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REVIEW

Thyroid function tests and serum anti-Müllerian hormone in various populations, is there any association? A systematic review and meta-analysis

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Abstract

Purpose This systematic review and meta-analysis aimed to evaluate the association between anti-Müllerian hormone (AMH) as a biomarker of ovarian reserve and various thyroid function tests, including thyroid-stimulating hormone (TSH), free T3 (FT3), free T4 (FT4), T3, T4, and thyroid autoantibodies, such as thyroid peroxidase antibody (TPOAb) and thyroglobulin antibody (TgAb), in various female populations.

Methods A comprehensive literature search was conducted across six electronic databases and original observational studies examining the correlation between AMH levels and at least one TFT, TPOAb, or TgAb.

Results Forty studies with 14,009 participants were included in the quantitative synthesis. The pooled results showed that AMH levels were not significantly correlated with TSH levels in the overall population or diverse subgroups. However, after adjusting for publication bias, a small, but significant, positive correlation was observed. Meta-regression analyses identified estradiol, FT3, and follicle-stimulating hormone (FSH) as significant moderators of AMH-TSH association in various populations. AMH was significantly positively correlated with FT3 (r = 0.177) and FT4 (r = 058), negatively correlated with T3 (r = − 0.202) and T4 (r = − 0.216) in the overall population, and significantly positively correlated with TPOAb in the normal population (r = 0.348). AMH levels were not significantly correlated with TgAb levels. Meta-regression revealed body mass index and FT4 as moderators in AMH-FT4 and AMH-TPOAb correlations.

Conclusion These findings highlight the complex relationship between AMH and thyroid function markers with potential moderators influencing these associations. Further well-controlled longitudinal studies are required to clarify the underlying mechanisms and clinical implications of these associations across reproductive stages and metabolic profiles.

Keywords Anti-Mullerian hormone · Thyroid stimulating hormone · Infertility · Thyroid function tests · Ovarian reserve · Thyroid hormones

Introduction

Anti-Müllerian hormone (AMH), or Müllerian inhibiting substance (MIS), is a dimeric glycoprotein of the transforming growth factor-beta family [1]. Granulosa cells of small antral and preantral growing follicles secrete AMH from fetal life to menopausal, independent of the follicle-stimulating hormone (FSH) [1, 2]. AMH levels decrease by 5–7 pmol/l every three–five years during the reproductive age and become undetectable after menopause [3, 4]. Unlike other hormones, serum AMH levels remain stable throughout menstruation, making it the most reliable biomarker for assessing ovarian reserve compared to inhibin B, FSH, and antral follicle count (AFC) [4, 5].

Thyroid hormones are necessary for the normal functioning of women's reproductive systems, and different components of this system, including the ovaries, contain thyroid hormone receptors [6]. These hormones are crucial during various pregnancy phases, including spermatogenesis, folliculogenesis, fertilization, implantation, fetal development, and placentation [5, 7]. Therefore, thyroid dysfunction is linked to adverse pregnancy outcomes and a risk factor for infertility in women [5, 8]. Epidemiological studies have

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shown that 20% of infertile women have subclinical hypothyroidism; among reproductive-aged women, the prevalence of subclinical and clinical hypothyroidism is 4–10% and 0.1–2%, respectively [9, 10]. In patients with ovarian failure, the prevalence of autoimmune thyroid disease (AITD) is reported to be 10–30%, suggesting an association with ovarian reserve [10]. Additionally, the presence of thyroid autoantibodies in pregnant women increases the risk of miscarriage and preterm delivery [11].

Several studies have investigated the association between thyroid function and serum AMH levels as biomarkers of ovarian reserve, with inconsistent results. For example, a cross-sectional study of infertile women found an inverse association between thyroid-stimulating hormone (TSH) and AMH levels [10], whereas another study found an insignificant association [12]. Another cross-sectional study revealed that serum levels of thyroid peroxidase antibody (TPOAb) were not significantly correlated with ovarian reserve [11].

Few systematic reviews and meta-analyses have investigated the correlation between AMH levels and thyroid function. In a meta-analysis, Hasegawa et al. found that AMH levels were significantly lower in euthyroid adults with AITD; however, this study did not adjust for the effect of age on AMH levels [13]. The association between Hashimoto's thyroiditis (HT) and AMH was analyzed in another study, which only observed a significant reduction in the ovarian reserve in a subgroup of reproductive women [14]. To the best of our knowledge, no systematic review or meta-analysis has been conducted to investigate the correlation between AMH and thyroid function tests (TFTs) and thyroid autoantibodies such as thyroglobulin antibody (TgAb) and TPOAb in infertile women and patients with thyroid disorders. Therefore, this review aimed to evaluate the association between AMH as a biomarker of ovarian reserve and different thyroid tests, including TSH, free T3 (FT3), free T4 (FT4), triiodothyronine (T3), and thyroxine (T4), and thyroid autoantibodies, including TPOAb and TgAb.

Methods

Protocol and registration

This study was conducted according to the Preferred Reporting Items for Systematic Reviews and Meta-analysis (PRISMA) guidelines [15] and was registered in the International Prospective Register of Systematic Reviews (PROSPERO) under registration number CRD42024609166 in 14/11/2024.

Search strategy and study selection

Three independent researchers (N. K., A. K., and S. Gh.) conducted a comprehensive literature search across six electronic databases, including Medline, Embase, Scopus, Web of Science, Cochrane Central Register of Controlled Trials, and ClinicalTrials.gov, covering all records from their inception to May 19, 2024. Additionally, a supplementary manual search was performed of the reference lists of relevant reviews and articles to ensure thorough coverage. The results from Google and Google Scholar were manually reviewed. Endnote 21 software was used to manage the retrieved literature; the detailed search query for this study can be found in Table S1. Original observational studies that evaluated the correlation between AMH levels and at least one TFTs (TSH, FT4, FT3, T4, and T3), TPOAb, or TgAb. Animal studies, case reports, reviews, commentaries, short surveys, non-English studies, and conference papers were also excluded. To exclude irrelevant citations, three authors (N. K., A. K., and S. G.) independently screened the titles and abstracts of all articles and resolved any disagreements through discussion with a third reviewer (M. H.). Subsequently, the full-text records of the selected studies were assessed for eligibility.

Data extraction

Three reviewers (B. D., R. A. B., and E. A. S.) independently extracted data from the selected articles. The data from each study were compiled in Microsoft Excel (Microsoft Excel v.2016), which included the following: (1) study details, including the first author's name, year of publication, country, and study design; (2) population characteristics including sample size, mean age, body mass index (BMI), baseline condition (e.g., overt hypothyroidism, subclinical hypothyroidism, HT, and Polycystic ovary syndrome (PCOS)); (3) laboratory data including TSH, FT4, FT3, T4, T3, TPOAb, TgAb, AMH, prolactin, FSH, Luteinizing hormone (LH), testosterone, progesterone, and estradiol; and (4) correlation coefficients (Pearson's *r* or Spearman's), risk ratios, or odds ratios. The results were documented and verified, and any discrepancies were addressed through a discussion with a third author (M. H.).

Quality assessment

According to a study by Gardani et al., a customized version of Newcastle Ottawa Scale was used to critically evaluate studies (17). The scale was adjusted to match the inclusion criteria to determine the quality of the potentially identified studies. The scale items measuring the "assessment of the

outcome" and "ascertainment of exposure" were eliminated. Second, the scale item controlling for confounding factors was eliminated, because the correlation and association coefficients were the effect size estimates selected for this meta-analysis. This tool, displayed in Table S2, contains two sets of criteria (methodology and analysis). Low quality was denoted by ratings between 0 and 2, moderate quality by ratings between 3 and 5, and high quality by ratings between 6 and 10 (17). Each study was independently reviewed by two authors (M. R. R. and Gh. Gh.), and discrepancies were resolved by a third reviewer (R. A. B.) to ensure consistency in the quality scoring.

Statistical analysis

This meta-analysis considered the correlation coefficient (r) and 95% confidence interval (CI) to be the desired effect size. Cochran's Q-test and I^2 statistics were used to assess heterogeneity among the included studies. An I^2 value > 50% indicated significant heterogeneity. In the presence of significant methodological heterogeneity among the included studies, a random effects model was used, otherwise, a fixed-effects model was used. Publication bias was analyzed using the Egger's and Begg's tests. The non-parametric trim-and-fill method was used to adjust for the publication bias. Subgroup analyses were conducted based on population disease types, and meta-regression was performed to explore sources of heterogeneity when sufficient data were available. In addition, sensitivity analysis using the leave-one-out method was performed. All analyses were performed using STATA software v.17, and a p-value less than 0.05 was considered statistically significant.

Results

Search result and study selection

The initial search yielded 2670 results, and after removing duplicates, 1216 studies were eligible for further screening. After removing studies based on the exclusion criteria, 178 studies remained for the full-text evaluation. Finally, 32 studies were included in the meta-analysis [3, 8–11, 16–43]. Eight additional studies were added from Google and Google Scholar engines [44–51], resulting in a total of 40 studies included in the quantitative synthesis. The detailed study selection process is illustrated in Fig. 1.

Characteristics of the included studies

The baseline characteristics and laboratory data of the included studies are summarized in Tables 1 and S3,

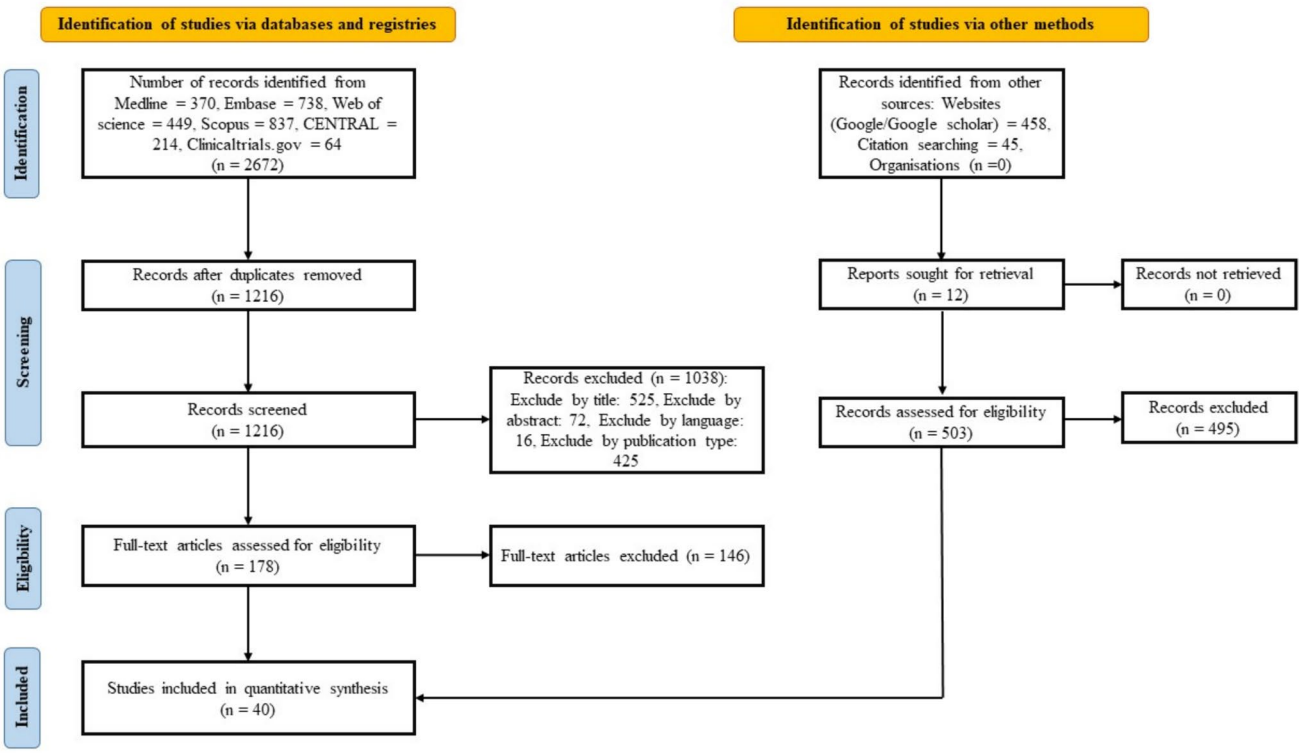

Fig. 1 PRISMA flow diagram of the literature search and selection of studies

Table 1 Baseline characteristics of the included studies

First author, Year	Country	Study design	Population	Sample size, n	Mean age	Main findings	Quality assessment
Aalpona et al. [16], 2023	Bangladesh	Cross-sectional	Women newly diagnosed with PCOS	150	21.5 [18.0–26.0]	There was no significant correlation between AMH and TSH ($p=0.22$)	3
Adamska et al. [17], 2020	Poland	Prospective cross-sectional	Euthyroid women with different PCOS phenotypes	141	26.8 [11, 24–28]	There was a significant correlation between AMH and TPOAb ($p=0.02$)	5
Adamska et al. [18], 2021	Poland	Prospective cross-sectional	Euthyroid Caucasian women with HT	39	26.8 [11, 24–28]	There was a significant correlation between AMH and fT3 in HT women ($p=0.04$) There was no significant correlation between AMH and TSH, fT4, TPOAbs or TgAbs in HT women, nor in the control group (all $p>0.05$)	3
			Euthyroid Caucasian women without HT (control group)	46	26 [11, 25, 26]		
Adamska et al. [19], 2021	Poland	Prospective cross-sectional	Women with papillary thyroid cancer before treatment with radioactive iodine	25	33 [30–37]	There was no significant correlation between AMH and fT4 ($p=0.8$) and fT3 ($p=0.05$)	5
Al-Azzawi et al. [44], 2015	Iraq	Cross-sectional	Infertile women	100	27.55 ± 5.95	There was a non-significant negative correlation between AMH and serum level of TSH ($p=0.898$), T4 ($p=0.232$), T3 ($p=0.229$), and TG ($p=0.095$)	3
Al-Jaff et al. [20], 2018	Iraq	Cross-sectional	Healthy fertile women aged between 26–40 years old (control group)	20	[25–39]	There was a significant negative correlation between AMH and TSH in PCOS ($p=0.003$), hypothyroid ($p=0.001$), and PCOS + hypothyroid groups ($p=0.003$) There was no significant correlation between TSH and AMH in control group ($P=0.087$)	4
			PCOS patients aged between 26–40 years old	20			
			Hypothyroid patients aged between 26–40 years old	20			
			Patients with both hypothyroidism and PCOS aged between 26–40 years old	20			

Table 1 (continued)

First author, Year	Country	Study design	Population	Sample size, n	Mean age	Main findings	Quality assessment
Alwahab et al. [21], 2015	Sudan	Retrospective cross-sectional	Women from the infertility outpatient clinic with different problems of infertility (primary and secondary) with low AMH (< 1.2 ng/ml) and TSH < 2.5 mIU/ml	23	NA	There was a significant negative correlation between normal AMH levels (1.2–3.0 ng /ml) and TSH levels < 2.5 mIU/ml (P = 0.002). Also, there was a significant positive correlation between low AMH levels (< 1.2 ng/ml) and TSH levels < 2.5 mIU/ml (P = 0.003)	3
			Women from the infertility outpatient clinic with different problems of infertility (primary and secondary) with low AMH (< 1.2 ng/ml) and TSH > 2.5 mIU/ml	4	NA		
			Women from the infertility outpatient clinic with different problems of infertility (primary and secondary) with normal AMH (1.2–3.0 ng/ml) and TSH < 2.5 mIU/ml	22	NA		
			Women from the infertility outpatient clinic with different problems of infertility (primary and secondary) with normal AMH (1.2–3.0 ng/ml) and TSH > 2.5 mIU/ml	3	NA		
			Women from the infertility outpatient clinic with different problems of infertility (primary and secondary) with high AMH (> 3.0 ng/ml) and TSH < 2.5 mIU/ml	39	NA		
			Women from the infertility outpatient clinic with different problems of infertility (primary and secondary) with high AMH (> 3.0 ng/ml) and TSH > 2.5 mIU/ml	6	NA		
Azziz et al. [22], 2019	Iraq	Cross-sectional	Women with PCOS	105	31 ± 6.6	There was a positive correlation between serum AMH and TSH in PCOS women (p = 0.001)	3

Table 1 (continued)

First author, Year	Country	Study design	Population	Sample size, n	Mean age	Main findings	Quality assessment
Bahri et al. [23], 2019	Iran	Cohort study	Reproductive age women, aged 20–50 years at the baseline, without any thyroid disease or ovarian dysfunction in the first quartile of age-specific AMH	203	38.3 ± 6.7	There was a significant annual decrease in mean changes of FT4 in all AMH quartiles (all $p < 0.05$). There was a significant annual increase in TPO Ab ($p < 0.05$) and TPOAb positivity ($p < 0.05$) in women of 1st quartile of AMH ($p < 0.05$) but not in other quartiles There was no significant difference in mean changes of TSH in all age-specific AMH quartiles	5
			Reproductive age women, aged 20–50 years at the baseline, without any thyroid disease or ovarian dysfunction in the second quartile of age-specific AMH	181	37.3 ± 6.5		
			Reproductive age women, aged 20–50 years at the baseline, without any thyroid disease or ovarian dysfunction in the third quartile of age-specific AMH	201	36.4 ± 6.8		
			Reproductive age women, aged 20–50 years at the baseline, without any thyroid disease or ovarian dysfunction in the fourth quartile of age-specific AMH	190	37.2 ± 6.7		
Bansal et al. [24], 2020	India	Prospective cohort	Females aged ≥ 25 years who presented with acne (PCOS group)	31	27.74 ± 4.86	There was no significant correlation between AMH and TSH in any of the groups (all $P > 0.05$)	5
			Females aged ≥ 25 years who presented with acne (non-PCOS group)	89	30.43 ± 4.95		
			Females aged ≥ 25 years who presented with acne (PCOS and non-PCOS groups)	120	29.73 ± 5.04		
Battikhi et al. [45], 2018	Canada	Prospective cross-sectional	Infertile women	36	24.50 ± 19.64	There was a Significant negative correlation between AMH and TSH ($p < 0.004$) There was no significant correlation between AMH and FT4 ($p = 0.32$)	6

Table 1 (continued)

First author, Year	Country	Study design	Population	Sample size, n	Mean age	Main findings	Quality assessment
Chen et al. [11], 2017	Taiwan	Cross-sectional	Chinese women (total population)	1044	36.01 ± 2.46	There was no significant correlation between AMH and TPOAb, whether for the whole study population (p = 0.685) or for patients without known cause compromising ovarian reserve (p = 0.413)	5
			Patients without known causes compromising ovarian reserve	933	35.97 ± 4.11		
Crawford et al. [25], 2017	USA	Cohort study	English-speaking women between 30–44 years of age, who were attempting to conceive for 3 months or less	99	33.3 ± 3	There was no significant correlation between AMH and TSH, T3, T4, or FT4 (p ≥ 0.05)	9
Das et al. [43], 2024	India	Cross-sectional	Patients aged 18–38 years old (reproductive age) with PCOS	20	24.50 ± 3.75	There was no significant correlation between AMH and TSH, T3, T4, or FT4 (all P > 0.05)	4
Demirci et al. [46], 2020	Turkey	Cross-sectional	Euthyroid and subclinical hypothyroid women, between the ages of 20–45, who were consulted to the endocrinology clinic for fertility evaluation	198	29.36 ± 6.43	There was no significant correlation between AMH and TSH (p = 0.608)	3
Giusti et al. [47], 2018	Italy	Cross-sectional	Pre-menopausal women, aged over 18 years, who were not on hormonal contraception and who had a history of differentiated thyroid cancer and previously undergone radioactive iodine ablative treatment	34	40.7 ± 6.7	There was no significant correlation between AMH and TSH (p = 0.91) and FT4 (p = 0.27) in the radioactive iodine group There was no significant correlation between AMH and TSH (p = 0.91) and FT4 (p = 0.60) in the control group	5
			Pre-menopausal women, aged over 18 years, who were not on hormonal contraception and who had a history of differentiated thyroid cancer without radioactive iodine ablative treatment (control group)	23	41.6 ± 7.4		
Giusti et al. [67], 2022	Italy	Retrospective cross-sectional	Women off or on L-T4 treatment for subclinical hypothyroidism (all participants)	250	34.1 ± 9.9	There was no significant correlation between AMH and TSH and, FT4 in any of the groups	5
			Sub-clinical hypothyroid women with normal thyroid function	171	31.5 ± 9.2		
			Sub-clinical hypothyroid women on L-T4 therapy	79	39.7 ± 9.5		

Table 1 (continued)

First author, Year	Country	Study design	Population	Sample size, n	Mean age	Main findings	Quality assessment
Halici et al. [26], 2023	Turkey	Cross-sectional	Women with a history of infertility for over a year, aged between 20 and 45 years old, and with a BMI between 18 kg/m^2 and 30 kg/m^2	1396	36.79 (median)	A correlation between TSH and AMH was found. Both high TSH and low TSH levels result in a low AMH level; thus, the association between TSH and AMH is not linear ($p=0.625$). This correlation was best described in a 2nd degree polynomial regression model	4
Hamilton et al. [48], 2016	Australia	Retrospective cohort	Women who attended for fertility treatment were recruited based on a positive pregnancy blood test (serum hCG > 25 IU/mL)	85	35.4 ± 0.5	There was a negative correlation between AMH and TSH ($p=0.001$) during gestation weeks 4 to 6.5 There was a negative correlation between AMH and TSH ($p=0.000$) and fT3 ($p=0.026$), When accounting for cycle types There was a negative correlation between AMH and TSH ($p<0.005$) and fT3 ($p<0.005$), when accounting for cycle type and gestation week combined	7
Kabodmehri et al. [68], 2021	Iran	Prospective cross-sectional	Women with infertility due to various causes (all participants) Women with infertility due to various causes with AMH < 1.1 (ng/ml) Women with infertility due to various causes with AMH ≥ 1.1 (ng/ml)	314 142 172	36.66 ± 6.12	In multivariate logistic regression, TSH was significantly associated with low AMH, so that with one unit increase in TSH level, the odds of having AMH < 1.1 ng/ml increases by 1.25 times or by 25% ($P=0.017$) In the partial correlation test, there was no significant correlation between AMH and TSH and FT4 in any of the study groups (all $p>0.05$)	4
Kuroda et al. [52], 2015	Japan	Case–control	Infertile patients after matching by age and BMI Total cohort of infertile patients	23 67	33.7 ± 3.4 35.0 ± 3.2	There was an inverse correlation between AMH and TSH whether in post-matched group ($p=0.036$) or in total infertile patients ($p=0.002$)	5

Table 1 (continued)

First author, Year	Country	Study design	Population	Sample size, n	Mean age	Main findings	Quality assessment
Lee et al. [27], 2022	Korea	Cross-sectional	Patients with previously diagnosed PCOS (total population)	233	25.34 ± 4.87	There was no significant correlation between AMH and TSH in any of the groups (total p = 0.368, HA-IM-PCO p = 0.337, HA-IM p = 0.206, HA-PCO p = 0.516, and IM-PCO p = 0.812)	3
			PCOS patients with hyperandrogenism, irregular menstruation, and polycystic ovaries	75	24.79 ± 4.73		
			PCOS patients with hyperandrogenism and irregular menstruation	60	24.62 ± 4.36		
			PCOS patients with hyperandrogenism and polycystic ovaries	4	21.00 ± 1.41		
			PCOS patients with irregular menstruation and polycystic ovaries	94	26.44 ± 5.17		
Li et al. [8], 2022	China	Retrospective cross-sectional	Infertile women aged 20–40 years with normal thyroid function before treatment	3501	32.63 ± 3.83	There was a significant negative correlation between AMH and TSH (p = 0.003)	5
Mittica et al. [28], 2020	Italy	Cross-sectional	Premenopausal women aged over 18 years with a history of differentiated thyroid cancer, who are not on hormonal contraception and had previously undergone radioactive iodine ablative treatment	59	41.2 ± 7.5	There was no significant correlation between AMH and TSH and FT4 in any of the groups (all p > 0.05)	4
			Premenopausal women aged over 18 years with a history of differentiated thyroid cancer, who are not on hormonal contraception and had not previously undergone radioactive iodine ablative treatment	30	42.4 ± 9.2		
			Premenopausal women aged over 18 years with normal menstrual cycles and without a history of neck surgery or radiation	141	33.1 ± 10.1		

Table 1 (continued)

First author, Year	Country	Study design	Population	Sample size, n	Mean age	Main findings	Quality assessment
Mohaisen et al. [29], 2019	Iraq	Cross-sectional	Females with PCOS belonging to age groups of (18–35) years (controls)	40	23.82 ± 4.27	There were non-significant negative correlations between AMH and TSH, T3, and T4 (all $p > 0.05$) in all three groups	4
			Females with PCOS and hyperthyroidism belonging to age groups of (18–35) years	40	33.02 ± 5.35		
			Females with PCOS and hypothyroidism belonging to age groups of (18–35) years	40	36.19 ± 4.94		
Osuka et al. [30], 2018	Japan	Retrospective-cohort	All participants either positive or negative for TPOAb and TgAb, aged < 40 years and with regular menstrual cycles (25–35 days)	203	NA	There was no significant correlation between serum AMH and TSH levels ($p = 0.664$), TgAb levels ($p = 0.2087$), or TPOAb levels ($p = 0.504$) of all participants	3
Pullaiah et al. [32], 2022	India	Cross-sectional	Newly diagnosed HT patients with Overt Hypothyroid	60	37 [30.75–39.25]	There was no significant correlation between the AMH values and TPOAb, FT4, and TSH in any of the three groups (all $p > 0.05$). Linear regression analysis in total Hashimoto's Thyroiditis cases showed AMH levels were not significantly correlated with TSH, TPOAb, and FT4 (all $p > 0.05$)	3
			Newly diagnosed HT patients with Subclinical Hypothyroid	60	35 [30.7–37.25]		
			Healthy euthyroid people (control group)	60	37 [33.75–39]		
Safarian et al. [34], 2023	Russia	Cross-sectional	Non-pregnant euthyroid women with infertility and verified autoimmune thyroiditis positive for anti-TPO	45	35 [30–36]	There was a negative correlation between follicular fluid AT-TPO and serum AMH ($p = 0.017$)	3
			Women with infertility without any thyroid disorder and negative for thyroid autoimmunity	45	33 [30–35]		
Saglam et al. [49], 2015	Turkey	Case–control	Women younger than 40 years old with or without autoimmune thyroid disease	165	35.19 ± 2.80	AMH was not significantly affected by TSH ($p = 0.859$)	5

Table 1 (continued)

First author, Year	Country	Study design	Population	Sample size, n	Mean age	Main findings	Quality assessment
Sammour et al. [51], 2017	Egypt	Case–control	Infertile women at reproductive age (20–35) years + normal fertile women aged (20– 35) years. (All participants)	128	26.45 ± 4.15	There was a significantly positive correlaton between AMH and TSH ($p < 0.0001$) and FT3 ($p < 0.0001$) in the control group. There was no significant correlation between AMH and TSH ($p = 0.378$) and FT3 ($p = 0.968$) in the case group. There was no significant correlation between AMH and FT3 ($p = 0.450$) in all participants. There was a significant positive correlation between AMH and TSH ($p = 0.007$) in all participants	6
			Infertile women at reproductive age (20–35) years	64	25.4 ± 4.5		
			Normal fertile women aged (20–35) years (control group)	64	27.5 ± 3.5		
Serin et al. [35], 2021	Turkey	Cross-sectional	PCOS married patients, aged 18–35	46	26.4 ± 3.8	In the PCOS + HT group, there was a significant negative correlation between AMH levels and TPOAb ($p = 0.047$) but not with TgAb ($p = 0.728$). In PCOS group, there was no significant correlation between AMH levels and TPOAb ($p = 0.804$) and TgAb levels ($p = 0.332$)	4
			PCOS married patients, aged 18–35 with HT	46	27.7 ± 3.2		
Sharma et al. [36], 2019	India	Cross-sectional	Women aged between 18 and 45 years attending the gynecology clinic who fulfilled the Rotterdam criteria for PCOS	45	24.1 ± 4.7	There were no significant correlation between AMH and TSH neither in PCOS patients ($p = 0.326$) nor in the control group ($p = 0.821$)	3
			Normo-ovulatory women between 18 and 45 years old (control group)	45	25.2 ± 4.6		
Shima et al. [37], 2023	Bangladesh	Cross-sectional	Hypothyroid infertile women aged 20–35 years old who had TSH level > 2.5 mIU/L with normal/low free T4	167	NA	There was a significant negative correlation between AMH and TSH ($p = 0.024$), TPOAb ($p = 0.011$), and TgAb ($p = 0.018$)	3

Table 1 (continued)

First author, Year	Country	Study design	Population	Sample size, n	Mean age	Main findings	Quality assessment
Soam et al. [38], 2022	India	Cross-sectional	Infertile women aged 20–40	78	30.59 ± 4.67	There was a significant negative correlation between AMH and TSH (p = 0.002). On bivariate regression analysis, it was concluded that with a unit rise in serum TSH value, the odds of having AMH value of less than 1 ng/mL increases by 61% (adjusted OR: 1.61 (1.17–2.21)	3
Swadi et al. [39], 2023	Iraq	Cross-sectional	Fifty subfertile couples from the intra-cytoplasmic sperm injection attenders	100	31.52 ± 5.91	There was a significant positive correlation between follicular fluid FT4 and AMH levels (p = 0.02), but there was not a significant correlation between follicular fluid FT3 and AMH levels (p = 0.05)	3
Tuten et al. [40], 2014	Turkey	Cross-sectional	Premenopausal women with HT aged between 18 and 45 with regular menstrual cycles	32	34.93 ± 8.87	There were significant positive correlations between AMH and anti-TPO (p = 0.010) and anti-TG (p = 0.001); but there was no significant correlation between AMH and TSH (p = 0.634), FT3 (p = 0.665), and FT4 (p = 0.748)	3
Ünsal et al. [31], 2021	Turkey	Retrospective case–control	Euthyroid HT patients, aged between 18 to 40, with regular menstrual cycles of between 25 and 35 days, both those using and not using levothyroxine	108	32 [27.3 – 38]	There was no significant correlation between AMH and TSH (p = 0.7), fT4 (p = 0.796), TPOAb (p = 0.173), and TgAb levels (p = 0.471)	5
Vidales et al. [50], 2016	Spain	Prospective Cohort	Women between the ages of 29 and 41 scheduled for In vitro fertilisation treatment using the GnRH-antagonist protocol	46	37.04 ± 3.1	There was no significant correlation between AMH and TSH and FT4 (all p > 0.05)	5
Wu et al. [41], 2021	China	Cross-sectional	Chinese infertile women with normal sex life without contraception and have not been pregnant for more than 12 months	496	30.31 ± 4.49	There was no significant correlation between AMH and TSH (p = 0.50), FT3 (p = 0.18), and FT4 (p = 0.44)	3

Table 1 (continued)

First author, Year	Country	Study design	Population	Sample size, n	Mean age	Main findings	Quality assessment
Zynat et al. [42], 2023	China	Cross-sectional	Women between 18 and 45 years old who had their TPOAb, TgAb, TSH, FT4, and AMH levels measures on the same day	483	33.77 ± 7.47	There was a significant negative correlation between AMH and TgAb (p = 0.013). There was no significant correlation between AMH and TSH, FT3, FT4, and TPOAb. In multivariate logistic regression, TgAb was significantly associated with low AMH (p = 0.032), but not with TSH and TPOAb	3

AMH Anti-Müllerian Hormone; *TSH* Thyroid-Stimulating Hormone; *PCOS* Polycystic Ovary Syndrome; *TPOAb* Thyroid Peroxidase Antibodies; *FT3* free T3; *FT4* free T4; *HT* Hashimoto thyroiditis; *TgAb* Thyroglobulin antibodies; *NA* not addressed; *L-T4* levothyroxine; *HCG* Human chorionic gonadotropin; *BMI* body mass index

respectively. Among the 40 studies included in this analysis, the majority were cross-sectional (n = 30) [3, 8, 10, 11, 16–22, 26–29, 32–47], followed by 6 cohort studies [23–25, 30, 48, 50] and 4 case–control studies [31, 49, 51, 52]. The publication year among the studies spanned from 2014 to 2024, with sample sizes ranging from 20 to 3,501 participants and a total population of 14,009 in the final analysis. The mean age of the participants varied from 21.0 to 42.4 years, and the population consisted of several groups of healthy women, women with infertility, PCOS, HT, papillary thyroid cancer, hypothyroidism, subclinical hypothyroidism, and premenopausal women. Study quality assessments yielded scores ranging from 3 to 9, with the majority of studies (n = 36) scoring between 3 and 5, indicating a moderate quality. Notably, four studies were rated as high-quality [25, 45, 48, 51].

AMH and TSH

The pooled results showed that AMH levels were not significantly correlated with TSH levels (r = 0.104; 95% CI: − 0.027, 0.232) using the random-effects model, with considerable heterogeneity among the included studies (I^2:97.02; p < 0.001) (Table 2). We found evidence of publication bias according to the results of Begg's test (p = 0.0025) but not Egger's test (p = 0.468). Therefore, we applied the random-effects trim-and-fill method to adjust for publication bias and observed considerable differences in the overall results (r = 0.188, 95% CI: 0.168, 0.209). Subgroup analysis based on disease type revealed no significant difference in the correlation between AMH and TSH levels between the groups (p = 0.132) (Table 2 and Figures S1–S6). According to the meta-regression analysis, estradiol level (pmol/dl) was a significant factor affecting the correlation between AMH and TSH in the total population (beta (SE) = 0.003 (0.001), p = 0.006) and in PCOs patients (beta (SE) = 0.047 (0.022), p = 0.036) (Table 3). Furthermore, the FT3 level (pmol/dl) was found to be a significant factor influencing the correlation between AMH and TSH in the overall population (beta (SE) = − 0.194 (0.060), p = 0.001) (Table 3). In the healthy population, FSH level (MIU/dl) was a significant factor influencing the correlation between AMH and TSH (beta (SE) = 0.061 (0.027), p = 0.026) (Table 3). Sensitivity analysis showed no considerable changes in the subgroup or overall results.

AMH and FT3

As shown in Table 4 and Figure S7, AMH levels were significantly correlated with FT3 levels (r = 0.177; 95% CI: 0.058–0.290) using the random-effects model with considerable heterogeneity among the included studies (I^2:76.73; p < 0.001). There was no evidence of publication

Table 2 Pooled correlation between AMH and TSH in the overall population and sub-grouped by disease type

AMH and TSH (number of the effect sizes)	Correlation Coefficient	95% CI		I^2%	P-value (for heterogeneity)
		Lower	Upper		
PCOS (13)	0.311	– 0.080	0.619	97.18	< 0.001*
Infertile (16)	0.061	– 0.221	0.334	98.57	< 0.001*
Hypothyroidism (4)	0.032	– 0.260	0.318	64.59	< 0.001*
Subclinical Hypothyroidism (4)	0.008	– 0.075	0.090	47.87	0.124
Hashimoto thyroiditis (3)	0.021	– 0.126	0.167	0	0.965
Normal (9)	0.006	– 0.216	0.228	90.38	< 0.001*
Overall (53)	0.104	– 0.027	0.232	97.02	< 0.001*

AMH Anti-Müllerian hormone; *TSH* thyroid-stimulating hormone; *CI* confidence interval; *PCOS* polycystic ovary syndrome

*p-value less than 0.05 considered as statistically significant

Table 3 Meta-regression analysis examines the potential moderators of the pooled correlation between AMH and TSH in the overall population and according to the sub-groups by disease type

Potential Moderators	Overall			PCOs			Infertility			Healthy		
	Beta	SE	p-value	Beta	SE	p-value	Beta	SE	p-value	Beta	SE	p-value
Age (year)	– 0.014	0.015	0.351	– 0.016	0.048	0.740	0.048	0.029	0.097	– 0.039	0.028	0.159
BMI (kg/m^2)	– 0.004	0.023	0.865	– 0.029	0.060	0.621	– 0.002	0.022	0.938	– 0.062	0.100	0.538
Prolactin (ng/ml)	0.012	0.019	0.548	0.011	0.030	0.703				– 0.272	0.323	0.400
FSH (MIU/ml)	0.025	0.039	0.508	0.125	0.179	0.482				0.061	0.027	0.026*
LH (MIU/ml)	0.045	0.028	0.106	– 0.044	0.110	0.687				0.080	0.044	0.068
Testosterone (ng/ml)	0.563	1.05	0.592	– 1.42	2.33	0.542						
Progesterone (ng/ml)	– 0.029	0.047	0.534	– 2.30	1.83	0.209						
Estradiol (pmol/dl)	0.003	0.001	0.006*	0.047	0.022	0.036*						
FT3 (pmol/l)	– 0.194	0.060	0.001*									
T3 (nmol/l)	0.017	0.036	0.632									
FT4 (pmol/l)	0.003	0.014	0.834									
T4 (nmol/l)	– 0.001	0.002	0.533									

AMH Anti-Müllerian hormone; *TSH* thyroid-stimulating hormone; *PCOS* polycystic ovary syndrome; *SE* standard error; *BMI* body mass index; *FSH* follicle-stimulating hormone; *LH* luteinizing hormone; *FT3* free triiodothyronine; *FT4* Free Thyroxine

*p-value less than 0.05 considered as statistically significant

Table 4 Pooled correlation between AMH and FT3 in the overall population and sub-grouped by disease type

AMH and FT3 (number of the effect sizes)	Correlation	95% CI		I^2%	p-value (for heterogeneity)
		Lower	Upper		
Infertile (3)	0.086	0.009	0.161	49.46	0.138
Overall (11)	0.177	0.058	0.290	76.73	< 0.001*

AMH Anti-Müllerian hormone; *FT3* free triiodothyronine; *CI* confidence interval

*p-value less than 0.05 considered as statistically significant

bias according to the results of Begg's test (p = 0.051) and Egger's test (p = 0.192). Subgroup analysis based on disease type revealed no significant difference in the correlation of AMH and FT3 levels between the groups (p = 0.083) (Table 4 and Figure S8). Similar results were observed in the infertile patient subgroup without considerable heterogeneity (r = 0.086; 95% CI: 0.009, 0.161–I^2:49.46; p = 0.138). No significant factors influenced the correlation between AMH and FT3 levels in the total population (p > 0.05) (Table 5). Sensitivity analysis showed no significant changes in the results (Figure S9).

AMH and T3

The fixed-effects model showed that AMH level was negatively correlated with T3 level (r = – 0.202; 95% CI: – 0.303, – 0.097), with non-significant heterogeneity among the included studies (I^2:22.69; p = 0.263) (Figure S10). There was no evidence of publication bias according to the results of the Begg (p = 0.840) or Egger tests (p = 0.613).

Table 5 Meta-regression analysis examines the potential moderators of the pooled correlation between AMH and FT3 in the overall population and according to the sub-groups by disease type

Potential Moderators	Overall		
	Beta	SE	p-value
Age (years)	– 0.013	0.018	0.457
BMI (kg/m^2)	0.038	0.040	0.339
FSH (MIU/ml)	– 0.065	0.038	0.085
LH (MIU/ml)	0.097	0.058	0.092

AMH Anti-Müllerian hormone; FT3 free triiodothyronine; *SE* standard error; *BMI* Body Mass Index; *FSH* follicle-stimulating hormone; *LH* luteinizing hormone

*p-value less than 0.05 considered as statistically significant

The sensitivity analysis showed no significant changes in the results (Figure S11).

AMH and FT4

According to the fixed-effects model, AMH levels were positively correlated with FT4 levels (r = 0.058; 95% CI: 0.013–0.103), with non-significant heterogeneity among the included studies (I^2:36.41; p = 0.168) (Table 6 and Figure S12). There was no evidence of publication bias according to the results of Begg's test (p = 0.254) and Egger's test (p = 0.316). The sensitivity analysis showed no considerable changes in the results. Subgroup analysis based on the disease types revealed no significant difference in the correlation of AMH and FT4 between groups (p = 0.116) (Table 7 and Figure S13-15). BMI was a significant factor influencing the correlation between AMH and FT4 levels (p > 0.05) (beta (SE): 0.047(0.015): p = 0.003). The sensitivity analysis showed no considerable changes in the results (Figure S16).

AMH and T4

The fixed effects model showed that AMH levels were negatively correlated with T4 levels (r = − 0.216; 95% CI: − 0.322, − 0.105), with non-significant heterogeneity among the included studies (I^2:15.17%; p = 0.442) (Figure S17). There was no evidence of publication bias according to the results of the Begg test (p = 0.312) or Egger test (p = 0.194). The sensitivity analysis showed no considerable changes in the results (Figure S18).

Table 7 Meta-regression analysis examines the potential moderators of the pooled correlation between AMH and FT4 in the overall population and according to the sub-groups by disease type

Potential Moderators	Overall		
	Beta	SE	p-value
Age (year)	0.009	0.007	0.178
BMI (kg/m^2)	0.047	0.015	0.003*
Prolactin (ng/ml)	– 0.018	0.011	0.082
FSH (MIU/ml)	0.008	0.012	0.539
LH (MIU/ml)	– 0.044	0.017	0.011
Progesterone (ng/ml)	0.018	0.017	0.300

AMH Anti-Müllerian hormone; *FT4* Free thyroxine; *SE* standard error; *BMI* body mass index; *FSH* follicle-stimulating hormone; *LH* luteinizing hormone

*p-value less than 0.05 considered as statistically significant

AMH and TPOAb

As shown in Table 8 and Figure S19, AMH levels were not significantly correlated with TPOAb levels (r = − 0.046; 95% CI: − 0.116–0.206), with considerable heterogeneity among the included studies (I^2:88.44%; p < 0.001). There was no evidence of publication bias according to the results of the Begg test (p = 0.367) or Egger test (p = 0.735). Subgroup analysis based on disease type revealed a significant correlation between AMH and TPOAb levels in the normal population (r = 0.348; 95% CI: 0.068, 0.577), with significant heterogeneity among studies (I^2:79.35; p = 0.008) (Table 8 and Figure S20-21). The sensitivity analysis showed no considerable changes in the results (Figure S22). In the meta-regression analysis, the FT4 level (pmol/L) was a significant factor influencing the correlation between AMH and TPOAb

Table 6 Pooled correlation between AMH and FT4 in the overall population and sub-grouped by disease type

AMH and FT4 (number of the effect sizes)	Correlation	95% CI		I^2%	p-value (for heterogeneity)
		Lower	Upper		
Infertile (4)	0.035	– 0.184	0.252	78.48	0.003*
Hashimoto thyroiditis (3)	– 0.018	– 0.165	0.129	0	0.822
Normal (3)	– 0.032	– 0.169	0.105	0	0.817
Overall (18)	0.058	0.013	0.103	36.41	0.168

AMH Anti-Müllerian hormone; *FT4* free thyroxine; *CI* confidence interval

*p-value less than 0.05 considered as statistically significant

Table 8 Pooled correlation between AMH and TPOAb in the overall population and sub-grouped by disease type

AMH and TPOAb (number of the effect sizes)	Correlation	95% CI		I^2%	p-value (for heterogeneity)
		Lower	Upper		
Hashimoto thyroiditis (3)	0.056	− 0.288	0.386	72.18	0.028*
Normal (3)	0.348	0.068	0.577	79.35	0.008*
Overall (12)	0.046	− 0.116	0.206	88.44	< 0.001*

AMH Anti-Müllerian hormone; *TPOAb* Thyroid peroxidase antibodies; *CI* Confidence interval

*p-value less than 0.05 considered as statistically significant

Table 9 Meta-regression analysis examines the potential moderators of the pooled correlation between AMH and TPOAb in the overall population and according to the sub-groups by disease type

Potential Moderators	Overall		
	Beta	SE	p-value
Age (years)	− 0.014	0.012	0.260
BMI (kg/m^2)	− 0.011	0.031	0.723
Prolactin (ng/ml)	0.094	0.145	0.515
FSH (MIU/ml)	0.030	0.038	0.428
LH (MIU/ml)	0.024	0.023	0.288
Estradiol (pmol/dl)	0.002	0.001	0.075
FT4 (pmol/L)	0.036	0.016	0.020*

AMH Anti-Müllerian hormone; *TPOAb* thyroid peroxidase antibodies; *SE* standard error; *BMI* body mass index; *FSH* follicle-stimulating hormone; *LH* luteinizing hormone; *FT4* free thyroxine

*p-value less than 0.05 considered as statistically significant

in the total population (beta (SE) = 0.036 (0.016), p = 0.020) (Table 9).

AMH and TgAb

The random-effects pooled model revealed that AMH levels were not significantly correlated with TgAb levels (r = 0.069; 95% CI: − 0.098, 0.231), with considerable heterogeneity among the included studies (I^2: 65.42%; p = 0.013) (Figure S23). There was no evidence of publication bias according to the results of the Begg test (p = 0.566) or Egger test (p = 0.706). The sensitivity analysis showed no considerable changes in the results (Figure S24).

Discussion

The present meta-analysis investigated the association between AMH and various thyroid function markers across diverse populations. Our pooled results indicated no significant correlation between AMH and TSH levels in overall populations, and diverse population groups, including females with PCOS, infertility, hypothyroidism, subclinical hypothyroidism, and HT, and normal population. Notably, meta-regression analyses identified estradiol and FT3 as significant potential moderators in the total population, estradiol levels in PCOs, and FSH levels in the healthy population.

Moreover, AMH showed modest correlations with other thyroid markers, including a significant positive correlation with FT3 and FT4 and a significant negative association with both T3 and T4. Among the potential moderators influencing the correlation between AMH and FT4 or FT3, only BMI was a significant moderator. Additionally, AMH levels did not significantly correlate with thyroid antibodies (TPOAb and TgAb), except for a significant positive correlation between AMH and TPOAb levels in the normal population. In addition, FT4 was a significant factor influencing the association between AMH and TPOAb.

Although the initial results of our meta-analysis found a non-significant association between TSH and AMH, after applying the trim-and-fill method to address publication bias, the adjusted results (r = 0.188; 95% CI: 0.168–0.209) suggested a small but statistically significant positive correlation, highlighting the potential impact of missing studies on initial findings. Moreover, the high heterogeneity could stem from variations in study populations, underlying thyroid disorders, diagnostic methodologies, and the need for standardized and larger-scale studies in the future with more robust methodologies to assess the power of these associations. These results are consistent with those of previous studies, such as those by Unsal et al., Wu et al., and Vidales et al., who reported no significant correlation between AMH and TSH in women with HT, infertility, and PCOs, respectively [27, 31, 41]. However, the power of these results was limited by the small and homogenous sample size and the study design. In a longitudinal study with a 12-year follow-up of a population of 775 reproductive-aged women without thyroid disease or dysfunction, no significant changes in the mean TSH levels across quartiles of AMH were observed [23].

On the other hand, various studies have reported contradictory results considering the hypothesis that TSH is associated with hypothyroidism and negatively affects ovarian function [53]. This hypothesis stems from the presence of

thyroxin-binding domains within the ovaries, which implies a potential effect of thyroid hormones on female ovarian tissue [54]. Women with unexplained infertility and premature failure of the ovaries have increased TSH levels compared to fertile women [55]. Moreover, hypothyroidism has been correlated with menstrual abnormalities and anovulatory cycles, implying that thyroid dysfunction may adversely affect the processes of follicular development and maturation [6, 56].

These hypotheses were reflected in the results of several studies. For instance, Soam et al. even identified a negative correlation between AMH and TSH in infertile populations, suggesting that thyroid dysfunction may impair the ovarian reserve [38]. In this study, with a unit increase in TSH levels, the odds of having AMH < 1 ng/mL increased by 1.57 (95% CI: 1.13, 2.19) [38]. Similarly, in another study by Kabodmehri et al., each unit increase in TSH level was significantly associated with 25% odds of AMH < 1.1ng/ml [10]. In addition, a retrospective cohort study found that even moderate-to-high levels of TSH (> 2.5 IU/mL) within the normal range could significantly decrease AMH levels and antral follicle counts [8]. However, as infertility and thyroid disease can co-occur at high rates in women, establishing causality between AMH and thyroid function remains challenging [6, 57]. Liang et al. used Mendelian randomization to investigate the causal relationship between AMH FT4 and TSH and found no conclusive relationship [6]. Similarly, Polyzos et al. found no significant differences in FT4 and TSH levels among groups with low, normal, or high ovarian reserves (p = 0.611 and p = 0.811, respectively) [12].

The lack of correlation between AMH and TSH or weak correlations between AMH and FT4 indicates that thyroid dysfunction alone does not influence ovarian reserve, potentially alleviating fertility concerns in cases of thyroid conditions [6]. However, treating thyroid disorders is crucial for improving fertilization chances and embryo quality, with recent guidelines recommending levothyroxine (LT4) therapy in cases of significant thyroid dysfunction or TSH levels > 4.0 mIU/L [58]. This guideline also recommends that LT4 supplementation be administered individually to subfertile females with TSH levels > 2.5 lIU/mL and thyroid autoimmunity to reach optimal ovarian reserve [58].

Regarding the correlation between TSH and AMH, several potential moderators such as estradiol, FT3, and FSH suggest that hormonal interplay should be considered when interpreting the relationship between AMH and TSH. Additionally, BMI was identified as a potential moderator of the correlation between AMH and FT4 levels in the overall population. This could be because obesity is correlated with diminished ovarian reserve and dysfunction in follicular development, potentially lowering the AMH levels [59]. The influence of obesity on AMH levels may be explained by how adiposity detrimentally affects the granulosa cells responsible for AMH synthesis or, alternatively, by a dilutional effect on circulating AMH concentrations [60]. Future studies should consider these moderators when adjusting for potential confounding factors.

AMH levels were positively correlated with FT3 levels in the overall population, with considerable heterogeneity among the included studies. These results were also observed in subgroups of infertile women with weaker correlations but insignificant heterogeneity, which suggests that the results in homogeneous or heterogeneous populations can be somewhat different. Moreover, no significant potential moderator influenced AMH-FT3 correlation in the overall population. Altogether, a reduction in thyroid hormones impairs folliculogenesis, which prevents the maturation of granulosa cells and facilitates apoptosis in atretic follicles, leading to diminished AMH levels [61]. However, in a recent meta-analysis, results showed that AMH levels were not significantly different between patients with subclinical hypothyroidism and the control group (mean difference (MD): − 0.50; 95% CI: − 1.11, 0.11), with similar results between patients with overt hypothyroidism and the control group (mean difference: − 0.60; 95% CI: − 1.34, 0.14 [61]. A possible explanation for these findings is that the natural age-dependent decline in AMH, a key marker of ovarian reserve, may overshadow or mask the potential influence of thyroid dysfunction on ovarian function [61].

Finally, our meta-analysis did not yield significant results regarding the correlation among AMH, TPOAb, and TgAb levels in the overall population. Our results were aligned with those of a meta-analysis of women with HT and ovarian reserve [14]. In this study, AMH levels were not significantly different between HT and non-HT women among the 13 studies with overall 3997 women (standardized MD: − 0.15, 95% CI: − 0.36, 0.06). Moreover, TgAb positivity was not significantly associated with ovarian reserve (OR, 3.17; 95% CI: 0.89, 11.38). However, conflicting results were observed in this meta-analysis. We found a significant positive correlation between TPOAb and AMH in the normal population, whereas in this study, significantly lower AMH levels were observed in reproductive-aged women with HT (standardized MD: − 0.35, 95% CI: − 0.51, − 0.19) [14]. In addition, Adamska et al. found that AMH levels were negatively correlated with TPOAb in the PCOS group (r = − 0.4), whereas Polyzos et al. did not report a significant correlation between AMH and TPOAb in a large-scale study of 5000 women [12, 17]. Ünsal et al., in a study of HT patients compared with a control group, did not find a significant correlation between AMH and TgAb or TPOAb [31].

This notable discrepancy may arise from differences in age groups, sample size and characteristics, or other confounding factors, emphasizing the complex and multifactorial nature of the relationship between thyroid antibodies and ovarian reserve. However, thyroid antibodies appear to affect ovarian tissue directly, and although the mechanism

underlying the effect of thyroid antibodies on ovarian reserve is still unclear, it is hypothesized that TPOAb may traverse the follicle barrier, leading to follicle and oocyte damage [62, 63]. Furthermore, thyroid autoimmunity (TAI) might be associated with ovarian autoimmunity, potentially leading to primary ovarian insufficiency (POI). A recent nationwide cohort study demonstrated that women with Hashimoto's and Grave's diseases are significantly more likely to develop POI and ovarian insufficiency compared to healthy populations [64]. One proposed autoimmune mechanism is the cross-reactivity or concurrent expression of ovarian and thyroid antigens, resulting in autoimmune-mediated ovarian damage [65]. Anti-thyroid antibodies, such as anti-TPOAb, can induce ovarian damage by initiating cytotoxic immune reactions within ovarian follicles, ultimately impairing follicular maturation and the ovarian reserve [65].

Although most studies included in our meta-analysis explicitly excluded women with overt primary ovarian insufficiency (POI), the potential autoimmune interplay between TPOAb and ovarian function remains noteworthy. A Recent study have suggested that the odds of having low ovarian reserve were 1.402 times higher in women with positive TPOAb compared to those with normal ovarian reserve (95% CI: 1.085, 1.812, p = 0.010) [66]. Moreover, TAI was strongly linked to overt POI in women with TSH levels above 2.5 μIU/ml, but showed no significant association with biochemical POI or overt POI in women with TSH at or below 2.5 μIU/ml. [66], highlighting an autoimmune process involving shared thyroid and ovarian antigens, thereby directly contributing to ovarian follicular damage and increased POI risk. Thus, the observed associations between thyroid antibodies and AMH might reflect a shared autoimmune process affecting ovarian and thyroid tissues concurrently, rather than a direct causal effect of thyroid antibodies on AMH levels alone. In this regard, these results should be interpreted with caution, and further research is essential to disentangle these correlations and to provide a more precise understanding of several populations of women with PCOS, HT, infertility, and the normal population.

Limitations and future directions

Our meta-analysis provides valuable insights but has several limitations. Significant heterogeneity across studies may reflect differences in population, thyroid status, and methodologies. Variations in AMH levels and thyroid assays may have caused inconsistencies. Most studies were observational, limiting causal inferences, with unmeasured confounders like lifestyle and genetic predispositions potentially affecting results. Due to limited regression analysis, only studies with correlation analysis were included. This review focused on linear associations, but some original studies using logistic regression suggested a nonlinear relationship. Future research should apply regression analysis with longitudinal methodologies to explore potential nonlinear associations, considering different reproductive stages and metabolic profiles. Identifying moderators that influence AMH and thyroid markers will offer a more comprehensive understanding of thyroid-ovarian interactions.

Conclusions

In conclusion, this meta-analysis underscores the complex relationship between AMH levels and thyroid function markers in a diverse population. AMH levels were not significantly correlated with TSH levels in the overall population or in diverse population subgroups. Meta-regression analysis identified estradiol, FT3, and FSH as key moderators of the association between AMH and TSH levels in various populations. In the overall population, AMH showed significant positive correlations with FT3 and FT4, negative correlations with T3 and T4, and significant positive correlation with TPOAb in the normal population. These findings highlight the need for further research to elucidate the underlying mechanisms, clinical implications, and strengths of these associations.

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