterization, including fertility assays, histologic analysis, and evaluation of testicular germ cell markers, this study aimed to provide novel insight into the physiologic significance of *Serinc4* in the male reproductive system and to expand our understanding of the broader functional repertoire of the SERINC family.

Materials and methods

Bioinformatic analysis

We investigated the expression patterns of *Serinc4* using publicly available scRNA-seq datasets from human and mouse testes. Data processing and visualization were performed using Seurat (v4.0). Dotplot and Featureplot functions were used to display *Serinc4* expression across testicular cell populations. Cell identities were annotated based on well-established marker genes.

Mice

Serinc4-KO mice were generated by CRISPR/Cas9-mediated genome editing, as previously described with modifications [14]. Four single-guide RNAs (sgRNAs) were designed to target exon 5 of the *Serinc4* gene, leading to a 330 bp deletion including this exon. The sgRNA sequences were as follows: gRNA-A_1: 5'-TGGGAACTTTTAGCTCTGGCTGG-3'; gRNA-A_2: 5'-CCATAGCACGTTTGCTCCCATGG-3'; gRNA-B_1: 5'-CCATTGGGAACTTTTAGCTCTGG-3'; gRNA-B_2: 5'-CCATGGGAGCAAACGTGCTATGG-3'. Genotyping of *Serinc4*-KO mice was performed by PCR using the following primers: *Serinc4*-F_1: 5'-TCAAGCTGTTGTTCCTGTTAGGTC-3'; *Serinc4*-R_1: 5'-AGGCTGACTTTGGAACTCCC-3'; *Serinc4*-R_2: 5'-GTGCTTCTTACAAATGTGTGTGTGTC-3'.

All animal experiments were conducted under protocols approved by the Animal Ethics and Welfare Committee of Nanjing Medical University (No. IACUC-2402015). Mice were humanely euthanized by CO_2 inhalation followed by cervical dislocation to ensure minimal suffering, in accordance with institutional guidelines.

Fertility test

Fertility testing was performed according to previously published methods [15]. Adult male *Serinc4*-KO and wild-type (WT) mice were paired with WT females at a 1:2 ratio for 12 weeks. Litter size, pup number per litter, and

birth dates were recorded for fertility assessment.

Computer-assisted sperm analysis (CASA)

Sperm were collected from the cauda epididymis of adult WT and *Serinc4*-KO mice, incubated in human tubal fluid medium supplemented with 10% bovine serum albumin (BSA), as described previously [16-18]. CASA was performed using a Ceros™ II sperm analyzer (Hamilton Thorne, USA) to assess sperm concentration, total motility, and progressive motility.

Histological analysis

Testes from adult WT and *Serinc4*-KO mice were fixed in modified Davidson's fixative, processed for paraffin embedding, and sectioned at 5 μm thickness. Sections were stained with hematoxylin-eosin (H&E) and periodic acid-Schiff (PAS) for histologic evaluation, as described previously [19-21].

Immunofluorescence

Following antigen retrieval in citrate buffer, paraffin-embedded testicular sections were blocked with 1% BSA and incubated overnight at 4°C with primary antibodies: anti-Lin28 (1:200), anti-γ-H2AX (1:400), PNA (1:400), anti-SOX9 (1:400), and anti-3β-HSD (1:200). After PBS washes, AlexaFluor-conjugated secondary antibodies were applied, followed by DAPI nuclear staining. Images were captured using an LSM800 confocal microscope (Zeiss), as described previously [22-24].

Terminal deoxynucleotidyl transferase-dUTP nick-end labeling (TUNEL) assay

Testicular cell apoptosis was assessed using a TUNEL assay kit (Vazyme, Nanjing, China). Sections were deparaffinized, rehydrated, and treated with proteinase K, followed by labeling with BrightRed Labeling Buffer. After counterstaining with DAPI, images were acquired using a confocal microscope.

Statistical analysis

Statistical analyses were performed using GraphPad Prism 8.0. Data are expressed as mean ± SD from at least three independent experiments. Comparisons between WT and *Serinc4*-KO groups were made using unpaired Student's t-test, with significance set at $P < 0.05$.

Results

Serinc4 predominantly localizes in testicular germ cells of human and mouse

To characterize the cellular distribution of *Serinc4*, we analyzed its expression using publicly available scRNA-seq datasets (GSE149512 for human, GSM5563668 for mouse). In human testicular samples, *Serinc4* was mainly enriched in spermatids (**Figure 1A**, **1B**), while in mouse testes, its expression was pronounced in spermatocytes (**Figure 1C**, **1D**).

Serinc4 deficiency does not impair male fertility

To assess the reproductive role of *Serinc4*, we created *Serinc4*-deficient mice using a CRISPR/Cas9 strategy. As illustrated in **Figure 2A**, four sgRNAs were designed to target exons 5 of the *Serinc4* gene, deleting a total of 330 base pairs. The resulting genotypes (*Serinc4*$^{+/+}$, *Serinc4*$^{+/-}$ and *Serinc4*$^{-/-}$) were validated by PCR amplification (**Figure 2B**). Over a three-month breeding period, we compared the reproductive performance of WT and *Serinc4*-KO males. No significant differences were observed in litter sizes or number of pups (**Figure 2C**). Testicular morphology (**Figure 2D**), testis-to-body weight ratio (**Figure 2E**), and sperm indices including count, motility, and progressive motility, measured using CASA (**Figure 2F-H**), showed no significant changes. Furthermore, H&E staining indicated no morphologic abnormalities in spermatozoa from *Serinc4*-KO mice (**Figure 2I**, **2J**). Collectively, these findings suggest that *Serinc4* is dispensable for male fertility under physiological conditions.

Spermatogenesis proceeds normally in the absence of Serinc4

To determine whether *Serinc4* deletion affects spermatogenesis, we performed H&E and PAS staining on testis sections. Both staining methods revealed structurally intact seminiferous tubules in *Serinc4*-KO mice, similar to WT controls (**Figure 3A**, **3B**).

Further, we evaluated the expression of germ cell markers by immunofluorescence. Markers

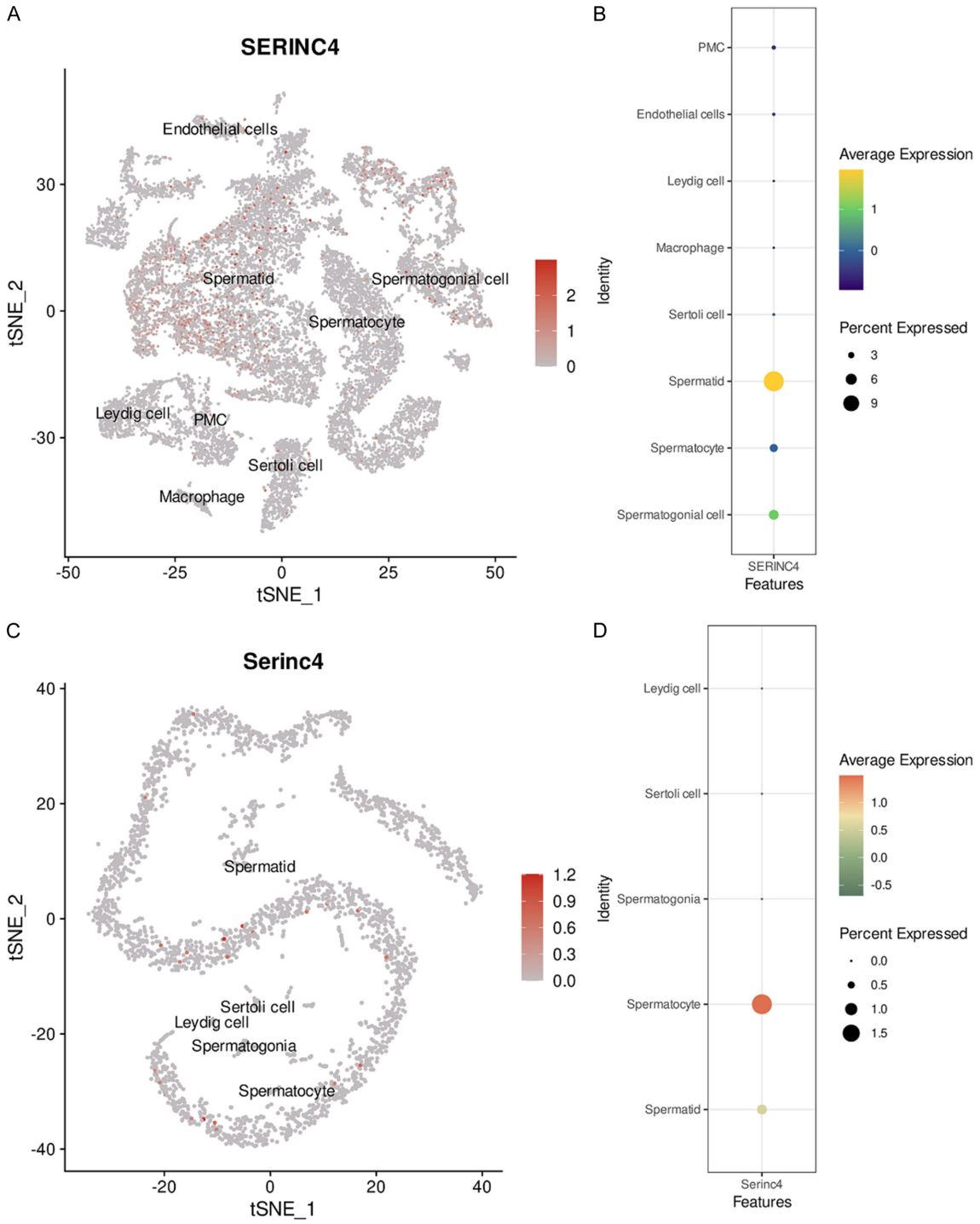

Figure 1. Expression profiling of *SERINC4* in human and *Serinc4* in mouse testis. A. Dot plot showing *SERINC4* expression across defined human testicular cell clusters. B. t-SNE plot depicting *SERINC4* spatial expression in human testicular cells. C. Dot plot represents *Serinc4* expression among mouse testicular cell types. D. t-SNE map illustrates *Serinc4* distribution in mouse testis cell populations.

including Lin28 (spermatogonial stem cell) [25], γ-H2AX (spermatocyte) [26], PNA (acrosome) [27], SOX9 (Sertoli cell) [28], and 3β-HSD (Leydig cell) [29], showed no significant difference between WT and *Serinc4*-KO testes (**Figure 4**). These results indicate that *Serinc4*

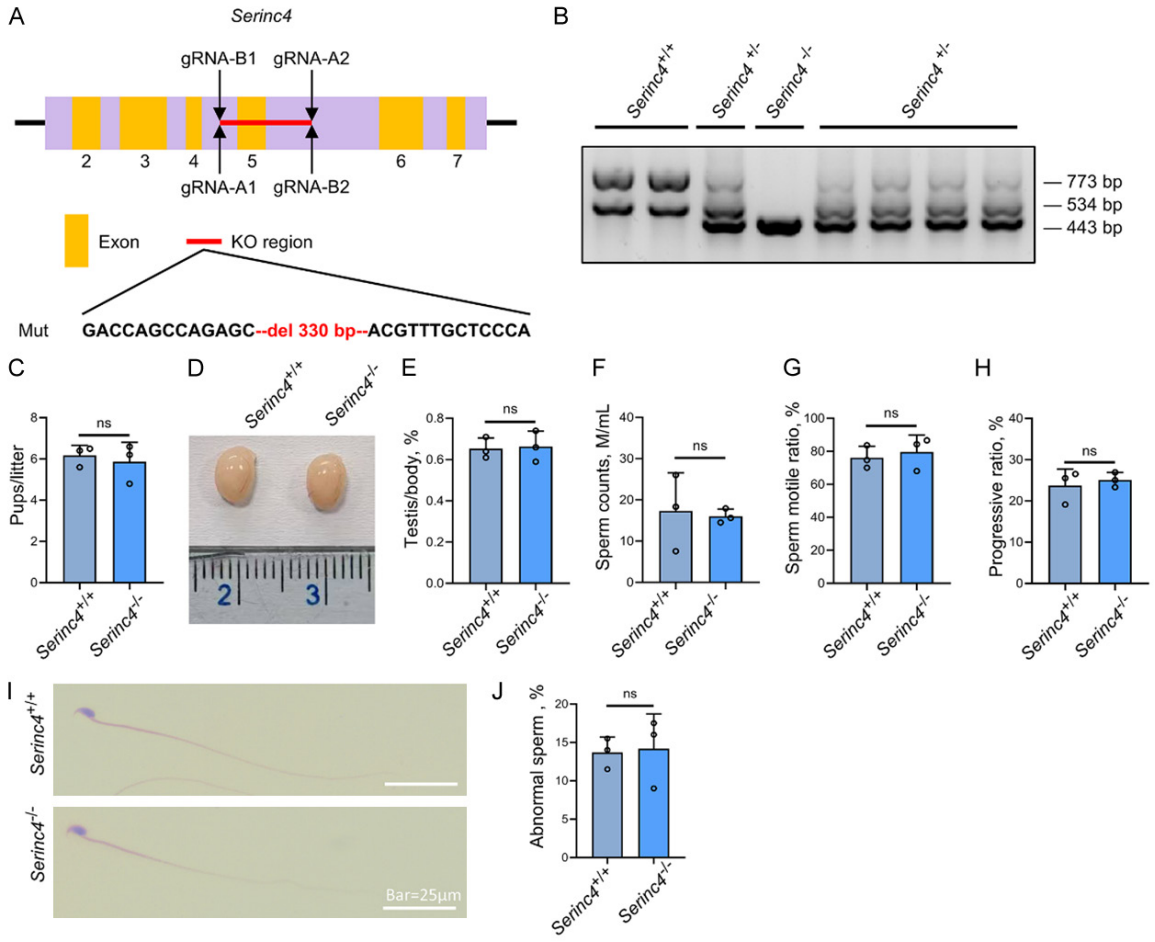

Figure 2. Generation of *Serinc4*-KO mice and fertility assessment. A. Schematic of the CRISPR/Cas9 deletion strategy targeting exon 5 of *Serinc4*. B. PCR validation of genomic DNA from *Serinc4*$^{+/+}$, *Serinc4*$^{+/-}$ and *Serinc4*$^{-/-}$ mice. C. Fertility test of WT and *Serinc4*-KO mice (n = 3, P > 0.05). D. Histologic analysis of testicular morphology in WT and *Serinc4*-KO mice. E. Comparison of testis-to-body weight ratios (n = 3, P > 0.05). F-H. Sperm count, motility, and progressive motility of WT and *Serinc4*-KO mice, evaluated by CASA (n = 4, P > 0.05). I. Representative sperm morphology observed by H&E staining (Scale bar: 20 μm). J. Quantification of sperm abnormalities (n = 3, P > 0.05).

knockout does not adversely affect spermatogenic progression or somatic cell populations within the testes.

Apoptotic activity remains unaltered in Serinc4-KO testes

Despite its notable expression in the testes, *Serinc4* ablation did not influence reproductive outcomes. To explore whether *Serinc4* affects germ cell survival, we conducted a TUNEL assay to assess apoptosis levels. The number of TUNEL-positive cells and the proportion of affected seminiferous tubules showed no significant difference between WT and *Serinc4*-KO mice (**Figure 5A-C**), suggesting no increase in germ cell apoptosis.

Discussion

In this study, we generated *Serinc4*-KO mice to investigate the physiological role of *Serinc4* in male reproduction. Although single-cell transcriptomic data showed that *Serinc4* is highly expressed in testicular germ cells - primarily in spermatids in humans and spermatocytes in mice - our data indicated that Serinc4 deletion did not affect spermatogenesis or fertility under normal physiologic conditions. This lack of phenotype may reflect functional compensation by other SERINC family members, such as SERINC5, or overlapping lipid-related pathways. Moreover, the potential effects of Serinc4 deficiency might only become apparent under aging or other stress conditions, which

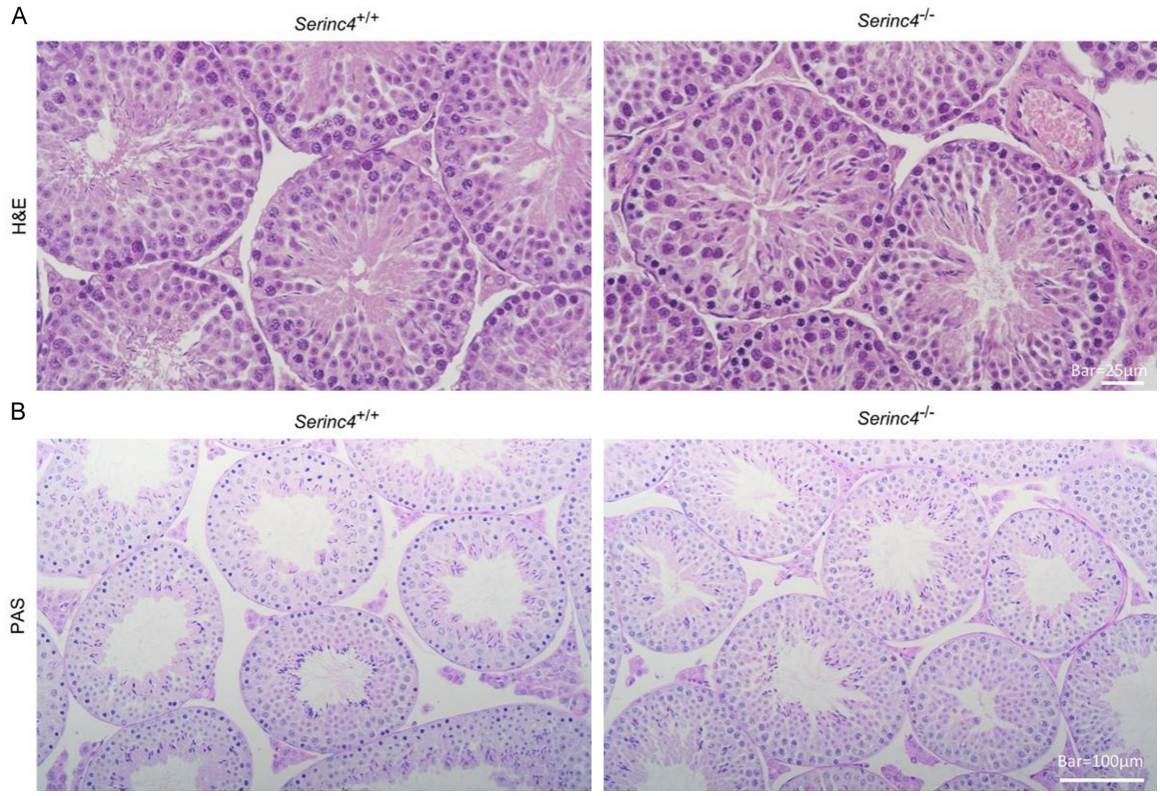

Figure 3. Testicular morphology in WT and *Serinc4*-KO mice. A. H&E staining of testis sections showing preserved seminiferous tubule structure (Scale bar: 25 μm). B. PAS staining confirming intact seminiferous architecture (Scale bar: 100 μm).

were not examined in the present study, and subtle age-related changes may not have been captured by the three-month fertility testing period. While *Serinc4* appears dispensable for normal spermatogenesis, the investigation of developmental toxicology biomarkers may help uncover subtle or context-specific roles that could be overlooked under standard physiologic settings [30].

Members of the SERINC family have drawn considerable attention for their roles in antiviral defense and lipid metabolism. Among them, SERINC3 and SERINC5 exhibit potent antiviral activity by enhancing type I interferon and NF-κB signaling and promoting TIM protein localization to the plasma membrane [31-33]. Notably, SERINC5 exerts a stronger inhibitory effect on HIV-1 membrane fusion than SERINC3 by interfering with the function of the viral envelope glycoprotein (Env) [34, 35]. In addition, SERINC2, SERINC3, and SERINC4 are involved in phosphatidylserine and sphingolipid biosynthesis [8]. However, their roles beyond viral inhibition remain largely unclear.

Previous studies have reported reduced *SERINC4* expression in Sertoli cells of NOA patients, suggesting a role in germ cell development. Despite this, our *Serinc4*-KO mice showed normal testicular histology (H&E, PAS), unaltered expression of germ cell markers (LIN28, γ-H2AX, PNA, SOX9, and 3β-HSD), and no increase in apoptosis (TUNEL assay). Sperm indices and fertility were also unaffected. These findings suggest that *Serinc4* is not essential for testicular homeostasis under basal conditions. Nevertheless, given the downregulation of SERINC4 in NOA patients, further validation in human testicular samples or in vitro models would be valuable to clarify its clinical relevance to male infertility. Additionally, more detailed functional analyses, such as meiotic chromosome spread assays or sperm acrosome reaction tests, were not performed in the present study and will be pursued in future experiments.

In conclusion, this study provides the first *in vivo* evidence that *Serinc4* deficiency does not impair spermatogenesis or male fertility in mice

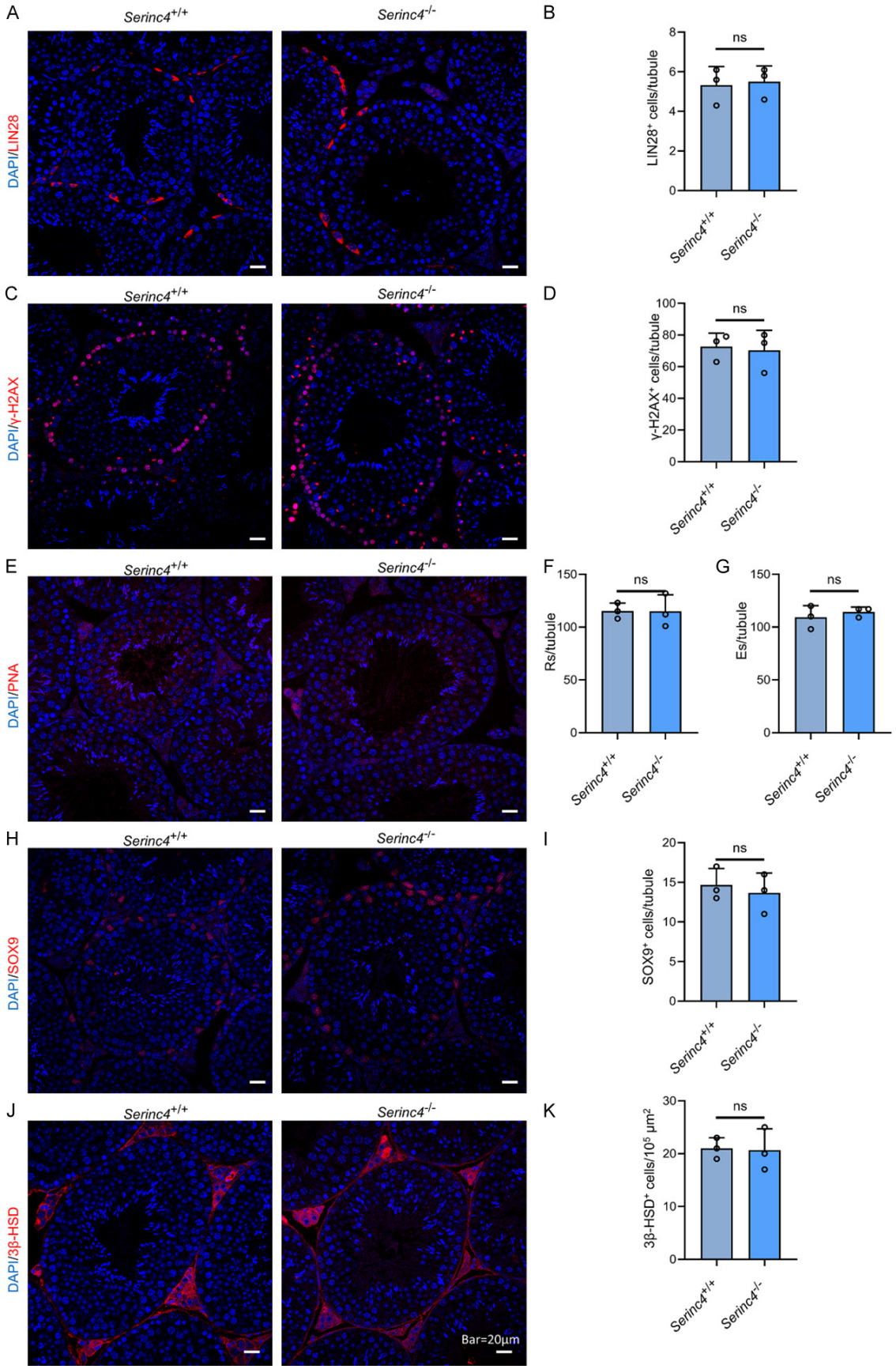
A
Serinc4+/+
Serinc4-/-
DAPI/LIN28
B
LIN28+ cells/tubule
ns
C
DAPI/γ-H2AX
D
γ-H2AX+ cells/tubule
E
DAPI/PNA
F
Rs/tubule
G
Es/tubule
H
DAPI/SOX9
I
SOX9+ cells/tubule
J
DAPI/3β-HSD
K
3β-HSD+ cells/10^5 μm^2
Bar=20μm

Figure 4. Immunostaining analysis of testicular markers of WT and *Serinc4*-KO mice. (A) Representative images of Lin28 immunostaining in testis sections from mice (Scale bar: 20 μm). (B) Quantification of Lin28$^+$ cells/tubule (n = 3, P > 0.05). (C) Immunostaining of γ-H2AX in testis sections from mice (Scale bar: 20 μm). (D) Quantitative analysis of γ-H2AX expression (n = 3, P > 0.05). (E) Representative images of PNA immunostaining in mouse testis (Scale bar: 20 μm). (F) Count of round spermatids (Rs) per tubule in (E) (n = 3, P > 0.05). (G) Count of elongated spermatids (Es) per tubule in the same sections (n = 3, P > 0.05). (H) Immunostaining of SOX9 in testis sections from mice (Scale bar: 20 μm). (I) Quantification of SOX9$^+$ cells/tubule (n = 3, P > 0.05). (J) Representative images of 3β-HSD immunostaining in testis sections (Scale bar: 20 μm). (K) Quantification of 3β-HSD expression (n = 3, P > 0.05).

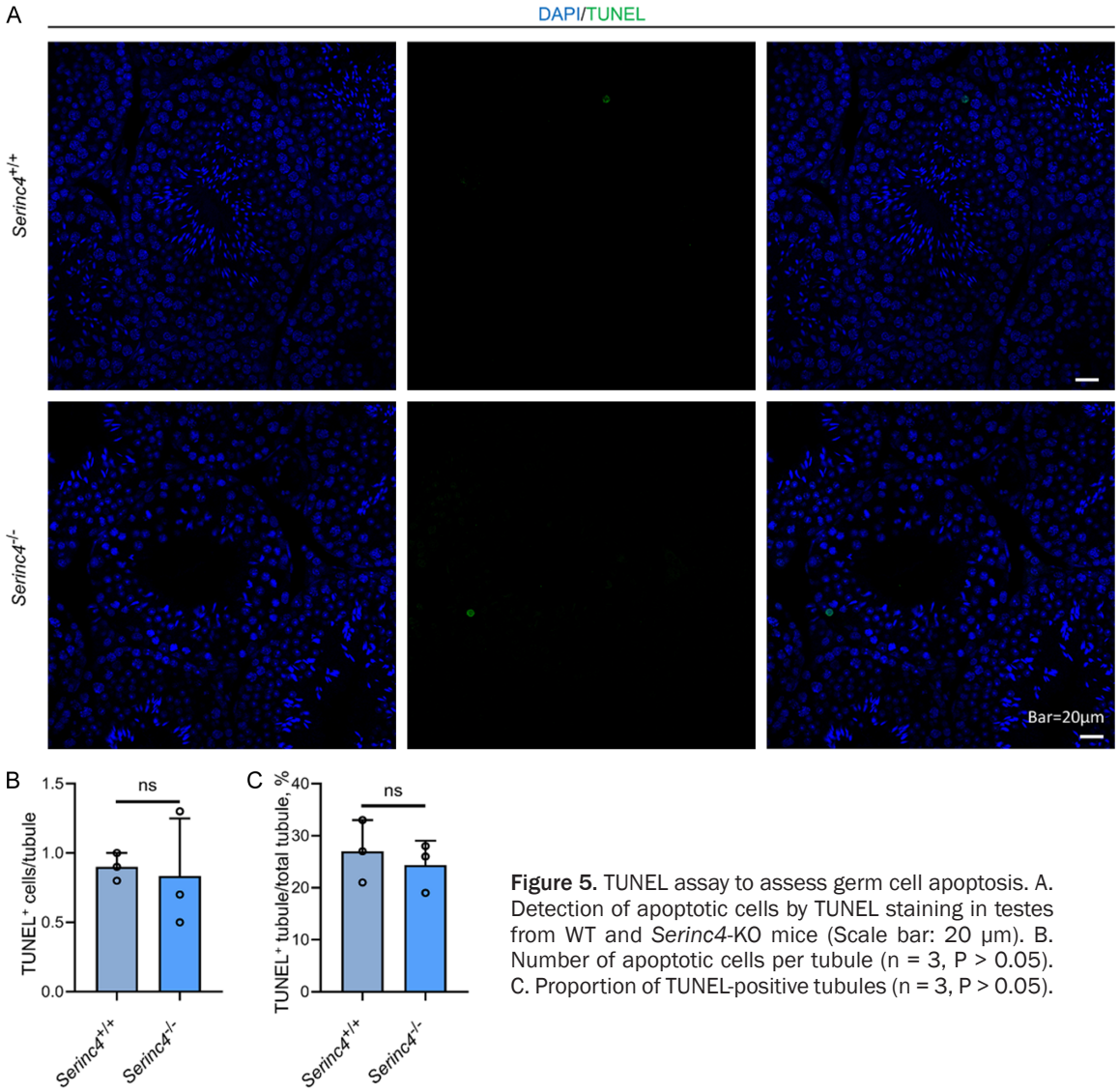

Figure 5. TUNEL assay to assess germ cell apoptosis. A. Detection of apoptotic cells by TUNEL staining in testes from WT and *Serinc4*-KO mice (Scale bar: 20 μm). B. Number of apoptotic cells per tubule (n = 3, P > 0.05). C. Proportion of TUNEL-positive tubules (n = 3, P > 0.05).

under physiological conditions. These findings offer new insight on the roles of SERINC family proteins in reproductive biology. Future investigations should focus on exploring the function of *Serinc4* under stress conditions, in pathological states, or in the context of functional interactions with other SERINC members. Possible experiments include generating double or multiple knockout models, evaluating Serinc4 function in models of testicular injury or aging, and employing human-derived testicular organoids or cell lines for mechanistic studies. Moreover, future studies integrating metabolomic approaches may provide further insight into the physiologic and pathologic roles of *Serinc4* in spermatogenesis [36].

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Disclosure of conflict of interest

None.

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