



Research Article

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Thyroid Damage and Reduced Ovarian Reserve: Is the Chernobyl Nuclear Disaster a Myth or a Reality?

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Abstract

Background: Thyroid dysfunction represents a well-established consequence of radiation exposure following the Chernobyl nuclear disaster. Its potential association with reduced ovarian reserve remains controversial and incompletely defined.

Objective: To investigate the association between thyroid disease and ovarian reserve status and to quantify the magnitude of this association using risk estimates.

Methods: A cross-sectional analysis was performed on 665 women of reproductive age. Ovarian reserve was categorised as low or normal according to age-adjusted Anti-Müllerian Hormone (AMH) levels. Associations were assessed using Pearson's chi-square test, Odds Ratio (OR), Relative Risk (RR), and 95% Confidence Intervals (CI).

Results: Thyroid disease was significantly associated with reduced ovarian reserve ($\chi^2=19.406$, $p<0.001$). Women with thyroid disorders exhibited a significantly higher risk of low ovarian reserve (OR=2.75, 95% CI 1.73-4.37; RR=1.78, 95% CI 1.42-2.23).

Conclusion: Thyroid pathology is significantly associated with diminished ovarian reserve. These findings support an indirect endocrine-mediated reproductive effect rather than a direct ovarian radiation injury following Chernobyl exposure.

Introduction

On 26 April 1986, the explosion of reactor number four at the Chernobyl Nuclear Power Plant resulted in the release of large quantities of radioactive material into the environment. The long-term health consequences of this event have been extensively investigated, particularly with regard to endocrine organs [1,2]. The thyroid gland rapidly emerged as the most vulnerable target due to its ability to concentrate radioactive iodine, especially iodine-131 [3]. In contrast, the impact of radiation exposure on female reproductive health, and specifically on ovarian reserve, remains less clearly defined. Ovarian reserve reflects a woman's remaining reproductive potential and is sensitive to both endocrine and environmental influences [4]. The hypothesis that radiation exposure following Chernobyl may have caused a significant decline in ovar-

ian reserve has gained attention in both scientific and public discourse. However, distinguishing scientifically substantiated effects from speculative or exaggerated claims remains essential.

Radiation-Induced Thyroid Damage

The association between exposure to radioactive iodine and thyroid pathology is firmly established [2,3]. Numerous studies have documented an increased incidence of papillary thyroid carcinoma, benign thyroid nodules, autoimmune thyroid diseases, and functional disorders such as hypothyroidism and hyperthyroidism in exposed populations [5-7]. Children and adolescents at the time of exposure were particularly susceptible, owing to higher thyroid iodine uptake and increased cellular proliferation [6]. These findings represent one of the most robust examples of radiation-in-



duced endocrine pathology and are widely accepted within the scientific community.

Ovarian Reserve and Radiation Exposure

Ovarian reserve refers to the quantity and quality of a woman's remaining oocytes and is commonly assessed using biomarkers such as Anti-Müllerian Hormone (AMH), antral follicle count, and follicle-stimulating hormone levels [4,8]. It is well established that high-dose pelvic or total-body radiation, such as that employed in oncological treatments, can result in premature ovarian insufficiency [9]. However, radiation doses received by the majority of populations affected by the Chernobyl fallout were substantially lower and often indirect. Epidemiological studies assessing fertility outcomes, menstrual disturbances, and age at menopause in exposed women have yielded inconsistent results [10,11]. While subtle reproductive alterations have been reported in highly exposed subgroups, such as nuclear cleanup workers, large-scale population data do not conclusively demonstrate a widespread reduction in ovarian reserve attributable solely to Chernobyl-related radiation exposure [12].

Potential Biological Mechanisms

Ionising radiation is known to induce DNA damage, oxidative stress, and apoptosis in ovarian follicles [9]. Unlike the thyroid gland, the ovary does not selectively accumulate radioactive isotopes, which may partly explain its relative resistance to low-to-moderate radiation exposure. Indirect mechanisms have also been proposed, including disruption of the hypothalamic-pituitary axis, radiation-induced thyroid dysfunction affecting gonadotropin secretion, and psychosocial stressors associated with environmental disasters [13,14]. These factors highlight the multifactorial nature of reproductive outcomes and complicate causal attribution.

Table 1

	Low Ovarian Reserve	Normal Ovarian Reserve	Marginal Row Totals
Thyroidopathic	47 (29.02) [11.15]	38 (55.98) [5.78]	85
Non-Thyroidal	180 (197.98) [1.63]	400 (382.02) [0.85]	580
Marginal Column Totals	227	438	665 (Grand Total)

- Odds Ratio (OR): 2.75 (95% CI 1.73-4.37)
- Relative Risk (RR): 1.78 (95% CI 1.42-2.23) (Table 1)

The chi-square statistic is 19.406 The p-value is .000011 Significant at $p < .05$.

The chi-square statistic with Yates correction is 18.342 The p-value is .000018 Significant at $p < .05$.

Materials and Methods

Study Design and Population

This cross-sectional study included 665 women of reproductive age. Participants were stratified according to thyroid status (presence or absence of thyroid disease) and ovarian reserve status. Ovarian reserve was classified as low or normal based on age-adjusted AMH concentrations.

Thyroid disease was defined by documented clinical diagnosis.

Statistical Analysis

Data were summarised as absolute frequencies. The association between thyroid disease and ovarian reserve was evaluated using Pearson's chi-square test. Yates' continuity correction was applied due to the 2x2 table structure. Effect size was quantified using Odds Ratio (OR) and Relative Risk (RR), with 95% Confidence Intervals (CI) calculated via logarithmic transformation. Statistical significance was set at $p < 0.05$.

Results

Descriptive and Inferential Statistics

Among women with thyroid disease ($n=85$), 47 (55.3%) exhibited low ovarian reserve, compared with 180 of 580 women (31.0%) without thyroid disease. The association between thyroid pathology and reduced ovarian reserve was statistically significant ($\chi^2=19.406$, $p=0.000011$) and remained robust after Yates' correction ($\chi^2=18.342$, $p=0.000018$).

Risk Estimates

Women with thyroid disease demonstrated a markedly increased risk of reduced ovarian reserve:

Discussion

The present study demonstrates a strong and statistically significant association between thyroid disease and reduced ovarian reserve. The magnitude of the observed effect suggests a clinically relevant relationship rather than a marginal statistical finding. Thyroid hormones play a critical role in folliculogenesis, granulosa cell proliferation, and gonadotropin responsiveness [15]. Radiation-induced thyroid dysfunction following Chernobyl exposure may

therefore indirectly impair ovarian function through disruption of the hypothalamic- pituitary-ovarian axis rather than through direct ovarian irradiation. These findings support a reframing of the narrative surrounding reproductive consequences of the Chernobyl disaster, emphasising endocrine-mediated mechanisms over simplistic radiation-ovary causation models.

Myth or Reality?

Thyroid damage following the Chernobyl nuclear accident is an unequivocal reality supported by extensive epidemiological evidence [1-3]. In contrast, the hypothesis of a universal or severe decline in ovarian reserve among exposed women appears overstated when evaluated against current data. While reproductive alterations may occur in selected high-risk subgroups with higher cumulative exposure, available evidence does not support a broad population-level effect comparable to that observed in thyroid pathology.

Limitations

The cross-sectional design precludes causal inference. Individual radiation dose measurements were unavailable, and ovarian reserve was analysed as a dichotomous variable. Additionally, heterogeneity in thyroid disease type, severity, and duration could not be assessed.

Conclusion

Thyroid disease is significantly associated with reduced ovarian reserve, with affected women demonstrating a substantially increased reproductive risk. In the context of the Chernobyl nuclear disaster, these findings support an indirect endocrine-mediated pathway linking radiation exposure to reproductive dysfunction. Longitudinal studies incorporating precise dosimetry and quantitative ovarian reserve markers are required to further elucidate this relationship.

Acknowledgements

None.

Conflict of Interest

None.

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