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Changes in extracellular collagen and caspase-3 expression in testis of male mice due to frequent exposure to magnetic resonance imaging (MRI); Histochemical and immunohistochemical studies

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ABSTRACT

Magnetic fields included in magnetic resonance imaging (MRI) technology are now utilized extensively in diagnostic medicine and healthcare applications. Simultaneously, it provides potential risks to human health, and their biological impacts on the human body are garnering heightened inquiry. The primary objective of the current study was to examine the biological effects of a high-strength magnetic field produced by 1.5 Tesla static magnetic fields on male mice spermatogenesis parameters in magnetic resonance imaging.

Material and methods: In this study, 20 adult male mice were exposed to a 1.5 T MRI field for four weeks (two sessions per week, 1 hour each). Animals were sacrificed at the end of 4th week. Histopathological, histochemical, and Caspase-3 immunohistochemical examinations were performed.

Results: Exposure to MRI fields in male mice led to structural and functional damage in the testes, including degeneration of germ and Leydig cells, heightened testicular cell apoptosis, elevated collagen deposition, and upregulated caspase-3 expression.

Conclusion: Exposure to MRI magnetic fields led to notable histological, histochemical, and immunohistochemical alterations in the mice testes at the conditions of the current experiment.

Keywords: MRI, mice, testis, histopathology, collagen, caspase-3.

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1. INTRODUCTION

Magnetic Resonance Imaging (MRI) is extensively used in clinical diagnostics due to its non-invasive technique and its ability to provide high-resolution images of soft tissues. Unlike ionizing imaging modalities such as X-rays and CT scans, MRI employs static magnetic fields (SMFs) and radiofrequency (RF) waves, both of which are classified as non-ionizing radiation. Although MRI is generally considered safe, especially when compared to ionizing radiation, recent studies have raised concerns about its potential biological effects, particularly when exposure is repeated or prolonged. These concerns relate primarily to changes occurring at the cellular and molecular levels **(Abtin et al., 2024)**, prompting closer examination of how MRI might affect sensitive biological systems. One such vulnerable system is the male reproductive system, especially the testes, which are highly sensitive to environmental stressors, including electromagnetic fields (EMFs). Research has shown that static magnetic fields similar to those used in clinical MRI machines (typically 1.5–3 Tesla) can lead to oxidative stress, disturb cellular homeostasis, and interfere with spermatogenesis and germ cell development **(Aitken & Roman, 2008; Bahaodini et al., 2015)**. These disruptions have the potential to impair male fertility, particularly under conditions of high-field strength or repeated exposure.

Several studies have begun to explore the specific effects of MRI-generated magnetic and RF fields on testicular structure and function. While much of the early MRI safety research focused on issues like tissue heating **(Sammet, 2016)**, electromagnetic interference, and genotoxicity **(Wilén et al., 2020)**, a smaller body of literature has addressed the impact on reproductive parameters. Some findings suggest that MRI exposure could alter sperm characteristics, damage testicular tissue, or disrupt hormonal regulation **(Rostamzadeh et al., 2019)**. Some studies report no significant effect on male fertility **(Møllerløkken et al., 2012)**, while others point to potential reproductive risks, especially under conditions of long-term or frequent exposure **(Kaur et al., 2023)**.

Despite the growing interest, the biological effects of MRI exposure on male reproductive health remain controversial. Inconsistencies among studies may stem from differing methodologies, sample sizes, exposure durations, or failure to control for thermal effects and confounding variables **(Lee et al., 2004)**. With MRI becoming increasingly prevalent in healthcare, there is a clear need for more targeted research to clarify whether non-ionizing radiation from MRI poses a genuine risk to reproductive health. In this context, the current study was designed to assess the structural and functional impact of a 1.5 Tesla static magnetic field—comparable to clinical MRI systems—on the testicular tissue of adult male mice. To achieve this, the study employed histological, histochemical, and immunohistochemical analyses to detect cellular degeneration, collagen deposition, and apoptotic changes that may arise as a result of MRI exposure.

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1. Material and methods:

1.1. Experimental animals:

Twenty adult male albino mice were used in this experiment, obtained from the Animal Breeding House of the National Research Center, Cairo, Egypt. The mice were maintained on a standard pellet diet with unlimited access to food and water. They underwent a two-week acclimatization period before the experiment commenced. The study received ethical approval from the Animal Ethical Committee of Zagazig University, with approval number ZU-IACUC/1/F/422/2022.

1.2. Magnetic field exposure:

For the MRI exposure, the mice were housed in specially designed perforated plastic cages that allowed for adequate ventilation while providing enough space for the exposure procedure. The animals were not sedated to ensure they could breathe and move freely within the cages during the exposure. Each cage was carefully positioned over the head coil of the MRI scanner to ensure that all mice were exposed simultaneously. The MRI scans were conducted using a closed superconductive MRI system (Philips Achieva scanner, Netherlands) located at the Children's Hospital of Zagazig University Hospitals. The MRI system had a 1.5 Tesla magnetic field strength, a 60 cm bore size, and an RF frequency of 64 MHz, equipped with a specialized head coil for optimized imaging. The imaging procedure began with a 3-plane localizer scan to confirm the correct positioning of the mice and to define the field of view. Following this, three-dimensional Axial FLAIR (Fluid-attenuated inversion recovery) images were acquired to assess the effects of the MRI exposure. The imaging parameters included an echo time (TE) of 567.12 milliseconds (ms), a repetition time (TR) of 4800 ms, and a field of view (FOV) of 290 x 335 x 183 mm. Ten acquisitions (NQ) were performed with a matrix of 580 x 674, resulting in a total scanning time of one hour. These detailed imaging parameters were carefully selected to obtain high-resolution images while ensuring the mice were safely exposed to the MRI field.

1.3. Experimental Design

After the acclimatization period, the mice were randomly divided into two groups, with each group consisting of 10 animals. Group I served as the control group and did not undergo any MRI exposure, while Group II was subjected to MRI scanning sessions for a period of four weeks. Mice in Group II underwent two MRI sessions per week, with each session lasting for one hour, to assess the potential effects of MRI exposure on their health and biological systems.

1.4. Preparation of Samples

Animals were euthanized post-exposure following anesthesia with 10 mg/kg xylazine and 115 mg/kg ketamine **(Pedrosa *et al.* 2021)**. Small testicular samples were excised and promptly preserved in a

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10% buffered formaldehyde solution (Merck, Germany) for 24 hours. The tissue samples were subsequently fixed in paraffin and sectioned to a thickness of 5 μm. Tissue sections were affixed to slides and stained with hematoxylin and eosin (H&E) **(Bancroft and Gamble 2008)**. The sections were examined microscopically for histological alterations using a light microscope. Collagen fibers deposition was assessed by Masson's trichrome staining **(Bancroft and Gamble 2008).**

1.5. Immunohistochemical method:

1.5.1. Immuno-staining of caspase-3:

Neutral buffered formalin (10%) fixed testicular sections (5 μ thick) were dewaxed, hydrated to phosphate-buffered saline (PBS; pH 7.5). Immunostaining of caspase-3 was carried out by using streptavidin/biotin immunoperoxidease method (LSAB kit, Dako Corp). For caspase-3, a rabbit polyclonal antibody was used as a key marker for apoptosis (**Bancroft** and **Gamble, 2008)**.

1.6. Image analysis

Caspase-3 immuno-expression in the testis of mice was analyzed using a light microscope, with images captured through a digital camera at 400x magnification. Image analysis software was employed to quantify the intensity of the staining by measuring the area of positive immunoreactivity. The software then calculated the mean optical density (MOD) for each sample, providing a precise assessment of caspase-3 expression levels. This quantitative analysis allowed for the evaluation of apoptotic activity in the testicular tissue based on the extent of caspase-3 positivity.

1.7. Statistical analysis

The data obtained in this study were analyzed using the Statistical Package for the Social Sciences (SPSS/PC) Version 20.0 (IBM Corp., Armonk, NY) for Windows. One-way analysis of variance (ANOVA) was used to analyze the data, and the results were presented as mean ± standard error (SE). A P-value of ≤ 0.05 was considered statistically significant, and a P-value of ≤ 0.01 was considered highly significant. Descriptive statistics were employed to assess both graphical and numerical representations of the data.

2. Results:

2.1. Histopathological observations:

Light microscopy examination of H&E-stained testicular sections from control mice revealed well-organized testicular parenchyma consisting of rounded to oval seminiferous tubules with uniform boundaries. The interstitial spaces between the tubules contained Leydig cells. The tubules were lined with a complete series of spermatogenic cells, including spermatogonia, primary spermatocytes, and early round spermatids. Sertoli cells were also present, supporting the developing spermatozoa **(Figs. 1, 2)**. In contrast, testicular sections from the MRI-exposed group displayed notable structural

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disruptions. There was a marked reduction in spermatogenic cells, presence of apoptotic spermatogonia, cytoplasmic vacuolization, and clumping of spermatozoa within the seminiferous tubules. Several germ cells appeared degenerated, showing pyknotic nuclei, and the Leydig cells in the interstitial tissue were smaller and showed signs of degeneration **(Fig. 3)**.

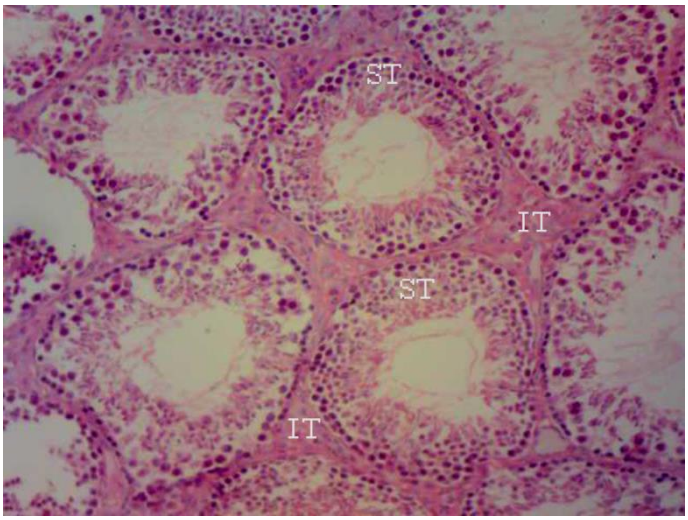

Fig. 1 a Photomicrograph of a testicular section from control mice displaying testicular parenchyma, which includes seminiferous tubules (ST) characterized by rounded or oval shapes with regular contours and lumens, alongside interstitial tissue (IT) composed of loose connective tissue interspersed with Leydig cells. (H and E, ×100)

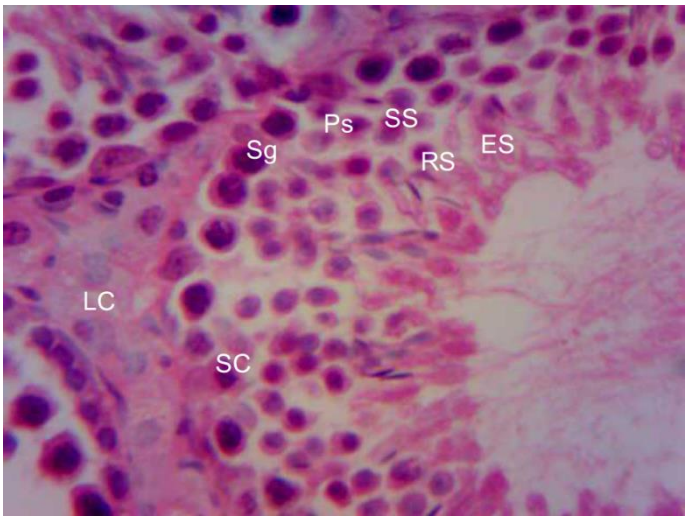

Fig. 2 a Photomicrograph of a testicular section from control mice revealing a seminiferous tubule lined with various spermatogenic cells including spermatogonia (Sg), primary spermatocytes (PS), secondary spermatocytes (SS), round and elongated spermatids (RS, ES). Sertoli cells (SC), The interstitial regions between the tubules show Leydig cells (LC) with vesicular nuclei. (H and E, ×1000)

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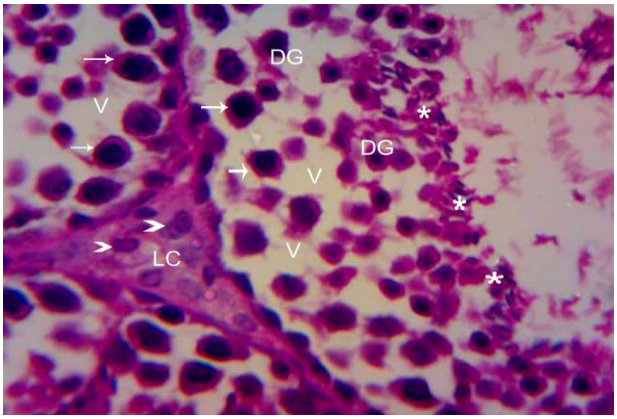

Fig. 3 a Photomicrograph of MRI-exposed rat testis revealing degenerated spermatogenic cells (DG), apoptotic cells (white arrows), vacuolization (V), and spermatozoa adhesion (stars). Leydig cells show signs of degeneration (arrowheads). (H and E, ×1000)

2.2. Histochemical observations:

Masson's trichrome staining of testicular sections from the control group revealed a minimal presence of collagen fibers, identifiable by their characteristic bluish-green coloration. These fibers were primarily localized to the tunica albuginea, the basal lamina encasing the seminiferous tubules, and around the perivascular areas within the interstitial tissue **(Figs. 4, 5)**. The collagen distribution appeared sparse and uniform, reflecting the normal histoarchitecture of healthy testicular tissue.

Conversely, testicular sections obtained from mice exposed to MRI showed a notable increase in collagen fiber deposition. Dense bluish-green collagen accumulation was observed in multiple regions, including the tunica albuginea, interstitial spaces, and around blood vessels. Additionally, the basal lamina of the seminiferous tubules, and intratubular localities exhibited thickened collagen layers, suggesting fibrotic changes potentially linked to tissue remodeling or pathological response to magnetic field exposure **(Figs. 6, 7)**.

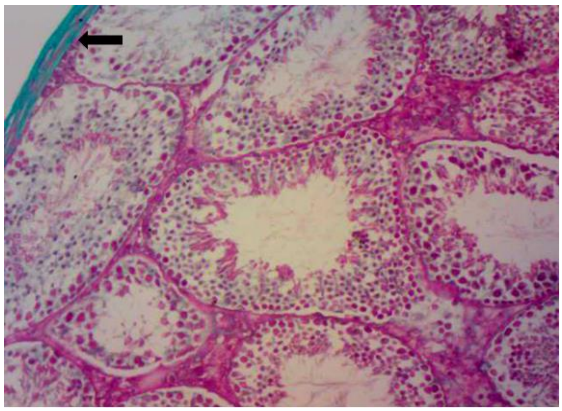

Fig. 4 a Photomicrograph of a section in testis of control mice showing a normal distribution of collagen fibers deposition in the testicular capsule (black arrow) and between the seminiferous tubules (Masson’s trichrome stain x400).

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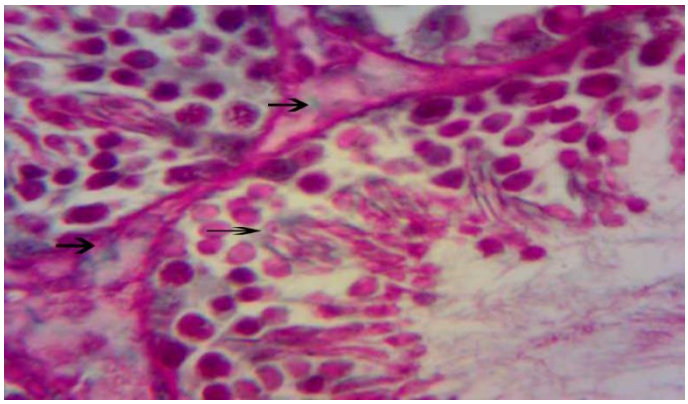

Fig. 5 a Photomicrograph of a section in testis of control mice showing a normal distribution of collagen fibers deposition in the in the basal lamina of seminiferous tubules, between the seminiferous tubules (arrows) (Masson's trichrome stain x1000).

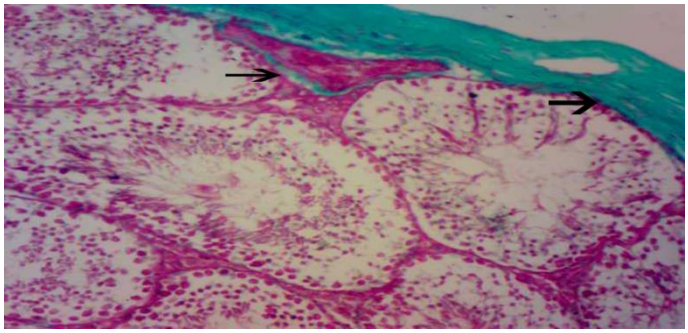

Fig. 6 a Photomicrograph of a section in testis of MRI-exposed mice showing increased collagen fibers deposition in the testicular capsule and around the blood vessels (arrows) (Masson's trichrome stain x400).

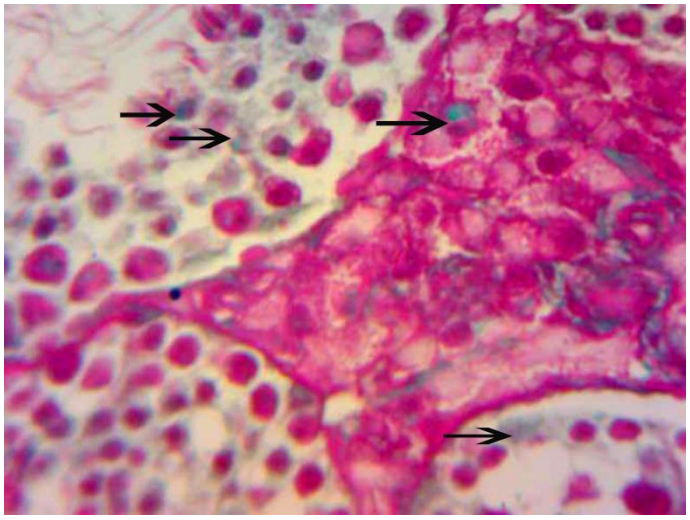

Fig. 7 a Photomicrograph of a section in testis of MRI-exposed mice showing increased collagen fibers deposition in the intratubular localities, interstitium and Leydig cells (Masson's trichrome stain x1000).

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2.3. Immunohistochemical observations:

2.3.1. The expression of caspase-3:

Immunohistochemical analysis of testicular tissue using anti-caspase-3 antibody revealed minimal apoptotic activity in the control group. The seminiferous epithelium exhibited only faint cytoplasmic staining, indicating a low level of caspase-3 expression under normal physiological conditions. Similarly, Leydig cells within the interstitial spaces showed weak or negligible immunoreactivity, consistent with limited apoptosis in healthy testicular tissue **(Fig. 8).**

In comparison, testicular sections from the MRI-exposed group demonstrated a marked upregulation of caspase-3 expression. A strong, diffuse cytoplasmic immunopositive reaction was evident in numerous cells of the germinal epithelium, suggesting enhanced apoptotic activity. Leydig cells in the interstitial tissue also exhibited prominent caspase-3 immunoreactivity, further indicating that MRI exposure may trigger apoptosis in both somatic and germ cells of the testis **(Fig. 9)**.

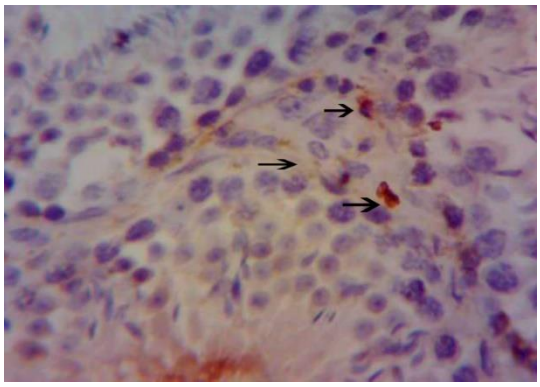

Fig. 8 a Photomicrograph of a section in testis of control mice showing weak immune reactivity of caspase-3 in the cytoplasmic areas of the spermatogonia and Leydig cells (arrows) (Caspase-3 immunostaining, X1000).

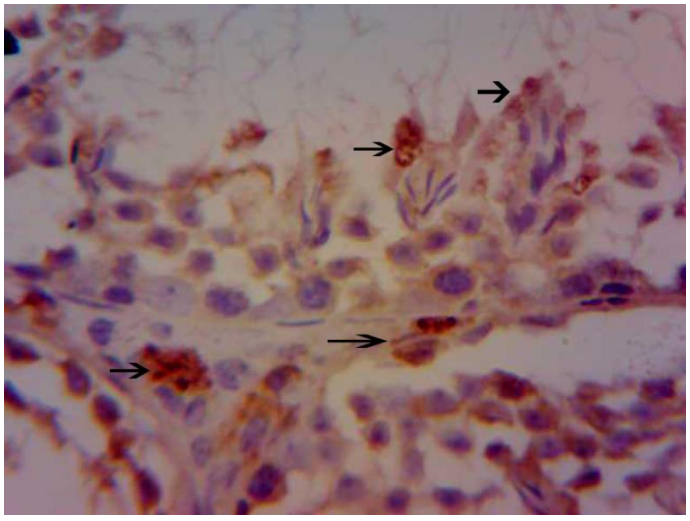

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Fig. 9 a Photomicrograph of a section in testis of MRI-exposed mice showing strong immune expression of caspase-3 in the cytoplasmic areas the of spermatogonia and Leydig cells (arrows) (Caspase-3 immunostaining, X1000).

3. Discussion

Magnetic Resonance Imaging (MRI) is an invaluable tool in both biological research and clinical diagnostics, offering high-resolution, non-invasive imaging of soft tissues, including the brain, muscles, and other organs, without the use of ionizing radiation. This ability to evaluate tissue structure and function has revolutionized the diagnosis of neurological disorders, cancer, and cardiovascular diseases **(Hussain et al., 2022)**. Additionally, MRI has proven to be essential in investigating cellular and molecular mechanisms across various biological systems, aiding in the development of therapeutic strategies **(Bai et al., 2023)**.

The results from the present study corroborate these findings, demonstrating that MRI exposure induces notable structural and cellular changes in the testes, such as a decrease in spermatogenic cells, the presence of apoptotic spermatogonia, and vacuolization in the cytoplasm of seminiferous tubules. Furthermore, several spermatogenic cells exhibited degeneration, including pyknotic nuclei, suggesting that MRI exposure may induce cellular oxidative stress and apoptosis in the germinal epithelium. Consistent with these results, previous studies have raised concerns about the effects of MRI exposure on male reproductive health, particularly in animal models. These studies have shown significant alterations in testicular structure and function, including changes in spermatogenesis **(Zaun et al., 2014; Rostamzadeh et al., 2019)**. Histopathological alterations observed in MRI-exposed testicular tissues, including cellular degeneration and vacuolation within the seminiferous tubules are consistent with these findings **(Rostamzadeh et al., 2019)**.

The testes are particularly sensitive to various stressors, such as heat, radiation, and inflammation, which can result in germ cell death. Exposure to EMFs has been shown to cause significant damage to the seminiferous tubules, reduce Leydig cell numbers, and lower testosterone levels in rodents **(Nassar, 2009; Saygin et al., 2011, Nassar et al, 2020)**. The present study further supports these observations, with evidence of reduced spermatogenic cell numbers and accumulation of spermatozoa, indicative of disrupted spermatogenesis. Similar degenerative changes have been reported in rodent models exposed to EMFs **(Lee et al., 2014; Yilmaz et al., 2025)**, reinforcing the idea that MRI exposure may contribute to cellular oxidative stress and apoptosis in the testes, thereby impairing spermatogenesis.

In the current study, marked collagen fiber accumulation was observed in the testicular tissue of MRI-exposed mice, further suggesting a maladaptive fibrotic response to injury. These results come in context with previous ones; where increasing evidence suggests that prolonged or repeated exposure to MRI may induce additional biological responses in testicular tissues, including increased collagen

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deposition. Collagen deposition is a key feature of fibrosis, which is typically triggered by tissue injury, inflammation, and cellular stress. MRI exposure, particularly through SMFs and RF waves, has been shown to cause oxidative stress, mitochondrial dysfunction, and cellular injury **(Schuermann and Mevissen, 2021).** These stressors can activate several intracellular signaling pathways, including the transforming growth factor-beta (TGF-β) pathway, which is well-known for its role in fibrotic processes **(Wynn and Ramalingam, 2012).** In the testis, TGF-β signaling is likely activated by oxidative stress resulting from MRI exposure, leading to the recruitment of fibroblasts to the site of injury. These fibroblasts then secrete collagen types I and III, contributing to an accumulation of extracellular matrix components in the testicular interstitium and around the seminiferous tubules **(Martinez-Vidal et al., 2021)**. This increased collagen deposition can disrupt the normal architecture of the testis, impairing blood flow and potentially compromising spermatogenesis **(Xu et al., 2024)**. Excessive collagen deposition can lead to tissue stiffness, impaired blood flow, and disrupted testicular architecture **(Martinez-Vidal et al., 2021)**. These factors may ultimately affect testicular function and spermatogenesis by hindering the necessary cellular interactions and nutrient supply within the testes. Therefore, although collagen deposition may initially serve as an attempt at tissue repair, when excessive, it can result in negative consequences for testicular function.

In addition to the fibrotic response, MRI exposure has been linked to increased activity of caspase-3, a critical mediator of apoptosis. Caspase-3 activation occurs in response to oxidative stress, DNA damage, and disruption of cellular homeostasis, all of which can be induced by non-ionizing radiation such as the SMFs and RF waves used in MRI **(López-Martín et al., 2024; Schuermann and Mevissen, 2021)**. In the testes, MRI exposure has been shown to cause damage to both spermatogenic and Leydig cells, leading to the activation of caspase-3 and subsequent impairment of spermatogenesis **(Kim et al., 2014; Lee et al., 2014)**. The present study corroborates these findings, revealing a marked increase in caspase-3 expression in both spermatogenic and Leydig cells in MRI-exposed mice, suggesting a heightened apoptotic response. The activation of caspase-3 leads to the cleavage of vital proteins, compromising cellular integrity and promoting cell death. This results in the loss of both germ cells and Leydig cells, which are critical for testosterone production and the maintenance of normal spermatogenesis **(Morgan et al., 2015)**. These findings underscore the potential for MRI-induced damage to male reproductive health, particularly with repeated or prolonged exposure. The increased caspase-3 activity in testicular tissues following MRI exposure highlights a significant mechanism through which non-ionizing radiation can contribute to cellular damage and disrupted spermatogenesis. One critical aspect of the observed testicular damage is the activation of apoptotic pathways, particularly the role of caspase-3, which plays a central role in mediating cell death **(Murphy and Richburg, 2015, Nassar et al, 2020))**. This suggests that the damage to the germinal

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epithelium, characterized by degeneration of spermatogenic cells and pyknotic nuclei, is likely the result of increased apoptotic activity triggered by MRI exposure

Conclusion: Although MRI remains a critical diagnostic tool, the biological effects on the testes, especially with prolonged exposure, warrant further investigation. Future studies should aim to elucidate the underlying mechanisms of MRI-induced testicular toxicity and explore strategies to mitigate potential risks associated with extended or frequent MRI exposure.

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