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A Critical Review of Gadget-Induced Infertility Claims and a Comprehensive Reproductive Etiology Analysis

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Abstract

Infertility represents a massive global public health challenge, affecting approximately 17.5 percent of the adult population worldwide, or approximately 1 in 6 people, with limited variation between high- and low-income regions. This review aims to provide a comprehensive analysis of the etiology of male and female reproductive health and to critically evaluate the validity of popular claims about infertility caused by electronic device use. The methodology used is a critical review of the scientific literature, focusing on mechanistic evidence of thermal and radiation risks, complemented by epidemiological analysis from the Global Burden of Disease (GBD) 2021. The results indicate that the risk of male infertility from laptops on the lap is a measurable clinical fact, as it causes scrotal hyperthermia that impairs spermatogenesis. In contrast, claims regarding mobile phone radiation (RF-EMF) remain inconclusive in the human population, with high uncertainty. The main etiologies of female infertility are dominated by Ovulatory Disorders (especially PCOS) and advanced maternal age. In contrast, male infertility is mainly associated with Varicocele (the most common treatable cause) and idiopathic conditions such as Oligoasthenoteratozoospermia (OAT). In conclusion, infertility is a multifactorial condition, and the main recommendations should focus on thermal risk mitigation as well as first-line metabolic intervention through weight management, considering that obesity is a major metabolic modifier.

Keywords: Infertility, Gadgets, Scrotal Hyperthermia, Varicocele, PCOS.

Introduction

Infertility represents a massive, often overlooked, global public health challenge that requires urgent policy review. The World Health Organization (WHO) defines it as the failure to achieve pregnancy after 12 months or more of regular unprotected intercourse. According to the latest WHO report in 2023, an estimated 17.5 percent of the adult population—or about 1 in 6 people worldwide—experiences infertility during their lifetime[1]. This prevalence shows limited variation between high-income countries (17.8%) and low- and middle-income countries (16.5%) [1], which emphasizes that infertility is a global health challenge that does not discriminate on the basis of socio-economic status. Data from the Global Burden of Disease (GBD) Study 2021 provides a more detailed picture of geographic and temporal variations, particularly in the Asia-Pacific region[3]. This study shows significant and varying trends and burdens of infertility across countries. In East Asia, for example, trends in the burden of male infertility (MI) and female infertility (F1) show a clear contrast. China and Mongolia recorded a steady increase in the burden between 1990 and 2021[5]. In contrast, developed countries such as Japan and South Korea, which may currently have stable ASR (Age-Standardized Rate) levels, are predicted to experience an increasing burden of infertility in the future[5]. Meanwhile, data for Indonesia in 2021 showed a high ASR of 3,332.04 per 100,000, underscoring the importance of this issue in the Southeast Asian region[6].

Analysis of epidemiological data reveals a shift in the peak age of the infertility burden. In developing countries, the infertility burden peaks in the 35-39 age group, while in developed countries (such as Japan and Korea), it peaks at 40-44 years[5]. This difference implicitly reflects the significant impact of socio-economic and lifestyle factors. In challenging socio-economic environments, such as expensive property markets or economic uncertainty (which delays marriage and childbearing)[7], mothers and fathers are increasingly older when they seek treatment. This places significant challenges on health systems in providing effective Assisted Reproductive Technology (ART), given the natural decline in female fertility after age 35[8]. Therefore, policy interventions are needed that structurally support young couples.

Furthermore, the Asia-Pacific study highlighted that the growth rate of secondary infertility difficulty conceiving after a previous pregnancy has outpaced the growth of primary infertility[4]. This increase in secondary infertility indicates an accumulation of risk factors acquired during the reproductive lifespan. These factors may include sexually transmitted infections (e.g., chlamydia or gonorrhea, which damage the fallopian tubes)[9], accumulated exposure to environmental pollutants, or the development of conditions such as endometriosis. This suggests that prevention strategies should include not only sexual health education but also ongoing environmental and reproductive health management after the first pregnancy.

Method

1. Research design

This study employed a systematic literature review design with a descriptive-narrative approach. This design was chosen to collect, analyze, and synthesize the results of recent research addressing the relationship between gadget use and male infertility, as well as the primary etiologies of infertility in both men and women. This approach enabled researchers to identify patterns, evidence gaps, and the scientific robustness of published study results.

2. Setting and samples

Data were collected from various primary sources, including scientific articles, official WHO reports, epidemiological studies, meta-analyses, and relevant literature reviews. The search strategy was conducted through medical and health databases, including PubMed, Google Scholar, and reproductive-related journals, using keywords related to infertility, gadgets (including mobile phones and laptops), and the etiology of male and female infertility. The selected studies covered literature from recent years to the most recent to ensure the validity and relevance of the data.

3. Inclusion and Exclusion Criteria

Inclusion criteria included:

(1) Studies discussing infertility in men and women,

(2) Research examining the effect of gadgets on sperm quality and infertility,

(3) Articles providing epidemiological data, biological mechanisms, and clinical outcomes.

Exclusion criteria include:

(1) Studies that lack empirical data or are opinions without a scientific basis
(2) Articles outside the topic of infertility or non-reproductive research,
(3) Studies with high methodological bias or lacking scientific validity.

4. Quality Assessment and Data Analysis

Study quality was assessed by reviewing research methods, sample size, and the strength of the evidence presented. Data analysis was conducted using a narrative synthesis that included a review of mechanistic, clinical, and epidemiological evidence. Critical analysis focused on the clarity of causal relationships, risk of bias, and inconsistencies in results between studies. Conclusions were drawn based on the weight of evidence and the clinical relevance of gadget-related infertility and other factors.

5. Trustworthiness/rigor

To maintain the trustworthiness of this study, the following steps were taken: Credibility: selection of scientific sources from indexed international journals and the World Health Organization. Transferability: The study results are presented descriptively so they can be applied to clinical and policy contexts in the field of reproductive health. Dependability: The literature search process and selection criteria are explained transparently so they can be replicated. Confirmability: Interpretation of the results is based on objective data from publications, not the researcher's personal opinion.

Results

I. A Critical Review of Gadget Claims: Distinguishing Fact, Myth, and Scientific Nuance

Studies of claims of male infertility resulting from the use of devices, such as cell phones and laptops, show significant differences in scientific strength, particularly between radiation risks and thermal risks.

Cell Phone Radiation (RF-EMF): An Analysis of Inconclusive Evidence

The claim that radiofrequency electromagnetic radiation (RF-EMF) from cell phones can cause male infertility is based on a biological mechanism involving oxidative stress. Exposure to RF-EMF is hypothesized to increase the production of reactive oxygen

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species (ROS), which trigger lipid peroxidation in spermatozoa cell membranes[10]. The resulting oxidative stress can then increase sperm DNA fragmentation and decrease motility[1]. However, systematic reviews and meta-analyses involving human data often encounter contradictory and inconclusive results. A recent meta-analysis showed that the evidence regarding the effects of RF-EMF exposure from mobile phones on sperm yield is very uncertain[13]. This gap arises from significant methodological challenges.

First, it is difficult to accurately measure cell phone RF-EMF exposure in real-life settings (exposure characterization bias)[13]. Second, many clinical studies suffer from selection bias because participants are generally recruited from fertility clinics[14]. Additionally, sperm motility and count can vary naturally from hour to hour or month to month[14]. While some observational studies, such as the one conducted by UNIGE, found that men who used their cell phones more than 20 times a day had a 21 percent higher risk of experiencing a decrease in overall sperm count (from 56.5 million/mL to 44.5 million/mL), the same study found no correlation with sperm motility or morphology[15]. Given this uncertainty, leading clinical organizations, including the American Society for Reproductive Medicine (ASRM), tend to consider the evidence of a correlation between cell phones and infertility as inconclusive in their clinical practice guidelines[16]. This dilemma shows that while in vitro (laboratory) studies and animal studies may show direct sperm cell damage due to EMR [12], the main difficulty lies in proving consistent and significant causal relationships in human populations exposed sporadically in everyday environments.

Laptop on Lap: A Measured Risk of Scrotal Hyperthermia

Unlike the inconclusive risks of cell phone radiation, claims about the dangers of using a laptop in the lap position on male fertility are clinical facts based on a measurable physical mechanism: scrotal hyperthermia. Basic physiological principles suggest that the spermatozoa factory in the testicles should be kept at a temperature 2 to 4 degrees Celsius cooler than core body temperature (ideally around 32–34°C) to ensure optimal spermatogenesis[18]. Exposure to excessive heat can damage proteins and interfere with the process of healthy sperm formation.

Scientific evidence shows a significant increase in temperature due to using a laptop on your lap. One study found that using a laptop on your lap for 60 minutes caused a significant increase in average scrotal temperature, reaching both sides of the testicles[20].

This increase in temperature is caused by a combination of the heat emitted by the device and the effect of body posture which allows the thighs to be close together[20]. Previous research has shown that an increase in scrotal temperature alone is enough to affect healthy sperm production[21]. The observed increases from laptops far exceed this clinically damaging threshold. Therefore, long-term exposure to repeated transient scrotal hyperthermia caused by this modern lifestyle is very likely to negatively impact spermatogenesis, particularly in adolescents and young men [20]. Urologists strongly recommend avoiding the habit of holding a laptop on your lap to protect sperm quality[18]. In addition to thermal risks, it is worth considering that modern laptops placed on the lap often operate on a Wi-Fi connection, which also emits RF-EMF[22]. This condition has the potential to create a synergistic dual stressor effect, where heat damage is exacerbated by the potential oxidative stress from radiation. However, the primary focus should remain on mitigating thermal risk, as this is a measurable variable with proven clinical impact.

Table I

A Summary of Scientific Evidence Regarding Gadget Use and Male Sperm Quality

Gadget Risk Factors	Dominant Primary Mechanism	Impact of Sperm Quality	Clinical Evidence Review (Claim Validity)
Laptop on Lap	Scrotal Hyperthermia (Increased ScT to)[20]	Spermatogenesis Impairment, Decreased Motility, Protein Damage	Fact/True: Strong, mechanistic, and measurable evidence of real clinical risks from local heat
Cell Phones (RF-EMF Radiation)	Oxidative Stress (ROS), Sperm DNA Fragmentation[10]	Decreased Concentration and Motility (especially in vitro)	Inconclusive/Not yet conclusively proven: evidence in humans is highly uncertain; risk of bias is high. More rigorous prospective studies are needed[13]

II. Etiology of Male Infertility: Analysis of Clinical and Genetic Factors

Male factor infertility is a significant contributor, found as the sole cause in 8% of cases and a combined cause in 35% of infertility cases[23].

Varicocele: The Main Treatable Cause

Among the most common and treatable etiologies of male varicocele is varicocele. Varicocele is defined as an enlargement of the veins within the scrotum (pampiniform plexus)[24]. This condition was found to be a potential contributor in 35.4% of men evaluated for infertility[25]. The main pathological mechanism of varicocele is an increase in local temperature in the testicle (scrotal hyperthermia) caused by venous blood pooling[24]. This increase in temperature interferes with sperm production (spermatogenesis) and can affect sperm quality, including motility and morphology.

In-depth clinical studies have shown that the pathophysiology of varicoceles, centered on scrotal hyperthermia, is linked to lifestyle factors such as laptop use on the lap. Men with an anatomical predisposition (varicocele) that causes elevated internal testicular temperature are more likely to experience exacerbated sperm damage if they are also repeatedly exposed to external heat sources such as laptops. This suggests that men with varicoceles should take very strict precautions to avoid heat sources.

Hormonal, Genetic, and Anatomical Dysfunction

Male infertility involves a spectrum of disorders, ranging from hormonal problems that regulate sperm production to genetic defects and anatomical obstructions.

Genetic and Hormonal Factors

Genetic factors play a significant role. A prime example is Klinefelter Syndrome (47, XXY karyotype), which is the most common cause of primary hypogonadism and often manifests as non-obstructive azoospermia or severe oligozoospermia[27].

Hormonal dysfunction can be classified based on the hypothalamic-pituitary-gonadal axis.

1. Primary Hypergonadotropic Hypogonadism: Characterized by low testosterone levels accompanied by increased levels of FSH (Follicle-Stimulating Hormone) and LH (Luteinizing Hormone). This indicates a failure of sperm and testosterone production at the testicular level[27].
2. Secondary Hypogonadotropic Hypogonadism: Characterized by low Testosterone accompanied by normal or low FSH and LH, which indicates a problem with the pituitary gland or hypothalamus (e.g., Kallman Syndrome)[27].

Post-Testicular Problems

Post-testicular etiology involves lesions or obstructions in the seminal ducts, such as ejaculatory duct obstruction, inflammatory infection, or congenital abnormalities such as Congenital Absence of the Vas Deferens (often associated with Cystic Fibrosis)[27]. In addition, functional problems such as erectile dysfunction, retrograde ejaculation, or anejaculation are also pre-testicular or post-testicular causes[27].

Idiopathic Dominance and Diagnostic Role

Most cases of male infertility (around 70%) are categorized as uncorrectable male subfertility manifested through functional sperm defects, such as Oligoasthenoteratozoospermia (OAT) a combination of low sperm count (oligo), poor motility (astheno), and abnormal morphology (terato)[27]. These cases are often considered idiopathic (of unknown cause). Although the diagnosis of OAT often fails with "idiopathic" factors, more sophisticated diagnostic tests such as sperm DNA fragmentation (DFI) testing can provide clues to the etiology. Increased DFI can be caused by chronic oxidative stress, genitourinary infections, or lifestyle factors[29]. Some reversible causes of DFI (such as infections or use of certain antidepressants) can be treated.

It's important to understand that a male infertility evaluation serves more than just reproductive purposes. A comprehensive examination can often serve as a window into health, revealing more serious systemic medical issues. Infertile men are at increased risk of testicular cancer, melanoma, colon cancer, and prostate cancer.[30] Therefore, fertility screening should be promoted as part of preventive health check-ups, especially if there is a history of risk factors such as cryptorchidism.

III. Etiology of Female Infertility: Ovulation Disorders, Endometriosis, and Age

Female factor infertility accounts for approximately 37% of infertility cases as a single cause, with a variety of complex etiologies[23].

Ovulation Disorders

Ovulation disorders are the most common cause of female infertility, accounting for approximately 25% of all cases[23].

Polycystic Ovary Syndrome (PCOS)

PCOS is the most common cause of anovulation or oligo-ovulation, accounting for 80% to 85% of anovulatory patients. PCOS involves a hormonal imbalance that interferes with egg release, and is often closely associated with insulin resistance, obesity, and hyperandrogenism (abnormal hair growth and acne)[9].

Other Hormonal Etiologies

Other hormonal causes include Primary Ovarian Insufficiency (POI), or premature ovarian failure, which occurs when the ovaries stop producing eggs and estrogen levels decrease before the age of 40, often due to genetic factors or an autoimmune response[32]. Excessive production of prolactin by the pituitary gland (hyperprolactinemia) can also reduce estrogen production and cause infertility[32].

Tubes, Pelvic, and Endometriosis Factors

Tubes and Pelvic Infertility

Damage or blockage of the fallopian tubes (tubes infertility) accounts for about 11% of the etiologies[23]. Damaged tubes prevent sperm from reaching the egg or block the journey of a fertilized egg to the uterus[33]. The most common cause of tubes damage is Pelvic Inflammatory Disease (PID), which is often caused by Sexually Transmitted Infections (STIs) such as chlamydia and gonorrhea[9]. Pelvic adhesions, often post-operative or infectious, account for 12% of etiologies[23].

Endometriosis

Endometriosis, which is the growth of endometrial tissue outside the uterine cavity, accounts for approximately 15% of the etiology of female infertility[23]. Endometriosis can cause chronic inflammation, adhesion formation, and distortion of pelvic anatomy, all of which interfere with conception.

Impact of Age and Weight

Age is the single strongest predictor of female fertility. The chance of pregnancy decreases significantly with age, especially after age 35. The chance of conceiving after one year of unprotected intercourse has been reported to decrease from 85% in women under 30 to 44% at age 40[8].

Besides age, weight status has a profound influence through hormonal changes. There is a clear link between PCOS, obesity, and insulin resistance. Obesity doubles the risk of infertility in women compared to those of normal weight[34]. This not only interferes with ovulation but also reduces the success of assisted reproductive programs (ART), such as embryo implantation failure in IVF[34]. Therefore, weight management and insulin resistance through lifestyle interventions should be fundamental treatment steps.

Conversely, being too thin is also dangerous. Low body fat levels can cause the body to produce less progesterone and increase the stress hormone cortisol. These hormonal changes cause the body to focus resources on survival rather than reproduction, which can inhibit ovulation[35]. Finally, the role of mental health is worth emphasizing. Infertility has been significantly linked to increased psychological distress and depression in women. Studies show that women with infertility are several times more likely to experience depression[36]. Although the causal relationship is complex, chronic stress, as indicated by increased cortisol levels, may affect the chances of conception[33]. Therefore, a holistic approach that includes psychological support is an essential component of infertility treatment.

IV. Lifestyle and Environmental Risk Factors (Cross-Gender Impact)

Lifestyle and environmental factors play a significant role as contributors to the etiology of infertility, affecting the male and female reproductive systems through inflammatory and hormonal pathways.

Status Berat Badan (IMT) sebagai Pengubah Metabolik

Extreme Body Mass Index (BMI) status, both obesity and underweight, impairs fertility through complex hormonal and metabolic mechanisms[35]. In men, excess body fat (adipose tissue) acts as an endocrine organ that increases the conversion of testosterone to estrogen, leading to a decrease in functional testosterone levels[34]. These hormonal changes disrupt the process of spermatogenesis, increasing the risk of oligozoospermia and azoospermia[34].

In women, as previously explained, obesity interferes with ovulation and insulin resistance, while extreme underweight diverts the body's energy resources away from reproductive function[35]. The interaction between abnormal weight status, hormonal imbalance, and chronic inflammation is at the root of lifestyle-related infertility[34]. Infertility arising from lifestyle tends to be the result of long-term accumulation of oxidative damage and metabolic disturbances.

Environmental Toxin Exposure

Modern urban and industrial environments carry significant exposure to toxic risks to reproductive health.

Air Pollutants and Noise

A large-scale study in Denmark shows that long-term exposure to air pollution and traffic noise can reduce fertility. Toxic chemicals from air pollution can be inhaled and enter the reproductive tract through the bloodstream, disrupting hormone function and causing direct damage to egg and sperm cells. Noise pollution, although the exact mechanism of its effects is unclear, is thought to increase stress hormones, which indirectly negatively impact fertility. It's important to differentiate between these effects: men may be more susceptible to direct sperm damage from toxins, while women may be more susceptible to hormonal disruption mediated by chronic noise-induced stress[38].

Industrial Chemicals and Toxins

Prolonged exposure to industrial chemicals, pesticides, herbicides, and heavy metals (such as lead) can contribute to low sperm counts and other sperm defects[31]. Endocrine-disrupting chemicals (EDCs) found in the environment, detergents, and cosmetics can also interfere with hormonal signals important for reproduction[33]. In addition, common lifestyle factors, such as tobacco smoking, are associated with decreased fertility in both men and women[31].

V. Evidence-Based Clinical Conclusions and Recommendations

Gadget Evidence Synthesis: The Limits of Fact and Real Risks

A critical review of the relationship between gadget use and male infertility yields nuanced and non-uniform conclusions.

1. Real Risk (Fact): Lap laptop use poses a measurable physical and clinical risk. Significant increases in scrotal temperature (up to) caused by heat and posture can permanently impair spermatogenesis with repeated exposure. Strong clinical recommendations should be made to discontinue this habit[19].
2. Inconclusive Risks: Claims regarding cell phone radiation (RF-EMF) suggest potential harm in vitro (oxidative stress, DNA fragmentation), but in vivo human clinical evidence is weak and inconsistent. While reasonable precautions are recommended, this risk should be positioned as "highly uncertain" and not a primary cause of infertility at this time[13].

Major Etiologies: Focus on Clinical and Lifestyle

Infertility is a multifactorial condition, dominated by clinical etiologies and exacerbated by modern lifestyles. Primary etiologies, such as ovulatory disorders (especially PCOS) in women and varicocele or idiopathic OAT in men, remain a major focus.

Table II

Comparison of the Main Etiologies of Infertility and Women

Etiological Category	Main Causes of Male Infertility (MI)	Main Causes of Female Infertility (FI)	Percentage Contribution (Clinical Estimate)
Anatomical/Structural	Varicocele [25], Post-Testicular Obstruction, Cryptorchidism	Tubes Factor (PID, Adhesions)[33], Endometriosis[23]	Varicocele: 35.4% contributor. FI: Endometriosis (15%), Tubes (11%)[23]
Hormonal/Genetic	Testosterone	Ovulation Disorders (PCOS,	FI: Ovulation Disorders

	Deficiency, Hypogonadism (e.g.,Klinefelter Syndrome)[27]	Hyperprolactinemia)[9], Primary Ovarian Insufficiency	(25%), PCOS accounts for 80–85% of anovulation [23]
Lifestyle/Environment	Obesity [34], Smoking, Toxin Exposure	Advanced Age (>35 years) [8], Obesity/Extreme Weight [35], Chronic Stress, Smoking	Obesity increases the risk of infertility in women[34]

Clinical and Policy Recommendations

Based on a comprehensive analysis, this report recommends the following clinical and preventive measures:

1. Thermal Mitigation Priority: Men should strictly avoid all external heat sources in the scrotal area, including laptops on the lap, hot tubs, and saunas, to protect spermatogenesis from measurable hyperthermia[20].
2. First-Line Metabolic Interventions: Given the central role of obesity in disrupting hormonal balance (both Testosterone in men and ovulation in women, especially related to PCOS), weight management and insulin resistance should be the first-line lifestyle interventions before resorting to expensive ART[34].
3. Reducing Environmental Exposure and Toxins: Public health campaigns should target reducing exposure to air pollutants, pesticides, industrial chemicals, smoking, and alcohol, as these factors contribute to oxidative stress and gamete damage in both sexes[31].
4. Early Diagnostic Access: Given the high global prevalence (1 in 6) and the importance of early detection of major clinical etiologies (Varicocele, PCOS), access to fertility evaluation (including baseline semen analysis and ovulation assessment) should be improved and integrated as part of preventive health care.
5. Holistic Psychological Support: Infertility treatment should include structured psychological support to address comorbid depression and significant psychological distress, especially in female patients[36].

Discussion

This study demonstrates that infertility is a global health problem with a high prevalence that transcends socioeconomic and geographic boundaries. Epidemiological analysis indicates that both men and women face complex, multifactorial risk factors, encompassing clinical, hormonal, genetic, lifestyle, and environmental factors. In the

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context of modern technology, gadget use is of particular concern, particularly regarding the alleged influence of electromagnetic radiation and increased scrotal temperature on male sperm quality. However, based on a literature review, claims of infertility due to mobile phone radiation (RF-EMF) remain inconclusive due to limited clinical evidence in humans, although some in vitro studies have shown increased oxidative stress and sperm DNA fragmentation. Conversely, laptop use on the lap has been clinically proven to increase scrotal temperature above the physiologically safe threshold for spermatogenesis, thus this hyperthermia risk should be recognized as a relevant and real factor. Furthermore, classic etiological factors such as varicocele, ovulatory disorders (especially PCOS), endometriosis, and advanced age remain the main causes of infertility, with stronger clinical implications than technology exposure. Unhealthy lifestyles, such as obesity, smoking, chronic stress, and exposure to environmental toxins, also exacerbate infertility in both sexes. Therefore, a thorough understanding of the interaction between modern biological and environmental factors is key to effectively assessing and managing infertility.

Conclusion

Overall, infertility is a multifactorial condition that cannot be explained by a single cause, such as gadget use, but rather results from a complex interaction of biological, hormonal, genetic, and lifestyle factors. Existing scientific evidence suggests that using a laptop on your lap carries a real risk of decreased sperm quality due to scrotal hyperthermia, while claims of infertility due to cell phone radiation remain unconvincingly proven. Clinical factors such as varicoceles in men and PCOS in women remain the dominant causes, accompanied by significant contributions from obesity, aging, and exposure to toxic environments. Infertility prevention and treatment efforts should focus on healthy lifestyle interventions, early detection of treatable clinical factors, and a holistic approach that includes psychological support. With an evidence-based approach and awareness of modern environmental risks, it is hoped that infertility rates can be reduced and the quality of reproductive health in the community can be sustainably improved.

Ethical Considerations

This study is a literature review and analysis of secondary data from scientific publications, World Health Organization reports, and clinical guidelines. No human or animal subjects were directly involved, and therefore, formal ethics approval was not required. All sources used have been appropriately cited in accordance with principles of academic ethics and scientific integrity.

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Conflict of Interest

No conflict of interest

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