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# Anti- Müllerian Hormone as a Biomarker of Ovarian Reserve after Chemotherapy

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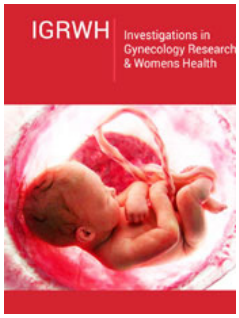


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## Abstract

Chemotherapy and related cancer therapies can injure the ovary, accelerate follicular depletion, and increase the risk of Premature Ovarian Insufficiency (POI) and fertility impairment. Anti-Müllerian Hormone (AMH), secreted by granulosa cells of growing preantral and small antral follicles, closely tracks the functional ovarian reserve and is relatively stable across the menstrual cycle, making it a practical biomarker for serial monitoring during and after cancer treatment. Across malignancies, AMH commonly falls to very low or undetectable concentrations during chemotherapy, with partial and age- and regimen-dependent recovery thereafter. Robust prospective data in early breast cancer show that undetectable AMH measured 6 months after chemotherapy can diagnose persistent ovarian failure at ≈30 months with high accuracy (AUROC ~0.89), and that pretreatment AMH adds predictive value beyond age for long-term ovarian activity. More recent individual-patient-data meta-analyses and trial-linked biomarker studies have refined these observations, confirming the prognostic contribution of pretreatment AMH while underscoring persistent challenges, including assay heterogeneity and difficulty establishing universally applicable cutoffs for clinical decision-making. Clinically, AMH supports fertility counselling and survivorship care; it also informs endocrine therapy choices when interpreted alongside estradiol, FSH, age, and treatment details. However, AMH is not a direct "fertility test," does not reflect oocyte quality, and pregnancies can occur despite low or even undetectable values. Standardization of assays is progressing but incomplete, and consensus on timing/frequency of testing is evolving-particularly in adolescents and young adult (AYA) survivors. This review synthesizes contemporary evidence on AMH trajectories by disease and regimen, diagnostic and prognostic performance before and after chemotherapy, guideline-anchored applications, practical implementation issues, and priority gaps for future research.

**Keywords:** Anti-Müllerian hormone; Chemotherapy; Ovarian reserve; Premature ovarian insufficiency; Breast cancer; Lymphoma; Fertility preservation; Ovarian protection; Survivorship

## Introduction and Scope

Premature Ovarian Insufficiency (POI) following cytotoxic therapy is a central survivorship concern for adolescents and premenopausal women with cancer, with ramifications for fertility, bone and cardiovascular health, sexual function, and quality of life. Reliable biomarkers that quantify ovarian injury and forecast the likelihood of ovarian function recovery are therefore crucial to personalize counselling and treatment decisions-from pre-treatment fertility preservation to post-treatment endocrine therapy selection. Anti-Müllerian Hormone (AMH) has emerged as a leading candidate because it reflects the pool of small growing follicles, declines predictably with age, shows minimal intra-cycle variation, and is technically amenable to automated immunoassays suitable for routine use [1,2]. A comprehensive systematic review (92 studies; ~9,200 patients) concluded that AMH typically falls dramatically after chemotherapy (often ≥90%), with at least partial recovery reported in many longitudinal series; recovery magnitude depends on regimen, age, and pretreatment AMH. Those findings, a cornerstone of the earlier evidence base, have since

been extended by analyses in specific disease settings (e.g., HER2-positive breast cancer) and by pooled individual-patient-data meta-analysis focusing on predictive performance [3].

Your original narrative review captured much of this arc through 2022-2025; the present rewrite updates the field to early 2026, incorporating fresh guidelines (ASCO 2025), new cohort and meta-analytic data, and practical consensus statements relevant to endocrine management-while maintaining a pragmatic lens for clinicians and counsellors working with cancer survivors [4-7].

## AMH Biology and What It Measures

AMH is secreted by granulosa cells in preantral and small antral follicles and is thought to inhibit excessive primordial follicle recruitment and modulate FSH sensitivity during early folliculogenesis. Serum AMH correlates with the quantity of small growing follicles and thus with functional ovarian reserve; however, it does not directly index oocyte competence or the probability of pregnancy in each cycle. These properties make AMH a useful indicator of ovarian quantity rather than quality-an especially important distinction in oncology [1]. From a laboratory perspective, modern automated platforms (e.g., Roche Elecsys) measure AMH with improved analytical precision. Public health laboratories (e.g., CDC/NHANES) also provide a method of documentation that summarizes AMH's clinical relevance and preanalytical nuances. Although cycle-phase influences are modest, hormonal contraception, BMI, and hypoestrogenic states can lower AMH and should be considered when interpreting values-emphasizing the need to view AMH in context, not isolation [2,8].

## Assay Standardization: Where We are in 2026

A major limitation has been inter-assay variability. The WHO evaluated candidate international standards (e.g., recombinant AMH 16/190) to harmonize calibration across methods; while initial efforts improved stability, issues of commutability remain, and true traceability to a universal reference is not fully realized. Clinicians should avoid mixing assay platforms in serial monitoring and interpreting published cutoffs with caution. Continued international work toward a second-generation standard is underway [9,10].

**Practical tip:** If serial AMH testing will inform decisions (e.g., endocrine therapy in early breast cancer), keep measurements on a single platform and-where available-apply laboratory specific reference ranges or decision thresholds validated against clinical outcomes rather than cross study numeric values alone.

## How Chemotherapy Affects AMH: Magnitude and Time Course

### General patterns

Across tumor types and regimens, AMH typically plunges to low or undetectable levels during or shortly after chemotherapy-consistent with damage to the growing follicle pool-followed by variable recovery over 1-3 years in those with sufficient residual reserve. The 2022 Human Reproduction Update meta-analysis documented reductions in 92% of studies, often ≥90%; partial recovery was common but heterogeneous. Recovery odds and amplitude decline with older age at treatment and more gonadotoxic regimens [4]. Building on these observations, a 2025 breast cancer-focused meta-analysis reported that >50% of survivors <40 years had very low AMH (≤0.5-1.0ng/mL) 12-24 months after chemotherapy, underscoring the high residual risk of diminished ovarian reserve even in younger patients [11].

### Disease and regimen specific signals

**Breast cancer:** In women aged 40-45, undetectable AMH at 6 months post-chemotherapy portends persistent ovarian failure at ≈30 months (AUROC ~0.89), with modest gains in discriminative accuracy when combined with age, pretreatment AMH/FSH, and exposure to taxanes. This reinforces AMH's value as a post-therapy diagnostic tool in endocrine decision-making. More recently, in HER2-positive disease treated on contemporary regimens, baseline AMH predicted ovarian function loss with good accuracy (AUC ~0.78-0.80), whereas adding FSH/E2 did not improve prediction-an important reminder that AMH may capture the most clinically salient variance for this endpoint [6,12].

**Lymphomas:** Prospective series differentiating ABVD (non-alkylating) versus alkylatingagent-heavy protocols demonstrate far greater AMH suppression and less recovery with the latter, in both AYA and nonAYA groups-even when GnRH agonists were co-administered. A 2023 cohort of 270 Hodgkin lymphoma patients treated with ABVD showed a sharp AMH drop at 6 months with near return toward baseline by 12 months, again highlighting regimen specific effects on trajectory.

**Pediatric and AYA survivors:** A 2025 mega-analysis (13 studies; n=608 female childhood cancer survivors) showed AMH correlates with established gonadotoxicity risk strata (O-PIN), increases with time since therapy in many survivors, and is particularly low after Hematopoietic Stem Cell Transplantation (HSCT)-irrespective of cyclophosphamide equivalent dose-supporting both treatment-intensity and developmental-stage effects on recovery. Early declines can be abrupt even after a single cycle of high-risk regimens (e.g., VDC-IE for Ewing sarcoma) [9]. Before treatment begins. Baseline AMH may already be lower in some malignancies (e.g., lymphomas) compared with age-matched controls, an observation relevant for counselling and interpreting subsequent declines. In contrast, baseline AMH in many breast cancer cohorts is similar to controls, emphasizing that the disease effect at diagnosis differs by tumor type [13].

## Pretreatment AMH as a Prognostic Tool

Pretreatment AMH generally adds prognostic information beyond age for the likelihood and timing of ovarian function resumption after chemotherapy. A 2025 prospective study found that prechemotherapy AMH yielded an AUC of 0.86 for resumption of ovarian function within 24 months, with only marginal incremental value from adding age; however, the optimal cutoff (~0.6-0.7μg/L in that study using a hypersensitive assay) remains assay dependent.

Complementing this, a 2026 individual patient data meta -analysis across 31 studies confirmed that higher pretreatment AMH is associated with ovarian function resumption (AUC ~0.79-0.83) but cautioned that cutoffs vary widely with follow-up time, age, and assay-limiting universal application for treatment choices such as endocrine therapy selection. In other words, pretreatment AMH is a strong risk stratifier but not yet a one size fits all decision threshold. In HER2 positive early breast cancer from randomized trials, both baseline and end of therapy AMH predicted ovarian function loss (AUC ~0.74-0.82), with modest gains when combining timepoints and age; importantly, baseline FSH/E2 added no predictive value-again elevating AMH as the principal biomarker for this endpoint.

## Post treatment AMH as a Diagnostic/Prognostic Test

Serial post chemotherapy AMH is clinically informative. In early breast cancer, undetectable AMH 6 months after chemotherapy strongly predicts persistent low estradiol levels 30 months later (AUROC ~0.89), offering a practical timepoint to identify women unlikely to recover ovarian function-information that can be integrated into endocrine therapy decisions (e.g., tamoxifen vs. AI + ovarian suppression).

However, two caveats are essential:

**a) AMH≠fertility test:** AMH correlates with ovarian quantity, not oocyte quality or fecundability. Conceived pregnancies-and resumption of menses-can occur even with low/undetectable AMH, so AMH alone must not be used to counsel against attempted conception if that is otherwise appropriate.

**b) Endocrine decision-making requires estradiol context:** For women considering Aromatase Inhibitor (AI) therapy after chemotherapy, estradiol remains the biomarker of record for confirming adequate ovarian suppression, with institutional consensus statements outlining monitoring strategies and troubleshooting "breakthrough" ovarian function. In practice, AMH can complement estradiol by characterizing the likelihood of sustained suppression, but estradiol drives near-term endocrine choices [14-16].

## How AMH Stacks Up Against Other Markers (AFC, FSH, Estradiol, Inhibin B)

AMH outperforms single time-point FSH and inhibin B as a quantitative indicator of ovarian reserve and is less operator-dependent than Antral Follicle Count (AFC), especially in oncology where repeated transvaginal ultrasound may be impractical mid-treatment. AMH and AFC are broadly concordant, but AFC adds cost, operator dependence, and limited feasibility during chemotherapy. Meanwhile, estradiol is essential for endocrine therapy decisions but does not measure ovarian reserve; AMH and estradiol answer different clinical questions. Notably, in the 2025 HER2-positive trials, adding FSH/E2 to AMH did not improve prediction of ovarian function loss, underscoring AMH's primacy for reserve while preserving estradiol's role for suppression verification [1,6,14].

## Modifiers of AMH Interpretation: Age, Regimen, and Genetics

**Age is the dominant clinical modifier:** Younger women display greater AMH recovery and higher probabilities of resumed ovarian function than older women, reflecting larger residual follicle pools at diagnosis.

**Regimen matters:** Alkylatingagent-intense or high dose regimens (including HSCT conditioning) cause more profound and durable AMH suppression than non-alkylating protocols such as ABVD. These patterns hold across AYA and non-AYA cohorts.

BRCA status may influence baseline AMH and on-treatment decline. A meta-analysis suggests lower AMH in BRCA1 (but not consistently BRCA2) carriers ≤41 years. Pilot longitudinal data indicate a steeper AMH decline during anthracycline-taxane chemotherapy among germline BRCA carriers, possibly reflecting heightened susceptibility of the ovary's DNA damage response pathways-although evidence remains preliminary and sample sizes small. Clinically, lower baseline or faster decline in AMH among carriers may signal heightened gonadotoxic vulnerability, supporting proactive fertility preservation and careful survivorship monitoring [17,18].

## Clinical Applications

### Fertility risk assessment and preservation

**Before treatment:** Baseline AMH informs individualized risk, counseling and can help prioritize fertility preservation-for example, flagging those at greatest risk for poor oocyte yield or for sustained POI after treatment. Updated ASCO guidelines (2025) recommend fertility counseling at diagnosis and in survivorship, offer established methods (oocyte/embryo cryopreservation, ovarian tissue cryopreservation, and ovarian transposition as indicated), and note that In-Vitro Maturation (IVM) may be offered as an emerging method in select cases. AMH should be interpreted in this broader counseling framework, not as a stand-alone gatekeeper [7,19].

**During urgent stimulation cycles:** Practical questions often arise about triggering ovulation and initiating GnRH agonists for ovarian suppression around the time of fertility preservation. Emerging data in oncology populations speak to the safety and logistics of long-acting GnRH agonist "triggers" in urgent cycles prior to chemotherapy, though caution and individualized planning remain prudent.

**After treatment:** Lower posttreatment AMH quantifies gonadotoxic impact and can guide expectation setting. For some survivors who deferred pretreatment preservation, AMH helps triage next steps (e.g., attempts at conception vs. assisted reproduction) while underscoring that natural conception remains possible despite low AMH. National and society guidelines from 2023-2025 continue to anchor best practices for oncofertility services, including attention to timing, consent, ethics, and access.

### Ovarian protection with GnRH agonists during chemotherapy

Randomized breast cancer trials-most notably POEMS/S0230-demonstrate that concurrent GnRH agonists reduce treatment-induced ovarian failure and are associated with more post-treatment pregnancies, establishing this strategy for ovarian function preservation in appropriate patients. Long-term follow-up supports safety with no evident detriment to cancer outcomes. Nonetheless, large real-world cohorts suggest that while GnRH agonists can mitigate POI risk, they do not guarantee preserved fertility when measured by post-cancer childbirth rates-emphasizing the need to combine pharmacologic protection with established preservation methods when pregnancy is a priority [20]. Mechanistically, the ovarian protective effect of GnRH agonists remains debated, and the magnitude of benefit outside breast cancer (e.g., in hematologic malignancies) is less certain. A 2022 textbook chapter and recent reviews summarize evidence and highlight practice nuances (e.g., timing of first injection, expectations by regimen).

### Endocrine therapy selection after chemotherapy

For premenopausal, hormone receptor-positive breast cancer, deciding between tamoxifen vs. aromatase inhibitor (AI) plus ovarian suppression demands a clear understanding of ovarian function status. Here:

a. **Estradiol is the key laboratory anchor for confirming adequate suppression on GnRH agonists:** Several expert statements and consensus documents outline practical thresholds, timing of monitoring, and actions when "breakthrough" ovarian function occurs on therapy [14,15].

b. **AMH can enrich risk stratification:** e.g., undetectable AMH 6-12 months post chemotherapy indicates a low likelihood of near-term ovarian recovery and may support AI based approaches in appropriately selected women. But current meta-analyses caution against using specific universal cutoffs to "optimize" endocrine therapy because assay variability and differing follow-ups limit portability; AMH should inform, not dictate, the plan.

c. Guidelines and pathways (e.g., NCCN updates through 2026) reinforce that patients premenopausal at diagnosis are candidates for ovarian function suppression and that estradiol-based monitoring is reasonable when AI therapy is used, especially in the first year.

## Special Populations and Survivorship Considerations

### Adolescents and Young Adults (AYAs)

Timing and frequency of AMH testing in childhood cancer survivors remain uncertain. A 2024 systematic review from the Oncofertility Consortium's Pediatric & Adolescent Committee concluded that evidence is insufficient to stipulate testing intervals and that AMH correlates with other reserve markers but not reliably with the ability to conceive or the exact timing of menopause-reinforcing a measured, individualized approach [21].

### HSCT recipients

AMH is often undetectable long-term after HSCT; this information is key to anticipatory guidance about hormone replacement, bone health, and, in some cases, consideration of experimental preservation at earlier life stages.

### Managing menopause after cancer

POI precipitated by treatment can produce more severe vasomotor, sexual, and mood symptoms than natural menopause. Updated reviews and national guidelines (e.g., British Menopause Society 2024; Lancet 2024 series) offer frameworks for symptom management, the selective use of menopausal hormone therapy by cancer type, and non-hormonal options-care pathways that should be integrated with biomarker-based ovarian function assessment [22].

## Practical Implementation: Timing, Frequency, and Interpretation

When to test:

### Baseline (pre-chemotherapy)

Captures pre-existing reserve, informs risk and fertility preservation planning, and provides a reference for subsequent changes. Baseline values may differ by cancer type (e.g., lower in lymphomas), so comparison to population norms should be cautious [13].

### During chemotherapy

Steep declines can be observed after the first cycles, particularly with highly gonadotoxic regimens; on-treatment measurements can contextualize expectations but rarely alter active cancer therapy [9].

**6-12 months post chemotherapy:** A pragmatic window for predicting sustained ovarian failure (e.g., undetectable AMH at 6 months → high PPV for failure at ~30 months in women 40-45. For AYA survivors, individualized schedules are reasonable given the lack of strong evidence for specific intervals.

### How to interpret:

Consider age, regimen, assay used (stick to one platform for serial monitoring), concomitant medications (e.g., hormonal contraception), and body habitus. View AMH alongside estradiol/FSH and clinical status, especially when endocrine therapy decisions hinge on ovarian suppression.

**Avoid rigid cutoffs:** Where thresholds are cited (e.g., 0.6-0.7μg/L in single assay studies), treat them as assay specific and hypothesis generating rather than universally portable.

Communicate clearly that pregnancy can occur with low/undetectable AMH and that AMH is not a direct measure of fecundability-mitigating misinterpretation and undue distress [1].

### Laboratory and standards

Use the same platform over time; if a change is unavoidable, interpret differences cautiously. WHO standardization is in progress but not complete; laboratories should disclose assay characteristics, including limits of detection and functional sensitivity [8-10]. Lean on national lab method documents (e.g., CDC) for preanalytical guidance.

## Limitations of AMH and Ongoing Controversies

a) Assay heterogeneity remains the Achilles' heel, constraining global cutoffs for clinical decision-making-especially for optimizing endocrine therapy. Consensus panels and methodologic work toward an internationally commutable standard are priorities [9,10].

**b) Outcome surrogacy:** AMH tracks ovarian quantity but is only a proxy for chances of pregnancy or age at menopause. Counseling should integrate patient goals, time horizons, and the full fertility/menopause management toolkit [1,7].

**c) Role with GnRH agonists:** While randomized data in breast cancer support ovarian protection during chemotherapy, large registry analyses question whether this translates into more childbirths, reminding clinicians to align expectations and combine modalities when parenthood is a primary goal.

**d) Genetic modifiers:** Data on BRCA carriers are mixed and still evolving; small cohorts suggest lower baseline AMH and steeper declines, but robust, assay-harmonized, regimen-specific studies are needed before genotype-tailored thresholds are used in practice [17,18].

## Research Priorities for the Next 5 Years

a. Assay harmonization and clinical thresholds: Achieve broad uptake of a commutable international standard and validate assay specific decision thresholds tied to outcomes (e.g., time to menopause, POI, estradiol confirmed ovarian inactivity) across diverse populations.

b. Longer-term trajectories (>5-10 years) by malignancy and regimen, including contemporary targeted and immune therapies, with attention to late recovery and predictors of resilience.

c. Integration models that combine AMH with estradiol, age, regimen, and genetic modifiers to generate validated, patient facing risk estimates (e.g., individualized risk of POI at 24-36 months).

d. AYA and pediatric survivorship: Define timing/frequency of AMH testing, correlate with fertility and menopause endpoints, and test care pathways that improve patient centered outcomes.

e. Comparative effectiveness of ovarian protection strategies (GnRH agonists±fertility preservation) on livebirth and parent reported outcomes-with AMH as a mechanistic intermediate but not the sole endpoint.

## Bottom line for practice in 2026

a. AMH is a practical, cycle-independent biomarker that quantifies ovarian reserve and mirrors chemotherapy's impact on the ovary. Expect dramatic declines during therapy and regimen- and age-dependent partial recovery thereafter [4].

b. Pretreatment AMH improves prediction of who will recover ovarian function post-therapy beyond age alone, but universal cutoffs remain elusive due to assay variability; treat thresholds as contextual, not absolute [4,5].

c. Post-treatment AMH at 6-12 months helps diagnose persistent ovarian failure, particularly in women ≥40 at diagnosis, and can support endocrine therapy choices alongside estradiol [12,14].

d. AMH should support, not replace, guideline anchored oncofertility counseling and estradiol-based verification of ovarian suppression in those receiving AI therapy [23-25].

e. Communicate that AMH is not a fertility test; pregnancies can occur despite low/undetectable AMH-planned care around patient goals, timing, and the full menu of fertility and survivorship options [26,27].

## Conclusion

Anti-Müllerian hormone has emerged as the most practical and informative biomarker for quantifying chemotherapy-associated ovarian injury and for estimating the likelihood of ovarian function recovery in premenopausal cancer patients and survivors. Its biologic specificity for the growing follicle pool, relative cycle stability, and feasibility for serial monitoring position AMH as a cornerstone of modern oncofertility and survivorship care. Across malignancies and treatment regimens, chemotherapy typically causes a profound early decline in AMH, with partial and highly variable recovery driven principally by age, pretreatment reserve, and gonadotoxic intensity [28,29].

The accumulated evidence supports two clinically distinct uses of AMH. Pretreatment AMH refines risk stratification beyond chronological age alone and can inform individualized counseling regarding fertility preservation and the probability of post-therapy ovarian function. Posttreatment AMH, particularly values obtained 6-12 months after chemotherapy, has strong diagnostic and prognostic utility for identifying persistent ovarian insufficiency-most convincingly demonstrated in early breast cancer-and can complement endocrine decision-making when interpreted alongside estradiol, FSH, and clinical context. However, assay heterogeneity and population-specific variation preclude universal AMH cutoffs, underscoring the importance of assay consistency and cautious interpretation. Crucially, AMH should not be misconstrued as a direct measure of fertility or reproductive potential. It reflects ovarian quantity rather than oocyte quality, and pregnancies may occur even with very low or undetectable concentrations. Accordingly, AMH must support rather than supplant guideline-anchored fertility counseling, established fertility-preservation strategies, and estradiol-based verification

of ovarian suppression in patients receiving aromatase inhibitor therapy. Clear communication around these limitations is essential to prevent misinterpretation, undue distress, or inappropriate clinical decisions. Despite substantial progress, gaps remain. International assay standardization is incomplete; validated, outcome-linked decision thresholds are still evolving; and long-term ovarian trajectories following contemporary targeted and immune-based therapies are incompletely characterized. Evidence is particularly limited for optimal testing strategies in pediatric, adolescent, and young adult survivors, as well as for genetically susceptible subgroups. Addressing these gaps will require harmonized assays, individual-patient-data analyses, and longitudinal survivorship cohorts that integrate biochemical, clinical, and patient-centered outcomes.

In summary, when used thoughtfully and in context, AMH is a powerful biomarker that enhances personalized counseling, informs survivorship planning, and bridges oncology and reproductive medicine. Continued methodological refinement and prospective validation will be essential to fully realize its potential as an integrated tool for risk prediction, shared decision-making, and long-term care of women exposed to chemotherapy.

## References


1. Cedars MI (2022) Evaluation of female fertility-AMH and ovarian reserve testing. J Clin Endocrinol Metab 107(6): 1510-1519.
2. Roche (2026) Elecsys AMH. method sheet for cobas e analyzers. pp. 1-6.
3. Anderson RA, Cameron D, Clatot F, Demeestere I, Lambertini M, et al. (2022) Anti-Müllerian hormone as a marker of ovarian reserve and premature ovarian insufficiency in children and women with cancer: a systematic review. Human Reproduction Update 28(3): 417-434.
4. Zwol-Janssens C, Rosmalen MM, Laven JSE, Nasserinejad K, Visser JA, et al. (2026) AMH as a marker for resumption of ovarian function after chemotherapy: An individual patient data meta-analysis and systematic review. Cancer Treatment Reviews 142: 103068.
5. Zwol-Janssens C, Rosmalen MM, Hoop EO, Drooger JC, Padt-Pruijsten A, et al. (2025) Early prediction of menopausal status after chemotherapy to optimize adjuvant endocrine therapy. The Breast 83: 104562.
6. Lambertini M, Allegranza D, Laubender RP, Harbeck N, Swain SM, et al. (2025) Predicting ovarian function loss after chemotherapy and anti-HER2 therapy in young breast cancer patients. J Natl Cancer Inst 117(11): 2317-2326.
7. H Irene Su, Lacchetti C, Letourneau J, Partridge AH, Qamar R, et al. (2025) Fertility preservation in people with cancer: ASCO clinical practice guideline update. J Clin Oncol 43(12): 1488-1515.
8. Xu J, Liu K, Xie L, Shi J, Tang X, et al. (2024) AMH levels after initial cycles of chemotherapy in female adolescents with ewing sarcoma and implications for preservation of fertility. J Clin Oncol 42(16_suppl): 10067.
9. Ciccarone M, Cavaceppi P, Tesei C, Brunetti S, Pulsoni A, et al. (2023) Effects of ABVD chemotherapy on ovarian function: Hormonal and ultrasound analyses in 270 patients with Hodgkin's disease. Frontiers in Oncology 13: 1059393.
10. Yano Maher JCY, Kumnick A, Sinaii N, Irene Su, Cameron KE, et al. (2025) A mega-analysis of AMH levels in female childhood cancer survivors based on treatment risk and time since treatment. J Adolesc Young Adult Oncol 14(2): 160-171.
11. Weidlinger S, Weidlinger M, Schramm R, Vidal A, Pape J, et al. (2025) High impact of chemotherapy on ovarian reserve in reproductive-age breast cancer survivors: A systematic review and meta-analysis. The Breast 82: 104514.
12. Anderson RA, Kelsey TW, Perdrix A, Olympios N, Duhamel O, et al. (2022) Diagnostic and predictive accuracy of AMH for ovarian function after chemotherapy in premenopausal women with early breast cancer. Breast Cancer Research and Treatment 192(2): 273-282.
13. Caringella AM, Stanziano A, Cantatore C, Vitti A, Cortone A, et al. (2025) Does the type of cancer influence anti-müllerian hormone levels in women of reproductive age? A cross-sectional study Biomedicines 13(10): 2542.
14. McCann KE, Goldfarb SB, Traina TA, Regan MM, Vidula N, et al. (2024) Selection of appropriate biomarkers to monitor effectiveness of ovarian function suppression in ER+ breast cancer. NPJ Breast Cancer 10(1): 8.
15. Dana-Farber Breast Oncology Center (2025) Consensus statement on ovarian function suppression and estradiol monitoring in ER+ early breast cancer. pp. 1-13.
16. (2024) NCCN Breast Cancer Guidelines (endocrine monitoring details). pp. 1-86.
17. Gasparri ML, Micco RD, Zuber V, Taghavi K, Bianchini G, et al. (2021) Ovarian reserve of women with and without BRCA pathogenic variants: A systematic review and meta-analysis. The Breast 60: 155-162.
18. Decanter C, Dassonneville A, D'Orazio E, Hélène Behal, Anne-Laure Gagez, et al. (2025) AMH decline during chemotherapy reflects breast cancer cell DNA Damage Response (DDR) proficiency: The ONCO AMH1 pilot study. Journal of Assisted Reproduction and Genetics 42(5): 1435-1441.
19. Saito S, Yamada M, Yano R, Takahashi K, Ebara A, et al. (2023) Fertility preservation after gonadotoxic treatments for cancer and autoimmune diseases. Journal of Ovarian Research 16(1): 159.
20. Moore HCF, Unger JM, Phillips K, Boyle F, Hitre E, et al. (2015) POEMS/S0230: Goserelin for ovarian protection during breast-cancer chemotherapy. N Engl J Med 372(10): 923-932.
21. Rotz SJ, Bjornard K, Hampanda K, Kumnick A, Maher JCY, et al. (2024) Limited recommendations and evidence for timing/frequency of AMH screening in female pediatric cancer survivors: A systematic review. J Adolesc Young Adult Oncol 14(3): 212-219.
22. Hickey M, Basu P, Sassarini J, Stegmann ME, Weiderpass E, et al. (2024) Managing menopause after cancer. Lancet 403 (10430): 984-996.
23. Decanter C, Delepine J, Behal H, Manier S, Bruno B, et al. (2021) Longitudinal study of AMH variations in 122 Adolescents and Young Adults (AYA) and non-AYA lymphoma patients to evaluate the chemo-induced ovarian toxicity to further personalise fertility preservation counselling. Human Reproduction 36(10): 2743-2752.
24. Breast Cancer (Version 3.2024) (2026) NCCN Clinical Practice Guidelines in Oncology. J Natl Compr Canc Netw 22(5): 331-357.
25. Rodriguez-Wallberg KA, Kieler H, Foukakis T, Jiong Li, Gissler M, et al. (2024) GnRHa during chemotherapy and post-cancer childbirths: A nationwide population-based cohort study. EClinical Medicine 67: 102335.
26. Massarotti C, Stigliani S, Gazzo I, Lambertini M, Anserini P (2023) Long-acting GnRH agonist trigger in fertility preservation cycles before chemotherapy. ESMO Open 8(4): 101597.
27. WHO Expert Committee (2019) International Standard for AMH (code 16/190).
28. Li HWR, Robertson DM, Burns C, Ledger WL (2021) Challenges in measuring AMH in the clinical setting. Front Endocrinol 12: 691432.
29. CDC NHANES (2023) Analytical procedure manual for AMH.