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The Impacts of Noise Pollution on Sperm Quality and The Suggestive Mechanisms

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Abstract

Chronic exposure to environmental noise is increasingly recognized as a significant stressor with potential detrimental effects on male reproductive health. Noise-induced stress can impact sperm quality through various mechanisms, including oxidative stress, mitochondrial dysfunction, endocrine disruption, genetic and epigenetic damage, and inflammation. These factors can lead to impaired sperm concentration, motility, morphology, and viability, thereby affecting male fertility. Emerging evidence suggests that noise exposure may also influence the success of assisted reproductive technologies (ART), such as in vitro fertilization (IVF), by reducing fertilization rates and embryo development. This review integrates current findings and highlights research gaps, emphasizing the need for comprehensive studies on the interaction between noise and other environmental stressors.

Keywords: Noise-induced stress, Sperm quality, Oxidative stress, Endocrine disruption

Introduction

There is some evidence that showed a great decline in sperm quality over the last 50 years, although, the controversy with this regard may be region-depended raising alarms regarding the environmental and lifestyle factors contributing to this trend. The data showed that the sperm quality decline continued in 21 century with a growing rate worldwide (1, 2). The evidence revealed that the incidence of acquired and congenital urogenital disorders has been increased partly due to the severe impact of environmental interrupters (3). These environmental interrupters include air pollution (4), unhealthy diets, lifestyles, chemotherapy (5), change in temperature (6), light pollution from devoices such as smart phones (7), microplastic exposure (8), electromagnetic radiation from electronic devices (9), and noise pollution (10). Besides the psychosocial and economic challenges experienced by the men with poor sperm quality, a correlation has been reported between the poor sperm quality and offspring mortality due to congenital malformation (11).

Male fertility, which is fundamentally determined by sperm quality, is increasingly recognized as being susceptible to environmental stressors (12). Sperm quality encompasses several important parameters, including sperm concentration, motility, morphology, and DNA integrity (13). Noise-induced stress is emerging as a potential, yet often overlooked, contributor to diminished sperm health (14).

Environmental noise has become a pervasive and often unavoidable feature of modern life, particularly in urbanized areas where traffic, industrial activities, and social noise levels contribute to chronic exposure. Unwanted anthropogenic noise can be considered as a kind of environmental pollution (15) that influences human life quality and health. WHO has a guideline for the safe level of noise that depends on the situations and times (16). Overall, environmental Protection Agency's (EPA's) has been suggested that the noise level higher than 70 db can be considered as health threatening for public (17). Noise pollution has been reported to reduce weight, brain size and weight as well as neuronal density (18), and induce embryotoxicity (19). It also induces some ultrastructural and molecular changes in organs such as heart (20,21), inner ear (22,23), and adrenal cortex (24). Recent research has started to uncover more subtle, yet significant, consequences of noise exposure in reproductive health.

Noise exposure, especially when it occurs at high or prolonged levels, can induce significant physiological changes that may disrupt normal reproductive functions (25). While much research has focused on the impact of noise on the auditory system, emerging studies suggest that noise stress also affects other systems in the body, including the endocrine (24, 26) and immune systems (27), which directly or indirectly influence sperm health.

This review aims to provide a comprehensive overview of the current understanding of noise-induced stress and its effects on sperm quality. We will focus on the key mechanisms through which noise exposure disrupts male reproductive health, including hormonal imbalances, oxidative stress, inflammation, and genetic damage.

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We will also review the potential long-term and transgenerational consequences of noise on fertility.

By synthesizing the most recent research, we hope to shed light on this emerging area of study and encourage further investigation into the role of noise in male reproductive health.

Mechanisms of Noise Stress on Male Reproductive Health

Noise stress as hormone interrupter

Noise interferes with primary stress hormones produced by adrenal glands including glucocorticoids, adrenocorticosterone, nor-epinephrine hormone (28). The glucocorticoides affects the hypothalamo-pituitary-gonadal axis. At the hypothalamic level, glucocorticoids elevate the production and releasing of gonadotropin-releasing hormone (GnRH). At the pituitary, it diminishes the production and releasing of LH and FSH. At the gonad level, it influence on the hormone production and gametognensis directly. Exposing the pregnant mice to glucocorticoids leads to permanent alternation in the offspring's hypothalamo-pituitary-gonadal axis. The receptor for glucocorticoids was found in the pachytene primary spermatocytes, sertoli cells, and the other male reproductive accessory tissues. Glucocorticoids also inhibit the androgen biosynthesis by Leydig cells and induce leydig cell death. (29). In vitro studies showed that administration of cortisol or corticosterone to mouse female pituitary cell culture inhibited the basal release of LH for the first 28 h of exposure, while at the same time, the production of the basal level of FSH increased. The response of the pituitary cells to GnRH to release LH increased by cortisol (30). In the other study, the daily administration of noise at 90db level (road traffic) for 5-6 hrs to pigeons led to a decrease in serum level of LH and FSH while the serum levels of corticosterone and thyroid stimulating hormone (TSH) increased (31). Despite all these studies, on systematic review concluded that cortisol does not significantly increase in infertile cases (32).

Feedback Mechanisms and Hormonal Imbalance

The interaction between cortisol and testosterone is also influenced by feedback mechanisms within the endocrine system. Cortisol injection has been shown to reduce testosterone level (33). Under normal conditions, the HPG axis maintains a delicate balance between gonadal hormone production and other signals. However, chronic noise-induced stress leads to a persistent elevation in cortisol, which may disrupt this feedback loop (34). Over time, this hormonal imbalance may result in a significant reduction in testosterone levels, exacerbating sperm dysfunction and fertility issues (35).

In addition to testosterone, noise-induced endocrine disruption can affect other reproductive hormones, such as LH and FSH (31). FSH plays a crucial role in regulating Sertoli cells in the testes, which support sperm development. A decrease in FSH levels due to hormonal dys-regulation can impair requirements for spermatogenesis, further compromising sperm health.

Other Hormonal Changes Due to Noise Exposure

In addition to cortisol, noise stress has been linked to changes in other hormones that affect reproductive health. Prolactin, a hormone involved in regulating reproductive function, has also been shown to increase in rats (36) and human (37) in response to stress such as noise exposure (38). Elevated prolactin levels can interfere with the secretion of GnRH, thus contributing to decreased testosterone production and further impairing spermatogenesis (39).

Noise stress also led to a decrease in the serum level of T4, T3, and TSH in rats expose to 60, 80 and 110db noise (40). Thyroid hormones have direct effects on gonad metabolism and it is important for maintaining normal fertility (41). Indirectly, the thyroid hormones also increase the level of hepatic sex hormone-binding globulin (42) that can bind to circulating steroid hormones and regulate the blood testosterone concentration level (43).

Moreover, chronic noise exposure has been associated with changes in the levels of estradiol, a form of estrogen that can influence the HPG axis. It was found that female are more resistance to hearing damage induces by noise (44,45), and eastradiol administration has been suggested to protect the noise induced hearing loss (46). Estradiol is typically found at low levels in males (47), but an imbalance in estrogen signaling due to noise-induced hormonal changes could interfere with the normal reproductive function and affect sperm quality.

Genetic Damage

Recent studies have suggested that environmental stresses such as noise exposure may lead to significant genetic damage in sperm DNA (48), which can severely impact male fertility. The integrity of sperm DNA is essential for successful fertilization and the healthy development of embryos. When sperm DNA is damaged, it can result in reduced fertilization rates, impaired embryo development (49), and an increased risk of pregnancy loss or birth defects (50). DNA fragmentation is one of the most commonly reported noise induces damages (51). DNA fragmentation refers to breaks or disruptions in the sperm's genetic material, and it is a key indicator of sperm quality and potential fertility issues (52).

The relationship between chronic noise exposure and DNA fragmentation is supported by increasing evidence, suggesting that oxidative stress plays a central role in this process (53). Sperm cells, due to their limited DNA repair capabilities, are particularly vulnerable to oxidative damage (54, 55). As a result, noise-induced ROS production can lead to increased DNA fragmentation in sperm, which in turn affects fertilization potential and embryonic development.

Inflammatory Response

Noise-induced stress is not only linked to oxidative damage but also triggers inflammatory responses (56) that can adversely affect the male reproductive system (57). Chronic exposure to high noise levels activates the body's immune system, resulting in the release of pro-inflammatory cytokines and other inflammatory mediators (27). These molecules can disrupt the delicate balance within the

testicular environment, impairing spermatogenesis and ultimately reducing sperm quality (56).

Studies have shown that noise-induced stress leads to elevated levels of pro-inflammatory cytokines, including interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α) in different organs (58,59) including testis (60). These cytokines are key mediators of the body's immune response and play significant roles in regulating inflammation (61). IL-6 deficiency has been demonstrated to elevate sperm and spermatids count, the testicular testosterone and dihydrotestosterone levels (62). IL-6 also declines sperm motility (63), inhibits the production of inhibin by sertoli cells (64), and interrupt the blood-testis barrier (65). Elevated TNF-α level has been also shown to affect sperm quality (66). Both IL-6 and TNF- α have receptors on the germ cells and induce apoptosis (67). TNF-α can also alter the blood-testis barrier, a critical structure that maintains the testicular microenvironment conducive to spermatogenesis (68).

Systemic vs. Local Inflammatory Responses

Noise stress induces systematic inflammation (69). While much of the inflammatory response occurs locally within the testes, systemic inflammation also plays a role in the overall effect of noise-induced stress on male fertility. The systemic inflammation affects other organs and tissues, including the brain, cardiovascular system, and reproductive organs (70). The interplay between systemic and local inflammation creates a feedback loop that exacerbates the negative effects of noise exposure on male reproductive health. Long time untreated inflammation induces DNA damage in spermatogenesis cell linages, and abnormality in the sertoli cells and leydig cells, that lead to reduces sperm quality (71). Besides, systemic inflammation can activate the hypothalamus-pituitary-adrenal (HPA) axis, leading to an increase in cortisol, a stress hormone (72). Elevated cortisol levels, in turn, suppress the production of reproductive hormones, including testosterone, further impairing sperm production (73).

Oxidative stress

Noise stress elevates the ROS production (74, 75) and leads to mitochondria dysfunction (76). One of the reasons for male fertility is oxidative stress. ROS production by dysfunctional mitochondrion reduces sperm motility and induces sperm apoptosis (53). ROS leads to DNA damage, induces mutation (77), and can cause breaks in one strand of the sperm DNA (78). Epigenetic changes have been reported by oxidative stress induction in male. Oxidative stress changes the DNA methylation, histone deacetylation as well as the expression of non-coding RNA that in turn impacts on the cellular gene expression and function. Epigenetic modification influence spermatogenesis and spermiogenesis processes (79).

Noise induce and aging of the mammalian gamete cells

Gamet cells have been reported to undergo age-related changes that decline the reproductive activities. Aging of the sperms influences the sperm quality such as number and motility. One of the aspects of aging is epigenetic changes. The methylation of 5-methyl-cytosine increases in aged sperms, and decrease on long interspersed nuclear elements (LINE) elements. In aged men, the histone metylation also changes in sperms. A reduction in protamine production and misbalance in the protamine P1 and protamine P2 were also reported in aged men (80). Noise pollution can induce epigenetic changes such as alternation in DNA methylation, histone and microRNA expression (81, 82). Changing in the DNA methylation has been demonstrated in several parts of the brain via response changing to stress hormones (83).

Consequences for Sperm Quality

Noise exposure has several negative outcomes for sperm quality including sperm count, motility, morphology and viability:

Sperm Concentration

Sperm concentration, defined as the number of sperm present in a given volume of semen, is a key parameter for assessing male fertility (84). Studies have indicated that chronic noise exposure is associated with a significant reduction in sperm concentration, in human populations (37, 85). A cohort study shows noise stress causes azoospermia (85). This decrease in sperm count is often attributed to a combination of oxidative stress, hormonal imbalances, and disrupted spermatogenesis (86). Excessive ROS leads to cellular damage, including the impairment of the Leydig cells and Sertoli cells. The oxidative damage to these cells can disrupt the normal production of sperm and reduce sperm concentration (87). Sperm cells themselves are also particularly vulnerable to ROS due to the presence of abundance of polyunsaturated fatty acids in their plasma membrane and cytoplasm (86, 87), further contributing to a decrease in sperm numbers.

Sperm Motility

Sperm motility, which refers to the ability of sperm to move efficiently, is a crucial determinant of male fertility. Effective sperm motility is necessary for sperm to reach and fertilize the egg. Reduced sperm motility can significantly impair fertilization and is often associated with male infertility (13). Chronic exposure to high levels of environmental noise has been consistently linked to a significant decline in sperm motility (88). This decline is believed to result from a combination of oxidative stress (89), hormonal dysregulation (37), and cellular damage (90) that affects the function and energy production of sperm.

Sperm Morphology

Sperm morphology, which refers to the size, shape, and structure of sperm, is another crucial parameter of male fertility (91). Abnormalities in sperm morphology can impair sperm function, including its ability to swim effectively and penetrate the egg for fertilization (92). Chronic exposure to high levels of noise has been shown to induce significant morphological defects in sperm (93). These defects are believed to result from oxidative stress-induced damage to the sperm's DNA, membrane, and structural proteins, which compromise sperm function and contribute to reduced fertility (54). Genetic modification that

produces by noise exposure can lead (94) to change in sperm morphology (95).

Sperm Viability

Sperm viability refers to the ability of sperm cells to remain alive and functional, which is essential for successful fertilization (96). Viable sperm must maintain their structural integrity and energy production systems to swim toward and penetrate the egg (97). Reduced sperm viability can significantly impair male fertility, leading to difficulties in conception. A negative correlation has been found between sperm viability and DNA fragmentation (98). Chronic exposure to noise stress has been shown to reduce sperm viability through a variety of mechanisms (14), primarily related to oxidative damage, mitochondrial dysfunction, (99), and membrane destabilization (100).

Role of Noise in Assisted Reproductive Technologies (ART)

A novel area of research that has recently emerged in the field of reproductive health is the potential impact of noise stress on the success of assisted reproductive technologies (ART), particularly procedures like in vitro fertilization (IVF). ARTs, which include IVF, intracytoplasmic sperm injection (ICSI), and egg freezing, have become crucial tools in overcoming infertility, particularly when faced with male factor infertility. However, the success rates of these procedures are influenced by multiple factors, including sperm quality, egg quality, and the environment in which fertilization occurs. The studies have suggested that noise-induced stress may have a significant impact on sperm quality, which, in turn, could influence the outcomes of ART procedures (25).

Impact of Noise on Sperm Quality in ART

The effects of noise exposure on embryo development and implantation rates (102) are also important to consider when examining the role of noise in ART. The embryos derived from noise-exposed sperm may exhibit impaired development, which could lead to a higher incidence of poor-quality embryos, early embryo arrest, or embryo fragmentation. These complications are known to reduce the chances of successful implantation in the uterine lining. However, further research is needed to fully understand the mechanisms through which noise stress impacts sperm quality and ART outcomes, and to evaluate the efficacy of these mitigation strategies.

Awareness of Noise Pollution and Its Link to Infertility Risks

Knowledge of infertility risk factors is crucial, as it empowers individuals to make informed decisions about their health and lifestyle (103, 104). Many factors such as lifestyle, diet, and environmental pollutants can affect the reproductive system (105, 106). Most people may not have enough awareness about the subtle yet significant impact of environmental stressors like on fertility (107). Noise pollution, often overlooked, has been linked to hormonal imbalances, oxidative stress, and reduced sperm quality, including lower sperm count, motility, and morphology (13). By understanding these connections, individuals can take proactive steps to reduce exposure to harmful noise levels, thereby safeguarding their reproductive health. Raising awareness about such risk factors is essential to promote healthier lifestyles and mitigate the growing challenges of infertility in modern society.

Gaps in Current Research

While recent studies have provided valuable insights into the individual effects of noise and other environmental stressors on sperm quality, several research gaps remain:

1. Combined Exposure Studies: There is a lack of longitudinal studies examining the combined effects of noise and multiple environmental stressors on male fertility. Most studies have investigated these stressors in isolation, which limits our understanding of their potential synergistic effects on sperm quality.
2. Molecular Mechanisms: The molecular mechanisms underlying the interactions between noise and other environmental factors are not yet fully understood. Future research should focus on elucidating how noise-induced oxidative stress, hormonal disruption, and inflammation interact with other stressors to impact sperm function at the cellular and molecular levels.
3. Human Studies: While animal studies provide important insights, more human studies are needed to confirm the findings related to noise and environmental stressor interactions. Research should explore how environmental pollutants, lifestyle factors, and psychological stressors interact with noise exposure in real-world settings, particularly in urban and industrial populations.
4. Potential Interventions: Future studies should also explore intervention strategies to mitigate the combined effects of noise and other environmental stressors. For example, antioxidant therapies, lifestyle modifications, and noise reduction strategies could potentially help protect male fertility in populations exposed to multiple environmental stressors.
5. Noise-induced damaged sperms and ART success: As the noise exposure reduces the sperm quality, it is unknown whether using noise-induces sperm damage has significant influence on the ART outcome.

Conclusion

Noise stress significantly impacts male reproductive health through various interrelated mechanisms. It leads to elevated cortisol levels, disrupts the hormonal balance within the hypothalamo-pituitary-gonadal (HPG) axis, that affect the secretion of key reproductive hormones like LH and FSH, and inhibit testosterone production. This hormonal imbalance contributes to reduced sperm quality, characterized by decreased sperm concentration, motility, morphology, and viability.

Additionally, noise exposure induces oxidative stress, leading to increased reactive oxygen species (ROS) production, mitochondrial dysfunction, and sperm DNA fragmentation. The inflammatory responses triggered by

chronic noise exposure further exacerbate these effects, impairing spermatogenesis and overall reproductive health.

Given the substantial evidence linking noise stress to adverse sperm characteristics, it is crucial to recognize and mitigate noise pollution's impact on male fertility. Strategies to limit exposure and protect reproductive health such as using antioxidant could be vital in addressing the rising rates of male infertility. Further research is needed to explore preventive measures and interventions that could alleviate the negative effects of noise on reproductive functions.

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